MEDICO-CHIRURGICAL
TRANSACTIONS.
73920

PUBLISHED BY

THE ROYAL
MEDICAL AND CHIRURGICAL SOCIETY
OF
LONDON.

VOLUME THE FORTY-FIRST.

LONDON:
LONGMAN, BROWN, GREEN, LONGMANS, AND ROBERTS,
PATERNOSTER-ROW.

1858.
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SECOND SERIES.

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PATERNOSTER ROW.

1858.
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ROYAL
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FROM ITS FORMATION.

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1806. WILLIAM SAUNDERS, M.D.
1808. MATTHEW BAILLIE, M.D.
1810. SIR HENRY HALFORD, BART., M.D. G.C.H.
1813. SIR GILBERT BLANE, BART., M.D.
1815. HENRY CLINE.
1817. WILLIAM BABBINGTON, M.D.
1819. SIR ASTLEY PASTON COOPER, BART., K.C.H. D.C.L.
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1855. CÆSAR HENRY HAWKINS.
1857. SIR CHARLES LOCOCK, BART., M.D.
FELLOWS
OF THE
ROYAL MEDICAL AND CHIRURGICAL SOCIETY
OF LONDON.

EXPLANATION OF THE ABBREVIATIONS.
P.—President. V.P.—Vice-President.
T.—Treasurer. S.—Secretary.
L.—Librarian. C.—Member of Council.
The figures succeeding the words Trans. and Proc. show the number of Papers which have been contributed to the Transactions or Proceedings by the Fellow to whose name they are annexed.

OCTOBER 1858.

Amongst the non-residents, those marked thus (*) are entitled by composition to receive the Transactions.

Elected
1841 *James Abercrombie, M.D., Cape of Good Hope.
1846 *John Abercrombie, M.D., Physician to the Cheltenham General Hospital, 13, Suffolk Square, Cheltenham.
1851 *Henry Wentworth Acland, M.D., D.C.L., F.R.S., Physician to the Radcliffe Infirmary; Regius Professor of Medicine, and Clinical Professor in the University of Oxford.
1847 Elias Acosta, M.D., New York, U.S.
1842 William Acton, 46, Queen Anne-street, Cavendish-square. Trans. 1.
1851 John Adams, Surgeon to, and Lecturer on Descriptive and Surgical Anatomy at, the London Hospital; 4, St. Helen's-place, Bishopsgate-street. Trans. 1.
Elected

1852 William Adams, Surgeon to the Royal Orthopedic Hospital; Lecturer on Anatomy and Surgery at the Grosvenor-place School of Anatomy and Medicine; 5, Henrietta-street, Cavendish-square. Trans. 1.


1837 Ralph Fawsett Ainsworth, M.D., Physician to the Manchester Royal Infirmary; 6, Piccadilly, Manchester.

1819 George Frederick Albert.


1826 James Alderson, M.D. F.R.S., Senior Physician to, and Lecturer on Clinical Medicine at, St. Mary’s Hospital; 17, Berkeley-square. S. 1829. C. 1848. T. 1849. V.P. 1852-3. Trans. 3.

1843 Charles James Berridge Aldis, M.D., Medical Officer of Health for St. George’s Hanover-square; Senior Physician to the Surrey Dispensary; and Physician to the St. Paul and St. Barnabas Dispensary; 1, Chester-terrace, Chester-square.

1850 Charles Revans Alexander, Assistant-Surgeon to the Royal Infirmary for Diseases of the Eye; 6, Cork-street, Bond-street.

1813 Henry Alexander, F.R.S., Surgeon-Oculist in Ordinary to H.M. the Queen, and Surgeon to the Royal Infirmary for Diseases of the Eye; 6, Cork-street, Bond-street. C. 1822, 1840. V.P. 1850.


1820 Thomas Andrews, M.D., Norfolk, Virginia.

1813 William Ankers, Knutsford.

1819 Professor Antommarchi, Florence.
Elected


1851  THOMAS JOHN ASHTON, Surgeon to the Blenheim-Street Dispensary; 31, Cavendish-square.

1825  BENJAMIN GUY BABINGTON, M.D. F.R.S., Physician to the Asylum for Deaf and Dumb, and Consulting Physician to the German Hospital, and to the City of London Hospital for Diseases of the Chest; 31, George-street, Hanover-square. C. 1829. V.P. 1845-6. T. 1848. Trans. 2.

1846  CORNELIUS METCALFE STUART BABINGTON, F.R.C.P., Physician to Queen Charlotte's Lying-In Hospital, and Assistant-Physician to the Hospital for Sick Children; 29, Hertford-street, May-fair.

1820  *JOHN H. BADLEY, Dudley, Worcestershire.

1838  FRANCIS BADGLEY, M.D., Toronto, Upper Canada.

1840  WILLIAM BAINBRIDGE, late of Kingston, Surrey.

1836  ANDREW WOOD BAIRD, M.D., Physician to the Dover Hospital; Dover, Kent.

1851  *ALFRED BAKER, Surgeon to the Birmingham General Hospital, and Lecturer on Surgery at Sydenham College; Congreve Street, Birmingham.


1848  EDWARD BALLARD, M.D., Medical Officer of Health for Islington; 42, Myddelton-square. Trans. 1.

1849  THOMAS BALLARD, 10, Southwick-place, Hyde-park.

1837  WILLIAM B Alert, M.D. F.R.S., Assistant-Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; and Physician to the Millbank Prison; 45, Queen Anne-street, Cavendish-square. C. 1845-6. L. 1847. S. 1848-9. V.P. 1855-6. Trans. 1.
Elected

1847 Andrew Whyte Barclay, M.D., Secretary, Assistant-Physician to, and Lecturer on Materia Medica at, St. George's Hospital; Medical Officer of Health for Chelsea; 23a, Bruton-street, Berkeley-square. S. 1857. Trans. 2.

1848 Edgar Barker, 9, Oxford-square, Hyde-park.

1833 Thomas Alfred Barker, M.D., Senior Physician to, and Lecturer on Clinical Medicine at, St. Thomas's Hospital; 71, Grosvenor-street. C. 1844-5. V.P. 1853-4. Trans. 5.

1843 Thomas Herbert Barker, M.D., Harpur-place, Bedford.

1847 George Hilaro Barlow, M.D., Physician to, and Lecturer on Clinical Medicine at, Guy's Hospital; Physician to the Magdalen Hospital; 5, Union-street, Southwark.

1840 Benjamin Barrow, Surgeon to the Royal Isle of Wight Infirmary; Ryde, Isle of Wight.

1844 William Richard Basham, M.D., Senior Physician to, and Lecturer on Medicine at, the Westminster Hospital; 17, Chester-street, Grosvenor-place. S. 1832-4. Trans. 2.

1841 George Beaman, M.D., 3, Henrietta-street, Covent-garden,

1856 Amos Beardsley, Ulverstone, Lancashire.

1836 William Beaumont, Professor of Surgery in the University of King's College, Toronto, Upper Canada. Trans. 2.

1840 Charles Beevor, 41, Upper Harley-street.

1858 William Chapman Begley, M.D., Middlesex County Lunatic Asylum; Hanwell.

1819 Thomas Bell, F.R.S. F.L.S., Professor of Zoology in King's College, London; Surgeon-Dentist to, and Lecturer on the Anatomy and Diseases of the Teeth at, Guy's Hospital; and President of the Linnean Society; 17, New Broad-street, City. C. 1832-3. V.P. 1854. Trans. 1.

1847 James Henry Bennet, M.D., Physician-Accoucheur to the Royal Free Hospital; 60, Grosvenor-street.

1845 Edwin Unwin Berry, 7, James-street, Covent-garden.

Elected

1855 Archibald Billing, M.D. F.R.S., late Senior Physician to the London Hospital; Member of the Senate, and Examiner in Medicine at the University of London; 6, Grosvenor-gate. C. 1825. V.P. 1828-9.


1850 James Bird, M.D., Lecturer on Military Surgery at St. Mary's Hospital Medical School; 27, Hyde Park-square.

1855 Peter Hinchers Bird, 1, Norfolk-square, Hyde-park.

1856 William Bird, Surgeon to the St. George's and St. James's, and to the Fulham and Hammersmith General Dispensaries; 11, George-street, Hanover-square.

1849 Edmund Lloyd Birkett, M.D., Physician to the City of London Hospital for Diseases of the Chest; 48, Russell-square.

1851 George Birkett, M.D., Lecturer on Medical Jurisprudence at the Charing Cross Hospital; 1, Gloucester-villas, Highbury New Park.

1851 John Birkett, F.L.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 59, Green-street, Grosvenor-square. L. 1856-7. Trans. 2.

1846 Hugh Birt, Bognor-on-Sea, Sussex.

1843 Patrick Black, M.D., Assistant-Physician to, and Lecturer on Forensic Medicine at, St. Bartholomew's Hospital; 49, Queen Anne-street, Cavendish-square. C. 1856.

1847 George C. Blackman, M.D., Professor of Surgery in the Medical College of Ohio; New York, U.S.


1840 Peyton Blakiston, M.D. F.R.S., St. Leonards-on-Sea.

1845 Henry Blenkinsop, Senior Surgeon to the Warwick Dispensary; Jury-street, Warwick.

1823 Louis Henry Bojanus, M.D., Wilna.
Elected

1810  JOHN KAYE BOOTH, M.D., late Principal of Queen's College, and Hon. Physician to the Queen's Hospital, Birmingham; Brush-house, near Sheffield, Yorkshire.

1846  PETER BOSSEY, 1, Queen's-terrace, The Common, Woolwich, Kent.

1846  JOHN ASHTON BOSTOCK, Surgeon-Major, Scots Fusilier Guards; 54, Chester-square, Belgravia.

1841  WILLIAM BOWMAN, F.R.S., Surgeon to King's College Hospital, and to the Royal London Ophthalmic Hospital, Moorfields; 5, Clifford-street, Bond-street. C. 1852-3. Trans. 3.


1857  WILLIAM BRINTON, M.D., Physician to the Royal Free Hospital, and Lecturer on Physiology and Forensic Medicine at St. Thomas's Hospital; 20, Brook-street, Grosvenor-square.

1851  BERNARD EDWARD BRODHURST, Assistant Surgeon to the Royal Orthopedic Hospital, and Surgeon to the Hon. Artillery Company; 20, Grosvenor-street. Trans. 1; Proc. 1.


1844  CHARLES BROOKE, M.A. F.R.S., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; 29, Keppel-street, Russell-square. C. 1855.
Elected

1848 WILLIAM PHILPOT BROOKES, M.D., Surgeon to the Cheltenham General Dispensary, and Medical Inspector of Lunatic Asylums for the Upper Division of Gloucestershire; Albion House, Cheltenham.

1854 *HENRY BROWN, Surgeon to H.M. the Queen, H.R.H. the Prince Consort, and the Royal Household; Windsor.

1857 *ROBERT BROWN, late House-Surgeon to the Cumberland Infirmary; 4, Devonshire-street, Carlisle.

1851 ALEXANDER BROWNE, M.D., Army and Navy Club, St. James's-square; and Twynholm, Kirkcudbright.

1855 WALTER JOHN BRYANT, 7, Bathurst-street, Hyde-park-gardens.

1823 B. BARTLET BUCHANAN, M.D.

1843 JOHN CHARLES BUCKNILL, M.D., Medical Superintendent of the Devon County Lunatic Asylum, Exminster, Devonshire.

1839 GEORGE BUDD, M.D. F.R.S., Professor of Medicine in King's College, London; Physician to King's College Hospital; Consulting Physician to the Seamen's Hospital Ship 'Dreadnought,' and to the Blenheim Free Dispensary; 20, Dover-street, Piccadilly. C. 1846-7. V.P. 1857. *Trans. 4.

1839 THOMAS HENRY BURGESS, M.D., Military Hospital, Portsmouth.

1853 PATRICK BURKE, 13, Upper Montagu-street, Montagu-square.

1854 PHILIP BURROWS, Surgeon to the London City Mission; 23, Gloucester-crescent north, Hyde-park.


1820 SAMUEL BURROWS.

1837 GEORGE BURK, F.R.S. F.L.S., Professor of Comparative Anatomy and Physiology at the Royal College of Surgeons; Surgeon to the Seamen's Hospital Ship 'Dreadnought'; 15, Harley-street, Cavendish-square. C. 1847-8. V.P. 1855. Trans. 4.
Elected

1818 **John Butter**, M.D. F.R.S. F.L.S., Physician Extraordinary to the Plymouth Royal Eye Infirmary; Plymouth.

1851 *William Cadge*, Surgeon to the Norfolk and Norwich Hospital; All Saints, Norwich. *Trans.* 1.

1851 **Thomas Callaway**, India.

1852 *George Canney*, Bishop-Auckland, Durham.


1825 **Harry W. Carter**, M.D., Consulting Physician to the Kent and Canterbury Hospital; Ashford, Kent.


1820 **Samuel Cartwright**, F.R.S. F.L.S., Nizell's House, near Tunbridge, Kent.

1845 **Samuel Cartwright**, Jun., Surgeon-Dentist to King's College Hospital; 32, Old Burlington-street.


1845 **William Oliver Chalk**, Surgeon to the St. Marylebone Eye and Ear Institution; 3, Nottingham-terrace, Yorkgate, Regent's-park [40, Marylebone-road].


1844 **Thomas King Chambers**, M.D., Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Examiner in Physiology, University of Oxford; 1, Hill-street, Berkeley-square. *Trans.* 1.

1849 **Frederick Chapman**, Richmond-green, Surrey.


1852 **George Borlase Childs**, Surgeon-in-Chief to the City Police Force, and Surgeon to the Metropolitan Free Hospital; 11, Finsbury Place South.

1842 **William Dingle Chowne**, M.D., Physician to, and Lecturer on Medicine and Midwifery at, the Charing Cross Hospital, Corresponding Fellow of the Royal Academy of Surgery of Madrid; 8, Connaught-place West, Hyde-park. C. 1853-4.
FELLOWS OF THE SOCIETY.

Elected

1839 Frederick Le Gros Clark, Surgeon to, and Lecturer on Surgical Anatomy at, St. Thomas's Hospital; Surgeon to the Magdalen Hospital; Consulting Surgeon to the Western General Dispensary, and to the London Female Penitentiary, Pentonville; 24, Spring Gardens. S. 1847-9. V.P. 1855-6. Trans. 3.

1845 John Clark, M.D., Staff Surgeon, 1st Class. Canada.

1848 John Clarke, L.R.C.P., Physician to the British Lying-in Hospital; 42, Hertford-street, Mayfair.

1850 Josiah Clarkson, New Hall-street, Birmingham. Trans. 1.

1842 Oscar Moore Passey Clayton, 87, Harley-street.

1853 Joseph Thomas Clover, 44, Mortimer-street, Cavendish-sq.

1857 Charles Coates, F.R.C.P. Edinb., Physician to the Bath General Hospital; 10, Circus, Bath.

1851 Edward Cock, Senior Surgeon to, and Lecturer on Clinical Surgery at, Guy's Hospital; 12, St. Thomas's-street, Southwark. C. 1857. Trans. 3.

1850 Daniel Whitaker Cohen, M.D., Headley-grove, near Dorking.

1835 *William Colborne, Chippenham, Wiltshire.


1855 Frederick Collins, M.D., Wanstead, Essex.

1828 John Conolly, M.D., D.C.L., Consulting Physician to the Middlesex County Lunatic Asylum, Hanwell.

1840 *William Robert Cooke, Burford, Oxfordshire.

1820 Benjamin Cooper, Stamford.

1819 George Cooper, late Consulting Surgeon to the Middlesex County Lunatic Asylum; Brentford, Middlesex.

1841 George Lewis Cooper, Surgeon to the Bloomsbury Dispensary; 7, Woburn-place, Russell-square.

1843 William White Cooper, Senior Surgeon to the North London Eye Infirmary, and to the Honorable Artillery Company, and Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Mary's Hospital; 19, Berkeley-square. C. 1858.

1854 Charles Thomas Coote, M.D., Physician to the Great Northern Hospital, King's Cross; 1, Gloucester-place, Hyde-park.

xli.
Elected

1841 Holmes Coote, Assistant-Surgeon to St. Bartholomew’s Hospital, and to the Royal Orthopedic Hospital; 26, New Bridge-street, Blackfriars. S. 1853-4. Trans. 1.

1835 George Ford Copeland, Cheltenham.

1822 James Copland, M.D. F.R.S., Consulting Physician to the Royal Infirmary for Children, and to the Great Northern Hospital, King’s Cross; Hon. Fellow of the Royal Academy of Sciences of Sweden, &c.; 5, Old Burlington-street. C. 1831. V.P. 1838-9. P. 1853-4.

1847 John Rose Cormack, M.D. F.R.S.E., 27, Ampthill-square, Hampstead-road.

1839 Charles Caesar Corsellis, M.D. F.L.S., Benson, Oxon.

1853 William Gillett Cory, M.D., Burgh Heath, Sutton, Surrey.

1847 Richard Payne Cotton, M.D., Physician to the Hospital for Consumption and Diseases of the Chest; 46, Clarges-street, Piccadilly.

1828 William Coulson, Senior Surgeon to, and Lecturer on Surgery at, St. Mary’s Hospital; 1, Chester-terrace, Regent’s-park. C. 1831. L. 1832-7. V.P. 1851-2. Trans. 1.

1841 Mervyn Archdall Nott Crawford, M.D., Wiesbaden; C. 1853-4.

1847 George Critchett, Senior Assistant-Surgeon to, and Lecturer on Surgery at, the London Hospital, and Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 46, Finsbury-square. Trans. 1.

1837 John Farrar Crookes, Farewell, near Faversham, Kent.

1849 William Edward Crowfoot, Bectles, Suffolk.

1851 James Cameron Cumming, M.D., 1, Cadogan-place, Sloane-street.

1846 Henry Curling, Surgeon to the Margate Royal Sea-Bathing Infirmary, and the Ramsgate Seamen’s Infirmary; Ramsgate, Kent.

Elected

1847 John Edmund Currey, M.D., Lismore, County Waterford.
1836 George Cushman, M.D., Treasurer; Physician to the Hospital for Consumption and Diseases of the Chest and to the Female Orphan Asylum; and Inspector of Anatomy for the Provinces; 5, Savile-row, Burlington-gardens. S. 1842-7. C. 1850-1. V.P. 1855. T. 1856-7.
1822 Christopher John Cusack, Chateau d'Eu, France.
1852 Thomas Cutler, M.D., Acting Physician at the Spa Waters; Spa, Belgium.
1828 Adolphe Dalmas, M.D., Paris.
1836 *James Stock Daniel, Ramsgate, Kent.
1850 John Bampfylde Daniell, M.D.
1820 George Darling, M.D., 6, Russell-square. C. 1841-2.
1818 *Sir Francis Sacheverel Darwin, Knt., M.D., Deputy-Lieutenant of Derbyshire; Breadsall Priory, near Derby.
1848 Henry Daubeney, 40, York-place, Portman-square.
1846 Frédéric Davies, 19, Upper Gower-street, Bedford-square.
1847 John Davies, M.D., Physician Extraordinary to the Hertford General Infirmary, and Visiting Physician to the Hadham Palace Lunatic Asylum, Hertford.
1853 Robert Coker Nash Davies, Rye, Sussex.
1852 William Davies, M.D., Senior Physician to the Bath United Hospital; 10, Gay-street, Bath.
1852 John Hall Davis, M.D., Physician to the Royal Maternity Charity; Physician-Accoucheur to the St. George's and St. James's Dispensary; and Consulting Physician-Accoucheur to the St. Pancras Infirmary; 11, Harley-street, Cavendish-square.
1820 Thomas Davis, 28, Spring-gardens. C. 1837, 1843.
1818 James Dawson, Liverpool.
1847 George Edward Day, M.D. F.R.S., Chandos Professor of Anatomy, and Examiner in Medicine in the University of St. Andrew's.
1858 Teofilo Delima, M.D., Caracas, Venezuela, South America.
Fellows of the Society.

Elected

1846  *Samuel Best Denton, Ivy Lodge, Hornsea, East Riding, Yorkshire.

1844  Robert Dickson, M.D. F.L.S., Physician to the Scottish Hospital, and to the British Orphan Asylum, Clapham; 16, Hertford-street, Mayfair.

1839  James Dixon, Vice-President; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; and Consulting Ophthalmic Surgeon to the Asylum for Idiots; 2, Portman-square. L. 1849-55. V.P. 1857. Trans. 4.

1845  John Dodd.


1853  Robert Druitt, L.R.C.P., Medical Officer of Health for St. George's Hanover-square; 37, Hertford-street, Mayfair. Trans. 2.

1846  John Drummond, Deputy-Inspector of Fleets and Hospitals; Melville Naval Hospital, Chatham, Kent. Trans. 1.

1843  Thomas Jones Drury, M.D., Physician to the Salop Infirmary; Quarry-place, Shrewsbury.

1845  George Duff, M.D., High-street, Elgin.

1845  Edward Williamson Duffin, 14, Langham-place. Trans. 1.


1843  Christopher Mercer Durrant, M.D., Physician to the East Suffolk and Ipswich Hospital; Ipswich, Suffolk.

1839  Henry Sumner Dyke, M.D., 37, Bryanston-sq. C. 1854-5.

1836  James William Eable, Norwich.

1834  Booth Eddison, Surgeon to the Nottingham General Hospital, and Medical Attendant of Broom House Asylum; High Pavement, Nottingham.

1833  *George Edwardes, Wolverhampton. Trans. 1.

1824  George Edwards.

1823  Charles Chandler Egerton, Kendal Lodge, Epping.

1848  George Viner Ellis, Professor of Anatomy in University College, London; 15, Gloucester-street, Regent's-park. Trans. 2.

1854  *James Ellison, M.D., 14, High-street, Windsor.

1835  William England, M.D., Ipswich, Suffolk.
Elected


1836 **George Fabian Evans**, M.D., Physician to the General Hospital, Birmingham.

1815 *Griffith Francis Dorsett Evans*, M.D., St. Mary’s, Bedford. C. 1838.

1845 **William Julian Evans**, M.D.


1831 **Robert Furguson**, M.D., Physician Extraordinary to H.M. the Queen, and Consulting Physician to King’s College Hospital; 125, Park-street, Grosvenor-square. C. 1839. V.P. 1847.

1841 **William Fergusson**, F.R.S., Surgeon Extraordinary to H.M. the Queen; Surgeon in Ordinary to H.R.H. the Prince Consort; Professor of Surgery in King’s College, London, and Surgeon to King’s College Hospital; Consulting Surgeon to the Hospital for Consumption; Examiner in Surgery at the University of London; 16, George-street, Hanover-square. C. 1849-50. *Trans. 4.*

1852 *Alfred George Field*, 28, Old Steine, Brighton.

1849 **George Tupman Fincham**, M.D., Physician to, and Lecturer on Medical Jurisprudence at, the Westminster Hospital; 28, Chapel-street, Belgrave-square.


1838 **George Lionel Fitzmaurice**, 97, Gloucester-place, Portland-square.

1842 **Thomas Bell Elcock Fletcher**, M.D., Physician to the Birmingham General Hospital; Waterloo-street, Birmingham. *Trans. 1.*

Elected

1852 **John Cooper Forster**, Assistant-Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Royal Infirmary for Women and Children; 11, Wellington-street, Southwark. *Proc.* 1.

1820 **Thomas Forster**, M.D., Hartfield Lodge, East Grinstead.

1856 **John F. France**, Lecturer on Ophthalmic Surgery at Guy's Hospital, and Surgeon to the Eye Infirmary attached to the Hospital; 24, Bloomsbury-square.

1816 **John W. Francis**, M.D., LL.D., Professor of Materia Medica in the University of New York, U.S.

1841 **John Christopher Augustus Franz**, M.D., 11, Old Steine, Brighton.

1843 **Patrick Fraser**, M.D., Physician to the London Hospital, and to the London Dispensary; 61, Grosvenor-street.


1849 **Robert Temple Freere**, M.A., F.R.C.P., Physician-Acoucheur to, and Lecturer on Midwifery at, the Middlesex Hospital; 9, Queen-street, May-fair.

1846 **Henry William Fuller**, M.D., Physician to, and Lecturer on Medical Jurisprudence at, St. George's Hospital; 13, Manchester-square. *Trans.* 1.

1815 **George Frederick Furnival**, Medical Attendant of Great Foster House Asylum for Lunatics; Egham, Surrey.


1851 **George Gaskoin**, 3, Westbourne-park.

1819 **Henry Gaultier**.

1840 **John Gay**, Senior Surgeon to the Great Northern Hospital; 10, Finsbury-place South.
Elected

1821  *RICHARD FRANCIS GEORGE, Senior Surgeon to the Bath General Hospital; 10, Royal Crescent, Bath.

1854  BERNARD GILPIN, Belle Vue House, Ulverstone, Lancashire.

1858  BENJAMIN GODFREY, M.D., Carlton House, Enfield, Middlesex.

1851  STEPHEN JENNINGS GOFELLOW, M.D., Physician to, and Lecturer on Medicine at, the Middlesex Hospital; 4, Russell-square.

1818  JAMES ALEXANDER GORDON, M.D. C.B. F.R.S., Burford Lodge, Box Hill. C. 1828. V.P. 1829. Trans. 1.

1851  PETER YEAMES GOWLAND, Assistant-Surgeon to the London and St. Mark's Hospitals, Senior Surgeon to the Islington Dispensary; 34, Finsbury-square.

1844  JOHN GRANTHAM, Crayford, Kent.

1850  HENRY GRAY, F.R.S., Lecturer on Anatomy at St. George's Hospital Medical School, and Surgeon to the St. George's and St. James's Dispensary; 8, Wilton-street, Grosvenor-place. Trans. 2.

1846  GEORGE THOMPSON GREAM, M.D., 2, Upper Brook-street, Grosvenor-square.

1816  JOSEPH HENRY GREEN, D.C.L. F.R.S., President of the Royal College of Surgeons; Consulting Surgeon to St. Thomas's Hospital; Hadley, Middlesex. C. 1820. V.P. 1830. Trans. 1.

1843  ROBERT GREENHALGH, M.D., Consulting Physician-Accoucheur to the St. John's Wood Dispensary; 11, Upper Woburn-place, Russell-square.

1814  JOHN GROVE, M.D., Salisbury.

1852  JOHN GROVE, West Hill, Wandsworth, Surrey.

1849  WILLIAM WITHEY GULL, M.D., Senior Assistant Physician to, and Lecturer on Medicine at, Guy's Hospital, and Member of the Senate of the University of London; 8, Finsbury-square. Trans. 2.

1837  JAMES MANBY GULLY, M.D., Holyrood House, Great Malvern, Worcestershire.
Elected


1842 Charles William Gardiner Guthrie, Surgeon to the Royal Westminster Ophthalmic Hospital; 18, Pall Mall East.

1854 Samuel Osborne Habershon, M.D., Assistant-Physician to, and Lecturer on Materia Medica and Therapeutics at, Guy's Hospital; 22, Wimpole-street, Cavendish-square.

1849 Hammet Hailey, Newport Pagnell, Bucks.


1848 Alexander Halley, M.D. F.G.S., 7, Harley-street, Cavendish-square.


1838 Henry Hancock, Surgeon to, and Lecturer on Surgery at, the Charing Cross Hospital, and Surgeon to the Royal Westminster Ophthalmic Hospital; 37, Harley-street, Cavendish-square. C. 1851.

1849 *Richard James Hansard, Surgeon to the Radcliffe Infirmary; 5, Broad-street, Oxford.

1848 *George Harcourt, M.D., Chertsey, Surrey.

1836 John Fosse Harding, Sandford House, Highbury New Park. C. 1858.

1856 Charles John Hare, M.D., Assistant-Physician to University College Hospital; 41, Brook-street, Grosvenor-square.

1858 William Warwick Harkness, Demonstrator of Anatomy at the London Hospital Medical College; 9, Finsbury Circus.


1846 John Harrison, 2, the Court-yard, Albany.
Elected

1841 **William Harvey**, Surgeon to the Royal Dispensary for Diseases of the Ear, and to the Freemasons' Female Charity, and Aural Surgeon to the Great Northern Hospital; 2, Soho-square. C. 1854.


1855 **Alfred Haviland**, Surgeon to the Cannington Dispensary; Bridgewater, Somerset.


1848 **Thomas Hawksley**, M.D., Physician to the Margaret-street Dispensary for Consumption and Diseases of the Chest; 26, George-street, Hanover-square.

1820 **Thomas Emerson Headlam**, M.D., Consulting Physician to the Newcastle Infirmary, Newcastle-upon-Tyne.

1848 **James Newton Heale**, M.D., Physician to the Winchester County Hospital; Winchester, Hants.

1850 **George Heaton**, M.D., Boston, U.S.

1829 **Thomas Heberden**, M.D., 43, Park-street, Grosvenor-square.


1849 **Amos Henriques**, M.D., Hon. Physician to the Spanish Embassy; 67, Upper Berkeley-street, Portman-square.

1848 **Mitchell Henry**, Surgeon to, and Lecturer on Medical Jurisprudence at, the Middlesex Hospital, and Surgeon to the North London Eye Infirmary; 5, Harley-street, Cavendish-square. *Trans.* 2.

1821 **Vincent Herberski**, M.D., Professor of Medicine in the University of Wilna.
Fellows of the Society.

Elected

1843 Prescott Gardner Hewett, Professor of Anatomy and Surgery at the Royal College of Surgeons; Assistant-Surgeon to St. George's Hospital; 1, Chesterfield-street, May-fair. Trans. 7.

1855 W. M. Grailey Hewitt, M.D., Physician to the Samaritan Free Hospital for Women and Children; Lecturer and Registrar at St. Mary's Hospital; 17, Radnor-place, Hyde-park.

1853 Thomas Hewlett, Surgeon to Harrow School; Harrow. Trans. 1.

1841 Nathaniel Highmore, Sherborne, Dorsetshire.

1854 Thomas Hillier, M.D., Medical Officer of Health for St. Pancras, and Assistant-Physician to the Hospital for Sick Children; 21, Upper Gower-street.

1842 William Augustus Hillman, Senior Assistant-Surgeon to, and Lecturer on Anatomy and Physiology at, the Westminster Hospital; 1, Argyll-st., Regent-st. C. 1858.

1841 John Hilton, F.R.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital, and Consulting-Surgeon to the St. Pancras Royal General Dispensary; 10, New Broad-street, City. C. 1851. Trans. 3.

1848 Martin Thomas Hiscox, M.D., Bath, Somersetshire.

1840 Thomas Hodgkin, M.D., Consulting Physician to the Hospital for Diseases of the Skin, and Member of the Senate of the University of London; 35, Bedford-square. C. 1842-3. Trans. 6.


1843 Luther Holden, Demonstrator of Anatomy at St. Bartholomew's Hospital, and Surgeon to the Metropolitan Dispensary; 54, Gower-street, Bedford-square.

1814 Sir Henry Holland, Bart., M.D., D.C.L., LL.D. F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince Consort; 25, Brook-street, Grosvenor-square. C. 1817, 1833-4. V.P. 1826, 1840. Trans. 1.

1856 Timothy Holmes, Curator of the Pathological Museum of St. George's Hospital; 39, Curzon-street, May-fair. Trans. 1.
Elected

1846 **Barnard Wight Holt**, Senior Surgeon to, and Lecturer on Clinical Surgery at, the Westminster Hospital; Medical Officer of Health for Westminster; 5, Parliament-street.

1846 **Carsten Holthouse**, Surgeon to, and Lecturer on Anatomy at, the Westminster Hospital; Surgeon to the South London Ophthalmic Hospital; 2, Storey's-gate, St. James's-park.

1853 **William Charles Hood**, M.D., Resident Physician and Medical Superintendent of Bethlem Hospital.

1828 *Edward Howell*, M.D., Senior Consulting Physician to the Swansea Infirmary; 2, South Hill-place, Swansea, Glamorganshire.


1857 **Edward Charles Hulme**, Assistant-Surgeon to the Central London Ophthalmic Hospital, Surgeon to the Blenheim-street Dispensary, and Examining Surgeon to the Marine Society; 19, Gower-street, Bedford-square.

1844 **Edwin Humby**, 1, Windsor-terrace, Maida-Hill.


1820 **William Hutchinson**, M.D.

1840 **Charles Hutton**, M.D., Physician to the General Lying-in Hospital, and to the Royal Infirmary for Women and Children; 26, Lowndes-street, Belgrave-sq. C. 1858.


1856 **Cornelius Inglis**, M.D., House-Surgeon, Taunton Hospital; Taunton, Somerset.
Elected

1826 William Ingram, Midhurst, Sussex.

1845 *Henry Jackson, Senior Surgeon to the Sheffield General Infirmary; St. James's-row, Sheffield, Yorkshire.

1841 Paul Jackson, 24, Wimpole-street, Cavendish-square.

1841 Maximilian Morris Jacobovics, M.D., Vienna.

1825 John B. James, M.D.

1847 *William Withall James, Surgeon to the Exeter Dispensary; Exeter, Devonshire.

1844 Samuel John Jeffreson, M.D., Physician to the Warwick Hospital, and Warwick Dispensary; Leamington, Warwickshire.

1839 Julius Jeffreys, F.R.S., Kingston, Surrey.

1840 *George Samuel Jenks, M.D.

1851 William Jenner, M.D., Professor of Pathological-Anatomy in University College, London, and Physician to University College Hospital; Physician to the Hospital for Sick Children; 8, Harley-street, Cavendish-square. Trans. 2.

1848 Athol Archibald Wood Johnson, Lecturer on Anatomy and Physiology at St. George's Hospital Medical School, and Surgeon to the Hospital for Sick Children; 37, Albemarle-street. Trans. 1.

1851 Edmund Charles Johnson, M.D., Corresponding Member of the Imperial Society of Florence; 6, Savile-row.

1821 Sir Edward Johnson, M.D., Weymouth, Dorsetshire.

1847 George Johnson, M.D., Professor of Materia Medica and Therapeutics in King's College, London, and Physician to King's College Hospital; 3, Woburn-square. Trans. 3.

1837 Henry Charles Johnson, Surgeon to St. George's Hospital; 6, Savile-row, Burlington-gardens. C. 1850-1.

1853 Henry Jones, 23, Soho-square.

1844 Henry Bence Jones, M.D., F.R.S., Physician to St. George's Hospital; 31, Brook-street, Grosvenor-square. C. 1855-6. Trans. 11.

1835 Henry Derviche Jones, 23, Soho-square. C. 1854-5.
FELLOWS OF THE SOCIETY.

Elected

1853 Thomas Wharton Jones, F.R.S., Professor of Ophthalmic Surgery in University College, London, and Ophthalmic Surgeon to University College Hospital; 35, George-street, Hanover-square. Trans. 1.

1837 Thomas William Jones, M.D., Physician to the City Dispensary; 19, Finsbury-pavement. C. 1858.

1829 *George Charles Julius, Richmond, Surrey.

1816 *George Hermann Kauffmann, M.D., Hanover.

1848 *Daniel Burton Kendall, M.D., Senior Physician to the Wakefield General Dispensary; Kettlethorpe Hall, Wakefield, Yorkshire.

1847 Alfred Keyser, 21, Norfolk-crescent, Oxford-square.

1857 Henry Walter Kiallmark, late Staff Surgeon, 2d class, attached to the Ottoman Army; 32A, Fitzroy-square.

1839 *David King, M.D., Medical Officer of Health for Eltham; Eltham, Kent.

1851 John Abernethy Kingdon, Surgeon to the City of London Truss Society, and to the City Dispensary; 2, New Bank-buildings, City.

1840 Samuel Armstrong Lane, Surgeon to St. Mary's Hospital, and Surgeon to the Lock Hospital; 1, Grosvenor-place. C. 1849-50.

1855 James Robert Lane, Surgeon to, and Lecturer on Anatomy and Physiology at, St. Mary's Hospital; and Assistant-Surgeon to the Lock and St. Mark's Hospitals; 1, Grosvenor-place.


1816 G. E. Lawrence.

Elected

1840 Thomas Laycock, M.D. F.R.S.E., Professor of the Practice of Medicine in the University of Edinburgh, and Physician to the Edinburgh Royal Infirmary; 4, Rutland-street, Edinburgh.

1843 *Jesse Leach, Heywood, near Bury, Lancashire.

1823 John G. Leath, M.D.

1822 John Joseph Ledsam, M.D.


1843 Henry Lee, Surgeon to King’s College and the Lock Hospitals; 9, Savile-row, Burlington-Gardens. C. 1856-7. Trans. 2; Pro. 1.

1822 Robert Lee, M.D. F.R.S., Physician to the British Lying-in Hospital; Obstetric Physician to, and Lecturer on Midwifery at, St. George’s Hospital; and Corresponding Member of the Imperial Academy of Medicine, Paris; 4, Savile-row, Burlington-gardens. C. 1829, 1834. S. 1830-3. V.P. 1833. Trans. 20.

1836 Frederick Leighton, M.D., Frankfort-on-the-Maine.

1854 Hananel de Leon, M.D., 6, Victoria-terrace, Bedford.

1856 David Lewis, M.D., Physician to the Royal General Dispensary, Bartholomew Close, and to the Royal Society of Ancient Britons’ Schools; 23, Finsbury-place.

1847 Sir John Liddell, M.D. C.B. F.R.S., Director-General of the Medical Department of the Navy; Somerset House.

1806 John Lind, M.D.

1845 William John Little, M.D., Physician to the London Hospital; 34, Brook-street, Grosvenor-square.

1819 Robert Lloyd, M.D.


1824 Sir Charles Locock, Bart., M.D., President, First Physician-Accoucheur to H.M. the Queen, and Consulting Physician to the General Lying-in Hospital; Member of the Senate of the University of London; 26, Hertford-street, May-fair. C. 1826. V.P. 1841. P. 1837. Trans. 1.
Elected

1852 Charles Lodge, M.D.
1846 Henry Thomas Lomax, Surgeon to the 2d Staffordshire Militia, Stafford.
1836 Joseph S. Löwenfeld, M.D., Berbice.
1846 William M’Ewen, M.D., Surgeon to Chester Castle; 27, Nicholas-street, Chester.
1823 George Macilwain, Consulting Surgeon to the Finsbury Dispensary and the St. Anne’s Society’s Schools; 3, Court-yard, Albany. C. 1829-30. V.P. 1848. Trans. 1.
1848 Frederick William MacKenzie, M.D., Physician to the Western General Dispensary; 11, Chester-place, Hyde-park-square. Trans. 2.
1854 *Draper Mackinder, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.
1822 Richard Macintosh, M.D.
1844 Daniel MacLachlan, M.D., Physician to the Royal Hospital, Chelsea, and Deputy Inspector-General of Hospitals; Royal Hospital, Chelsea. Trans. 1.
1851 Samuel Maclean, 10, Conduit-street, Bond-street.
1849 Duncan MacLachlan Maclure, 16, Harley-street, Cavendish-square.
1842 John Macnaught, M.D., Bedford-street, Liverpool.
1837 Andrew Melville M’Whinnie, Assistant-Surgeon to, and Lecturer on Comparative Anatomy at, St. Bartholomew’s Hospital; and Assistant-Surgeon to the London Hospital for Diseases of the Skin, Blackfriars; 5, Crescent, New Bridge-street, Blackfriars. C. 1851-2. Trans. 1.
1855 William Marcet, M.D. F.R.S., Assistant-Physician to, and Lecturer on Medical Jurisprudence at, the Westminster Hospital; 36, Chapel-street, Belgrave-square. Trans. 1.
Elected

1848 William Orlando Markham, M.D., Physician to, and Lecturer on Physiology and General and Morbid Anatomy at, St. Mary's Hospital; 33, Clarges-street, Piccadilly. Trans. 1.

1824 Sir Henry Marsh, Bart., M.D., Physician to H.M. the Queen in Ireland, Consulting Physician to the City of Dublin Hospital, and Physician to Steevens's Hospital; 9, Merrion-square North, Dublin.

1838 Thomas Parr Marsh, M.D., Consulting Physician to the Salop Infirmary, Shrewsbury.

1851 John Marshall, F.R.S., Assistant-Surgeon to University College Hospital; 10, Savile-row, Burlington-gardens. Trans. 2.

1841 James Ranald Martin, F.R.S., 71a, Grosvenor-street. C. 1853.

1849 George Bellasis Masfen, Surgeon to St. Mary's Hospital, Manchester; 78, Oxford-street, Manchester.

1853 William Edward Masfen, Surgeon to the Staffordshire General Infirmary, Stafford.

1818 J. P. Maunoir, Professor of Surgery at Geneva. Trans. 4.


1852 James Merryweather, 57, Brook-street, Grosvenor-square.

1847 Edward Meryon, M.D., 14, Clarges-street, Piccadilly. Trans. 1.

1815 Augustus Meyer, M.D., St. Petersburgh.

1840 Richard Middlemore, Consulting Surgeon to the Birmingham Eye Infirmary; Temple Row, Birmingham.
Elected
1854 Edward Archibald Middleship, late of Richmond, Surrey.
1818 *Patrick Miller, M.D. F.R.S.Edin., Senior Physician to the Devon and Exeter Hospital, and to St. Thomas's Hospital for Lunatics; the Grove, Exeter, Devonshire.
1852 James Monro, M.D., Surgeon-Major, Coldstream Guards; Churton House, Churton-street, Belgrave-road. Trans. 1.
1844 Nathaniel Montefiore, 36, Hyde-park-gardens.
1848 Charles Hewitt Moore, Librarian, Surgeon to, and Lecturer on Anatomy at, the Middlesex Hospital; 35, Montague-place, Russell-square. Trans. 3.
1836 George Moore, M.D., late Physician to the Hastings Dispensary; Brighton.
1854 George Moseley (late of Sandgate).
1851 Frederick John Moutat, M.D., Professor of Medicine at the Medical College of Calcutta, and Secretary of the Council of Education in India; Calcutta.
1856 Charles Murchison, M.D., Assistant-Physician to King's College Hospital, and to the London Fever Hospital; 31, Sackville-street, Piccadilly. Trans. 2.
1847 Simon Murchison, Bicester, Oxon.
1845 Thomas D. Müttet, M.D., Emeritus Professor of Surgery in Jefferson Medical College; Philadelphia.
1835 Thomas Andrew Nelson, M.D., 10, Nottingham-terrace, York-gate, Regent's-park [54, Marylebone-road].
1843 Edward Newton, 30, Fitzroy-square.
1851 James Nicholls, 13, Savile-row, Burlington-gardens.
1819 *George Norman, Consulting Surgeon to the Bath United Hospital, and Surgeon to the Puerperal Charity; Circus, Bath. Trans. 3.
1849 Henry Burford Norman, Portland Lodge, Southsea, Hants.
1845 Henry Norris, Charmouth, Dorset.
1847 *William Edward Charles Nourse, Eltham, Kent.
1849 *Arthur Noverre, Great Stanmore, Middlesex.
1847 Thomas O'Connor, March, Cambridgeshire.
1843 William O'Connor, M.D., Physician to the Royal Free Hospital; 30, Upper Montagu-street, Montagu-square.

XLI.
Elected

1846 Francis Odling, 52, Devonshire-street, Portland-place.
1858 William Mackay Ogilvie, Boughton Blean, near Faversham, Kent.
1858 John William Ogle, M.D., Assistant-Physician to St. George’s Hospital; 13, Upper Brook-street, Grosvenor-square. Trans. 1.
1855 William Ogle, M.A. L.R.C.P., Physician to the Royal Pimlico Dispensary; 9, Lower Belgrave-street, Eaton-square.
1850 Henry Oldham, F.R.C.P., Obstetric Physician to, and Clinical Lecturer on Midwifery at, Guy’s Hospital; and Obstetric Physician to the Tower Hamlets Dispensary; 26, Finsbury-square. Trans. 1.
1842 William Piers Ormerod.
1846 *Edward Latham Ormerod, M.D., Physician to the Sussex County Hospital; 14, Old Steine, Brighton. Trans. 2.
1847 *William Bousfield Page, Surgeon to the Cumberland Infirmary; Carlisle. Trans. 2.
1840 James Paget, F.R.S., Surgeon Extraordinary to H.M. the Queen; Assistant-Surgeon to, and Lecturer on General and Morbid Anatomy and Physiology at, St. Bartholomew’s Hospital; 1, Harewood-place, Hanover-square. C. 1848-9. Trans. 8.
1806 *Robert Paley, M.D., Consulting-Physician to the Ripon Dispensary; Bishopston Grange, near Ripon, Yorkshire.
1836 S. W. Langston Parker, Surgeon to the Queen’s Hospital, Birmingham; Colmore-row, Birmingham.
1847 Nicholas Parker, M.D., Assistant-Physician to, and Lecturer on Medicine at, the London Hospital; 22, Finsbury-square.
1841 John Parkin, M.D., Paris.
1828 Richard Partridge, F.R.S., Professor of Anatomy to the Royal Academy of Arts; Surgeon to King’s College Hospital, and Professor of Anatomy in King’s College, London; 17, New-street, Spring-gardens. S. 1832-6. C. 1837-8. V.P. 1847-8.
Elected

1845  Thomas Bevill Peacock, M.D., Assistant-Physician to, and Lecturer on Materia Medica at, St. Thomas's Hospital; Physician to the City of London Hospital for Diseases of the Chest, Victoria-park; 20, Finsbury-circus. S. 1855-6. Trans. 2.

1856  Richard King Prince, Surgeon in Diseases of Women and Children to the Blenheim-street Dispensary; 16, Norfolk-place, Notting-hill.

1830  Charles P. Pelechini, M.D., St. Petersburgh.

1855  Oliver Pemberton, Surgeon to the Birmingham General Hospital, and Demonstrator of Anatomy at Queen's College; 11, Temple-row, Birmingham.

1844  William Vesalius Pettigrew, M.D., Surgeon to the Female Orphan Asylum, Lambeth; 7, Chester-street, Grosvenor-place.


1814  Edward Phillips, M.D., Consulting-Physician to the Haute County Hospital; Winchester, Hampshire.

1848  Edward Phillips, M.D. F.L.S., Physician to the Coventry and Warwickshire Hospital; Coventry, Warwickshire.

1852  Richard Phillips, Winchester-place, Claremont-square, Fentonville [68, Fentonville-road].

1854  Thomas Bacon Phillips, 36, Lansdown-place, Brighton.

1846  Francis Richard Philp, M.D., Nice.

1851  James Hollins Pickford, M.D. M.R.I.A., Physician to the Brighton Eye Infirmary; 1, Cavendish-pl., Brighton.

1851  John Picton, M.D.

1836  Isaac Pidduck, M.D., Physician to the Bloomsbury Dispensary; 22, Montague-street, Russell-square. Pro. 2.

1852  Henry Pilleau, Staff Surgeon, 1st Class; 21, Kensington-square. (India.)

1841  Henry Alfred Pittman, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital; 28, Montague-place, Russell-square. L. 1851-3.

1850  Alfred Poland, Assistant-Surgeon to Guy's Hospital, and Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Bolton-row, May-fair.
XXXIV FELLOWS OF THE SOCIETY.

Elected
1845 GEORGE DAVID POLLOCK, F.L.S., Assistant-Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; 27, Grosvenor-street. C. 1856-7. Trans. 1.
1843 CHARLES POPE, M.D. F.L.S., Glastonbury, Somersetshire.
1842 JAMES POWELL, M.B. 77, Guildford-street, Russell-square.
1851 ROBERT FRANCIS POWER, M.D., 7, Lower Grosvenor-place.
1857 WILLIAM OVREND PRIESTLEY, M.D., Physician-Accoucheur to the St. Marylebone Infirmary, and to the St. George's and St. James's Dispensary; 31, Somerset-street, Portman-square.
1839 JOHN PROPERT, Consulting-Surgeon to the Society of Ancient Britons; 6, New Cavendish-street, Portland-place.
1845 JOHN FYLE, 56, Oxford-terrace, Hyde-park.
1830 JONES QUAIN, M.D., Paris.
1850 RICHARD QUAIN, M.D., Physician to the Hospital for Consumption and Diseases of the Chest; 23, Harley-street, Cavendish-square. Trans. 1.
1852 CHARLES BLAND RADCLIFFE, M.D., Physician to, and Lecturer on Materia Medica at, the Westminster Hospital; 4, Henrietta-street, Cavendish-square.
1857 HENRY RANKE, M.D.
1854 WILLIAM HENRY RANSOM, M.D., Physician to the General Hospital; Nottingham.
1858 FREDERICK GEORGE REED, M.D., 46, Hertford-st., May-fair.
1821 HENRY REEDER, M.D., Ridge House, Chipping Sodbury.
Elected

1857  George Owen Rees, M.D. F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; Examiner in Materia Medica at the University of London; 26, Albemarle-street, Piccadilly. Trans. 1.

1835  G. Regnoli, Professor of Surgery in the University of Pisa.

1855  John Russell Reynolds, M.D., Assistant-Physician to the Westminster Hospital; 38, Grosvenor-street.

1847  Samuel Richards, M.D., 36, Bedford-square.

1852  Christopher Thomas Richardson, M.B., Physician to the Metropolitan Free Hospital; 16, Hinde-street, Manchester-square.


1849  *William Richardson, M.D., 9, Ephraim-road, Tunbridge Wells, Kent.

1845  Benjamin Ridge, M.D., 21, Bruton-street, Berkeley-square.


1852  Charles Ridley, Surgeon to the Royal Society for Protection of Life from Fire; 6, Charlotte-street, Bedford-sq.

1852  John Roberts, L.R.C.P., 75, Grosvenor-street.

1829  *Archibald Robertson, M.D. F.R.S., Hon. Physician to the Northampton General Infirmary, Northampton.

1855  Charles Alexander Lockhart Robertson, M.D., Medical Superintendent of the Sussex County Lunatic Asylum, and Hon. Secretary to the Association of Medical Officers of Asylums and Hospitals for the Insane; Hayward's Heath, Sussex.

1857  John George Robertson, Assistant Medical Officer, County Lunatic Asylum; Exminster, Devonshire.

1843  George Robinson, M.D., Physician to the Newcastle-on-Tyne Dispensary; Eldon-square, Newcastle-on-Tyne. Trans. 2.

1843  William Roden, M.D. F.L.S., the Grange, Kidderminster, Worcestershire.

1835  George Hamilton Roe, M.D., Senior Physician to the Hospital for Consumption and Diseases of the Chest; 57, Park-street, Grosvenor-square. C. 1841-2. Trans. 1.
Elected

1836 **Arnold Rogers**, Consulting Surgeon-Dentist to St. Bartholomew's Hospital; 16, Hanover-square.
1846 **William Richard Rogers**, M.D., Physician to the Farringdon General Dispensary; 56, Berners-street.
1819 **Henry Shuckburgh Roots**, M.D., Consulting Physician to St. Thomas's Hospital; 2, Russell-square. C. 1833, 1845. V.P., 1834-5. *Trans.* 1.
1829 **William Sudlow Roots**, F.L.S., Surgeon to H.M.'s Establishment at Hampton Court and to the Kingston Dispensary; Kingston, Surrey.
1850 **George Rofer**, 180, Shoreditch.
1836 **Richard Roscoe**, M.D.
1855 **Thomas Tattersall Roscow**, M.D., Physician to the Chelsea, Brompton, and Belgrave Dispensary; 1, Summer-place, Brompton.
1857 **Henry Cooper Rose**, M.D., High-street, Hampstead.
1845 **Henry Mortimer Rowdon**, Member of the Court of Examiners of the Society of Apothecaries; 29, Nottingham-place, [Marylebone-road].
1845 **James Russell**, M.D., Lecturer on Pathology and Therapeutics at Sydenham College; 91, New Hall-street, Birmingham.
1851 **Henry Hyde Salter**, M.D. F.R.S., Assistant-Physician to, and Lecturer on Physiology and Pathology at, the Charing Cross Hospital; 6, Montague-street, Russell-square.
1856 **Samuel James A. Salter**, F.L.S., Surgeon-Dentist to, and Lecturer on Dental Surgery at, Guy's Hospital; 17, New Broad-street, City. *Trans.* 1.
Fellows of the Society.

Elected

1849 Hugh James Sanderson, M.D., 26, Upper Berkeley-street, Portman-square.
1855 John Burdon Sanderson, M.D., Medical Officer of Health for Paddington; Lecturer on Medical Jurisprudence at St. Mary's Hospital; 9, Gloucester-place, Hyde-park.
1847 William Henry Octavius Sankey, M.D., Middlesex County Lunatic Asylum, Hanwell.
1845 Edwin Saunders, Surgeon-Dentist to H.M. the Queen and to H.R.H. the Prince Consort; 13A, George-street, Hanover-square.
1834 Ludwig V. Sautan, M.D., Warsaw.
1840 Augustin Sayer, M.D., Physician to the Lock Hospital; 28, Upper Seymour-street, Portman-square.
1853 Maurice Schulhof, M.D., Physician to the Royal General Dispensary, Bartholomew-close; 14, Brook-street, Grosvenor-square.
1858 George Scratchley, M.D., New Orleans, Louisiana, U.S.
1856 Edwin Sercombe, Surgeon-Dentist to St. Mary's Hospital; 49, Brook-street, Grosvenor-square. Trans. 1. Pro. 1.
1836 Alexander Shaw, Treasurer, Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 25, Henrietta-street, Cavendish-square. C. 1842. S. 1843-4. V.P. 1851-2. Trans. 3.
1848 Edward James Shearman, M.D., Rotherham, Yorkshire.
1849 Francis Stibson, M.D. F.R.S., Physician to, and Lecturer on Medicine at, St. Mary’s Hospital; 40, Brook-street, Grosvenor-square. Trans. 1.
FELLOWS OF THE SOCIETY.

Elected

1848 Edward Henry Sieveking, M.D., Physician to, and Lecturer on Materia Medica at, St. Mary's Hospital; 17, Manchester-square. *Trans. 1.*

1839 Thomas Hookham Silvester, M.D., Medical Officer to the Clapham General Dispensary; High-street, Clapham. C. 1854–5. *Trans. 1.*

1842 John Simon, F.R.S., Surgeon to, and Lecturer on Pathology at, St. Thomas's Hospital, Medical Officer of the Privy Council; 44, Cumberland-street, Bryanston-square. C. 1854–55. *Trans. 1.*

1857 James Lewis Siordet, M.B., Physician to the Blenheim Free Dispensary and Infirmary; 36, Queen Anne-street, Cavendish-square.

1827 George Robert Skene, Bedford.


1852 Charles Case Smith, Consulting Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk.

1854 Edward Smith, M.D. LL.B., Assistant-Physician to the Hospital for Consumption and Diseases of the Chest; 63, Grosvenor-street. *Trans. 4.*

1835 John Gregory Smith, Harewood, Leeds, Yorkshire.

1843 Robert William Smith, M.D., M.R.I.A., Professor of Surgery in the University of Dublin; Surgeon to the Richmond Hospital; 63, Eccles-street, Dublin.

1838 Spencer Smith, Secretary, Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital; 48, Sussex-gardens, Hyde-park. C. 1854. S. 1855–7.

1845 William Smith, Chesterfield, Derbyshire.

1847 William Smith, M.D., Consulting Physician to the Weymouth Infirmary, Weymouth, Dorsetshire.

1850 William Tyler Smith, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Mary's Hospital; 7, Upper Grosvenor-street. *Trans. 2.*

1851 John Soden, Surgeon to the Bath United Hospital, and Consulting Surgeon to the Bath Eye Infirmary; 24, Circus, Bath. *Trans. 2.*
Elected

1844 Frederick Robert Spackman, M.D., Harpenden, St. Alban's.
1834 James Spark, Italy.
1851 Robert John Spitta, M.B., Medical Officer to the Clapham General Dispensary; Clapham, Surrey. Trans. 1.
1843 *Stephen Spranger, 27, Henrietta-street, Bath.
1858 Joshua Harrison Stallard, L.R.C.P., Physician to the St. George's and St. James's Dispensary; 12, Welbeck-street, Cavendish-square.
1857 John Stanton, M.D., 7, Upper George-street, Bryanston-square.
1851 James Startin, Surgeon to, and Lecturer on Cutaneous Disorders at, the Hospital for Diseases of the Skin, Blackfriars; 3, Savile-row, Burlington-gardens.
1858 Edward Stephens, M.D., Consulting Surgeon to the Manchester Lying-in Hospital; 58, Bridge-street, Manchester.
1854 Henry Stevens, M.B., Resident Medical Officer, St. Luke's Hospital for Lunatics, Old-street.
1842 Alexander Patrick Stewart, M.D., Physician to, and Lecturer on Medicine at, the Middlesex Hospital; 74, Grosvenor-street. C. 1856-7.
1856 Alonzo Henry Stocker, M.D., Resident Medical Superintendent of Grove Hall Lunatic Asylum, Bow.
Fellows of the Society.

Elected

1858 J. F. Streatfeild, Assistant-Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 15, Upper Brook-street, Grosvenor-square.


1855 John Maule Sutton, M.D., Kent House, Tenby, South Wales.

1842 James Syme, F.R.S.E., Professor of Clinical Surgery in the University of Edinburgh, and Surgeon to the Edinburgh Royal Infirmary; 2, Rutland-street, Edinburgh. Trans. 3.

1854 Frederick Symonds, Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the Oxford Dispensary; 32, Beaumont-street, Oxford.

1844 Richard William T Amelia, Surgeon to the Royal Orthopaedic Hospital; 33, Old Burlington-street.

1848 Thomas Hawkes Tanner, M.D. F.L.S., 10, Charlotte-street, Bedford-square.

1852 Robert Taylor, Surgeon to the Central London Ophthalmic Hospital, and to the Cripple's Home, Hill-street; 10, George-street, Hanover-square.

1845 Thomas Taylor, Lecturer on Chemistry at the Middlesex Hospital Medical School; 4, Vere-street, Cavendish-square.


1845 *Evan Thomas, Chester-hill-road, Manchester.

1857 Henry Thompson, M.D., Assistant-Physician to, and Lecturer on Materia Medica at, the Middlesex Hospital; 75, Harley-street, Cavendish-square.

1852 Henry Thompson, Surgeon to the St. Marylebone Infirmary, and Assistant-Surgeon to University College Hospital; 16, Wimpole-street, Cavendish-square. Trans. 2.
Fellows of the Society.

Elected.

1839 Seth Thompson, M.D., 16, Lower Berkeley-street, Portman-square. C. 1849. S. 1850-1. V.P. 1857.

1842 Theophilus Thompson, M.D. F.R.S., Physician to the Hospital for Consumption and Diseases of the Chest; 3, Bedford-square. C. 1855-6. Trans. 4.

1835 Frederick Hale Thomson, Consulting Surgeon to the Westminster Hospital, and to the West London Institution for Diseases of the Eye; 4, Clarges-street, Piccadilly.


1850 Robert Dundas Thomson, M.D. F.R.S., Lecturer on Chemistry at St. Thomas's Hospital, Examiner in Chemistry at the University of London, and Medical Officer of Health for St. Marylebone; 41, York-terrace, Regent's-park. Trans. 2.

1836 John Thurnam, M.D., Resident Medical Superintendent of the Wilts County Asylum, Devizes, Wiltshire. Trans. 4.

1848 Edward John Tilt, M.D., Physician to the Farringdon General Dispensary and Lying-in Charity; 11, York-street, Portman-square.

1834 Robert Bentley Todd, M.D. F.R.S., Physician to King's College Hospital; 26, Brook-street, Grosvenor-square. L. 1842-6. T. 1850-1. V.P. 1854. Trans. 3.

1828 James Torrie, M.D., Aberdeen.

1843 Joseph Townsend, F.R.S., Aural Surgeon to, and Lecturer on Aural Surgery at, St. Mary's Hospital, Consulting Aural Surgeon to the Asylum for the Deaf and Dumb, and to the St. George's and St. James's General Dispensary; 18, Savile-row, Burlington-gardens. Trans. 6. Pro. 1.

1850 Samuel John Tract, Surgeon-Dentist to St. Bartholomew's and Christ's Hospitals; 28, Old Burlington-street.

1855 James Tulloch, M.D.

1835 John Cusson Turner, M.D., Bexley Heath, Kent.
Elected

1845 THOMAS TURNER, F.L.S., Consulting Surgeon to the Royal Manchester Infirmary, and Lecturer on Anatomy and Physiology at the Manchester Royal School of Medicine; 77, Mosley-street, Manchester.

1846 ALEXANDER URE, Surgeon to, and Lecturer on Clinical Surgery at, St. Mary's Hospital, and Consulting Surgeon to the Westminster General Dispensary; 18, Upper Seymour-street, Portman-square. Trans. 1.

1857 PHILIP JOHN VAN DER BYL, M.D., 1, Oxford-square, Hyde-park.

1819 BARNARD VAN OVEN, M.D., Consulting Surgeon to the Charity for Delivering Jewish Lying-in Women; 22, Manchester-square.

1806 BOYER VAUX, M.D.

1810 JAMES VOSE. Trans. 1.

1828 BENEDETTO VULPES, M.D., Physician to the Hospital of Aversa, and to the Hospital of Incurables, Naples.

1854 EDWARD WADDINGTON, Surgeon to the King's Own Staffordshire Rifles; Wakefield, Yorkshire.

1841 ROBERT WADE, Senior Surgeon to the Westminster General Dispensary; 68, Dean-street, Soho. Trans. 1.

1823 WILLIAM WAGNER, M.D., Berlin.

1820 THOMAS WALKER, M.D., Physician to the Forces; Morro Velhio, Brazil.

1852 WALTER HAYLE WALSHE, M.D., Professor of the Theory and Practice of Medicine in University College, London, and Physician to University College Hospital; Consulting Physician to the Hospital for Consumption; 40, Queen Anne-street, Cavendish-square. Trans. 1.

1851 HENRY HAYNES WALTON, Surgeon to the Central London Ophthalmic Hospital, and Surgeon to St. Mary's Hospital; 69, Brook-street, Hanover-square. Trans. 1. Pro. 1.

1852 DANIEL WANE, M.D., Obstetric Physician to the Blenheim-street Dispensary; 20, Grafton-street, Berkeley-square.
Elected


1821 William Tillbeard Ward, Duncannon House, Brighton.
1838 John Richard Wardell, M.D., St. John's Lodge, Regent's park.
1846 James Thomas Ware, Surgeon to the Finsbury Dispensary, and Hon. Surgeon to the Metropolitan Convalescent Institution; 51, Russell-square.
1818 John Ware, Clifton, near Bristol.
1814 Martin Ware, 51, Russell-square. C. 1844-5. T. 1846. V.P. 1853.
1829 Elias Taylor Warr, M.D., Yeovil, Somerset.
1837 Thomas Watson, M.D., Consulting Physician to King's College Hospital; 16, Henrietta-street, Cavendish-square. C. 1840-1, 1852. V.P. 1845-6.
1847 Thomas Watson, Holbeach, Lincolnshire.
1854 William Webb, M.D., late Resident Medical Officer of the Stafford General Infirmary; Wirksworth, Derbyshire.
1840 William Woodham Webb, M.D., Lowestoft, Suffolk.
1842 Frederick Weber, M.D., Assistant-Physician to the Middlesex Hospital, and Physician to the St. George's and St. James's Dispensary; 44, Green-street, Park-lane. C. 1857.
1857 Hermann Weber, M.D., Physician to the German Hospital; 49, Finsbury-square.
1835 John Webster, M.D. F.R.S., Physician to the Scottish Hospital, and Consulting Physician to the St. George's and St. James's Dispensary; 24, Brook-street, Grosvenor-square. C. 1843-4. V.P. 1855-6. Trans. 5.
1844 William Wegg, M.D., Librarian; Physician to the St. George's and St. James's Dispensary; 49, Maddox-street, Hanover-square. L. 1854-7.
1854 Thomas Spencer Wells, Lecturer on Surgery at the Grosvenor-place School of Anatomy and Medicine, and Surgeon to the Samaritan Free Hospital for Women and Children; 3, Upper Grosvenor-street. Trans. 1.
Elected

1816 Sir Augustus West, Knt., M.D., Deputy-Inspector of Army Hospitals to the Portuguese Forces; Paris.

1842 Charles West, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Bartholomew's Hospital; and Physician to the Hospital for Sick Children; 61, Wimpole-street, Cavendish-square. C. 1855-6. Trans. 2.

1841 Thomas West, M.D., Daventry, Northamptonshire.

1828 John Whatley, M.D.

1849 John White.

1852 John Wiblin, Medical Inspector of Emigrants and Recruits; 73, Morland-place, Southampton.

1824 *William John Wickham, Consulting Surgeon to the Haute County Hospital; Winchester. Trans. 1.

1844 Frederick Wildbore, 1, Trafalgar-place-east, Hackney-road.

1837 George Augustus Frederick Wilks, M.D.

1840 Charles James Blasius Williams, M.D. F.R.S., Consulting Physician to the Hospital for Consumption; 49, Upper Brook-street, Grosvenor-square. C. 1849-50.

1829 Robert Willis, M.D., Barnes, Surrey. L. 1838-41.

1839 Erasmus Wilson, F.R.S., Consulting Surgeon to the St. Pancras Infirmary; 17, Henrietta-street, Cavendish-square. Trans. 2.

1839 James Arthur Wilson, M.D., Vice-President; 28, Dover-street, Piccadilly. C. 1846-7. Trans. 4.

1850 *Robert Stanton Wise, M.D., Consulting Physician to the Southern Eye and Ear Infirmary; Banbury, Oxfordshire.


1851 John Wood, Assistant-Surgeon to King's College Hospital; 4, Montague-street, Russell-square.

1841 George Leighton Wood, Surgeon to the Bath General Hospital; 27, Queen-square, Bath.


1843 John Ward Woodfall, M.D., Physician to the West Kent Infirmary; Maidstone, Kent.
Elected

1833 Thomas Wormald, Assistant-Surgeon to St. Bartholomew's Hospital, and Surgeon to the Foundling Hospital; 42, Bedford-row. C. 1839. V.P. 1854.

1842 William Collins Worthington, Senior Surgeon to the Infirmary, Lowestoft, Suffolk. Trans. 3.

1848 Edward John Wright, 13, Montague-place, Clapham-road.

1855 Henry G. Wright, M.D., Physician to the St. Pancras Royal General Dispensary; 23, Somerset-street, Portman-square.

[It is particularly requested, that any change of Title or Residence may be communicated to the Secretaries before the 1st of August in each year, in order that the List may be made as correct as possible.]
HONORARY FELLOWS.

(Elected)

1841 WILLIAM THOMAS BRANDE, D.C.L. F.R.S., Hon. Professor of Chemistry at the Royal Institution of Great Britain, Examiner in Chemistry, and Member of the Senate of the University of London; Royal Mint, Tower-hill.


1853 BENJAMIN COLLINS PRODIE, B.A. F.R.S., Aldrichian Professor of Chemistry in the University of Oxford.

1847 EDWIN CHADWICK, late Commissioner of the Board of Health.

1835 MICHAEL FARADAY, D.C.L. F.R.S., Corresp. Memb. Institute of France, Member of the Senate of the University of London, and Fullerian Professor of Chemistry in the Royal Institution.


1841 SIR JOHN FREDERICK WILLIAM HERSCHEL, Bart., D.C.L. F.R.S., Corresp. Memb. Institute of France; Collingwood, near Hawkhurst, Kent.


1847 RICHARD OWEN, D.C.L. LL.D. F.R.S., Corresp. Memb. Institute of France; Superintendent of the Natural History Department in the British Museum; Sheen Lodge, Mortlake.

1835 The Rev. ADAM SEDGWICK, A.M. F.R.S., &c., late Woodwardian Professor of Geology, Cambridge.

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected

1841 G. ANDRÉ, M.D., Member of the Institute and of the Imperial Academy of Medicine, Physician in Ordinary to the Emperor of the French, Professor of Pathology in the Faculty of Medicine, and Physician to the "Hôpital de la Charité;" Paris.

1856 BARON PAUL DUBOIS, Commander of the Legion of Honour, Member of the Imperial Academy of Medicine, Dean of, and Professor of Clinical Midwifery in, the Faculty of Medicine, Paris.

1835 CARL JOHAN EKSTRÖMER, M.D. C.M. K.P.S. and W., Physician to the King of Sweden, President of the College of Health, and Director General of Hospitals; Stockholm.

1841 CHRISTIAN GOTTFRIED EHRENBERG, Berlin.

1835 BARON ALEXANDER VON HUMBOLDT, Member of the Institute of France, &c., Berlin.

1841 JAMES JACKSON, M.D. LL.D., Emeritus Professor of Medicine in the University of Cambridge, Boston, U.S.

1856 BERNHARD LANGENBECK, M.D., Professor of Surgery in the University of Berlin.

1843 BARON JUSTUS VON LIEBIG, M.D., Professor of Chemistry in the University of Munich.

1841 P. C. A. LOUIS, M.D., Honorary Physician to the Hôtel-Dieu, Member of the Imperial Academy of Medicine, Paris.

1847 CARLO MATTEUCCI, Professor in the University of Pisa, Member of the Institute of France.

1853 VALENTINE MOTT, M.D., Emeritus Professor of Surgery in the University of New York, late President of the New York Academy of Medicine; New York.

1841 BARTOLOMEO PANIZZA, M.D., Pavia.
Elected
1850 Carl Rokitansky, M.D., Curator of the Imperial Pathological Museum and Professor at the University of Vienna.
1856 Louis Stromeyer, M.D., Director-General of the Medical Department of the Army of Hanover; Hanover.
1835 Friedrich Tiedemann, M.D., Frankfort-on-the-Maine.
1856 A. Vulpian, Member of the Institute, and of the Imperial Academy of Medicine, Professor in the Faculty of Medicine, Surgeon to the "Hôpital de la Charité;" Paris.
1856 Rudolph Virchow, M.D., Professor of Pathological Anatomy in the University of Berlin.
CONTENTS.

List of Officers and Council .............................................................. v
List of Referees ........................................................................... v*
List of Presidents of the Society ..................................................... vi
List of Fellows of the Society .......................................................... vii

I. An Account of a Case of Arterio-venous Aneurism of the Temporal Vessels, which was treated by Ligature of both the Artery and the Vein. By Charles H. Moore, Surgeon to the Middlesex Hospital .................................................. 1

II. Case of Communication with the Stomach, through the Abdominal Parietes, produced by Ulceration from External Pressure; with Observations on the Cases of Gastro-cutaneous Fistula already recorded. By Charles Murchison, M.D. Edin., L.R.C.P., Assistant-Physician to King's College Hospital, and to the London Fever Hospital ................................................................. 11

III. Therapeutic Communications.—No. I. On the Influence of Liquor Potassae, and other Caustic Alkaline Solutions, upon the Therapeutic Properties of Henbane, Belladonna, and Stramonium. By Alfred B. Garrod, M.D., F.R.S., F.R.C.P., Professor of Materia Medica, Therapeutics, and Clinical Medicine in University College; Physician to University College Hospital ................................................................. 53

IV. Therapeutic Communications.—No. II. On the Influence of Liquor Potassae, and other Caustic Alkaline Solutions, upon the Therapeutic Properties of Henbane, Belladonna, and Stramonium. By Alfred B. Garrod, M.D., F.R.S., F.R.C.P., Professor of Materia Medica, Therapeutics, and Clinical Medicine, in University College; Physician to University College Hospital ................................................................. 61


VI. On some Points in the Pathology and Morbid Anatomy of Glaucoma. By J. W. Hulke, Assistant-Surgeon to King's College Hospital ........................................................................ 111
VII. On the Analysis and Immediate Principles of Human Excrements in the Diseased State. By W. Marcet, M.D., F.R.S., Assistant-Physician to the Westminster Hospital, &c. 119

VIII. On the Membrana Decidua which surrounds the Ovum in Cases of Tubal Gestation. By Robert Lee, M.D., F.R.S., Physician to the British Lying-in Hospital, and Obstetric Physician to St. George’s Hospital 137

IX. Supplement to a Paper on the Membrana Decidua which surrounds the Ovum in Cases of Tubal Gestation. By Robert Lee, M.D., F.R.S. 153

X. On the Action of Galvanism upon the Contractile Structure of the Gravid Uterus, and its Remedial Powers in Obstetric Practice. By F. W. Mackenzie, M.D., L.R.C.P., Physician to the Western General Dispensary and to the Paddington Maternity 157

XI. A Case of Complete Inversion of the Uterus, of nearly Twelve Years’ Duration, successfully treated. By W. Tyler Smith, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Mary’s Hospital 183

XII. On Excision of the Knee. By George Murray Humphry, Surgeon to Addenbrooke’s Hospital, and Lecturer on Anatomy and Surgery in the Medical School, Cambridge 193

XIII. Contributions to the Etiology of Continued Fever: or an Investigation of various Causes which influence the Prevalence and Mortality of its different Forms. By Charles Murchison, M.D. Edin., L.R.C.P., Assistant-Physician to King’s College Hospital, and to the London Fever Hospital 219

XIV. Case of Hydatids of the Tibia. By William Coulson, Senior Surgeon to, and Lecturer on Surgery at, St. Mary’s Hospital 307

XV. A Case of Fibrous Polypus of the Urinary Bladder, with Observations, and a Table of the Recorded Cases. By John Birkett, Surgeon to Guy’s Hospital; Corresponding Member of the Société de Chirurgie de Paris 311

XVI. Researches on Gout.—Part I. The Urine in the Different Forms of Gout.—Part II. The Influence of Colchicum upon the Urine. By Alfred B. Garrod, M.D., F.R.S., F.R.C.P., Professor of Materia Medica, Therapeutics, and Clinical Medicine in University College; Physician to University College Hospital 325

XVII. Contributions to the Pathology of the Glandular Structures of the Stomach. By Wilson Fox, M.D., B.A. Lond., of Newcastle-under-Lyme, Physician to the North Staffordshire Infirmary. (Communicated by A. B. Garrod, M.D. 361
CONTENTS

XVIII. On the Influence of the Cervical Portions of the Sympathetic Nerve and Spinal Cord upon the Eye and its Appendages, illustrated by Clinical Cases, with Observations. By John W. Ogle, M.D., Assistant-Physician to St. George's Hospital

XIX. An Account of a Case of Calculus in the Bladder removed by Lithotrity, in which a Communication existed between the Bladder and Intestine. By Charles Hawkins, Vice-President of the Society; Consulting Surgeon to Queen Charlotte's Hospital; Inspector of Anatomy, &c.

XX. On a Case of Dislocation of the Humerus, upwards and inwards, with Fracture of the Coracoïd Process of the Scapula; accompanied by a Dissection of the Parts involved in the Injury. By T. Holmes, M.A. Cantab., F.R.C.S., Curator of St. George's Hospital Museum

XXI. On a Case of Premature Puberty. By Robert Bath Smart, M.R.C.S. (Communicated by Richard Quain, F.R.S., &c.)

Index

Page

397
441
447
455
463
AN ACCOUNT OF A CASE
OF
ARTERIO-VENOUS ANEURISM
OF THE
TEMPORAL VESSELS,
WHICH WAS TREATED BY LIGATURE OF BOTH THE ARTERY AND THE VEIN.

BY
CHARLES H. MOORE,
SURGEON TO THE MIDDLESEX HOSPITAL.

Received October 31st, 1857.—Read November 11th, 1857.

Exactly a century has elapsed since Dr. William Hunter first recognised and described those aneurisms, in which a direct communication exists between the artery and a vein. The propriety of abstaining from surgical interference, which he laid down as the ordinary rule of practice in such cases, has since been amply tested; and many a patient has in consequence been spared the suffering and the danger attending the performance of an unnecessary operation. When the communication between the vessels is direct, there is, indeed, no reason for expecting danger to threaten either life or limb; the vein may be greatly distended and tortuous, but, unlike an aneurism, it does not burst; for the arterial blood, finding in the vein an open channel, better fitted for its conveyance than for its coagulation,
may and does continue for years to make its way harmlessly back to the heart. The longest period during which such a condition of the vascular system of a part has existed in any case is, so far as I can ascertain, in the instance I am about to relate to the Society, in which the arterio-venous communication had been made thirty-six years before the patient came under my care.

Under what circumstances a disorder, usually long innocuous, shall become so changed as to imperil life, and demand prompt surgical treatment, has been but partly ascertained. Most frequently, perhaps, the disastrous change has been produced by inefficient treatment, as when a ligature has been placed on but one (the upper) orifice of the artery. Other causes of danger are spontaneously engendered in the affected parts, and arise slowly from alterations of nutrition. Thus sometimes, as life advances, the coats of the varicose vein become so expanded and thin as to be no longer capable of resisting the ceaseless pulse of the arterial current; sometimes degeneration and softening occur in coagula, which may have formed, and the cavity of the vein is opened by suppuration. One or both of these last changes appear to have been the cause of the dangerous hæmorrhage which took place in the following case.

Thomas A—, a labourer, aged 60, in ordinary general health, but pale, very feeble and depressed in consequence of hæmorrhage, was brought from Northamptonshire, on the 30th September, 1857, by Mr. Jones, surgeon, of Brackley.

The patient's left temporal artery had been opened thirty-six years before, by a surgeon in Australia, for pain in the head. The bleeding returned two or three times after the operation, and was ultimately stopped by constant pressure; but a small swelling, which had formed in the site of the wound, never again subsided. It always pulsed synchronously with the heart, and the patient often observed that he could stop the beating of the tumour by pressing with
his finger behind the left ramus of the jaw. For the two
or three succeeding years, an enlargement, as of vessels,
took place, reaching the size of the little finger, and a
peculiar sound was noticed in the swelling, which was
audible by the bystander and by the patient. In the course
of years the swelling attained the size of the man’s fist;
but, although the vessels of the temple had assumed a most
formidable appearance, the tumour never occasioned him
any material suffering until last July. An abscess then
formed in the diseased part; the skin covering a large part
of it became thin, and at length gave way at a small spot.
Hæmorrhage did not take place until three weeks after the
first discharge of matter, but when it did occur, a quantity
of blood was rapidly lost, which there is reason for estimating
at four pints. Mr. Jones removed an immense quantity of
coagula, and applied pressure, under which the hæmorrhage
ceased. A fortnight afterwards the bleeding returned, and
extremely exhausted him, but it was again arrested; and as
soon as he could bear the journey, he was removed to
town, and admitted under my care into the Middlesex
Hospital.

I found the left half of the scalp much swollen, partly
by œdema, but above and behind the ear by a large abscess.
On removing the bandages, an ulcer was seen in the temple,
equal in size to a crown-piece, and formed of flabby granu-
lations. A stream of pus issued at a small opening near its
posterior border: towards the front a clot of blood adhered,
and showed the spot at which the blood had escaped.
Above, below, and in front of the ulcer, as well as beneath
the clot of adherent blood at its base, tortuous blood-vessels,
as large as the brachial artery, could be felt beating with a
bounding, soft pulsation. When pressure was made in the
parotid space, they ceased to beat, and their soft walls could
be but indistinctly felt among the loose and flaccid tissues
in which they lay. No purring tremor was felt in the veins
of the scalp; if any such movement existed, it was effectually
concealed by the general œdema of that part. In the neck,
however, two enormously dilated veins descended in the
course of the external jugular, and in them an arterial pulsation, which was most marked in the higher parts of the neck, and also a continuous vibratile thrill, were distinctly visible. Neither arteries nor veins were enlarged in the frontal, posterior auricular, or occipital regions.

Although there had been no haemorrhage for a fortnight, yet as the man suffered severe pain from the pressure which was employed, as he was under great apprehension of a recurrence of the bleeding, and as there was good reason to think a third gush of blood might place him beyond help, I had no hesitation as to the propriety of preventing such haemorrhage by ligature. The usual and proper operation on wounded arteries, viz., that of tying the artery immediately above and below the aperture in it, did not appear applicable in this case, as it was most unlikely that vessels which had been so long diseased, and had become so thin, would bear a ligature. Two considerations diverted me from tying the common carotid artery; one of which was the importance of the internal carotid stream to the nutrition of the brain; the other, that as none but the temporal artery was involved in the disease, it would suffice to secure that vessel, or the external carotid, at the highest point at which a healthy condition of its coats and the practicability of tying it coincided. That point appeared, on examination, to be in the parotid gland, a little below the external meatus of the ear.

A vertical incision behind the ramus of the jaw needed to be carried to a considerable depth before the vessel which had been felt beating was reached. It lay a little deeper than the edge of the ramus. I carried a ligature round it, and, before tying the knot, felt the pulsating vessel in the loop. When my finger was pressed into the loop, the vessels in the temple ceased to beat; but after I had tied the knot, they continued to pulsate as freely as before. Having repeated the operation with the same result, I examined the vessel again, and found it beating only on the distal side of the ligatures. This singular circumstance removed all doubt of the vessel having been
satisfactorily tied; it also disclosed the fact that the tied yet pulsating vessel must be the vein on the cardiac side of its communication with the temporal artery. The latter vessel, which led to the aneurism, was then felt for elsewhere, and was found in front of the wound, near the posterior edge of the masseter muscle. It was undoubtedly the temporal artery, tortuous and consequently much displaced; and it was situated where it might readily have been compressed, when the finger was introduced into the wound. I passed a ligature round it; and upon my tying the knot, the pulsation in the temple, and in the vessel previously tied, immediately ceased. Upon examining the jugular veins, I found them diminished in size, and entirely without their former pulsation and thrill.

The patient complained of pain in the evening, but after taking an opiate he passed a good night. His jaw was tied up with a bandage; he was fed freely with beef tea or minced meat, and took a little wine; and he went on very favorably. The discharge from the abscess rapidly diminished; the ligatures came away on the tenth and thirteenth days; the ulcers assumed a healthy appearance, and contracted to the size of a shilling; the oedema of the scalp disappeared; and the temple, no longer swollen with pulsating vessels, looked shrivelled, and shrank beneath the level of the ulcer. He did not, however, gain strength rapidly, and on the 15th October he had a slight attack of erysipelas, which commenced at the wound, and spread across the face. Nevertheless, though still feeble, he was able to leave the hospital on the 19th. No pulsation then existed in any of the diseased temporal vessels.

In November the man was at work as an agriculturist, and perfectly well.

The condition of the circulation in cases of circumscribed varicose aneurism is peculiar, and modifies the treatment admissible in them. Two parallel tubes, having their currents unequal in force and opposite in direction, communicate with one another. The arterial blood flows into the
vein with varying freedom in different cases, a freedom perhaps dependent on the size and directness of the opening by which the two vessels communicate. From the opening onward the force of the current in the artery is diminished, whilst in the vein the stream is reversed below the wound, and augmented and accelerated above it. The state of the circulation in the distal parts of the vein is well shown in Larrey's case of a sword-wound at the root of the neck, by which the subclavian artery and vein were opened into one another. When venesection was performed on the external jugular of the same side, the blood which issued was of scarlet colour. Upon this circumstance probably depends the character of the nutrition sometimes observed in the parts beyond the disease. Far from being wasted, as might be expected from the weakness of the circulation in the distal arteries, and from the proneness of the limb to gangrene upon the application of a single ligature on the higher part of the artery, a part affected with varicose aneurism is rather over-nourished. Broca mentions that the hair is unusually abundant on limbs whose trunk-vessels communicate, and that in some instances the limbs themselves grow to an unnatural length.

The treatment which is admissible in these cases, is governed by the peculiar state of the circulation. So far as I have ascertained, the modes of successful treatment which have been adopted are excision of the whole diseased vessels, compression, and the ligature. By excision the disease was in one instance removed from the scalp. The swelling was of course small, and the cure complete. Compression is found to produce its effects upon the vein. In one instance that vessel became obliterated below the point of its communication with the artery; in another instance the entire vein was closed. By means of the ligature, the artery has been tied above the unnatural aperture in it. It has also been tied both above and below that point, the ligatures having sometimes been placed close to the orifice in the artery, sometimes at a distance from it. In one instance the vein
also was included in one of the ligatures. In the case re-
lated in this paper, the artery and vein were both tied
on the cardiac side of their communication with one
another.

The results of these various combinations of treatment
are also varied. A brief survey of them will serve to show
what surgical value may attach to the case I have
detailed.

When the artery is tied above and below the opening, the
disease is cured; unless, indeed, an anastomosing branch
join the artery between the ligatures, in which case arterial
blood still continues to pass into the vein, and the operation
is unsuccessful.

The entire obliteration of the vein at the seat of the
opening suffices equally with the closure of the whole artery
for the cure of the disease. M. Nélaton undertook the
treatment of a case of arterio-venous aneurism at the bend
of the elbow by direct compression upon the wounded
vessels, aided by pressure upon the artery in the higher part
of the arm. After several months of treatment he suc-
cceeded in curing the disease, and at a post-mortem examina-
tion of the patient, two years afterwards, the vein was found
obliterated at the site of the aneurism, the artery was healed
and pervious.¹

There are but two classes of case in which one orifice
only of either vessel has been closed. They are that in
which the artery has been tied by a single ligature above
the aperture, and that in which, by compression, the vein
has been obliterated below it. Both modes of treatment
are unsuccessful. In the former case the collateral cir-
ocation fails to nourish the limb; not because it is de-
ficient either in amount or in force, but because, so long as
the artery and vein communicate, the blood continues to
find a more ready retrograde passage through the opening
into the vein, than into distant and narrow capillaries.
The ligature weakens the impulse of the arterial current,

¹ 'Gazette des Hôpitaux,' Sept. 22, 1855, and Sept. 12, 1857.
the very force which, up to the time of the operation, had secured the conveyance of the blood to the remoter parts of the limb. A frequent issue of the treatment, consequently, is gangrene of the limb; and, if that should be escaped, the disease is in no way improved. In one case, indeed, after the single ligature above the disease had been thrice unsuccessfully employed, a cure was effected by direct compression; whilst in another case the arterial current regained so much freedom and force through collateral channels, as to produce an aneurism in the lower part of the limb.

Two cases which have been reported in the Society's 'Transactions,' stand in interesting contrast to one another. Mr. Perry employed pressure over a part of the femoral artery, at which that vessel opened into the femoral vein. The lower orifice of the latter vessel became obliterated, and the thrill in the veins of the limb diminished. No cure of the disease, however, followed this partial closure of the vein, and an aneurism which had previously existed in the posterior tibial artery, suddenly enlarged, and required the ligature of the femoral artery. Mr. Cock, on the other hand, relates a case in which, eleven years after the ligature of the femoral artery on account of a wound of the popliteal artery and vein, the consequence of the persistence of a communication between the trunk vessels in the ham was seen in the formation of an aneurismal dilatation of the posterior tibial vein, which led to suppuration and arterial hemorrhage, and required amputation of the limb. Both cases illustrate the insufficiency of any treatment which obliterates one orifice only of either vessel.

The only remaining mode of successful treatment, but a mode which could hardly be recommended for varicose aneurisms of the limbs, is that of obliterating both the arterial and venous trunks on the cardiac side of the

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1 Lallemand, 'Gaz. Méd. de Paris,' 1837, p 347.
unnatural communication between them. That communication does, indeed, still exist, yet, since the direct access of blood to it by the artery, and the return by the vein, are prevented by the ligature of those vessels, the success of the procedure may reasonably be expected to be permanent. The unnatural aperture, being beyond both ligatures, is reduced in importance to that of a passage between subordinate and branch vessels, and will probably be entirely obliterated.
CASE

OF

COMMUNICATION WITH THE STOMACH,

THROUGH THE

ABDOMINAL PARIETES,

PRODUCED BY ULCERATION FROM EXTERNAL PRESSURE;

WITH OBSERVATIONS ON THE CASES OF GASTRO-

CUTANEOUS FISTULÆ ALREADY RECORDED.

BY

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Received 9th November.—Read 24th November, 1807.

In May of the present year, when on a visit to Aberdeen, I had frequent opportunities of seeing, and making observations on, the patient whose extraordinary case is detailed in this paper. She was then in the Aberdeen Royal Infirmary, under the care of my friend, Dr. W. Keith, the senior surgeon. I am indebted for the following details as to the patient's history to Dr. Keith, who has also kindly permitted me to bring the case before the notice of the Royal Medical and Chirurgical Society.

I have also appended to the paper a collection, arranged in a tabular form, of twenty-five cases of fistulous communications between the stomach and external surface, with some general observations deducible from these cases.
A. Case of Catherine Ross.

Catherine Ross, set. 34 (born June 16th, 1822), a native of Aberdeen, was admitted into the Aberdeen Royal Infirmary, under the care of Dr. Keith, February 19th, 1857, with a large fistulous opening in the walls of the abdomen, situated between the scrobiculus cordis and umbilicus, and communicating in the freest possible manner with the interior of the stomach.

1. Family history.—Her father and mother are both alive. They have had five sons and five daughters. Two of the sons and two of the daughters died in infancy. She has a married sister subject to some convulsive affection, probably hysterical. She has also a brother afflicted with epilepsy, and an unmarried sister little better than an idiot. The parents themselves are in good health of mind and body—always sober and industrious.

2. Previous individual history.—This woman has on more than one occasion made herself noted by feigning diseases, so as to deceive her friends and medical attendants. In 1840, she was, for a period of three months, under medical treatment for hysteria in its most exaggerated form, being often convulsed and frequently cataleptic. She would pass eight, ten, or even fourteen hours in a seemingly unconscious state of mind, frequently conjoined with a constrained and unusual position of body. In 1844, after various attempts on her lower limbs, she succeeded in producing such a solid edematous enlargement of the left upper extremity, as to deceive her friends, and mislead for two years more than one medical attendant, into the belief that she laboured under elephantiasis. During that period, severe and long-continued blistering was employed, and every conceivable mode of treatment had recourse to, with no perceptible alteration in the size of the limb. The cicatrices resulting from the blisters, becoming distended with effused fluid, and consequently rugged looking and elevated above the level of the surrounding skin, did more than anything else to complete the vraisemblance of actual elephantiasis. Wearied
out by a fruitless attendance, her medical advisers readily acquiesced in a proposal, emanating from herself, that the limb should be amputated at the shoulder-joint; and Dr. Keith was requested to see the case, with a view to her removal to the hospital. He pointed at once to the abrupt termination of the swelling at the insertion of the deltoid muscle, as demonstrative of an artificial enlargement; but as it would scarcely have been credited that the patient could, for two whole years, have kept up such a deception in the very midst of her family, no suspicion was betrayed in their presence, and she was admitted into a small ward of the Aberdeen Infirmary, on the 25th of August, 1846, the entry of her disease made in the Case Book being "Feigned elephantiasis." Being in the hospital at seven next morning, Dr. Keith walked suddenly into her ward, and found her hurriedly concealing a garter under her pillow. The fact of her having been in bed for two years, with no stockings on all that time, left no room to doubt that the garter had been tightly applied, at the insertion of the deltoid, on her left arm, by her teeth and right hand; and, accordingly, on exposing the arm, there was the deep sulcus, marked by the very ribbing of the worsted band. Dr. Keith gave her one chance, and promised not to expose the cheat, provided she would now consent to be cured: but in vain. She found means to strangulate the arm every night; so that, after a trial of three days, both her hands and arms were restrained in the sleeves of a strait waistcoat. After this, the cure advanced with great rapidity, and in five days the skin hung quite loosely upon the arm. The waistcoat was not removed for three weeks; indeed, not until her own entreaties, coupled with a promise of better behaviour, gave hope that she was wearied of deceit. On the 26th of October, 1846, after sixty-two days' treatment, she was dismissed cured; both arms being much alike; her spirits good; her mind active and intelligent; and she herself professing great gratitude for what she was pleased to designate "the wonderful cure!"

For two years after this she continued well, keeping
house for an uncle; when at length she took a fancy that she had heart disease, for which a surgeon inserted a seton in the vicinity of the scrobiculus cordis, and kept it open for some years. It was in November, 1853, that Dr. Keith again, and only by accident, saw her. The seton had at length ulcerated out; but he was told that the wound would not heal, and his opinion was asked. He examined a deep, round, fiery-looking ulcer, exactly midway between the umbilicus and the ensiform cartilage, of the exact shape and size of an old copper penny of the reign of George III, which she had very nimbly nipped out with her nail, along with the dressings. Dr. Keith warned her of her folly, and explained to her the risk she ran of ulcerating a passage into her belly, which might prove fatal. She evidently paid little attention to what was said, and Dr. Keith refused to take charge of the case. At length, after fully three years' pressure by a belt, of a copper coin over the ulcerating surface, she was seized with irritability of the stomach, bringing up bile and blood. Along with these symptoms, she had much fever for five days; and on one of these days, viz., March 2d, 1854, she discovered, on removing the dressings from the wound, that a piece of biscuit and a portion of orange peel, along with a quantity of fluid, escaped from the opening. For many days after, the whole food swallowed was discharged by the opening, and she became greatly exhausted. By the 23d of March, however, her own shrewdness taught her to fit a plug of gutta percha into the wound, so as actually to cork it up. This plug, somewhat enlarged on two subsequent occasions, as the opening has become a little larger, and kept in situ by a belt round the waist, has ever since sufficed to retain the great bulk of her food within the stomach, where it would appear to be well and quickly digested; her flesh, for a naturally spare person, being well kept up. Her general health has improved, and her hysterical symptoms have, for the most part, disappeared, from the period that the opening was established; she having now something special with which to occupy her mind and take up her
attention. After a delay of nearly three years, she sought admission into the hospital, as already stated, on February 19th, 1857, her professed wish being to have the opening closed.

3. Observations on her condition while in hospital.—She has jet-black hair and dark eyes, pale skin, prominent lips, melancholic countenance, and although spare, is not much emaciated. She has kept her bed for nine years; and consequently she is quite unable to walk or even to stand, and even feels faint and weak when she sits up in bed. There is no great stiffness of the joints, however, and the limbs are wonderfully well nourished; so that the chief reasons for her keeping her bed appear to be habit and her own dogged obstinacy.

In the epigastric, and upper part of the umbilical region of the abdominal parietes, is an oval opening communicating with the interior of the stomach. The long diameter is transverse, and measures four inches, the vertical diameter being about three inches. When the patient sits up or stands, or, even when reclining, if she coughs, or makes an effort as if to vomit, the walls of the stomach fall out through the opening, the whole organ becoming everted. She can take the stomach out, and push it in again, without feeling the slightest pain, even on free manipulation. Three fingers can be introduced into the cavity of the stomach with ease, and one of them passed down into the pylorus, or upwards into the cardiac orifice. She commonly keeps the opening closed by a gutta percha plug, covered with chamois leather and oil silk, and retained by a circular bandage. When this plug is removed, everything she swallows is almost immediately ejected through the opening, and even when the plug is in situ, some of the liquid portions of the ingesta ooze away by its sides, and, in a slight degree, irritate the surrounding skin. The margins of the opening, for some inches, are red, smooth, glistening, and tender. The mucous membrane of the stomach is of a bright vermilion-red colour, and occasionally bleeds a little, at isolated spots, when irritated. Although handling of the mucous mem-
brane and other irritating causes seem to produce no pain, they are always followed by a feeling of sinking and great faintness for a few minutes. A piece of blue litmus paper applied to the moist surface of the empty stomach, remains unaltered; but after food has been swallowed, or if the litmus paper is introduced into the pulpy food undergoing digestion, it is immediately turned red. The mucous membrane is disposed in rugæ, arranged longitudinally as regards the long axis of the stomach, but assuming a circular direction around the everted organ. Undulating movements may often be seen passing along the surface, which are increased by the contact of food. The edges of the opening are fully three quarters of an inch thick, red, and glazed; not at all ulcerated, but firmly cicatrized. The mucous membrane of the stomach appears gradually to lose itself in the surrounding skin, so that the margins of the opening are not unlike those of the human lip; and, indeed, the whole opening bears a coarse resemblance to a large mouth, half open.

Her tongue is clean and soft all day, but dry and parched towards night. She is usually very thirsty, and drinks a great deal of water. Her appetite is seldom at all impaired; and, at times, is so keen as to amount to pain. She often requires to swallow a piece of bread on the instant, to remove an unbearable feeling of sinking. She can eat and digest any kind of food, but she complains of pain at the pit of the stomach after eating solid food. Her preference is for eggs, and boiled fish, tea and bread, and vegetable soups. No pain follows the use of any of these; but she always feels great annoyance from the increase of irritation around the opening, owing to the escape of the fluid contents of the stomach, by the side of the plug, after each meal. She has only about one stool in the twelve days, consisting of numerous, hard, rounded scybala, like, but larger than, the droppings of sheep.

Her pulse is 80, regular, soft, and of fair volume. On auscultation there is nothing abnormal heard about the heart's sounds. The impulse of the heart cannot be dis-
tinctly felt from the stomach, on introducing the fingers and pressing them upwards, but the attempt to do this occasions considerable pain. It is very noticeable, however, how very close to the posterior wall of the stomach is the abdominal aorta, and how very immediately the tip of the finger meets its pulsation, when inserted into the opening.

Her respirations do not exceed twelve in a minute. She has no cough, nor any symptom of chest disease.

Her urine presents normal characters, but is rather scanty.

She has never been married, nor had any family.

She began to menstruate at the age of 14; and continued regular up to the time at which the opening in her belly took place. Since then the menses have entirely ceased. She has no leucorrhœa.

Her temper is placid, and she is affectionate to her parents. She at times has fits of crying, but not of laughing, as she once had; but, occasionally, "she flies off into a rapture," as her mother calls it. Her sleep is disturbed, but little seems to suffice: for a long time she has been in the habit of taking the equivalent of fully an ounce of laudanum a day. She is intelligent, and well informed for one in her station of life; she speaks rationally on every subject, but is silent when her own misdeeds are alluded to. She has never attempted self-destruction, but is calm in the prospect of death, and says she prays for it. Had she succeeded in her original wish of getting an arm amputated, so as to make herself an object of commis- sation, and afford a sufficient excuse for living on her friends in perfect idleness, the probability is that she never would have fancied heart-disease and never have commenced the process of continuous pressure, by which, after a period of three years, she succeeded in safely effecting an entrance into her stomach. Three years of the consequences may have somewhat sickened her of the experiment, and when she sought admission into the hospital, it was, as already stated, with the professed object of having the opening closed. Dr. Keith was inclined to think that this
might be effected by bringing the edges together after effectual paring, provided that the patient was really wishful to have it so. But, after some delay to weigh the question, he came to the conclusion that she would render every such attempt fruitless. She has gained her point; lives an idle life; eats, drinks, reads a book, and is satisfied. She feels herself to be an object of great interest; and one has only to witness the perfect complacency with which she uncorks her stomach, allows her dinner to gush out, and invites a visitor to search with his finger for the cardiac or pyloric orifice, to be convinced, that however much she might relish the *fuss* of an active attempt to close the opening, she has no wish nor intention that it ever should be closed.

Some weeks ago I wrote to Dr. Keith, to inquire if Catherine Ross ever vomited, and if any observations had been made of the stomach during the act of vomiting. The replies which I received, with an account of a very interesting experiment undertaken by Dr. Keith, were to the following effect: at intervals of a month or two she has a feeling of nausea, accompanied by a burning sensation at the scrobiculus cordis; but this ends only in an escape of bilious fluid at the side of the plug. Prior to the formation of the opening she vomited very frequently; but, in a period of three years and seven months, *i.e.*, since the 2d of March, 1854, she has only about ten or twelve times brought up one or two mouthfuls of yellow-tinged mucus, and that only when the plug was in the opening. On the 30th of last October, Dr. Keith made the following experiment: the plug was removed, and as much of the contents of the stomach (consisting of vegetable soup recently swallowed) as the ordinary protrusion of the stomach would eject, were allowed to escape. The sauces were then tickled with a feather held in the patient's own hand. Immediately the whole remaining contents of the stomach were projected from the opening. This appeared to depend, in the first place, on a strong spasmodic contraction of the stomach itself, and principally of its pyloric extremity, for
there was squirted from the pylorus, as if from a syringe, about a teaspoonful of light yellow bile. Simultaneously, however, with this, the abdominal walls were observed to become rigid, and this became more apparent on continuing the irritation of the fauces; for the next effect was seen to be violent contraction of the recti and other abdominal muscles, producing complete and almost instant eversion of the stomach. This organ was turned fairly inside out, evidently owing to the downward pressure of the contracting diaphragm meeting the upward pressure of the contracting muscles of the abdominal parietes. The upper wall of the stomach, indeed, could be seen and felt to be pressed downwards by the diaphragm. After a few minutes the patient herself restored the stomach to its place, pushing it in with the gutta-percha plug; and within ten minutes she partook of another plate of soup, as if nothing had happened, feeling neither nausea nor pain from the recent experiment.

Of the numerous points of interest connected with this case, the most important is undoubtedly the circumstance of the existence of a fistulous communication with the stomach, existing for a lengthened period during life. Every member of the profession is acquainted with the case of the Canadian, Alexis St. Martin; but few, I believe, are aware that the records of medicine contain accounts of many other such cases, resulting either from injury or disease; and that, in not a few instances, the patients have lived for many years with the open fistula. I have, therefore, been induced to make the following collection of cases of gastro-cutaneous fistulae.

1 I have had another opportunity (June, 1858) of seeing Catherine Ross, who is now living with her parents in Aberdeen. She is, on the whole, in better health than last year, having gained somewhat in flesh. Nothing will persuade her to leave her bed; but there appears little to prevent her, except her own perverse determination. The dimensions of the opening in the abdominal parietes have slightly increased.—C. M.

The cases of fistulous communications with the stomach through the abdominal parietes, which I have been able to collect from the records of medicine and surgery during the last 300 years, amount to twenty-five. For the sake of brevity, I have arranged them in a tabular form, so as to illustrate the following points:

1. The date at which the opening occurred.
2. The patient's name and residence.
3. The patient's age.
4. The cause which gave rise to the fistula.
5. The situation and characters of the external opening.
6. The size of the external opening.
7. Situation of the opening as regards the walls of the stomach, and any other morbid appearances of this organ.
8. Observations made as to the escape of food from the opening.
9. Duration of the fistula, and its consequences — whether the patient died or was cured—and the effects on the general health.
10. A brief notice of any physiological observations made as to the appearance of the mucous membrane of the stomach, the movements of its walls, and the nature and properties of the gastric juice.
11. The names of the observers of the cases, &c.
12. References to published accounts of them.
## Fistulae, Arranged Chronologically

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<tr>
<td>Food escaped from it, immediately after it was swallowed.</td>
<td>Two months in fistulous condition — then closed up.</td>
<td>None.</td>
<td>M. Lassalé.</td>
<td>M. Hévin, in Mem. de l'Acad. Roy. de Chir., tom. i, p. 594, 1743.</td>
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## CASES OF GASTRO-CUTANEOUS FISTULE.

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<thead>
<tr>
<th>No. of Case</th>
<th>Date of occurrence of Fistula</th>
<th>Patient’s Name and Residence</th>
<th>Age</th>
<th>Case</th>
<th>Characters, Situation, &amp;c., of External Opening</th>
<th>Size of External Opening</th>
<th>Situation of Opening in Stomach, and other morbid appearances of that organ</th>
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<tr>
<td>VIII</td>
<td>1712 to 1739</td>
<td>Marguerite Biguerin, of Nortlingen.</td>
<td>19 to 46</td>
<td>Necrosis of ribs with abscess, which at first only opened externally, and healed. Four years after, a second abscess, from which, on opening, food escaped. Was there originally simple ulcer of stomach?</td>
<td>Between eighth and ninth ribs, four inches from xyphoid cartilage, and two from left nipple. Ninth costal cartilage and portion of tenth rib necrosed.</td>
<td>Two inches in diameter. Easily admitted thumb. Cartilaginous margins. Skin excoriated.</td>
<td>Opening at middle of great curvature; between this and pylorus, stomach much contracted.</td>
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<td>IX</td>
<td>1715 May 16</td>
<td>A widow.</td>
<td>36</td>
<td>Cancer. A scirrhous mass, 5 inches by 2½, involved stomach and arch of colon; intimately uniting these with abdominal parietes and one another. Opening through skin for 11 days before food came from it.</td>
<td>In umbilical region.</td>
<td>Easily admitted finger.</td>
<td>A cancerous ulcer in middle of great curvature, two inches in diameter, in centre of which a perforation, the size of a shilling.</td>
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<td>X</td>
<td>1719</td>
<td>A virgin at Leipsic.</td>
<td>20 to 30</td>
<td>Blow in epigastrium from a carriage shaft—followed by abscess, which opened in six months.</td>
<td>Left of epigastrium.</td>
<td>Size of a large pea or haricot.</td>
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<td>XI</td>
<td>1740.7</td>
<td>A female.</td>
<td>Adult</td>
<td>Simple ulcer of stomach. First a tumour at epigastrium — this disappeared — skin became drawn in and like a cicatrix. More than a year after, fluid first seen to escape from this.</td>
<td>In epigastrium.</td>
<td>?</td>
<td>Apparently small.</td>
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<tr>
<td>XIII</td>
<td>1768. May, Wensaston, near Norwich</td>
<td>Mrs. Tovell, of Wensaston, near Norwich</td>
<td>60</td>
<td>Simple ulcer of stomach. Twenty years before had a slight injury over stomach — after this liable to pains in left side. In ten years a tumour at epigastrium, with fever, &amp;c.; and in ten years more this opened.</td>
<td>In epigastrium.</td>
<td>Of some size, as solid food escaped.</td>
<td>Near pylorus. Firm adhesion of stomach and liver round about orifice.</td>
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<td>Observations on Escape of Food, Prolapse of Stomach, &amp;c.</td>
<td>Duration of Fistula and Residual General Health of Patient</td>
<td>Physiological Observations</td>
<td>Observers</td>
<td>References</td>
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<td>When plug removed after meals, food immediately escaped by it.</td>
<td>Eight and a half years at least, as he was not dead when case reported. Enjoyed good health.</td>
<td>Interior of stomach seen to be &quot;d'un rouge très vif, et plissé dans tous les sens.&quot; Undulations of its surface occasionally observed, increased by contact of air or food. On making an effort to swallow, a lighted candle held before opening was agitated.</td>
<td>M. Percy.</td>
<td>Roux, Journ. de Méd. de Roux et Corv., tom. iii, p. 510, 1802. Gérard, Perf. Spont. de l'Estomac, Paris, 1803, p. 70.</td>
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<td>XV</td>
<td>1793.</td>
<td>Magdelaine Goré, of Roussigny, Dept. of La Manche, a patient in &quot;La Charité,&quot; Paris.</td>
<td>39 to 48</td>
<td>Simple ulcer of stomach. At age of 20, received an injury on epigastrium from fall on door-step. After this, liable to attacks of severe pain in stomach. Eighteen years after, an abscess, which opened on twenty-first day.</td>
<td>Opening oval, situated at upper and left part of epigastrium, one-third in epigastrium, and two-thirds in thoracic walls. Ninth, tenth, and part of eighth left costal cartilages wanting, and seventh sloped away. Surrounding integuments red and scaly.</td>
<td>1½ inch by 1¼.</td>
<td>Opening in anterior wall, eight inches from fundus, and four from pylorus. Extended from upper to lower margin.</td>
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<td>XVI</td>
<td>June 6, 1822, till present time, 1837.</td>
<td>Alexia St. Martin, a Canadian, a &quot;voyageur&quot; in Hudson's Bay Compy., wounded at Michillimackinac, in Michigan.</td>
<td>18 to 53</td>
<td>Gun-shot wound. Charge of powder and duck-shot, at distance of only one yard. Blew off integuments and muscles over space, size of man's hand, carried away anterior half of sixth rib, perforated stomach, and exposed left lung.</td>
<td>Opening situated two inches below left nipple, in a line drawn from this to spine of left ilium. One year after injury, parts were all sound, and firmly cicatrized, except opening into stomach.</td>
<td>2½ inches in circumference.</td>
<td>Opening three inches to left of cardia, near left superior termination of great curvature.</td>
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<td>ing plug, stomach would protrude several lines, forming a pad across opening.</td>
<td>stool in four days. Rounded scybala. One pint urine in twenty-four hours. Died of marasmus after an attack of diarrhoea.</td>
<td>washed out with infusion of chamomile. Gastric juice described and analysed. Fluid collected in morning before meals “neither acid nor alkaline.”</td>
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| At first, all food and drink discharged by it, on withdrawal of artificial plug; but one year and a half after injury, an accurate natural plug was formed by a protrusion of the mucous membrane, and food only escapes on the depression of this. | Thirty-five years, and is still alive (1857). Has enjoyed perfect health; has married, become the father of a family, and earned a livelihood for them by a laborious occupation. | Mucous membrane of a pale pink, varying in hue according to full or empty state of stomach. Temperature, 100° F. Ruge, Vermicular movements seen to be increased by food. Irrigation of stomach produced faintness, not pain. Physical characters of gastric juice described by Dr. Beaumont; and more recently its chemical nature investigated by Professor Dunglison and Dr. F. S. Smith. Fluid from empty stomach always of neutral reaction. Numerous observations on digestibility of different articles of diet, &c. Acidity of gastric juice due to hydrochloric acid (Dunglison) to lactic acid (Smith). | Dr. Beaumont, 1822 to 1833. Professor Dunglison, 1833. Dr. F. S. Smith, of Pennsylvania, 1856. | Beaumont, Experiments and Observations on Gastric Juice, Pittsburgh, U. S., 1833. Dr. Combe's Edin. Ed. of above, 1836. References in most modern works on Physiology and Practical Medicine. Dr. F. S. Smith, Philadelphia Medical Examiner, July and Sep., 1856, and Brit. and For Med.-Chir. Rev., Jan., 1857, p. 241. Dr. Bunting, Lancet, July 19th, 1856. Dunglison's 'Human Physiology,' Seventh Ed., vol. i, pp. 386-6.
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<td>XVII</td>
<td>July 1, 1828</td>
<td>M. A. W., a widow, Worcester Infirmary</td>
<td>39</td>
<td>Simple ulcer, producing abscess, adhesions, and external opening</td>
<td>Between seventh and eighth ribs, below right mamma. Lower margin of seventh rib, and upper of eighth, carious.</td>
<td>“A slit” 1 inch in length.</td>
<td>Immediately beyond pylorus, at very commencement of duodenum, two and a half inches from external opening.</td>
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<td>XVIII</td>
<td>June, 1832, and Jan., 1835</td>
<td>Madame G.</td>
<td>77 and 80</td>
<td>Simple ulcer of stomach. Symptoms for thirty years. At age of 77, an opening over stomach, from which “clear water” escaped for three days, and which then closed. At age of 80, this re-opened, discharging water and food.</td>
<td>In a transverse furrow of the skin in left hypochondrium, two inches below margin of ribs. Margins of opening red. Surrounding integuments drawn in like a funnel.</td>
<td>“Size of a pea.” “4 or 5 lines in diameter.”</td>
<td>In middle of anterior surface a rounded opening, one and a half inch in diameter, forming large end of a funnel, of which small at external opening. Thickening of pylorus.</td>
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<td>XIX</td>
<td>Sept. 3, 1832</td>
<td>Mary O’Neill, Meath Hospital, Dublin</td>
<td>50</td>
<td>Cancer of pyloric end of stomach.</td>
<td>To left of umbilicus.</td>
<td>Size of a large pea. A gum bougie passed readily.</td>
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<td>Gruel, &amp;c., escaped immediately after being swallowed. For two days the opening was closed.</td>
<td>Three weeks. Died exhausted.</td>
<td>None.</td>
<td>Dr. Stokes and Mr. Hamilton.</td>
<td>Hamilton, Lancet, 1832, vol. i, p. 612; Archiv. Gén. de Méd., 1st Ser., tom. xxviii, p. 264, 1832.</td>
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<td>XXI</td>
<td>Oct. 22, 1844, to Aug. 11, 1851</td>
<td>Isabella Davidson, Kelso, Scotland, and Edinburgh Royal Infirmary</td>
<td>30 to 37</td>
<td>Simple ulcer of stomach (?), inducing an abscess, which first communicated with stomach; then extension of inflammation and perforation of abdominal parietes.</td>
<td>Opening close to cartilage of eighth left rib, 2½ inches from median line, and 3½ from umbilicus. Oval. Margin rounded, depressed, hard, and red; with specks of ulceration. Surrounding integuments red, indurated, and excoriated.</td>
<td>Admitted thumb. Shortly before death, as large as a crown piece.</td>
<td>In anterior wall of stomach, about four inches from cardia.</td>
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<td>XXII</td>
<td>1850.</td>
<td>A married female, at Dorpat.</td>
<td>35</td>
<td>Simple ulcer of stomach.</td>
<td>Opening below left nipple, between ninth and tenth ribs, 2½ inches in an oblique line from the xiphoid cartilage. Margin red, and indurated. Surrounding skin erythematous, from trickling over it of gastric juice.</td>
<td>Half an inch in diameter.</td>
<td>?</td>
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<td>XXIII</td>
<td>Sept. 7, to Oct. 8, 1851.</td>
<td>Mrs. R., Braemar, Scotland.</td>
<td>50</td>
<td>Cancer of stomach</td>
<td>Left part of epigastrium.</td>
<td>At first size of a shilling; latterly increased to that of a half-crown piece.</td>
<td>No post mortem allowed; but on introducing finger during life, distinct carcinomatous hardness felt around opening.</td>
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<td>All food escaped freely by it, unless prevented by mechanical appliances. At first, the opening was closed by a valve of mucous membrane.</td>
<td>Nearly seven years; but during this time opening was twice closed, viz., from Jan., 1845, to Jan., 1846; and from March, 1846, to June, 1847. Was in bed for last three or four years of life. Tongue clean. Appetite good. No thirst. During digestion, pain at orifice. Urine scanty. Bowels confined. Menstruation regular.</td>
<td>A committee was appointed to make observations; but in consequence of patient’s state of health, it did nothing. Mucous membrane of stomach was visible, but is not described. Milk swallowed could be seen flowing over it. Contents always highly acid, during digestion. Introduction of a catheter caused faintness, but not pain.</td>
<td>Dr. W. Robertson, Robertson, Edin. Monthly Journ. of Med. Sc., Jan., 1851, p. 1. Archiv. Gên. de Méd., IV Ser., tom. xxv, p. 339. Schmidt’s Jahrbücher, vol. lxxxii, p. 35. Private letter from Dr. Hamilton, Nov., 1857. A plate showing the external opening accompanies Dr. Robertson’s paper.</td>
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<td>Contents of stomach freely escaped by it.</td>
<td>Three years; and patient still alive in 1853. Is otherwise healthy and strong; suckling an equally healthy infant. Has a ravenous appetite.</td>
<td>Numerous observations made as to the quantity, physical characters, and chemical composition of the gastric juice; also as to its digestive powers over different nutritive principles. Acidity of gastric juice due to butyric, and probably lactic acid; it contained no free hydrochloric acid. The secretion from the empty stomach was always neutral.</td>
<td>Professor Schmidt, Otto v. Grünwaldt, &quot;Succisa gastrici humani indolephysica et chemica et vis digestiva,&quot; Inaug. Dissert. Dorpat, 1853. Schmidt’s Jahrbücher, vol. lxxxiv, p. 1, 1854.</td>
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<td>Everything taken by mouth passed out at once by opening, unless prevented by gutta-percha plug.</td>
<td>Thirty-one days. Suffered great pain. Bowels confined. Died.</td>
<td>Introduction of laxatives through opening into stomach induced great pain and tendency to vomit.</td>
<td>Dr. James Cameron, of Braemar, N. B.</td>
<td>Private communication from Dr. Cameron through Sir James Clark.</td>
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XLI.
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<td>XXIV</td>
<td>Oct., 1854</td>
<td>A female, France</td>
<td>52</td>
<td>Cancer of stomach</td>
<td>Near umbilicus. Tumour, formed many months before ulcerating, had been in epigastrium, but then descended to umbilicus. Margin of opening gangrenous.</td>
<td>Enlarged rapidly by gangrene of integuments.</td>
<td>Cancer at pylorus, in all probability.</td>
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<td>XXV</td>
<td>March 2, 1854, to present time, June, 1855</td>
<td>Catherine Ross, Aberdeen, Scotland</td>
<td>32 to 36</td>
<td>Ulceration, produced by pressure from without of a copper penny piece, applied voluntarily by patient.</td>
<td>Opening in epigastric and upper part of umbilical region. Transversely oval. Edges of opening 4 inch thick, red, glazed, not ulcerated, showing a gradual transition from skin to mucous membrane. Integuments, for some inches around, red, smooth, glistering and tender</td>
<td>Transverse diameter, 4 inches; vertical, 3 inches. Three fingers can be passed into stomach with ease; and one into pylorus or cardia.</td>
<td>Opening about middle of anterior surface.</td>
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<td>When gutta-percha plug removed, everything she swallows is immediately ejected from opening; and even when that is in situ fluids ooze out. On sitting up, coughing, or making an effort to vomit, whole stomach becomes erected.</td>
<td>More than four years, and still alive and well. Tongue clean, parched at night. Great thirst. Appetite at times so keen, as to amount to pain. One stool in twelve days. Amenorrhoea.</td>
<td>Mucous membrane of stomach of a bright vermilion colour—will bleed at isolated points when irritated. Handling stomach causes no pain; indeed, she can take it out and put it in again with ease; but such manipulations generally produce a feeling of sinking, and great prostration. When stomach empty, blue litmus paper applied to mucous membrane is not turned red. Waves of contraction seen passing along rugae, increased by contact of food. Observation on mechanism of vomiting.</td>
<td>Dr. W. Keith, Aberdeen, and Dr. Murchison.</td>
<td>Account of case read before Royal Medical and Chirurgical Society of London, November 24th, 1857.</td>
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Gastro-cutaneous fistulae may be divided into those which are the consequences of mechanical injuries, and those which result from disease.

I. Mechanical Injuries.—Seven of the cases enumerated appear to have resulted from mechanical injury of some sort, and no doubt a careful search of the records of military surgery might increase this number. Wounds of the abdominal parieties involving the stomach are in most cases mortal. There are several instances, however, on record, in which they have healed up, and a speedy recovery has resulted. ¹ In other cases, again, the margin of the wound in the stomach has contracted adhesions to that in the abdominal parieties, so as to constitute a permanent fistula.

Wounds or mechanical injuries, which may give rise to such fistulae, appear to be of four sorts:

1. Incised wounds of the abdomen penetrating the stomach. Three of the seven cases were of this nature (1, 5, 6).

2. Gun-shot wounds (two cases, 14 and 16); the discharge either directly penetrating the stomach, as in the celebrated case of Alexis St. Martin, or inducing inflammation and subsequent sloughing of a portion of the wall of the stomach bordering on the original wound, as in the case of Maillot, a French lieutenant of infantry.

3. A blow over the stomach, without any laceration of the surface, may give rise to an abscess, which may open both into the stomach and also externally. This appears to have been the cause in Case 10. This patient received a blow in the epigastrium from a carriage shaft; an abscess followed, which in six months opened externally, and food swallowed was observed to escape from the opening. Case

¹ See, for instance, several remarkable instances mentioned by M. Hévin in the ‘Mém. de l’Acad. Roy. de Chirurgie,’ tom. i, part ii, p. 349, 1743; Gooch’s ‘Treatise on Wounds,’ vol. i, p. 397; and Chelius’s ‘Surgery,’ South’s ed., vol. i, p. 475.
15, also, was referred to an injury of this nature; but, inasmuch as eighteen years elapsed between its infliction and the appearance of the abscess, this explanation is doubtful.

4. Ulcerations from external pressure. The case of Catherine Ross is the only one recorded resulting from such a cause. The means adopted were not novel. Soldiers and sailors have been known to tie copper coins over open ulcers in their legs, to prevent the healing of these, and so elude their duty, or obtain a discharge from the service. But that a female should have recourse to such a proceeding, at such a part of the body, and when fully warned of its danger, is certainly a unique mode of courting notoriety. Yet if one really desired to obtain such a fistula, a safer process could hardly be adopted, than that followed by Catherine Ross. Its slowness gives ample time for the stomach to contract adhesions to the abdominal parietes, and thus the escape of its contents into the peritoneal cavity is prevented.

5. It seems possible that an injury of the stomach from within, might end in the formation of an external fistula. There are many instances on record of persons having swallowed large foreign bodies, such as knives and other cutting instruments, and surviving; the foreign bodies, after the lapse of a considerable period, escaping by abscesses through the abdominal parietes. I have not been able, however, to find any case in which food is stated to have escaped from the external opening; so that probably the wound in the gut in most cases closes before the abscess opens externally.

II. Disease.—The morbid conditions, of which we can speak with certainty as capable of giving rise to external fistulae of the stomach, are only two, viz., cancer and simple perforating ulcer of the stomach. In both of these affections the escape of the contents of the stomach into

1 Gavin, on 'Feigned and Fictitious Diseases,' p. 337.
the peritoneal cavity, on the occurrence of perforation, may be prevented by the previous contraction of adhesions to the abdominal walls, which last may also be perforated by a continuation of the original disease.

Opinions seem divided as to the relative frequency of these causes. Thus, Cruveilhier brings forward several cases depending on simple ulcer, but says he is not acquainted with an instance resulting from cancer, although he observes, it is not rare to meet with a cancerous pylorus adherent to the abdominal wall;¹ and Dr. Brinton states, as the result of his researches, that he has found about six cases resulting from simple ulcer, but only one from cancer.² Rokitansky, on the other hand, says a cancerous ulcer may force its way through the abdominal parietes, but makes no mention of such an occurrence when speaking of the simple perforating ulcer.³ Of the cases collected by myself:⁴

Six appear to have resulted from cancer (7? 9, 12, 19, 23, and 24); and 12 from simple perforating ulcer (2? 3, 4, 8, 11, 13, 15, 17, 18, 20? 21, 22).

Although, with regard to one or two of the older cases, the distinction between cancer and simple ulcer may be a matter of question, yet from a careful reference to the history, symptoms, and post-mortem appearances of these cases, I believe the statements made in the preceding table to be correct. Gastro-cutaneous fistula would thus appear to be twice as frequently the result of simple ulcer as of cancer. Now this is exactly the reverse of what has been found to hold good in the case of gastro-colic fistula.⁵ This is probably owing partly to the difference in the relative situation of the two diseases. Simple ulcer, as shown by Dr. Brinton, is very rare in that part of the stomach

¹ 'Traité d'Anat. Path.,' ii, 566.
⁴ The figures refer to the number of the cases in the table, and will be repeated in the course of the paper, in order to facilitate comparison.
nearest the colon, whereas of simple ulcers ending in perforation a very large proportion are found upon the anterior surface.¹ Again, as the length of time necessary to perforate the abdominal muscles and integuments must be considerable, in many cases of adherent cancerous stomach the patient no doubt sinks under the disease before the perforation is complete.

Of the six cases of cancer, in three the disease was situated at the pylorus (12, 19, and 24); in two (7 and 23) it was probably near the great curvature, but in one of these (7) there was also "scirrhus of the pylorus." In the sixth case (9) there was a large cancerous mass between the stomach and the arch of the colon, which had almost established a communication between these two portions of the digestive canal, as well as between the stomach and external surface.

In some of the cases of simple ulcer the fistula appears to have resulted from a gradual extension of the ulcerative process through the adherent abdominal parietes; but in many, perhaps most, the perforation of the stomach would seem to have excited a limited inflammation of the peritoneum, ending in chronic abscess, which has ultimately discharged its contents through the abdominal wall. Several cases have been recorded illustrative of the various stages of this mode of formation. Amongst these there is one remarkable instance which was brought before the notice of the Royal Medical and Chirurgical Society, by Mr. Lloyd, on November 14th, 1843, of an abscess pointing in the abdomen, which it had been resolved to open. The patient, however, died suddenly before this was done, and the abscess was found to communicate with an ulcer of the stomach.²

In some of the cases it may be questioned whether this abscess has not originated external to, and quite independently of, the stomach, ultimately bursting into this viscus, and also opening externally. Thus, in Case 3 the

² 'Lancet,' 1843-4, vol. i, p. 273. See also Cruveilhier, op. cit., ii, 566.
abscess may possibly have originated in the liver; and in Case 8, in the abdominal parietes, from necrosis of the ribs. Hepatic and other abdominal abscesses, we know, may burst into the stomach, and there seems no reason why they should not at the same time open externally. Should this explanation apply to any of the above cases, of course the number referred to simple ulcer of the stomach would be somewhat diminished.

Case 4 was ascribed to the erosion of a worm, but was probably a case of simple ulcer, in which a round worm may have come away through the opening.

As regards the situation of the opening in the stomach in the cases of simple ulcer, in

2 cases this was doubtful (2, 11);
3 cases it was near the fundus (4, 21, 22);
3 cases it was near middle of anterior surface (8, 15, 18);
4 cases it was at or near the pylorus (3, 12, 17, 20).

In one of these last cases the opening was situated rather on the duodenal side of the pylorus; and perhaps it should have been excluded from this list. The ingesta, however, were observed to escape as readily from the opening in this case, as if it had been in the stomach itself.

It is a curious fact, that all the patients with cancerous fistula, and all but two in which it resulted from simple ulcer, were females; and in one of the two exceptional cases the sex is not recorded.

D. Situation, Size, and other characters of the External Opening.

1. Situation. The situation of the external opening in these cases resulting from disease of course depends, in a great measure, on the region of the stomach from which the fistula originates. Thus, if this be the neighbourhood of the pylorus, the external opening is generally near the umbilicus; or, if the disease commences in the anterior surface or near the fundus of the stomach, the external opening will occupy the epigastric or left hypochondriac region.

1 Dr. Graves's 'Lectures on Clinical Medicine,' vol. ii, p. 232.
In four cases (8, 15, 16, and 22) the opening was situated between some of the left ribs, portions of which, or of their cartilages, had been removed. In Case 16 (St. Martin) this was the result of a gun-shot wound. In 15 and 22, and perhaps also in 8, the ribs appear to have been destroyed in the progress of a simple ulcer of the stomach. In Case 17, which originated in a perforating ulcer on the duodenal margin of the pylorus, the external opening was between the seventh and eight right ribs, below the right mamma. In connexion with this, it is important to allude to a case mentioned by Cruveilhier, in which a simple ulcer, commencing in the stomach, was found to have eroded the posterior surface of the ensiform cartilage.¹

2. The margins of the opening are generally described as rounded, hard, or of cartilaginous consistence, in those cases resulting from either wound or simple ulceration. In two cases (11 and 21) the margins are noted as having been depressed or drawn in; and in a third (18) this retraction of the margins was so great, that the opening resembled a funnel. All these three were cases of simple ulcer of the stomach. In cases resulting from cancer, the margins of the opening may be ragged, or even gangrenous. In Catherine Ross they are red, glazed, and firmly cicatrizied, and there appears to be a gradual transition between the skin and mucous membrane.

3. The integuments surrounding the opening for some inches are usually red, tender, and more or less excoriated, owing to the irritation from the constant escape of the fluid contents of the stomach.

4. The size of the external opening varies greatly.

Thus—

In 7 cases it was doubtful (3, 4, 5, 7, 11, 17, 24);
In 6 cases it was less than 1 inch (10, 14, 18, 19, 20, 22);
In 7 cases it was about 1 inch in diameter, or easily admitted finger (1, 2, 6, 9, 12, 13, 16);
In 1 case it was 1 ¼ by 1 ¼ inch (15.)

In 1 case it was 2 inches in diameter (8);
In 2 cases it was at first about 1 inch in diameter, but, shortly before death, increased to the size of a half-crown, or a whole crown piece (21, 28);
In 1 case it was 4 inches by 8 (25).
It will thus be seen that in none of the cases hitherto recorded did the opening at all equal in size that which exists in Catherine Ross (25).

E. Escape of Food swallowed by Artificial Opening.

In all of the cases this phenomenon was observed. In six cases (7, 11, 17, 18, 19 and 20) fluids only are stated to have escaped, and in these the opening was either very small, or no mention is made of its size. In all the other cases, whatever the patient swallowed immediately escaped from the opening. This escape of food has usually been prevented by some artificial contrivance, such as a plug of linen, gutta percha, &c.; but it has always been found difficult to prevent the fluid ingesta from oozing out by the sides of the plug. In the case of Alexis St. Martin, the necessity for such an artificial plug became, after a time, superseded by a natural one, consisting of a slight protrusion of the coats of the stomach, so that food only escapes on the depression of this. It is probably to this provision of nature for preventing the escape of food, that this individual's good health and long life are in no small degree to be attributed. A tendency to the formation of a similar natural plug was also observed in Cases 15 and 21; but in the case of Catherine Ross the opening is so large that the whole stomach becomes everted through it.

It is to be remembered, in reference to the diagnosis of such cases, that a fistulous opening in the abdominal walls, from which food escapes immediately on being swallowed, does not necessarily communicate directly with the stomach. We have already seen that in Case 17 the opening was rather on the duodenal side of the pylorus. Again, in the museum of Charing Cross Hospital there is a preparation
(G. 21) of a cancerous stricture of the arch of the colon, in which the colon above the stricture communicated with the duodenum, and also, through the abdominal parietes, with the external surface. In this case, any fluid taken into the stomach, flowing by the duodenum into the colon, made its appearance, in a few minutes, at the umbilicus.

F. Duration of the Fistula, and possibility of Cure.

The length of time during which an individual may live with a fistulous opening into the stomach, varies very greatly with the cause on which the fistula depends.

1. As might be expected, all the cases dependent on cancer have proved very speedily fatal, three months being the longest period during which a person has lived with a cancerous fistula. Of the six cases of cancer, the duration of life, after the formation of the fistula, was as follows:

   In 1 case it was doubtful (7).
   In 2 cases it was 3 weeks (12, 19).
   In 1 case it was 4 weeks and 3 days (23).
   In 1 case it was 5 weeks (24).
   In 1 case it was 3 months (9).

2. Wounds of the stomach, as already stated, are generally mortal; or, in a few instances, become speedily cured. The fistula, however, to which they now and then give rise, may continue for years. Thus—

   In Case 6 it lasted 2 months, and was cured.
   In Case 1 it lasted many years.
   In Case 10 it lasted 10 years, and was cured.
   In Case 5 it lasted 11 years.
   In Case 14 it lasted 8½ years at least; as the patient was not dead when the case was reported.
   In Case 25 it lasted 4 years, and still alive.
   In Case 16, it lasted 35 years, and still alive.

3. Of the cases resulting from simple ulcer—

   In Case 17 the patient lived 26 days, but died with tubercles in lungs.
   In Case 11 opening lasted a "short time," and closed up spontaneously.
In Case 18 opening closed after 3 days; 3 years after, it opened again, and on the 4th day patient died.
In Case 13 patient lived 8½ months, and died from effects of cold.
In Case 20 opening lasted 6½ months, and was cured.
In Case 8 patient lived 1½ year.
In Case 21 opening lasted 7 years, but was twice closed during this period for upwards of a year.
In Case 15 opening lasted 8½ years.
In Case 4 opening lasted 12 years.
In Case 8 opening lasted 27 years.
In Case 2 opening lasted "many years."
In Case 22 opening lasted 3 years, and still alive in 1858.

Thus, if we except the first four cases, in which either the opening closed up spontaneously or death resulted from independent causes, the most of the others lived for many years. It is to be observed that in three cases (11, 18, and 21) the opening closed up spontaneously. In Case 11 the cure appears to have been permanent; in Case 18 the fistula reopened after an interval of three years; and in Case 21 the opening was twice closed for upwards of a year, and as often reopened.

This leads us to consider the question of the possibility of curing such cases. In six of the cases the opening became closed (2, 10, 11, 18, 20, 21). Two of these cases (2, 10) resulted from wound; the other four from simple ulcer. In four of the six cases the opening appears to have closed spontaneously (2, 11, 18, 21); and in two of these (18 and 21) the fistula reopened. In two cases only does the obliteration of the fistula appear to have been the result of treatment. In Case 10 a fistula, resulting from an injury, of ten years' duration, was closed by simply making the patient, who had been in the habit of always going about, keep quiet in her bed for a few weeks; and in Case 20 a cure was effected by ingenious local appliances, of such a nature as to prevent the contact of the irritating contents of the stomach with the surrounding skin, and at the same time approximating the edges of the opening by means of
the gradually increased compression of a circular bandage, while at the same time the patient was nourished chiefly per rectum. In none of the cases does any cutting or paring of the edges of the wound appear to have been attempted. Dr. Keith seems to consider such a procedure practicable in the case of Catherine Ross, provided that she herself would throw no obstacle in the way. It is much to be feared, however, that in any such operation the contact of the gastric juice and the distention of the stomach by food would very much interfere with union by the first intention, while, at the same time, there would be a risk of exciting dangerous inflammation of the stomach.


It is astonishing to observe how little influence the existence of a gastro-cutaneous fistula has upon the general health. In most of the cases resulting from wound or simple ulcer the patients are stated to have enjoyed excellent health. This was particularly remarkable in Wrencher's case of a woman, who lived for twenty-seven years with such a fistula, following her ordinary avocations; and is still more so in the case of Alexis St. Martin, who, during the last thirty-five years that the fistula has existed, has married and become the father of several children, earning a livelihood for his family by a laborious occupation. In Catherine Ross the state of general debility is more to be attributed to a general derangement of the entire nervous system than to the effects of the fistula.

The principal abnormal symptoms which have been observed as dependent upon the fistula are, great thirst, increased appetite, obstinate constipation, deficient secretion of urine, and amenorrhoea. The most of these symptoms are directly traceable to the constant escape of the ingesta.

1 For a fuller account of the treatment in this case, see South's ed. of Chelius's 'Surgery,' vol. i, p. 477.
from the abnormal opening. This continual drain creates a craving for an additional supply, while at the same time it cuts off the material necessary for the production of feces and urine. In Case 15 the patient is said to have eaten as much as any three other women, and in Catherine Ross the feeling of hunger sometimes amounts to actual pain. There may be but one stool every three, four, or even twelve days; and this has, in several instances, been observed to consist of hard rounded scybala, like the droppings of a sheep.

Vomiting appears to have been a very unusual symptom in all of the cases, although it may have been very urgent before the formation of the fistula. In Case 15, however, there was an irresistible desire, three or four hours after each meal, to remove the plug, and evacuate the contents of the stomach. This same person also could never sleep unless she had previously washed out the stomach with an infusion of chamomile.

In Case 8 a vicarious discharge of pure blood from the mucous membrane of the stomach occurred at every menstrual period.

H. Physiological observations on the Stomach, &c.

Cases such as those we have been considering afford the opportunity of observing during life in the human subject—
1. The physical characters and properties of the coats of the stomach, such as its peculiar movements, the colour, &c., of the mucous membrane, &c.; and
2. The physical and chemical characters of the gastric juice, and its digestive power over different nutritive principles and articles of diet.

1. Observations of the former class have been made in six of the cases (8, 14, 15, 16, 21, and 25).

In Case 8, when food was swallowed the stomach could be seen occasionally to contract violently so as to expel the food with force. No mention, however, is made of the condition of the diaphragm and abdominal muscles.
In Case 14 the surface of the stomach is described as "d'un rouge très vif, et plissé dans tous les sens." Undulating movements could also be seen passing along the surface, which were increased by the contact of food. When the patient made the effort of swallowing, a lighted candle held to the fistula became agitated.

In Case 15 the interior of the stomach in the morning, before any food was taken, was said to be "d'un rouge vermeil, enduit d'un mucus luisant." It was disposed in longitudinal plissées; and two sets of undulatory movements were observed, one towards the pylorus and the other towards the fistula.

In Case 16 Dr. Beaumont describes the mucous membrane as of a pale pink colour, increased in hue when food entered the stomach. Vermicular movements were observed, which were augmented into a churning motion by the contact of food. Mechanical irritation of the stomach produced faintness, but not pain.

This last observation was also noted in Case 21.

In Catherine Ross (25) the mucous membrane is of a vermilion red colour, disposed in rugae, along which undulating movements can be observed. The freest manipulation of the stomach only produces faintness, not pain.

The observation made upon Catherine Ross, as regards the mechanism of vomiting, is of peculiar interest. During the last century there was a great controversy as to the precise mode in which this act is effected, and even at the present day we find the greatest variety of opinions expressed by different physiologists concerning it. These various opinions may be reduced to the four following:

1. Down to the end of the seventeenth century the almost universal belief appears to have been, that vomiting was the result of the convulsive contractions of the stomach itself; and, more recently, Haller, Rudolphi, and others, have considered the contraction of the surrounding muscles as unnecessary, or, at most, only auxiliary.

2. Another class of physiologists, commencing with Bayle
of Toulouse\(^1\) (a.d. 1681), and including John Hunter,\(^2\) have maintained that the stomach is nearly or entirely passive during vomiting, and that this action is excited by extraneous pressure of the diaphragm and other abdominal muscles. The greatest supporter of this theory has been Majendie, who drew his conclusions from observing the non-occurrence of vomiting after dividing the phrenic nerves, so as to paralyse the diaphragm, and removing the abdominal parietes; and the fact that it did take place when a pig's bladder was substituted for the stomach, the muscles remaining intact.

3. A third class, while they have admitted that the act has a double origin, depending on the contraction of the stomach, assisted by that of the abdominal muscles, have yet maintained that it is only the muscles of expiration which by their contraction compress the stomach; and that, after the first inspiration, the diaphragm becomes relaxed. This opinion, that vomiting is mainly an expiratory act, was pronounced by Lieutaud, so long ago as 1752;\(^3\) and in modern times has been strongly advocated by the late Dr. Marshall Hall.\(^4\) It is also the view which is taught by Dr. Carpenter in his 'Principles of Physiology.' "It is not true," Dr. Carpenter observes, "that the diaphragm actively cooperates in the effect of vomiting;"\(^5\) although he admits that this muscle, by its being passively fixed, may supply a firm surface, against which the stomach is pressed.

4. Lastly, there are others who believe that, while the stomach itself contracts, the vomitive act is in the main effected by both the muscles of expiration and inspiration; in other words, that the diaphragm remains contracted after the expiratory muscles come into play, and that, by the simultaneously opposing forces, the stomach is so compressed as to be evacuated of its contents. Dr. David Anderson, in an inaugural thesis presented to the medical faculty of the

\(^1\) 'Dissert. sur quelques points de Physique et de Médecine,' Toulouse, 1681.
\(^3\) 'Mém. de l'Acad. Roy. des Sciences,' tom. lxviii, p. 230.
\(^5\) 'Principles of Human Physiology,' fifth ed., p. 69.
University of Edinburgh, in 1842, proved by experiment that the diaphragm is rigidly contracted during vomiting. This he ascertained by laying open, and introducing his hand into, the abdomen of dogs which were vomiting from the effects of tartar emetic.¹ The correctness of this view is also supported by the powerful advocacy of Mesrs. Todd and Bowman,² and is corroborated by what has been observed in the case of Catherine Ross. This case shows that although the coats of the stomach itself do undoubtedly contract, the principal part of the act is played by both the diaphragm and abdominal muscles. The upper wall of the stomach can be distinctly seen to be pressed down by the diaphragm. The contraction of the stomach appears to be confined to the pyloric region, and to have for its object chiefly the closure of the pylorus, so that when the stomach becomes compressed between the diaphragm and abdominal parietes, the only exit for its contents, in the natural condition of the parts, must be through the oesophagus. From all the observations which have been made on the matter, the following would appear to be the mechanism of an act of vomiting:

1. A long inspiration followed by partial closure of the glottis.
2. Contraction of the pyloric end of the stomach, with relaxation of the cardia and oesophagus.
3. Persistent contraction of the diaphragm, with rigid contraction of the recti and oblique muscles.

In connection with this subject, I may be allowed to correct an erroneous statement of facts, which is to be found in several English works on physiology. Dr. Carpenter, in quoting a case, observed by a French surgeon, M. Lépine, to prove that the walls of the stomach contract during the act of vomiting, observes: "In this case, the abdominal parietes having been accidentally laid open in the human

² 'Physiological Anatomy,' vol. ii, p. 213.
subject, and the stomach having wholly protruded itself, it was seen to contract itself repeatedly and forcibly, during the space of half an hour, until by its own efforts it had expelled all its contents except gases. 1 A similar account of the case has been given by Mr. Paget; 2 and by Messrs. Todd and Bowman; 3 the latter, however, quote from Mr. Paget. How completely different were the phenomena really presented by this case, will be at once apparent on reading the following extract from the original account.

"Pendant tout le temps que l’estomac fut hors de l’abdomen, M. Lépine ne l’a ni vu, ni senti se contracter, quoique, dans le but de provoquer ces contractions, il ait appliqué sur cet organe ses mains, prèablement trempées dans l’eau froide. A peine la reduction était elle opérée, qu’aux nausées, et aux efforts inutiles pour vomir succédèrent de veritables vomissements, qui amenèrent au dehors les aliments, que le malade avait pris une demi-heure avant la blessure." 4

II. There are only three cases which have hitherto been taken advantage of for making observations on the chemical and physical properties and the digestive powers of the gastric juice. (15, 16, and 22.)

1. In the first of these (15), that of Magdelaine Goré, several chemical analyses of the gastric juice, and of the food undergoing digestion, were made by M. Clarion, more than half a century ago. M. Clarion found the gastric juice to contain 9 parts in 1000 of muriates, and 16 parts of an animal principle, precipitated by both alcohol and tannin. He also ascertained that, although the contents of the stomach during digestion were always acid, the fluid collected in the morning before breakfast was perfectly neutral, and had a strong analogy to saliva.

2. Every one is familiar with the valuable observations made by Dr. Beaumont, in the case of Alexis St. Martin (16), as regards the relative digestibility by the gastric juice of different articles of diet, &c. It would be quite apart from the object of this paper to enter into these here, I would merely call attention to some chemical analyses of the gastric juice obtained from St. Martin, which have been made since Beaumont's observations, and which are less generally known. These analyses have been performed in 1833, by Professor Dunglison, of Philadelphia; and within the last two years, by Dr. F. S. Smith, of the Pennsylvania College. The results arrived at by these two experimenters are, however, somewhat conflicting. Thus Professor Dunglison obtained hydrochloric acid with great readiness; and concluded that this acid was the principal, if not the sole, source of the gastric juice: whereas Dr. Smith states, as the result of his experiments, that if hydrochloric acid exists at all, it is in very small quantity, and "that the main agent in producing the acid reaction, is lactic acid." Dr. Smith found the fluid from the empty stomach always neutral.

3. Within the last few years a valuable series of observations, as regards the physical and chemical properties of the gastric juice, have been made on Case 22, and recorded in a thesis by Otto v. Grünwaldt. Professor Schmidt obtained the following results from his analyses: first, that the human gastric juice contains about 36 parts in 1000 of pepsin; secondly, that it contains butyric, and probably also lactic acid; and thirdly, that it contains no free hydrochloric acid, although, according to the experiments of the same chemist, this acid exists in considerable quantity in the gastric juice of some of the lower animals. Lastly, the fluid obtained from the empty stomach, early in the morning, before a meal, was always neutral.

3 See References in Table; also Carpenter's 'Principles of Human
4. One observation, which has been made in all these cases, has been confirmed in Catherine Ross. This is the non-acidity of the fluid obtained from the empty stomach. No other experiments have been undertaken upon her. As far as regards the size of the opening, no case could be more favorable for examining the interior of the stomach, or for introducing or removing food. Indeed, it would not be a difficult matter, by means of an elastic catheter, to draw off the contents of the duodenum for examination. The patient, however, is in such a debilitated state of health—bed-ridden for nine years and unable to stand, that it has been feared that any observations made on the properties of her gastric juice would at present be of small value. In addition to this, Catherine Ross's propensity to practise deceit would require careful vigilance on the part of the experimenter. In reference, however, to her general health, it may be observed, that since she has been under Dr. Keith's observation, she has gained considerably in flesh and strength; and if she could only be induced to get up and go about, the infatuated act of this poor woman might yet prove a benefit to science.

Physiology, fifth ed., p. 81. The analysis, as given by Dr. Carpenter, does not agree with the original.
THERAPEUTIC COMMUNICATIONS.—No. I.

ON

THE INFLUENCE OF

LIQUOR POTASSÆ,

AND OTHER CAUSTIC ALKALINE SOLUTIONS,

UPON THE THERAPEUTIC PROPERTIES OF HENBANE,
BELLADONNA, AND STRAMONIUM.

BY

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Should it be in accordance with the wishes of the Royal Medical and Chirurgical Society, I purpose laying before the Fellows, from time to time, a series of results obtained during a course of clinical investigations on the action of drugs—a subject which I believe of the highest importance in the present state of medicine, and to which I have paid, and hope still to be able to devote, some little attention. It will be impossible, in such communications, to observe any prescribed order or arrangement; for, as all must be aware
who have studied clinical therapeutics, a long period may often elapse before opportunities offer themselves for completing any one investigation. This, however, I do not regret, feeling convinced that at present the aim of the therapeutist should be to collect accurate facts on the action of individual remedies, rather than attempt to classify and generalize upon the few and imperfect data already in his possession.

Among other remedial agents I have examined the action and therapeutic uses of several plants belonging to the order Solanaceae; but as yet my observations upon them have been too few and incomplete for me to venture to bring them forward. Having, however, arrived at some points of considerable practical importance, I have thought it desirable at once to lay them before the Society, and these form the subject of my present communication.

Before discussing the action of liquor potassae upon the preparations of henbane it will be of some interest to ascertain if such a combination be frequently administered; for should it be found that the drugs are not often so prescribed, the remarks I shall have to make will be of but little value to the physician or surgeon. Should it, however, be ascertained that they are very frequently so given, it then becomes of great importance that the following facts should be known to all practitioners. From personal knowledge, from what I can gather from medical and surgical friends, and from inquiries at some of the principal dispensing establishments, I am convinced that combinations of liquor potassae and tincture or extract of henbane are very frequently prescribed and dispensed; and on referring to several works on surgery, such prescriptions will be commonly met with, and recommended as peculiarly valuable in irritable conditions of the bladder and urethra. Nor will a reference to the various publications on Materia Medica and Therapeutics show that mixtures of this kind are generally considered incompatible. For example, in the valuable and comprehensive treatise, by the late Dr. Pereira, no mention is made of the incompatibility of such com-
pounds; neither is the subject alluded to by Dr. Royle. Dr. Christison, in his 'Edinburgh Dispensatory,' makes no mention of the subject; neither does Dr. Neligan place liquor potassa among his list of the substances incompatible with henbane.

I have searched in vain through many other works, British and foreign, as the 'Traité de Thérapeutique,' of Drs. Trouseau and Pidoux; the 'Manuel de Matière Médicale,' of M. Bouchardat; the 'United States' Dispensatory,' of Drs. Wood and Bache; Phillips's 'Pharmacopoeia,' &c., &c., but cannot discover that any of these authors were at all impressed with the importance of avoiding the mixture of caustic alkalies with the preparations of henbane. However, in the late Dr. A. T. Thomson's 'London Dispensatory,' a statement is made to the effect that hyoscyamus should not be prescribed with alkalies, as these destroy their narcotic powers in the course of twenty-four hours; and allusion is also made to the fact in the 'Elements of Materia Medica' of the same author, subsequently published; but from the manner in which it is introduced, if the suggestion had been acted upon, it would probably, by most practitioners, have been supposed to apply to all alkaline remedies, and not particularly to the caustic alkalies.1

1 In my communication read before the Society it will be seen that I alluded to the fact of Dr. A. T. Thomson having noticed the destructive action of the alkalies upon henbane, and I have since found that Gieger, the discoverer, in conjunction with Hesse, of the crystallized principles in several solanaceous plants, refers to this property of the fixed caustic alkalies upon these bodies; and his statement, which is comprised in a very few words, has been copied into several works on chemistry, as those of Berzelius and Liebig. That therapists had not paid much attention to the subject, or regarded it of importance, is evident from the fact that in the principal works on materia medica and therapeutics alluded to in the paper, it is not spoken of.

I may also mention that my attention was drawn to the fact, when making observations on the action of a very dilute solution of atropine upon the pupil of the eye, being at that time quite ignorant of Gieger's statement. The present communication, as also the second paper, read subsequently before the Society, contain the results of my observations
I may here state, that until I had made most of the observations which I shall have to relate in the present communication, I was not myself aware of the above statements: that the fact has not been recognised by the profession I think I have already brought forward ample proof.

The effects of henbame on the animal economy can be appreciated either when it is applied externally or given internally; the local action is seen when any liquid preparation is dropped into the eye or rubbed upon the lids, and in these cases, unless an excessive quantity is made use of, that eye only to which the fluid has been applied becomes affected. Good tincture of henbane of the Pharmacopoeia diluted with seven parts of water will cause dilatation of the pupil, but the phenomenon is better observed when a watery solution of the extract is employed, in the proportion of two grains to the fluid ounce; a few drops of such a solution induces a very powerful effect in the course of from ten to twenty minutes, and the dilatation may last for several hours or even days, attended with more or less affection of the vision. When the preparations of henbane are administered internally in large medicinal doses, dryness of the mouth and throat is experienced, the vision becomes impaired, the pupils dilated, and a presbyopic condition produced; and should the drug be persevered in, other symptoms, as redness of the skin from the occurrence of an erythematous eruption, nervous trembling, and slight delirium are exhibited. In very large quantities henbane causes wild delirium, followed by coma, and even death.

These effects of the drug have been alluded to in order that the influence of liquor potasse upon it may be readily appreciated; for should it be found that a combination of the two fails to produce the above phenomena, either when topically applied or given internally, we shall, I think, be made to ascertain the influence of such combinations in a therapeutic point of view, and will, I trust, be the means of preventing, for the future, the exhibition of such incompatible combinations.
fully justified in asserting that liquor potassæ has the property of entirely neutralizing the effects of Henbane, and rendering it useless as a remedial agent, and that a combination of the two drugs should never be prescribed, being therapeutically incompatible.

The following observations were made to ascertain the influence of a solution of caustic potash upon the preparations of henbane. One part of good tincture of henbane was mixed with seven parts of water, this solution dilated the pupil; liquor potassæ was afterwards added, in the proportion of ten minims to the ounce, and, an interval of at least two hours having elapsed, it was applied to several eyes, and found to have lost all power of causing dilatation.

The solution next made use of consisted of two grains of the extract of henbane rubbed up with a fluid ounce of water; it was much more powerful in its action upon the eye than the diluted tincture, but after fifteen minims of liquor potassæ had been added, and the same interval allowed, in twelve cases in which it was applied, not the slightest effect upon the pupil was observed. The results of these experiments were perfectly conclusive to my own mind, and fully convinced me that the caustic alkali destroyed the activity of henbane, seeing that it prevented the occurrence of a phenomenon most characteristic of hyoscyamus and certain other solanaceous plants, viz., the dilatation of the pupil when topically applied. On mentioning the subject, however, to some friends, I found that in order to make my conclusions satisfactory to other minds, it would be necessary to carry my experiments further, and demonstrate, that not only is the local action of henbane destroyed, but also its activity when internally administered. To effect this, other and somewhat more difficult trials were required.

A medical friend feeling confidence in the correctness of my views, kindly volunteered to be the subject of the first experiment, and early one evening took a draught prepared in the following manner; a scruple of good extract of henbane was rubbed up with an ounce and a half of water,
to which two and a half fluid drachms of liquor potasse had been added, and, after a few hours, an addition of some citric acid was made, to remove the causticity of the draught. Not the slightest symptom was produced by the dose, although carefully watched for. The only effect of the citric acid, was the formation of a citrate of potash, and it was not added until the alkali had exerted its influence upon the henbane.

Emboldened by this last result, I proceeded further, and gave the extract of henbane in doses gradually increased from half a drachm to one drachm, in all cases mixed with a proper proportion of potash, and, I may state, that in no one instance was any effect produced, even by the largest dose.

Tincture of henbane, united with potash, was also given in quantities varying from two fluid drachms to a fluid ounce and a half, and in no case were symptoms exhibited. Some experiments were next made to ascertain the time required for the destructive influence of the potash to be completed, and when the extract of henbane was employed, about two hours were found sufficient.

It next became a matter of interest to determine if the carbonate of potash had any effect upon henbane, and for this purpose, a solution of the extracts of the same strength as that used in former experiments, had the salt added to it, in the proportion of five grains to the ounce, and, after some hours, it was applied to the eyes of several individuals; in all instances it exhibited full activity. Carbonate of potash, therefore, does not injure the therapeutic properties of henbane, and may be advantageously administered in conjunction with preparations of this drug. The same remarks apply to the bicarbonate of potash.

The Atropa Belladonna, or deadly nightshade, and the Datura Stramonium, or thorn-apple, were afterwards examined as to the effect of caustic potash upon their officinal preparations.

A grain of belladonna extract was dissolved in an ounce of water, the fluid acted powerfully upon the eye, but
after the addition of twenty minims of liquor potassæ, it ceased to have any influence.

The power of a solution of atropine upon the pupil was equally destroyed by the same agent. Solutions of extract of stramonium, and of the crystallized principle, daturine, were finally tried, and liquor potassæ exerted the same destructive influence upon their activity.

The remarks which have been made upon the solution of potash, and the carbonate and bicarbonate of potash, apply equally to the corresponding preparations of soda. The liquor sodæ producing the same effects as liquor potassæ upon henbane, belladonna, and stramonium; whereas the carbonate and bicarbonate are devoid of any influence.

When liquor potassæ has been added to any of the preparations of the above-named plants, their peculiar odour and taste is rather heightened than diminished, showing that the odorous and vapid principles have no connexion with their physiological action, or therapeutic properties.

The results arrived at in the present communication may be thus summed up:

1. Caustic fixed alkalies, such as exist in liquor potassæ or liquor sodæ, entirely destroy the activity of henbane, preventing its action on the pupil when topically applied, and its influence upon the system when internally administered; and, combined with a proper amount of these alkalies, the largest doses of the preparations of henbane may be given without the production of any symptom.

2. The same influence is exerted by the fixed caustic alkalies upon belladonna and stramonium.

3. The carbonates and bicarbonates of potash and soda produce no injurious effects upon the preparations of any of the three above-named plants.

The deductions naturally to be drawn from these results are:

a. That neither liquor potassæ nor any caustic fixed alkali should be prescribed with tincture or extract of henbane, as the virtues of the latter drug are thereby completely neutralized.
β. That when it is desirable to administer an alkaline remedy with henbane, either a carbonate or bicarbonate should be selected, which would probably be equally efficacious upon the stomach, if such influence be required, and certainly as efficient in altering the condition of the urine, and the mucous membrane of the urinary passages.

γ. That the same precautions should be observed with regard to belladonna and stramonium, if at any time prescribed in conjunction with alkalies.

In carrying out the numerous observations required to arrive at these facts, I have been much assisted by Mr. Walter Morgan, physician's assistant at University College Hospital.
THERAPEUTIC COMMUNICATIONS.—No. II.

ON

THE INFLUENCE OF
LIQUOR POTASSÆ,
AND OTHER CAUSTIC ALKALINE SOLUTIONS,
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It was my original intention to have added a dated postscript to the first therapeutic communication, containing the results of some fresh observations and more recent experiments; circumstances,¹ however, have altered this inten-

¹ Among others, I may mention that, since the abstract of my paper appeared, Mr. France has published a paper in the Lancet, February 27th, 1858, in which he attempts to show that no destructive influence is exerted by potash upon the active properties of belladonna and henbane. If his communication is examined, it will be at once seen how he has fallen into error. In one of his experiments, the amount of potash employed was not more than half sufficient to neutralize the natural acidity of the extract; and a comparison of his results with those detailed in the present and first papers will show that his observations may be perfectly true, and my deductions at the same time absolutely correct. It is to be re-
tion, and induced me to bring before the Society a second paper on the same subject, in which it will be my endeavour:

First: To prove that the active principles of the plants under consideration, are absolutely destroyed by the influence of the caustic potash.

Secondly: To show the ratio which must exist between the different preparations of the plants and the alkali, for the neutralization to be perfect.

Thirdly: To ascertain the time demanded for the decomposition to be complete.

Fourthly: To illustrate clinically the influence of the alkali in preventing the occurrence of symptoms, when large medicinal doses of these solanaceous drugs are administered.

Before proceeding to discuss any of these different heads, it will be necessary, for the right understanding of the subject, to have a clear conception of the nature of the drugs employed in the various experiments. And, in the first place, it must be remembered, that although liquor potassae is strongly caustic and alkaline in reaction, still it is a very dilute solution, containing but 6.7 per cent. of alkali, and, consequently, its power of neutralizing acids is exceedingly limited. Again, it will be found that the preparations of henbane, belladonna, and stramonium (the extracts and tinctures) are acid in reaction, as tested by litmus paper, and to such a degree is this the case, that two fluid drachms of the potash solution are not more than sufficient to neutralize the natural acidity of an ounce of the extracts. Nor must it be forgotten, that in the plants the alkaloids or active principles do not exist in a free state, but combined with acids in the form of salts, and that consequently, before any alkali can act upon them as a destructive agent, they must first be separated from such combinations; and to produce this effect, a portion of alkali necessarily becomes
greeted that attempts should be made to throw a doubt upon the accuracy of an observer, before the experiments from which his results have been obtained are made known, and such must have been the case in the present instance.
neutralized, or rendered inert, as far as ulterior action is concerned; and, lastly, it must be borne in mind, that the most minute quantity of the active principles, or even of the preparations containing them, will cause dilatation of the pupil, and that the effect, at least for a time, will be much the same, whether $\frac{1}{100}$ grain, or $\frac{1}{10000}$ grain of atropine or daturine be put into the eye, and, therefore, we may sometimes neutralize $\frac{1}{100}$ths of the power of a preparation, without being able to ascertain the fact by the local physiological action.

1. The active principles of the plants are absolutely destroyed by the caustic alkali.

In my first communication I did not attempt to show the manner in which the activity of the various plants became neutralized by the action of the caustic potash, resting satisfied with the mere statement of the fact. I have, however, since that period, made several experiments, which throw considerable light upon the subject.

The first consisted in making a solution of atropine in distilled water, with the aid of a few drops of rectified spirit, dividing it into two equal portions, to one of these adding a little liquor potasse, sufficient to effect the desired purpose, and, allowing it to remain for some hours; afterwards shaking each portion with chloroform, and, on the subsidence of this body, pouring off the supernatant fluid, washing the chloroform, and allowing the two quantities to evaporate spontaneously in glass dishes; from the solution to which the liquor potasse had been added, a very slight white-looking residue remained, having a powerful odour; when moistened with water, and a drop of the solution put into the eye, no effect was produced, and it evidently contained no atropine; from the other quantity, however, to which no addition of potash had been made, a gummy residue remained, and a drop of the watery solution produced intense dilatation of the pupil, which did not recover its original size for a fortnight.
In the next experiment, I made a solution of atropine in distilled water, employing one grain of the alkaloid to two fluid ounces; to one half liquor potassae was added, in the proportion of twenty minims to the ounce, to the other a strong solution of the carbonate of potash in the same proportion; after allowing each to remain during four or five hours, chloroform was employed in the same manner as in the first experiment, and the products exposed to spontaneous evaporation; in the case where the carbonate of potash had been added, the product was first gummy in nature, but afterwards became distinctly crystalline; the slightest particle dilated the pupil intensely, and, when a solution was made with water acidulated with hydrochloric acid, and chloride of gold added, a beautiful plumose crystallization of the double chloride of gold and atropine took place.

In the case where liquor potassae had been used, a whitish product was found, having a peculiar odour, causing no action on the eye, even when applied in large quantities, and giving rise to no crystalline salt on the addition of chloride of gold. By these experiments it is proved, that liquor potassae destroys the alkaloid atropine, even in very weak solutions, and at the ordinary temperatures of the air. The nature of the compounds resulting from such decomposition I have not ascertained, it being a subject purely chemical, and one which would probably take some months to investigate fully. Proof is, at the same time, afforded by the second experiment, that the carbonate of potash is devoid of this destructive power on atropine. It might perhaps be imagined that this action of potash on the alkaloids is common, and not at all confined to the group of solanaceous plants; that such, however, is not the case is easily proved, by performing the above experiments with quinine, cinchonine, strychnine, &c., in place of atropine or daturine, when it will be found that no alteration has taken place in the alkaloids, and that they can be recovered in their crystalline forms. It is also a well-known fact, that morphia, when dissolved in caustic alkaline
solutions, can be separated in its original state. Possibly, however, there may be a few of the alkaloids, besides those derived from the solanaceous plants under consideration, which are also broken up by means of dilute solutions of caustic potash.

2. The necessary ratio between the alkali, and the various preparations of the plants.

In my former communication I detailed the result of experiments made with certain definite quantities of the preparations of the plants, without determining the limit to the destructive power of the potash solution; since this time, I have endeavoured to arrive at something like quantitative determinations, which, if not absolutely correct, will still be found sufficiently so for all practical purposes; these results I have given in tabular form, and it will be observed, that the observations have been made upon the tincture and extract of henbane, the extract of stramonium, the tincture and extract of belladonna, and, lastly, upon pure crystallized atropine.

Table exhibiting the amount of Liquor Potassae required to destroy the activity of the preparations of Henbane, Belladonna, and Stramonium, as determined by at least Sixty Experiments.

On Henbane.

| Tincture Hyoscyami, 3i. | Dilated the pupil. |
| Liq. Potassae, 1Mv. | ............. |
| Tincture Hyoscyami, 3i. | Dilated the pupil. |
| Liq. Potassae, 1Mx. | ............. |
| Tincture Hyoscyami, 3i. | No dilatation. |
| Liq. Potassae, 1Mx. | ............. |
| A second tincture of same proportions | No dilatation in two instances. |

XLI.
Extracti Hyoscyami, gr. v. .......... No dilatation in two trials.
Aquæ Destillatæ, ʒi. .......... Liq. Potassa, m̄xxv.

Ext. Hyoscyami, gr. z. (Hospital
Extracts.) .......... No dilatation.

Ext. Hyoscyami (Bell’s Ext.) gr.z
Aquæ Destillatæ, ʒi. .......... A moderate amount of dilatation.
Liq. Potassa, m̄xx.

Ext. Hyoscyami (Bell’s Ext.) gr.z
Aquæ Destillatæ, ʒi. .......... No dilatation in seven cases.
Liq. Potassa, m̄x.

Ext. Hyoscyami, gr. ix
Aquæ Destillatæ, ʒi. .......... No dilatation in six cases.
Liq. Potassa, m̄xxx.

**On Stramonium.**

Ext. Stramonii, gr. v. .......... No action on five eyes.
Aquæ Destillatæ, ʒi. .......... Liq. Potassa, m̄x.

**On Belladonna.**

Tincturae Belladonnae, ʒi. .......... Some (moderate) dilatation of
Aquæ Destillatæ, ʒvi. .......... the pupil.
Liq. Potassa, m̄x.

Tinct. Belladonnae, ʒi. .......... No dilatation in five cases.
Aquæ Destillatæ, ʒvi. .......... Liq. Potassa, m̄xxv.

Ext. Belladonnae, gr. i.j
Aquæ Destillatæ, ʒi. .......... No dilatation in two eyes.
Liq. Potassa, m̄x.

Ext. Belladonnae, gr. v
Aquæ Destillatæ, ʒi. .......... No effect on pupil.
Liq. Potassa, m̄xxx.

Ext. Belladonnae, gr. v
Aquæ Destillatæ, ʒi. .......... No dilatation in four cases.
Liq. Potassa, m̄xxv.

**On Atropine.**

No. 1. Atropinae, gr. ʒ. .......... Dilated the pupil intensely.
a. The above (No. 1), with \( \text{M}XX \). of Liq. Potassae. No action on pupil.

b. The above (No. 1), with \( \text{M}xijas \). of Liq. Potassae. No action on pupil.

c. The above (No. 1), with \( \text{M}vijas \). of Liq. Potassae. Some dilatation of pupil. The same next day.

No. 2. Atropinæ, gr. j. In four cases produced no dilatation.

\begin{align*}
\text{Spt. rect., } & \text{ } 3j. \\
\text{Aq. destill., } & \text{ } 3vij. \\
\text{Liq. Potassae, } & \text{ } \text{M}XX
\end{align*}

a. The same (No. 2), with Atropinæ, gr. j. No action in two cases.

b. The same (No. 2), with Atropinæ, gr. j. No action in two cases.

c. The same (No. 2), with Atropinæ, gr. j. No action in two cases.

d. The same (No. 2), with Atropinæ, gr. j. Produced some dilatation of pupil.

No. 3. Atropinæ, gr. j. No dilatation in two cases.

\begin{align*}
\text{Spt. rectif., } & \text{ } \text{jas.} \\
\text{Aq. destill., ad } & \text{ } 3j. \\
\text{Liq. Potassae, } & \text{ } \text{M}XX
\end{align*}

No. 4. Atropinæ, gr. j. No effect produced; tried in two cases.

\begin{align*}
\text{Spt. rectif., } & \text{ } \text{jas.} \\
\text{Aq. destill., ad } & \text{ } 3j. \\
\text{Liq. Potassae, } & \text{ } \text{M}XX
\end{align*}

A caustic soda solution tried upon atropine and some of the preparations of hembana, was found to destroy their activity; but the requisite quantities were not determined.

From the above table it will be seen, that when atropine is operated upon by liquor potassae, the destructive influence of the latter is so great, that less than 20 minims are required to neutralize one grain of the active principle; and, I believe that a single grain of pure potash will, even in dilute solutions, destroy an equal quantity of the alkaloid.

A grain of caustic potash constitutes but a small medicinal dose of the drug, a twentieth of a grain of atropine,
on the other hand, is often found to act powerfully upon the human subject.

When belladonna preparations are employed in place of atropine, the power of the potash becomes weakened from the causes before alluded to, namely, the natural acidity of the drugs, and the necessity of first displacing the alkaloid from the acid with which it is combined, before the potash can act upon it as a destructive agent. Still, however, the table shows us that 15 minims of liquor potassae will destroy a fluid drachm of the tincture, and that 25 minims are sufficient to produce the same change in 5 grains of the extract, at once demonstrating, that quantities very greatly beyond the medicinal doses of these drugs, nay, even poisonous doses, are rendered quite inert by a moderate amount of the alkaline solution.

The same remarks hold good with regard to daturine, and the preparations of stramonium.

When henbane preparations are examined, the amount of liquor potassae required to neutralize the effect of a fluid drachm of the tincture, is ascertained to be about 10 minims, and the activity of 9 grains of the extract is destroyed by half a fluid drachm. Ten grains of the extract will frequently, when treated in this manner, produce distinct dilatation of the pupil.

This phenomenon, however, need not surprise us, as a single grain of the extract, when properly prepared, will produce the effect on the eye, when diluted with a fluid ounce of water. There are, however, many extracts of henbane found in commerce, which are neutralized when united in the proportion of ten grains to the half-drachm of liquor potassae, and, in the table, experiments illustrating this fact are exhibited.

When the superior extracts are employed in the proportion of ten grains to the half drachm of the potash solution, although the local effect on the eye may be visible, still it is evident, that at least 8ths of the activity of the combination is rendered inert, and no caustic potash can possibly exist in any such mixtures until
the whole of the active principles have been broken up, or disintegrated.

*The preservative power of ammonia salts.*—Certain salts containing ammonia possess a peculiar power, when present in the solution of the active principles, of acting as preservative agents, and of preventing their destruction by the potash. To illustrate this, a solution of atropine was made, and a sufficiency of liquor potassae added, it was then divided into two parts, to one of which a few grains of hydrochlorate of ammonia were added; after a few hours both portions of the solution were tested as to their power of dilating the pupil of the eye; the solution to which the ammonia salt had been added remained active, the other had lost all power. The explanation of this phenomenon is simple; in the presence of an ammonia salt, the potash unites with the acid, and liberates the ammonia, so that free ammonia, and not free potash, exists in the solution, and, consequently, the destructive change does not ensue.

If an alkaline bicarbonate were present, the same protective power would probably be exercised, as the effect of such a salt would be to convert the caustic alkali into a proto-carbonate, which possesses no power of disintegrating the active principle. When the potash solution is added in quantities more than sufficient to neutralize the acid of the ammonia salt, a destructive influence is immediately exerted.

3. *The time required for complete decomposition of the active principles.*

I mentioned in my first paper that about two or three hours appeared to be required, in order that liquor potassae should neutralize the effects of henbane; recently I have determined by more accurate experiments, the time demanded in the case of atropine. A solution of the alkaloid was made by dissolving it in water, with the aid of
a few drops of spirit, in the proportion of a grain to the fluid ounce; liquor potassae was afterwards added, in quantity known to be more than sufficient to destroy it, and a little put into different eyes every quarter of an hour.

After $\frac{1}{2}$ hour, full dilatation was produced.
" $\frac{3}{4}$ hour, full dilatation was produced.
" 1 hour, full dilatation was produced.
" 1$\frac{1}{4}$ hour, full dilatation was produced.
" 1$\frac{1}{2}$ hour, full dilatation was produced.
" 2$\frac{1}{2}$ hour, slight dilatation was produced.
" 2 hours, slight dilatation was produced.
" 2$\frac{1}{2}$ hours, no effect was produced.
" 2$\frac{3}{4}$ hours, very slight dilatation was produced (?)

The same solution gave a negative result when tested the next day. The dilatation remained in proportion as the solution was applied, sooner or later after the addition of the potash. In another experiment no perceptible dilatation was observed after 2$\frac{1}{2}$ hours, although the solution was tried upon several eyes. We may hence conclude, that, after two hours, solutions of atropine containing potash lose their power almost entirely, and, at 2$\frac{1}{2}$ hours, the activity may be said to be extinct. Probably the exact time varies, being dependent on several minor circumstances, as temperature, the strength of the solutions, and the ratio between the potash and atropine, for such influences are found, in other decompositions, to have considerable effect.

4. **Clinical illustrations of the influence of liquor potassae in rendering henbane and belladonna inert.**

Within the last few months it has occurred to me to see several cases in which patients have been very distinctly brought under the influence of these drugs, and in whom the symptoms have vanished by the addition of a few drops of liquor potassae to their medicine, the full quantity of the original and active ingredient being persevered in. The first case was that of a man suffering from some stomach
and intestinal affection, for whom a draught containing nitrate of bismuth, suspended by tragacanth, and a drachm of tincture of henbane, was ordered to be taken three times a day; on the third day he complained of dryness of mouth and throat, the pupils of the eyes were very large, and scarcely acted upon by light. Ten minims of liquor potassae were then added to each dose, and, at the next visit, the pupil was found to contract freely, and the distinctness of vision to be restored.

The second case was that of a young woman suffering from some painful inflammatory affection of the left ankle, and for whom iodide of potassium and bicarbonate of potash with a fluid drachm of tincture of henbane, were prescribed, three times a day. After continuing this medicine for seven days, with much relief to the ankle, she complained of imperfect vision as if a mist were before her eyes; on examination, the pupils were found enlarged, and contracted but slightly with light; she had also dryness of throat. Fifteen minims of liquor potassae were ordered to be added to each draught, in lieu of the bicarbonate of potash. At the next visit, three days afterwards, the patient could see well, and the dryness of the throat had passed off.

On omitting the liquor potassae, and increasing the henbane tincture to a drachm and a half, the eyes in two or three days became again much affected, but the symptoms rapidly vanished when only ten minims of liquor potassae were added to the medicine.

The next case was that of a young woman, for whom, on account of very troublesome cough, was ordered tincture of belladonna, at first in ten-minim doses, but afterwards increased to fifteen. In a short time there was considerable dryness of the throat, imperfect vision, and dilated pupils, which were scarcely affected by light.

The tincture of belladonna was increased to twenty minims, and, at the same time, fifteen minims of liquor potassae added: two days afterwards the pupils contracted freely, the vision was good, and there was no dryness of
the throat. I could relate two or three other cases illustrating the same fact.

Shortly after the abstract of my first paper appeared, an interesting occurrence took place, bearing strongly upon the present subject. A medical gentleman was prescribing for a lady the extract of belladonna, for the purpose of allaying a nervous or spasmodic cough; he commenced with \( \frac{1}{4} \) grain doses, and gradually increased the quantity to \( \frac{1}{2} \) grain, which was ordered to be taken several times in the day. The extract was rubbed up into a draught containing liquor potassae. He was surprised to find that the belladonna neither allayed the cough, nor produced any other symptom; but, on seeing the abstract of my paper in one of the journals, found an explanation of the phenomenon. The next medicine was prescribed with the bicarbonate of potash, in place of the caustic solution; and the result was that he was soon called to see his patient, who was then suffering under the effects of a rather large medicinal dose of the drug, having dryness of the mouth and throat, dilated pupils, and indistinct vision.

These experiments and observations prove beyond doubt the accuracy of the results arrived at in my first communication, and demonstrate that the caustic fixed alkalies possess the peculiar power of destroying the active principles of henbane, stramonium, and belladonna, even when in dilute solution, and that such combinations are therefore both chemically and therapeutically incompatible, and should never be prescribed.
A
CONTRIBUTION
TO THE

SCIENCE OF TERATOLOGY,

BY

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(Communicated by Dr. Silvester.)

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In the following paper it is proposed to bring before the notice of the Society a remarkable instance of congenital malformation of the two upper extremities.

The subject of monstruosities has been ably investigated by Otto St. Hilaire, Vrolik, and many others, but the precise nature of deviations from the normal state has not yet been satisfactorily explained. Descriptive anatomy, as applied to the science of teratology, is still defective; yet it appears to be well calculated to repay investigation. Moreover, accurate dissections, carefully recorded, would be of value for reference, since cases of similar deformity are constantly presenting themselves.
These considerations, together with the importance of the subject as a branch of pathology, and the peculiarities of the example about to be described, make me desirous of bringing before the Society the subject of the present communication.

According to the theory which was first propounded by Meckel, the case about to be described is one of malformation produced by "retardment" or "arrest of development." Otto would have placed it in his Third Order, "Monstra peromelas artubus defectivis," and the Second Genus, "Monstra pereochiras artubus anterioribus defectivis."

I hope that in the course of this inquiry we shall be able to point out, not only that there is a deficiency of development, but that there is evidence of excessive formation to compensate to a certain extent for the original defect, but differing somewhat from the "quantitative antithesis" of Geoffrey St. Hilaire, which he denominates "loi de balancement." This will be fully explained hereafter.

For the following particulars relating to the mother of the child I am much indebted to Mr. McLanglin, who attended at the confinement.

On the 10th January, 1857, Mrs. M—, aged 35, residing near Clapham, London, was safely delivered of the present child. At the period when the labour came on, Mrs. M— believed herself to be at least four weeks from the full period of utero-gestation. The evidence of this was, however, doubtful. She has had ten well-formed children born alive, and two miscarriages. The previous child to this one was a girl, born at the sixth month, who appeared free from deformity, but lived only twenty-four hours. There is no history of accident or illness during pregnancy. On neither maternal nor paternal side could there be discovered any hereditary tendency to congenital malformation. The dietary had been in no respect peculiar. The presentation was cranial, the placenta small, and the cord thin. The mother can assign no cause for the deformity, except her having heard of the birth, in her neighbourhood, of a child
without either arms or legs. This news probably was received before she became pregnant. The child (a male) breathed and cried, but lived about ten minutes only.

On examination, both upper extremities were found to be deformed—the left more so than the right.

*The Right Arm.* (Pl. I, fig. 1.)—The shoulder and upper arm deviate but little from the regular form. The forearm appears shorter than usual. This is occasioned by the situation of the hand, which is fixed in a prone position, and flexed to such an extent that the radial side of the index finger lies adjacent to the radial side of the forearm. The forearm itself is directed a little outwards, so as to form an angle with the outer side of the upper arm. The hand presents four fingers, without the slightest indication of a thumb.

Upon dissection, the radius is found to be entirely absent. The upper part of the ulna and the lower extremity of the humerus are modified to compensate for the deficiency. The defective parts of the hand are the scaphoid trapezium, and the metacarpal bone and phalanges of the thumb. The pronators, supinators, flexors, and extensors of the radial side of the carpus and of the thumb are absent. The flexors and extensors in the middle of the arm are abnormal, but those on the ulnar side are normal. The radial artery is absent.

*The Left Arm* is more deformed. (Pl. I, fig. 6.)—The shoulder is flattened, and the limb tapers gently to the wrist, and hangs down by the side of the chest, with the hand directed backwards and upwards, and fixed in such a position that the palm of the hand faces the back of the forearm. The upper arm seems to be nearly absent, and the prominence of the elbow is felt close to the posterior fold of the axilla. The hand presents only two fingers, the ring and the little finger. Upon more close examination the scapula is found to be well formed, except that the glenoid
cavity has grown into a hemispherical eminence, becoming an example of "excessive formation."

The humerus is so mal-formed that little more than the lower extremity exists.

The only portion of the radius which has been developed is a small cartilaginous nodule, which appears to represent the head of that bone.

The ulna is but slightly modified.

The carpus presents the three inner bones only, viz., pisiform, cuneiform, and unciform.

The metacarpus presents the three inner bones.

The phalanges of two inner fingers only are present.

The muscles of the upper arm are all either absent or very abnormal, and those of the forearm are quite as defective as those of the right arm.

Some interesting peculiarities are met with in the vessels and nerves of both arms.

The integument of both upper extremities is well supplied with fat, and does not present the slightest appearance of a scar.

**RIGHT ARM.**

*The Bones, (see Pl. I.)*

*Scapula.*—Not peculiar.

*Humerus* (Pl. I, fig. 2).—Abnormal at the lower extremity. The capitellum is well marked, but less rounded off than usual; no fossa anterior minor or radial depression exists above it, and the semicircular ridge, which usually is lodged in the space intervening between the radius and ulna, is entirely absent.

*Radius.*—Absent.

*Ulna* (Pl. I, fig. 2).—The shaft does not taper gradually from above downwards, but slightly increases in size from about half an inch (two-fifths) below the articular surface...
(in front) to the lower extremity, the breadth augmenting from one tenth to one fifth of an inch.

It has a uniform gentle curve, the concavity directed forwards. The inner side is rendered slightly concave by the projection inwards of the upper extremity. The outer side is nearly straight. No oblique line is met with on this surface. The olecranon process is well marked.

The outer surface of the coronoid process does not present the lesser sigmoid cavity, but is uniformly convex and rough.

The greater sigmoid cavity is divided, as usual, into two portions, an internal and an external. The internal is small, and concave transversely, as well as from above downwards, directed upwards and inwards; into this the outer part of the trochlea of the humerus accurately fits. The external is rendered larger than usual by projecting forwards as well as outwards; being also concave in the same way, and directed upwards, outwards, and forwards, it is exactly adapted to the capitellum of the humerus, which normally articulates with the head of the radius.

Lower extremity. The ulna ends by a rounded cartilaginous epiphysis. There is no styloid process, but on the outer side there is a slight projection, which, however, does not descend so low as the cartilaginous extremity.

The transverse section of the shaft of the ulna is triangular. The medullary foramen is large, and runs upwards.

The ulna (Pl. I, fig. 2) is set on the humerus very obliquely outwards. This is occasioned by the outer concave portion of the greater sigmoid cavity, which articulates with the capitellum of the humerus, being on a lower level than the inner part of the sigmoid cavity, which is applied to the inner part of the trochlea.

The ulna forms an angle of from thirty to forty degrees with the produced line of the axis of the shaft of the humerus; usually the angle does not exceed ten degrees.

Carpus (Pl. I, fig. 3).—The carpus is unossified. The
scaphoid and trapezium entirely absent. Trapezoid abnormal; it is free above; and on the outer side, where it is convex and smooth, it rests upon the base of the metacarpal bone of the fore finger; the inner side of the trapezoid articulates with the magnum.

Metacarpus and Phalanges (Pl. I, fig. 3).—The thumb is absent; the other fingers are well formed.

The carpus is loosely attached to the lower part of the ulna by ligamentous fibres, in such a way that the hand is at right angles with the anterior surface of the ulna. These fibres are attached to the ulna above the cartilaginous epi-

physis, and descend to the carpus, where they unite with
bands of ligament uniting the bones of the carpus with one another. No synovial cavity appears to exist.

The distance of the carpus from the ulna was one tenth of an inch, when the ligaments were put on the stretch.

Upper Arm.—Defective muscles, nerves, vessels, &c.

The biceps muscle (Pl. 1, fig. 4 a) is thin and flattened. The inner head arises from the coracoid process by a broad ligamentous attachment. The fibres have a direction downwards and outwards, and rest upon the coraco-brachialis muscle with which, to its insertion, they are inseparably united.

About the middle of the arm the biceps is joined by a slender slip of muscle which arises, by a pointed tendinous process, from the ligamentous investment of the humerus, immediately below the insertion of the latissimus dorsi. It passes down between the attachment of the coraco-brachialis, on the inner side, and the pectoralis major and deltoïd, on the outer side. Its extreme length is about half an inch, and its breadth, at the point of junction, one eighth of an inch. It does not extend up to the joint, nor even up to the groove of the humerus, which is usually occupied by the long head of the biceps.

The muscle thus formed is thin, and more or less united with the brachialis anticus, and, a little above the elbow-joint, terminates, partly by joining a piece of tendinous
fascia which passes into the forearm, and will be described hereafter, and partly by attachment to the outer condyle.

The brachialis antebrachii (Pl. I, fig. 4 c) is not well developed. It appears chiefly as a small mass of muscle at the inner side of the biceps, at its lower part. It arises as usual, and is inserted below into the piece of fascia which has been mentioned as the tendon of the biceps, to be traced into the forearm, and also into the coronoid process of the ulna.

There is nothing remarkable about the brachial artery. The nutritious and other branches are not defective.

The brachialplexus. The only nerves requiring special notice are the musculo-cutaneus and the musculo-spiral.

The musculo-cutaneus leaves, as usual, the outer cord of the plexus at the lower border of the pectoralis minor, and soon divides into two, and afterwards into several branches, which pass into the muscles arising from the coracoid process; they descend among the muscular fibres as far as the middle of the upper-arm, and appear to mark the distinction between the coraco-brachialis and the biceps, and the brachialis anticus and the biceps.

The musculo-spiral nerve takes its usual course, but when it arrives in front of the outer condyle it is very small, and, instead of giving off the radial and posterior interosseous nerves, it divides into several very minute branches, which are soon lost in the adjacent muscular structures, particularly the anconeus. It is accompanied by the profunda artery, which has a similar course and distribution.

The supinator radii longus, extensor carpi radialis, longior and brevior muscles are absent.

The inner condyle of the humerus gives origin to muscular fibres which probably represent, or are the imperfect rudiments of, the pronator radii teres and the flexor carpi radialis. They form a small mass of muscle (r), which crosses the elbow-joint obliquely, outwards and downwards: the upper fibres become lost among the descending fibres of the biceps and brachialis anticus,—the lower fibres are attached to the inner surface of the sheet of fascia, which has been mentioned as the prolongation of the tendon of
the biceps, and passes into the forearm as an intermuscular septum.

The shoulder-joint is rendered defective by the absence of the tendon of the biceps muscle; in other respects it appears well formed.

*Front of the forearm.*—The flexor carpi ulnaris, palmaris longus, and flexor sublimis are on the same level, and, with the ulnar artery and nerve, rest upon the profundus.

The flexor carpi ulnaris and palmaris longus muscles are quite normal.

The pronator radii teres and flexor carpi radialis are mentioned above as presenting their usual origin, their further development only being arrested.

The flexor sublimis (Pl. I, fig. 5 a) arises from the inner condyle of the humerus, between the "rudiments of the pronator radii teres and flexor carpi radialis," and the flexor carpi ulnaris, by a common origin, and from that rudimentary mass as far outwards as the tendinous slip from the biceps, and also from the coronoid process of the ulna. It soon divides into two parts; the inner part supplies tendons to the inner two fingers in the usual way; the outer part also supplies two tendons, one of which passes to the fore finger, but the other could not be traced to the second finger, but appeared to be lost in cellular tissue on the outer side of the fore finger. The outer edge of the muscle is free and rounded, and the vessels and nerves for the outer side of the hand emerge from under it. The inner edge, also free, is adjacent to the palmaris longus and flexor carpi ulnaris, with the ulnar nerve and artery between them. It rests upon the profundus.

The flexor digitorum profundus (Pl. I, fig. 5 r) arises from the anterior and inner surface of the ulna for three-quarters of the length of the bone, from the inner part of the olecranon, and from an aponeurosis common to this muscle and the flexor carpi ulnaris. The fibres have a downward and outward direction, and divide beneath the annular ligament into four tendons, whose destinations are the last phalanges
of the fingers. The outer border of the muscle is free, thick, rounded, and fleshy, and touches the vessels and nerves for the outer side of the hand. The cutaneous aspect of the muscle forms an expanded surface, and is in contact with the ulnar nerve and vessel, the flexor sublimis, and the flexor carpi ulnaris.

Length of origin, from inner side of ulna, one inch. Length of ulna, one inch and three quarters.

The flexor sublimis and profundus tendons pass through the same compartment in the annular ligament.

Ulnar nerve (Pl. I, fig. 5, 5).—Quite regular in its course and distribution, both in the hand and arm.

The brachial artery enters the forearm beneath the mass of muscular fibres called the "rudiments of the pronator teres," &c., and the flexor sublimis; it may be considered to become ulnar when it enters the forearm.

The ulnar artery is the direct continuation of the brachial, and, as usual, gives off the anterior and the posterior ulnar recurrent branches, and then pursues its accustomed course until it reaches the wrist. (See Pl. I, figs. 5, 7.)

Palmar part of the ulnar artery.—After passing beneath a slip of tendon from the annular ligament, the ulnar artery is directed outwards in the palm of the hand, and divides into two digital branches for the supply of the little and ring-fingers.

The profunda branch takes its usual course, and appears to unite with the profunda branch of the radial, after passing beneath the abductor indicis.

Branches of ulnar artery (Pl. I, figs. 4 and 5).—At the outer border of the flexor profundus, and covered by the flexor sublimis, the ulnar artery gives off a branch (the interosseous) nearly equal in size to itself, which divides immediately into two vessels—the anterior and the outer.

1. The anterior (median?) passes downwards, at first concealed by the flexor sublimis, but it soon emerges, and takes the course of the outer border of the flexor sublimis, and is accompanied by the median nerve, which at first rests upon it and then passes to its inner side. The artery divides
near the wrist into two branches—deep and superficial palmar.

The superficial palmar branch passes into the hand on the outer side of the tendons of the flexor sublimis, and at the base of the fore finger divides into two branches, for the supply of the fore and the next finger (outer side), and probably communicates with the palmar part of ulnar artery. These palmar digital branches are superficial to the tendons of the sublimis. The superficial branch gives off also a dorsal branch to the index finger.

The deep branch, or carpal, passes into the hand at the base of the metacarpal bone of the fore finger, and is distributed to the deep structures.

2. The outer vessel appears to represent the anterior interosseous branch of the ulnar artery. It is directed backwards, and is overlapped by the profundus, and rests upon the ulna, between the profundus and the extensor digitorum communis. It is accompanied by a branch of the median nerve (interosseous?). Near the wrist it gives off three branches—the outer one to the extensor communis, the middle to the structures about the wrist-joint, and the inner to the profundus, as well as muscular branches to the profundus.

The median nerve (Pl. I, figs. 4 and 5) passes into the forearm beneath the little mass of muscle arising from the inner condyle called "rudiment of pronator teres," &c., and the flexor sublimis; thence its course to the wrist is natural.

The nerve dips beneath the annular ligament to enter the palm of the hand, where it is thus distributed—

1st. An undivided nerve is given off to the outer side of the fore finger.

2nd. It divides at the base of the fore and middle fingers for their opposed sides.

3rd. A connecting branch is given to the ulnar nerve, and probably supplies the opposed sides of the middle and ring-fingers.

Branches.—Whilst the median nerve is beneath the flexor sublimis and the muscular mass arising from the inner
condyle, it gives off its first branch, which turns outwards and soon divides into three branches.

The first has a course directly outwards towards the outer condyle, and then, dividing into two minute branches, is distributed to the muscular slip which appears to be a portion of the profundus muscle arising from the outer side of the ulna.

The middle branch has the same course and distribution as the interosseous artery, and at length enters the structures at the base of the metacarpal bone of the fore finger with the deep palmar vessel mentioned above.

The last nerve gives off what appears to be a cutaneous branch for the outer side of the palm of the hand.

The radial nerve is supplied by a branch of the median (Pl. I, fig. 4), for just as the median nerve is emerging from the outer border of the flexor sublimis, it gives off a branch which takes the same course as the median down to the wrist, where it becomes cutaneous on the back of the hand. It then divides into three parts for the supply of the fingers.

One is an undivided nerve for the radial side of the fore finger; the other two divide at the base of the fingers for the opposed sides of the fore and middle, and outer side of ring, and communicate with the dorsal cutaneous branch of ulnar nerve. Length, from origin to back of hand, half an inch.

At this stage of the dissection we are able to trace the slip of fascia from the biceps into the forearm to its termination (Pl. I, fig. 4). This slip or process of fascia, after covering the outer surface of the lower part of the brachialis anticus mass, passes down as an intermuscular septum into the forearm, between the profundus and the extensor communis digitorum. It then attaches itself to the free margin of the portion of the extensor communis digitorum muscle which arises from the ulna, and, passing down on its outer surface as a thin membrane, is fixed to the greater part of the length of the ulna between the extensor carpi ulnaris and the aforesaid portion of the extensor communis digitorum.
Back of the Forearm.—The extensor communis digitorum, extensor carpi ulnaris, and anconeus, are the only muscles of the superficial layer present.

The extensor communis digitorum (Pl. I, fig. 4 u) is a slender muscle arising from the fascia covering the outer condyle, immediately on the outer side of the biceps, by a pointed process, and the fleshly fibres descend to a tendon which spreads out as a fascia over the back of the hand. Processes may be traced up the back of the middle, ring, and little fingers. The muscle crosses the arm obliquely downwards and backwards, nearly touching the flexor sublimis above; they are separated below from each other by a distance of a quarter of an inch. In this interval may be seen part of the flexor profundus, and the nerves and vessels of the limb.

Along the posterior border is the extensor carpi ulnaris.

Beneath it is the remnant of the extensor communis digitorum.

The extensor carpi ulnaris (Pl. I, fig. 4 w) arises from the outer condyle of the humerus, and from the upper half of the posterior border of the ulna; in other respects it is normal, but there is no extensor of the little finger on the outer side, and no annular ligament on back of the wrist.

The anconeus muscle is present, but requires no special description.

The muscle which has been referred to as the remnant of the extensor communis digitorum, arises from the posterior edge of the ulna, below the origin of the extensor carpi ulnaris (?) and from the outer surface of the ulna down to within one eighth of an inch of the wrist-joint. Pointed above, and becoming wider as they descend, the muscular fibres are inserted into the outer and anterior aspects of the carpus; a few fibres pass round the outer side of the carpus, above the base of the metacarpal bone of the fore finger, and are attached to the fascia covering the back of the hand.

The muscle is situated between the flexor profundus and the extensor carpi ulnaris, and is covered by the extensor communis digitorum.
The anterior edge would be free but for the attachment of the slip of tendon from the biceps and brachialis; and as it descends to the ulna it coats the outer surface of the muscle.

**The Left Arm.** (Pl. I, fig. 6, and Pl. II.)

**The Bones.**

The only part of the scapula which is abnormal is that usually called the head of the bone; this presents a hemispherical eminence, slightly flattened at the end, and having a small depression representing the glenoid cavity. The whole is cartilaginous, and enters into the formation of the shoulder-joint. (Pl. II, fig. 6.)

The humerus (Pl. II, figs. 1, 2, 3) is very diminutive in size, being little more than half an inch in length and rather less in breadth. It is composed principally of cartilage, but a small piece of bone, having somewhat the shape of a truncated cone, is situated at the upper and outer part.

The lower extremity, cartilaginous. Is flattened from before backwards, so that its transverse exceeds its anteroposterior diameter. It is not curved forward so as to present to view any of the articular surface.

The condyles.—The outer condyle is absent. The inner condyle is a rough conical eminence, projecting one fifth of an inch from the inner part of the humerus, to which it is united by its base; at the upper part the juncture is made with the bony portion of the humerus, and there a canal is formed for the transmission of the median nerve.

The articular surface is longest in the transverse direction and is divided by a slight groove into two lateral portions, viz., an external, small, globular eminence, or capitellum for articulation with the upper extremity of the
radius, and situated at the outer and lower angle of the humerus, and an internal pulley-like surface for articulation with the ulna.

The groove which usually intervenes between the capitellum and the trochlea is scarcely marked.

The trochlea turns over behind the bone, but is not apparent before when the bones are in situ; it is convex from before backwards, concave from side to side. The trochlea has no outer margin, the concavity passing without any ridge into the capitellum. The inner margin of the trochlea is well marked. The fossae do not exist, and the elbow-joint is incapable of flexion.

The body.—An irregular bony truncated cone, the axis directed upwards and outwards, and free, the base united with the remaining cartilaginous part of the humerus at its upper and outer angle.

It is flattened in front, convex behind and at the side, so as to be somewhat prismatic in form.

The borders.—The outer runs from above the capitellum towards the apex; the inner from the inner condyle also towards the apex; and the posterior produces the prominence of the posterior surface.

The junction of the bony portion with the cartilaginous takes place anteriorly by a curved line commencing at one tenth of an inch above the capitellum, turning upwards to nearly one third of an inch from the free extremity of the inner condyle, and posteriorly at a distance of about one tenth of an inch from the top of the olecranon.

The radius (Pl. II, figs. 1, 2, 3) is very defective; it appears as a small rounded cartilaginous nodule, slightly flattened from before backwards, and articulating with the capitellum of the humerus and the lesser sigmoid cavity of the ulna. The head of the radius seems to be the part represented. Its greatest diameter is less than half an inch. The upper extremity is composed of two parts, one on the inner side which corresponds with the "cartilaginous circumference," semicircular and convex, for articulation
with the lesser sigmoid cavity of the ulna; the other, terminal, circular, and concave, having a direction upwards and inwards, represents the "expanded articular summit" of the bone; it articulates with the capitellum of the humerus. The junction of these articular surfaces corresponds with the line of contact of the humerus and ulna.

The ulna (Pl. II, fig. 3) deviates but little from the regular form. The olecranon is well formed; the triceps is attached to its inner edge rather than to its summit; it is ossified. The lesser sigmoid cavity appears to be less concave than usual. The annular ligament is absent.

The lower extremity or head of the ulna is cartilaginous, and presents a rounded, slightly concave, articular surface on its inner side, which articulates with the upper part of the cuneiform bone, without the interposition of a fibro-cartilage.

The outer side of the head is smooth, convex, and prominent.

The shaft is much more nearly cylindrical than usual, and does not present its accustomed curves.

The borders are rounded and indistinct, and not flexuous in their course. The posterior border is the more prominent.

The surfaces are slightly convex, and the medullary foramen is situated on the inner instead of the anterior surface, at the junction of the upper and middle thirds.

The carpus (Pl. II, fig. 4).—Two wedge-shaped pieces of cartilage, together with the pisiform, represent the carpal bones. The upper, which answers to the cuneiform, is the smaller, and is convex superiorly for articulation with the ulna; concave inferiorly, where it is opposed to the carpal element below. The inferior and larger, which is the representative of the unciform, is also convex superiorly, and is flattened below, and upon it rest the bases of the three metacarpal bones.
The Fingers.—To the hand belong only two perfect fingers, the ring- and the little finger.

A third metacarpal bone is found on the outer side of the metacarpal bone of the ring-finger, but is considerably smaller in every respect than any of the others, and has no phalanges resting upon it.

The shoulder-joint (Pl. II, fig. 6) is formed between a fibro-cartilaginous cap covering the convex glenoid extremity of the scapula, and the upper and posterior part of the cartilaginous element of the humerus above the summit of the olecranon, and the posterior surface of the bony portion between the posterior and inner borders.

A capsular ligament surrounds the articulation and holds the parts together, and is strengthened by some ligamentous fibres from the contiguous tendons, especially from the pectoralis major and the triceps.

The capsular ligament at the upper part is fixed around the neck of the above-mentioned cartilaginous cap, and at the lower part the ligament is attached to the humerus at its upper and posterior surface.

The tendons of the pectoralis major, deltoid, and triceps muscles are attached just behind the inner border of the bony portion of the humerus, and thus intervene between it and the capsule of the joint.

A synovial membrane lines the capsular ligament. The following muscles surround the articulation, on the inner side; coraco-brachialis, and pectoralis major; above and on the outer side, deltoid and triceps.

The nerves belonging to the outer side of the arm pass beneath the capsular ligament.

Elbow-joint.—The bones are so firmly bound together in situ by fibrous tissues that no movement is permitted.

The pectoralis major (Pl. II, fig. 5 a) has its usual origin from the chest and clavicle, and ends in a tendon, which, instead of being inserted into the bicipital groove of the
humerus, is attached to the inner border of the conical bony portion of the humerus from the base nearly to the summit. Its posterior surface, after crossing the coraco-brachialis, lies upon the capsule of the shoulder-joint. To the upper border of the tendon, near its insertion, are attached fibres from the deltoid.

A process of membrane from the tendon of the pectoralis major passes upwards to the coracoid process, and assists in strengthening the joint and in retaining the arm in position. The tendon of this muscle is a strong flat membrane, three tenths of an inch broad at its insertion.

The pectoralis minor (Pl. II, fig. 5 b) is attached to the tip of the coracoid process, but is not united with the short head of the biceps, this being absent.

The axillary artery (Pl. II, fig. 5 c) commences at the lower border of the first rib, and terminates by the vessel entering the forearm beneath the origin of the flexor carpi ulnaris. The length of the artery is not more than one inch. Above and beneath the small pectoral muscle its connexions with surrounding parts is normal. Beyond that muscle the artery is concealed in part by the lower border of the pectoralis major; but thence to its entrance into the forearm it is covered only by integuments and fascia. Behind it is the lower part of the subscapularis and its tendon. To the outer side is the coraco-brachialis. The artery is surrounded by the nerves of the brachial plexus, and the axillary vein is on the thoracic side.

Just before the vessel enters the forearm it is overlapped by the coraco-brachialis on the outer side, and by the ulnar nerve on the inner side, and it rests upon the inner side of the semi-cartilaginous cap, covering the glenoid extremity of the scapula. All the branches of the axillary artery are represented except the circumflex; those belonging to the limb come off by one trunk just before the artery enters the forearm. This branch soon divides into the scapular, which runs along the lower costa; the profunda and muscular, which wind backwards behind the joint to the triceps and outer side of the arm with the musculo-spinal nerve.
The latissimus dorsi (Pl. II, fig. 5 p) arises as usual from the trunk of the body, and crosses the lower angle of the scapula. But the latissimus dorsi and the teres major tendon, instead of being inserted into the bottom of the bicipital groove of the humerus, are arrested in their course, for, meeting with the long head of the triceps as it descends from the inferior costa of the scapula near its neck, they join it and are conducted upwards on its tendon to its origin, and are inserted into the neck of the scapula. A tendinous intersection marks the line of junction. The deltoid muscle also sends down fibres to this point.

The serratus magnus and intercostal muscles are normal.

The subscapularis muscle (Pl. II, fig. 5 e) arises, as is usual, from all the concave surface of the ventral aspect of the scapula; but the tendon, instead of being inserted into the small tuberosity of the humerus, unites with the tendons of the other scapular muscles, viz., the supra-spinatus, infra-spinatus, and teres minor, and forms with them a smooth white cap, of cartilaginous hardness, fitting upon the glenoid extremity of the scapula, which, instead of being a cavity, is a smooth globular head covered with cartilage. The concavity of the cap is smooth, and freely moveable upon this rounded end of the scapula, whilst the convexity of the cap projects into and forms the scapular element of the shoulder-joint. The capsular ligament of the joint is attached around the neck of this cap. In other respects the subscapularis has its normal connexions.

The supra- (Pl. II, fig. 6 f) and the infra-spinatus (g) have their regular origin, and their fibres converge to a tendon which unites with the teres minor and subscapularis, as described above.

The teres minor (Pl. II, fig. 6 a) is a narrow fleshy slip, which is inseparably united with the infra-spinatus, along whose lower border it lies. Its origin is normal. (For insertion, see above.)

The teres major (Pl. II, fig. 6 i) arises from the rough surface on the dorsum of the scapula, near the inferior costa for some distance; the fibres are united with the
latissimus dorsi at the common point of tendinous intersection for these muscles, the triceps and the deltoid.

The deltoid (Pl. II, fig. 6 k) has its usual origin and form, and its fibres converge to a very extended insertion—the inner portion into the outer part of the tendon of the pectoralis major, just at its attachment to the inner border of the bony element of the humerus, between it and the capsule of the joint; it is, however, separated from the humerus by the origin of the triceps—the middle portion into the upper end of the humerus, between the capsular ligament and the triceps, and also inseparably united with them—the outer fleshy fibres to the upper edge of the teres major for its whole length, and especially to the common point of union for the teres major, latissimus dorsi, and triceps.

The upper arm.—The coraco-brachialis muscle passes from the coracoid process to the inner condyle of the humerus; at its inner side are the artery and nerve for the arm. The triceps covers all the humerus except the inner condyle.

The coraco-brachialis (Pl. II, fig. 5 l) arises from the tip of the coracoid process, and is inserted into the upper part of the inner condyle of the humerus; its length is half an inch. The tendon of the pectoralis major covers it, except at its insertion, which is subcutaneous. It rests upon the subscapularis. Perforating it just below the coracoid process is the musculo-cutaneous nerve; the median nerve rests upon the muscle, as well as a small communicating branch. Along the inner border are the axillary artery and the ulnar nerve.

The triceps (Pl. II, fig. 6 m) covers the whole humerus, except the inner part of the inner condyle, and is the only muscle at the back of the upper arm. It arises from the neck of the scapula, and from the inferior costs of that bone. It is here inseparably connected with the tendons of the latissimus dorsi and teres major, as before described; from this origin the fibres pass nearly vertically downwards to
be inserted into the olecranon process of the ulna, which is just below. The rest of the muscle arises from the anterior surface of the inner condyle, also from the inner and outer borders and posterior part of the apex of the bony body of the humerus, but separated from the capsule of the joint by the attachment of the tendons of the pectoralis major and deltoid, and also from the whole of the anterior, outer, and posterior surfaces of the humerus. From this extensive origin some of the fibres are directed to the tendon of insertion which is fixed to the end of the olecranon; the rest are connected with the fascia or muscles of the forearm, particularly with the extensor communis and anconeus. The triceps is quite superficial. It conceals the musculo-spiral, the median nerve, the profunda vessel, and the rudimentary radius.

Brachial plexus (Pl. II, fig. 5.)—There appears to be nothing peculiar about the three large cords composing the brachial plexus, except the absence of the circumflex nerve. It consists, as is usual, of three large cords—one is on the outer side, one on the inner, and one behind the artery.

1. The outer cord gives origin to one anterior thoracic, the musculo-cutaneous, and the outer head of the median.

2. The inner cord produces a second anterior thoracic, the inner head of the median, the ulnar nerve, and two cutaneous nerves.

3. The posterior furnishes the subscapular branches, and ends in the musculo-spiral.

The anterior thoracic, subscapular, upper, lower, and long, and the posterior thoracic (ext. respiratory, Bell), are quite regular.

The median nerve (Pl. II, fig. 5, 4) arises by two roots from the brachial plexus, one from the outer, the other from the inner cord. Commencing on the outer side of the artery, the nerve crosses over the vessel and passes across the coraco-brachialis muscle where it is joined by a fasciculus from the musculo-cutaneous nerve; it immediately changes its direction, and, proceeding downwards to the forearm, it
traverses a narrow foramen in the upper part of the inner condyle of the humerus (within the one tenth of an inch from the bony portion), and then enters the forearm between the origin of the flexor sublimis and the extensor digitorum communis.

The ulnar nerve (5) is derived from the inner cord of the brachial plexus, and lies close to the inner side of the axillary artery, and parallel to the coraco-brachialis, and rests upon the tendon of the subscapularis; it passes from the axilla into the forearm beneath the tendinous arch which stretches between the inner condyle of the humerus and the olecranon, and from which arises the flexor carpi ulnaris. Here it rests upon the line of junction of the olecranon and the humerus;—one twelfth of an inch broad.

The musculo-cutaneous nerve (6) leaves the outer cord of the plexus at the lower border of the pectoralis minor. It perforates directly the coraco-brachialis, and, upon emerging, gives off a fasciculus to join the median; it is then directed obliquely; passing from under the pectoralis major tendon, it becomes cutaneous at the back of the upper arm. Length, from origin to perforation, half an inch. Diameter, one twentieth of an inch.

The musculo-spiral nerve (17) arises from the posterior cord of the brachial plexus, of which it is the largest trunk. At first the nerve is behind and close to the main artery of the limb, but at the lower border of the tendon of the subscapularis it winds to the outer side of the arm, between that tendon and the upper part of the humerus, and behind the shoulder-joint; it enters the arm by passing through the triceps at its attachment to the upper extremity of the humerus, just behind its bony apex. It then, resting on that bone and covered by the triceps, winds from the back of the humerus to its outer and anterior surface, crossing in its course the ligamentous fibres between the humerus and the rudimentary radius. The body of the humerus is set on rather obliquely, so that with the projecting radius a groove is formed, in which the musculo-spiral nerve lies. This nerve enters the forearm between the extensor com-
municis and the extensor minimi digiti, after giving off
branches to the triceps.

The superior profunda, a small artery, accompanies the
musculo-spiral nerve.

The Forearm (Pl. II, figs. 7, 8, 9).—On the anterior
aspect of the forearm are two superficial muscles, viz., the
flexor carpi ulnaris and the flexor digitorum sublimis, and
between them the ulnar vessel and nerve. The flexor digi-
torum profundus lies under them, but at the inner side of
the flexor carpi ulnaris it is subcutaneous, and its tendons
near the wrist may be observed in the interval between the
tendons of the more superficial muscles.

The flexor carpi ulnaris (Pl. II, fig. 7 r) appears to be
perfectly normal as to its origin, insertion, and relations.
Its length is two inches; its breadth, a quarter of an inch.

The flexor digitorum sublimis (Pl. II, figs. 7 and 8 s)
arises from the anterior and outer surface of the inner
condyle of the humerus, from the coronoid process of the
ulna, and from the ulna for a short distance below, where
it is inseparably united with the profundus, upon which it
rests. Rather below the middle of the forearm the muscle
ends in a small round tendon, which passes beneath the
annular ligament, and divides in the palm of the hand into
two tendons, which are continued across the hand, to be in-
serted into the middle phalanges of the two fingers in the
usual way, after being perforated by the deep flexor tendons.
The flexor sublimis is quite superficial. Along the inner
border is the flexor carpi ulnaris, the ulnar vessel and nerve
between them. To the outer side is the extensor com-
munis digitorum. The superficial flexor rests upon the flexor
profundus.

Just above the annular ligament the tendons of the sub-
limis and flexor carpi ulnaris are separated by an interval
of one eighth of an inch, which allows the tendons of the
profundus to come into view.

The ulnar artery (Pl. II, fig. 7 7) is the direct continua-
tion of the axillary; its course is in a straight line from its
origin to the wrist. The vessel passes with the ulnar nerve into the arm beneath the tendinous arch between the inner condyle of the humerus and the olecranon, which gives origin to the flexor carpi ulnaris, but it soon becomes quite superficial between the flexor carpi ulnaris and the flexor sublimis, and is concealed only by the common integuments and the fascia of the limb, though the former muscle somewhat overlaps it. The ulnar nerve at first lies upon, but soon passes to its inner side. Beneath the vessel is the flexor profundus. On the annular ligament the artery lies close to the tendon of the flexor carpi ulnaris and is crossed by a band of fibres from the tendon of that muscle. The ulnar nerve is still on the inner side.

The palmar part of the ulnar artery (two digital branches), after passing the annular ligament, is continued into the palm of the hand, where it divides into two principal branches, one for the radial side of each finger, i.e., little and ring-finger. There is also a profunda artery.

Branches,—the anterior interosseous and the metacarpal.

—The anterior interosseous arises immediately after the ulnar artery has entered the forearm. It is at first directed outwards and downwards under the flexor sublimis to the interval between that muscle and the extensor communis digitorum. It then runs down between these muscles, resting at first on the profundus and afterwards on the ulna, until within a half an inch from the wrist, when, passing under the extensor communis, it pursues its course to the back of the hand between the tendons of the extensor communis and extensor minimi digitii. It is accompanied by two interosseous nerves, terminal branches of the median.

The metacarpal branch, arising from the ulnar artery about a quarter of an inch above the lower end of the ulna, is directed obliquely backwards beneath the flexor carpi ulnaris, and, winding round the inner side of the hand, it divides into three branches—one for the inner side of the little finger, the other two for the opposed sides of the little and ring-fingers. It has the same course and distribution as the dorsal cutaneous branch of the ulnar nerve. It anastomoses freely with the former branch.
The ulnar nerve (Pl. II, fig. 7, 5) enters the forearm between the attachments of the flexor carpi ulnaris to the olecranon and inner condyle of the humerus with the ulnar artery (that is, behind the inner condyle). It has a straight course through the arm under cover of the flexor carpi ulnaris. It at first rests upon the ulnar artery, but about the middle of the forearm it is continued on the inner side of the vessel to the hand.

It supplies muscular branches to the flexor carpi ulnaris and profundus. The only irregularity of this nerve so far is its first relations with the ulnar artery.

The dorsal cutaneous branch for the hand arises about a quarter of an inch above the end of the ulna, and passes, as usual, obliquely backwards beneath the flexor carpi ulnaris; finally, perforating the aponeurosis of the limb, it is found on the back of the hand, where it supplies the fingers in the following way:

One undivided branch passes to the inner side of the little finger, another branch to the space between the little and ring-fingers, and divides for the supply of their opposed sides. No branch of nerve is found for the outer side of the ring-finger. The dorsal cutaneous branch ought to communicate with a branch of the radial nerve that should supply the space between the ring- and middle-fingers; in other respects it is normal.

The palmar part of the ulnar nerve is normal in every respect, except that it gives a distinct branch to the outer side of the ring-finger. This is usually supplied from the median nerve.

The median nerve (Pl. II, fig. 8, 4), after traversing a canal in the humerus, as before mentioned, descends into the forearm under cover of the triceps, and then passes down deeply between the flexor sublimis and extensor communis, and, resting on the flexor profundus, it gives off the anterior interosseous nerve which accompanies the interosseous artery. The nerve itself, becoming very minute, passes under the annular ligament between the tendons of the flexor profundus and extensor communis, and lies on the
ulna. It next enters deeply into the palm of the hand and divides into three small filaments, thus:

The first was traced into the small interosseous muscle between the rudimentary metacarpal bone for the middle finger and ring-finger. The second divides into two branches—to the lumbricales (?) The third sends off two branches to the structures about the base of the ring and little finger, and each gives a thread to the interosseous muscle.

The flexor profundus digitorum (Pl. II, figs. 7, 9 t) arises from the anterior and inner surfaces of the ulna for three fourths of the length of the bone, and as far outwards as the origin of the extensor digitorum communis, and from the inner part of the olecranon. The muscle is thick and fleshy, and ends in three tendons, which are not separate above the annular ligament.

The cutaneous surface of the muscle is in contact with the ulnar nerve and vessels, the flexor sublimis, and the flexor carpi ulnaris, and the deep surface rests on the ulna. The outer border touches the extensor digitorum communis and the interosseous vessels and nerve, and the median nerve. The inner is subcutaneous, and is connected by the aponeurosis to the posterior margin of the ulna. So that its only irregularities are that it does not arise from the inner half of the interosseous ligament, and that the outer border does not touch the flexor pollicis longus and the quadratus, these parts being absent. The presence of the median nerve is also abnormal, as well as the relation with the extensor communis.

The tendons.—The two inner tendons have the usual insertion into the last phalanges of the fingers; but a third, a small slip of tendon on the outer side, after forming an expansion which covers the rounded head of the small rudimentary third metacarpal bone, is inserted into the fascia at the base of the ring-finger. (First phalanx.)

Palm of the hand.—The parts which belong generally to the little and ring-finger only are present, and require no special description.
The back of the left forearm. (Pl. II, figs. 8, 9.) The following muscles are present:

Extensor digitorum communis.
Extensor minimi digitii.
Extensor carpi ulnaris.
Anconeus.

The continuation of the musculo-spiral nerve enters the forearm between the common extensor of the fingers and the extensor of the little finger.

The extensor digitorum communis (Pl. II, fig. 8, u) arises from the whole of the outer surface of the ulna and from its posterior border to within half an inch of the end of that bone, between the attachments of the flexor profundus and the extensor minimi digitii. It is connected at the upper part with the triceps. Its fibres end in a tendon, which passes through a distinct compartment in the annular ligament at the outer side of the wrist, and then divides into two, which are directed along the back of the hand to be inserted in the usual way into the two last phalanges of the little and ring-fingers.

On the back of the hand the tendon of the ring-finger is united by an oblique band with the tendon of the little finger.

This muscle is situated between the sublimis and the extensor minimi digitii, and partly conceals the profundus. But there exists a small triangular interval in the upper part of the arm, between the sublimis and the extensor communis, in which may be seen the prolongation of the median nerve and the anterior interosseous artery resting on the profundus.

The extensor minimi digitii (Pl. II, figs. 8, 9 v) arises from the lower extremity of the rudimentary radius and from the ulna just below it, from about the upper one third of the posterior border, and from the intermuscular septum situate between itself and the extensor carpi ulnaris. Its direction is downwards and a little forwards, and it passes through a distinct sheath of the annular ligament on the outer side of the ulna, and its tendon, which is split into two
directly afterwards, ends by joining the common expansion on the first phalanx of the little finger.

The continuation of the musculo-spiral nerve in the forearm (Pl. II, figs. 8, 17). It enters the outer side of the forearm between the extensor communis digitorum and the extensor minimi digiti, and, after giving off branches to the extensor carpi ulnaris and extensor minimi digiti, ends in filaments distributed to the extensor communis.

The extensor carpi ulnaris (Pl. II, fig. 9, w) arises from the end of the rudimentary radius and from the posterior border of the ulna, middle one third, below the anconeus, between the attachment of the flexor profundus on the inner side and the extensor minimi digiti on the outer side. Its course is downwards, forwards, and inwards, having on its inner side, successively, the anconeus, the flexor profundus, and the tendon of the flexor carpi ulnaris. It passes through a distinct sheath of the annular ligament on the inner side of the ulna, to be inserted into the base of the metacarpal bone of the little finger.

At their origin the extensor carpi ulnaris and the extensor minimi digiti are in contact, but, as these muscles descend, they diverge from each other, one passing forward and inward, the other forward and outward, so that their tendons have a corresponding position on opposite sides of the extremity of the ulna. By this divergence a triangular portion of the ulna, about the lower quarter of its posterior surface, becomes subcutaneous. The rounded extremity of this bone forms the projection at the back of the wrist.

The anconeus (Pl. II, fig. 9 x) arises from the end and outer side of the rudimentary radius in common with the extensor carpi ulnaris, and from the adjacent part of the humerus; in other respects it requires no special notice.

The annular ligament, after arching over the tendons, passes round behind the lower extremity of the ulna, and, connected with the fascia of the back of the hand below and with the aponeurosis of the forearm above, forms a sheath which encloses the small cartilaginous representatives of the carpal bones.
The back of the left forearm. (Pl. II, figs. 8, 9.) The following muscles are present:

Extensor digitorum communis.
Extensor minimi digiti.
Extensor carpi ulnaris.
Anconeus.

The continuation of the musculo-spiral nerve enters the forearm between the common extensor of the fingers and the extensor of the little finger.

The extensor digitorum communis (Pl. II, fig. 8, u) arises from the whole of the outer surface of the ulna and from its posterior border to within half an inch of the end of that bone, between the attachments of the flexor profundus and the extensor minimi digiti. It is connected at the upper part with the triceps. Its fibres end in a tendon, which passes through a distinct compartment in the annular ligament at the outer side of the wrist, and then divides into two, which are directed along the back of the hand to be inserted in the usual way into the two last phalanges of the little and ring-fingers.

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The extensor minimi digiti (Pl. II, figs. 8, 9 v) arises from the lower extremity of the rudimentary radius and from the ulna just below it, from about the upper one third of the posterior border, and from the intermuscular septum situated between itself and the extensor carpi ulnaris. Its direction is downwards and a little forwards, and it passes through a distinct sheath of the annular ligament on the outer side of the ulna, and its tendon, which is split into two
directly afterwards, ends by joining the common expansion on the first phalanx of the little finger.

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The extensor carpi ulnaris (Pl. II, fig. 9, w) arises from the end of the rudimentary radius and from the posterior border of the ulna, middle one third, below the anconeus, between the attachment of the flexor profundus on the inner side and the extensor minimi digiti on the outer side. Its course is downwards, forwards, and inwards, having on its inner side, successively, the anconeus, the flexor profundus, and the tendon of the flexor carpi ulnaris. It passes through a distinct sheath of the annular ligament on the inner side of the ulna, to be inserted into the base of the metacarpal bone of the little finger.

At their origin the extensor carpi ulnaris and the extensor minimi digiti are in contact, but, as these muscles descend, they diverge from each other, one passing forward and inward, the other forward and outward, so that their tendons have a corresponding position on opposite sides of the extremity of the ulna. By this divergence a triangular portion of the ulna, about the lower quarter of its posterior surface, becomes subcutaneous. The rounded extremity of this bone forms the projection at the back of the wrist.

The anconeus (Pl. II, fig. 9 x) arises from the end and outer side of the rudimentary radius in common with the extensor carpi ulnaris, and from the adjacent part of the humerus; in other respects it requires no special notice.

The annular ligament, after arching over the tendons, passes round behind the lower extremity of the ulna, and, connected with the fascia of the back of the hand below and with the aponeurosis of the forearm above, forms a sheath which encloses the small cartilaginous representatives of the carpal bones.
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directly afterwards, ends by joining the common expansion on the first phalanx of the little finger.

The continuation of the musculo-spiral nerve in the forearm (Pl. II, figs. 8, 17). It enters the outer side of the forearm between the extensor communis digitorum and the extensor minimi digiti, and, after giving off branches to the extensor carpi ulnaris and extensor minimi digiti, ends in filaments distributed to the extensor communis.

The extensor carpi ulnaris (Pl. II, fig. 9, w) arises from the end of the rudimentary radius and from the posterior border of the ulna, middle one third, below the anconeus, between the attachment of the flexor profundus on the inner side and the extensor minimi digiti on the outer side. Its course is downwards, forwards, and inwards, having on its inner side, successively, the anconeus, the flexor profundus, and the tendon of the flexor carpi ulnaris. It passes through a distinct sheath of the annular ligament on the inner side of the ulna, to be inserted into the base of the metacarpal bone of the little finger.

At their origin the extensor carpi ulnaris and the extensor minimi digiti are in contact, but, as these muscles descend, they diverge from each other, one passing forward and inward, the other forward and outward, so that their tendons have a corresponding position on opposite sides of the extremity of the ulna. By this divergence a triangular portion of the ulna, about the lower quarter of its posterior surface, becomes subcutaneous. The rounded extremity of this bone forms the projection at the back of the wrist.

The anconeus (Pl. II, fig. 9 x) arises from the end and outer side of the rudimentary radius in common with the extensor carpi ulnaris, and from the adjacent part of the humerus; in other respects it requires no special notice.

The annular ligament, after arching over the tendons, passes round behind the lower extremity of the ulna, and, connected with the fascia of the back of the hand below and with the aponeurosis of the forearm above, forms a sheath which encloses the small cartilaginous representatives of the carpal bones.
### Table of the Muscles

<table>
<thead>
<tr>
<th>Right Arm</th>
<th>Left Arm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Abnormal</td>
</tr>
<tr>
<td>Triceps.</td>
<td></td>
</tr>
<tr>
<td>Palmaris longus.</td>
<td>Flexor carpi ulnaris.</td>
</tr>
<tr>
<td>Flexor carpi ulnaris.</td>
<td>Flexor profundus.</td>
</tr>
<tr>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Extensor communis digitorum.</td>
</tr>
<tr>
<td>Anconaeus.</td>
<td></td>
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<td></td>
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<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>Right Arm</td>
</tr>
<tr>
<td>--------</td>
<td>-----------</td>
</tr>
<tr>
<td><strong>Ulnar Artery.</strong></td>
<td><strong>Ulnar Artery.</strong></td>
</tr>
<tr>
<td>The continuation of the brachial in size, but not in direction. Has a curved course. Enters the forearm under cover of superficial muscles arising from the inner condyle. Comes in contact with ulnar nerve at juncture of upper and middle one-third of arm.</td>
<td>Ditto.</td>
</tr>
<tr>
<td><strong>Branches:</strong></td>
<td><strong>Branches:</strong></td>
</tr>
<tr>
<td>1. Anterior ulnar recurrent.</td>
<td>1. Ditto.</td>
</tr>
<tr>
<td>2. Posterior ulnar recurrent.</td>
<td>2. Ditto.</td>
</tr>
<tr>
<td>3. Common interosseous.</td>
<td>3. Ditto.</td>
</tr>
<tr>
<td>(a.) Anterior interosseous.</td>
<td>(a.) Ditto.</td>
</tr>
<tr>
<td>(γ.) Median artery.</td>
<td>(γ.) Median supplies—</td>
</tr>
<tr>
<td>(δ.) Posterior interosseous.</td>
<td>Carpal.</td>
</tr>
<tr>
<td>Two digital (palmar) to fore and middle finger.</td>
<td>Dorsal to index finger.</td>
</tr>
<tr>
<td><strong>5. Carpal.</strong></td>
<td><strong>5. —</strong></td>
</tr>
<tr>
<td>(α.) Anterior.</td>
<td>See (γ.) Median artery.</td>
</tr>
<tr>
<td>(δ.) Posterior.</td>
<td></td>
</tr>
<tr>
<td>Metacarpal artery.</td>
<td></td>
</tr>
<tr>
<td><strong>6. Superficial palmar arch.</strong></td>
<td><strong>6. Superficial palmar.</strong></td>
</tr>
<tr>
<td>(α.) Profunda.</td>
<td>(α.) Profunda.</td>
</tr>
<tr>
<td>(δ.) Four digital.</td>
<td>(δ.) Two digital.</td>
</tr>
<tr>
<td>1. Inner side of little finger.</td>
<td>1. Little finger.</td>
</tr>
<tr>
<td>3. Contiguous sides of ring- and middle fingers.</td>
<td></td>
</tr>
<tr>
<td>4. Contiguous sides of middle and index fingers.</td>
<td></td>
</tr>
<tr>
<td><strong>Radial Artery.</strong></td>
<td>Absent.</td>
</tr>
</tbody>
</table>
### Table of the Nerves

**NORMAL.**

<table>
<thead>
<tr>
<th>Ulnar Nerve</th>
<th>Right Arm</th>
<th>Left Arm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regular</td>
<td>Regular</td>
<td>Regular</td>
</tr>
</tbody>
</table>

**Median Nerve:**
1. Enters the forearm between two heads of pronator radii teres.
2. Is placed between the superficial and deep flexors.
3. At the lower part of arm is covered only by integuments, and lies between the flexor carpi radialis and the superficial flexor of the fingers.
4. Takes the middle line of the arm, and enters the palm beneath the annular ligament.

**Branches:**
- *Muscular.*—To pronators and flexors, except flexor carpi ulnaris and part of profundus.
- *Interosseous.*—Course of interosseous artery.

**Cutaneous Palmar.**

<table>
<thead>
<tr>
<th>Palmar part</th>
<th>Five digital</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 and 2. Thumb.</td>
<td>3. Outer side of index finger and 1st lumbrical.</td>
</tr>
<tr>
<td>4. Index and middle fingers and 2d lumbrical.</td>
<td>5. Middle and ring-fingers, and communicating with ulnar.</td>
</tr>
</tbody>
</table>

**Radial Nerve I (Back of hand):**
1. Radial side of fore finger.
2. Opposed sides of fore and middle fingers.
3. Opposed sides of middle and ring-fingers, and communicating with ulnar.

**Ditto.**

**Palmar part:**
1. Outer side of fore finger.
2. Opposed sides of fore and middle fingers.
3. Communicates with ulnar, and probably supplies middle and ring-fingers.

**Median Nerve:**
1. Enters the forearm through a canal in the humerus.
2. Is placed between the flexor sublimis and extensor communis, and rests upon the flexor profundus.
3. Lies deeply between the tendons of flexor profundus and extensor communis, and rests on the ulna.
4. Enters the palm beneath the annular ligament.

**Branches:**
- *Muscular.*—To profundus also.
- *Interosseous.*—Ditto.

**Interosseous.**—Course of interosseous artery.

---

**Palmar part.**—Three branches to lumbricales and interossei muscles.
CONTRIBUTION TO THE SCIENCE OF TERATOLOGY. 103

**Absent Bones.**

<table>
<thead>
<tr>
<th>RIGHT ARM</th>
<th>LEFT ARM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Radius</td>
<td>Radius, except the head.</td>
</tr>
<tr>
<td>Scaphoid</td>
<td>Scaphoid semilunar.</td>
</tr>
<tr>
<td>Trapezius</td>
<td>Trapezius trapezoid magnum.</td>
</tr>
<tr>
<td>Metacarpus and phalanges of thumb</td>
<td>Metacarpal bones of thumb and fore finger.</td>
</tr>
<tr>
<td></td>
<td>Phalanges of thumb, fore and middle fingers.</td>
</tr>
<tr>
<td></td>
<td>Upper part of humerus.</td>
</tr>
</tbody>
</table>

**Measurements.**

<table>
<thead>
<tr>
<th></th>
<th>RIGHT ARM</th>
<th>LEFT ARM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Length of humerus</td>
<td>2 5/6ths</td>
<td>5/6th of inch more</td>
</tr>
<tr>
<td></td>
<td>than 1 1/4 inch.</td>
<td>5/6th of inch less</td>
</tr>
<tr>
<td></td>
<td></td>
<td>than 2 inches.</td>
</tr>
<tr>
<td>Length of ulna</td>
<td>1/4</td>
<td>5/6ths</td>
</tr>
<tr>
<td>Length of hand from wrist to tip of fingers</td>
<td>2</td>
<td>...</td>
</tr>
<tr>
<td>Circumference of upper arm</td>
<td></td>
<td>2 1/2</td>
</tr>
<tr>
<td>Circumference of forearm</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Conclusions.**

1. The deformity appears to be the result of—first, the original malformation of the germ; secondly, the subsequent deformation of the embryo and fetus, by causes operating on its development; and, thirdly, certain *compensations* and vital accommodations having a conservative tendency.

2. The arrest of development reacts on various parts of the body, and particularly on such parts as have either a casual or a natural connexion with the original malformation.

3. A *law of compensation* prevails during the growth of the body in monsters. It consists in a certain tendency to render the parts as nearly normal as possible, and to make up by excessive formation for the defective development of an adjoining part.

This principle may be shown to exist equally in the vegetable kingdom. If the leading shoot of a plant be cut off, its place is supplied by the development of a lateral branch. So also when a portion of the carpel of a fruit is abortive, the defect is remedied by the excessive development of a similar or corresponding part; for instance, in the common walnut,
when one shell is abortive, the gap is closed by the growth of the remaining shell.

In the animal kingdom, when one bone is arrested in development, a neighbouring bone has the power of excessive increase, so as to make up, to a certain extent, for the deficiency. Thus, in the subject before us, the outer part of the great sigmoid cavity of the ulna is rendered larger than usual by projecting forwards and outwards. It is concave, and the concavity is directed upwards and outwards, and it becomes exactly adapted to the capitellum of the humerus, or that portion of the lower articular extremity of the humerus which usually articulates with the head of the radius. In this way the integrity of the elbow-joint is to a certain extent maintained, notwithstanding the entire absence of the radius.

The same law, when applied to the vascular system, leads to increase of size and to the distribution of a usually small branch of artery to compensate for the deficiency of the normal trunk. For instance, the superficial palmar arch, generally supplied by the ulnar artery, is defective in the right arm. The median artery, which is usually a small branch of the interosseous artery, accompanying the median nerve, compensates for the deficiency, and, passing with the median nerve into the palm of the hand, gives off digital branches to the fingers which require them, and ends by completing the superficial palmar arch.

This peculiarity of the median artery is noticed in Dr. Quain’s ‘Elements of Anatomy,’ in which the vessel is regarded as a ‘reinforcing vessel.’

In the left arm, the metacarpal branch, arising as a separate artery and supplying digital branches, affords an illustration of the same law.

An instance occurring in the nervous system may be mentioned. In the right arm, a branch of the interosseous nerve, given off from the median trunk, compensates by its distribution for the absence of the radial nerve, and supplies digital branches, usually supplied by the radial, to the back of the hand.
Possibly the very remarkable state of the left shoulder-joint may be explained by reference to this law of compensation. The upper portion of the humerus which enters into the formation of the articulation is concave; the glenoid extremity of the scapula appears to compensate for this deficiency by becoming convex and prominent, projecting into the capsular ligament so as to meet the concavity of the humerus, and thus to form a ball-and-socket joint. The ball in this case is transferred from the humerus to the scapula.

The law of compensation differs entirely from the "quantitative antithesis," which Geoffroy St. Hilaire denominates "loi de balancement." According to this law, the excessive development of one part of the body is often connected quantitatively with checked formation of another. For anencephalia, cyclopia, spina bifida, are often attended by excessive multiplication of fingers and toes; sireno-melia, by superfluous vertebrae and ribs; and frequently there occur in double monsters malformations of the head. Meckel saw, in one single instance, this "antithesis" exhibit itself in different children of the same mother. A girl had on each extremity a superfluous digit, whilst one hand of her sister wanted four fingers, being the precise number of digits which her sister had in excess, reckoning the four extremities together. (Article "Teratology," Dr. Todd's 'Cyclopædia,' by Vrolrik.)

4. The present inquiry confirms, in some measure, the opinion held by Vrolik, Müller, Bischoff, Stannius and others, that the several parts of the body are formed and developed independently of one another. An example may be taken from the osseous system. The radius may be defective in both arms, although the condition of both arms differs as to the humerus and carpus, while the ulna remains in its normal state in both arms. But it must be admitted that the presence or absence of the radius or ulna seems to exert an influence on the presence or absence of their respective sides of the hand. Probably the absence of the trapezium causes the disappearance of the thumb, as is known to be the case in comparative anatomy. Although the several parts of the
body may appear to be formed originally independently of one another, and no doubt their normal form is from the first impressed upon them, still the principle of mutual compensatory adaptation seems ultimately to prevail.

5. The absence, arrest of development, or defective condition of the radius, which appears to be the rule in congenital malformations of the human forearm, is not the normal state in beasts: the ulna is the bone which in them is atrophied. In the horse or the ox the ulna is retained only in so far as it is required to strengthen the radius, make the joint of the elbow secure, and give advantageous attachment to certain muscles, and forms what in human anatomy would be called a process of that bone. In the bat the ulna is similarly atrophied, and reduced to its olecranon and proximal half, which is ankylosed to the radius. (See Owen's 'Nature of Limbs'.)

6. "As the transient forms of the human foetus are for the most part comparable to the persistent forms of the lower animals, the malformations occasioned by impeded development often acquire a brute appearance." (Vrolik.) For example, in this case the arrest of development of the humerus suggests a resemblance to the fin of a fish, in which we find one segment of the limb abrogated, and its framework attached to the scapular arch by the two bones answering to the radius and ulna. With regard to the convex head of the scapula, we know that some animals have the socket on the humerus and its articulating ball on the scapula, so that instead of a convex head moving on a concave socket, a concave surface, formed on the head of the humerus, moves on a convex articulating surface on the scapula. There may be found in animals an example of this kind. "C'est à dire qu'une concavité se meut en tout sens sur une convexité; mais l'homme ne presente point cette disposition." (Bichat, 'Anatomie générale,’ p. 170.) But, as a whole, in man the organization is always essentially human.

7. The muscles are directed to fixed points of attachment,
and in the most nearly regular way possible under the altered circumstances. When a bony insertion is unattainable, they unite together by their tendons; there is a vital accommodation to the exigence of the case. The following are illustrations of this fact: In the right arm, where the radius is absent, the biceps tendon passes into the forearm and is inserted into the ulna. The latissimus dorsi is attached to the neck of the scapula, the usual place of insertion being absent, and the scapula being adjacent; the latissimus dorsi is first united with the tendon of the triceps muscle, and is by it directed to the above-mentioned bony attachment. The union of muscles by their tendons, so as to obtain as firm an attachment as possible, is well exemplified by the scapular muscles of the left shoulder. The tendon of the subscapularis, instead of being inserted into the small tuberosity of the humerus (which is absent), unites with the tendons of the other scapular muscles, viz., the supraspinatus, infraspinatus, and teres minor, and forms with them a tendinous cap, which fits upon the glenoid extremity of the scapula.

8. The absence of the usual bony attachment, or the want of a firm point of insertion, exerts a material influence upon the development of a muscle. This dissection furnishes many examples of this fact. There is one so remarkable that it requires to be mentioned. The long head of the biceps in the right arm is undeveloped; the shoulder-joint appears well formed in other respects; but the radius is absent, and the tendon of the biceps below exhibits a very unsatisfactory attachment in the forearm. These causes may perhaps operate upon the nutrition of the muscle.

9. The absence or defective state of an organ reacts unfavorably upon the formation of the nerves and vessels which supply it, even at a distance. This proposition is similar to that of the preceding paragraph, but is applied to the nervous and vascular systems. The absence of the radial artery and nerve in both arms affords an example, the radial side of both arms being defective.
The facts pointed out in the last three propositions, together with the law of compensation (No. 3), indicate an increased divergence from the regular anatomy of the part, but have nevertheless a most beneficial and conservative tendency, rendering the capacity for life as great as possible, notwithstanding the malformation.

10. The hand is maintained in its proper position by the radius. If the radius be defective in length, absent, or in excess, the posture of the hand is abnormal. The following cases are recorded:

First. Congenital deficiency in the length of the radius. There are certain cases of what have been called "congenital luxations of the wrist-joint," in which the bones of the forearm are placed on the dorsum of the carpus, which, I think, are explicable on this supposition.

Cruveilhier, in his 'Pathological Anatomy' (liv. ix, 1833), has published an example of this deformity, occurring in an adult female, although he was not aware of the true nature of the case. In this case the forearm was preternaturally short, and it formed a right angle with the hand, which besides was inclined to the radial side of the forearm; extension was impossible; flexion, to a certain degree, was permitted; the inferior extremities of the radius and ulna were dislocated backwards, and formed a very considerable prominence beneath the skin posteriorly. The extremity of the radius was less salient, and descended much less (half an inch) than that of the ulna. (See the article "Wrist-joint," in Dr. Todd's 'Cyclopedia,' by Robert Adams, Esq.)

Case 2, from the same source.—"The lower extremities of the bones of the right forearm could be seen and felt on the dorsum of the carpus, where they formed a very remarkable projection. The lowest extremity of the ulna could be seen to descend below the level of the lowest extremity of the dislocated radius. The hand inclined to the radial side; it could be extended on the forearm freely, but flexion was incomplete. The forearms appear scarcely more than half their normal length. The radius was only four
inches and a half in length; the ulna, six inches in length, was prolonged below the radius nearly half an inch."

Case 3 presents similar appearances.

Secondly. Congenital absence of the radius, the source of malposition of the hand.

_Vide 'Monstrorum Sexcentorum Descriptio Anatomica,'_
A. G. Otto.

No. CCXXXV.—"Manus simul in parte radiali ita inflexe sunt, ut pars radialis digitis indicis partii radiali antibrachii adjaceat."—"Radium cum parte musculorum vicinorum et cum parte ossium carpi adhuc cartilagineorum in radiali carpi latere deesse apparebat. Ulna normalis est."

No. CCXXXVI.—"Artus superiores eodem fere vitio laborant atque in monstro superiore."—"Et manus, quarum margines radiales ad antibrachia adducti sunt; pollicibus carent."

No. CCXXXVII.—"Manus vero et contorta sit, ita ut margo ejus radialis antibrachio incumbat, et pollice careat."—"Radius et cum eo omnes musculi, qui ei affixi sunt, prorsus desiderantur."

No. CCXXXVIII.—"Manus autem incurvata, quatuor tantum digitis instructa, quam pollice careat, preterea adeo introrsum et deinque sursum directa est, ut radiale manus et digitis indicis ipsum marginem radialem antibrachii attingat."—"In antibrachio utriusque lateris radius plane desideratur pariter atque pars ossium cartilagineorum radialis carpi lateris. Ulna-normalis est neque solito crassior."—"Supinator et pronatores desunt; etiam pars inflexorum et extensorum radiiulium desiderari videtur."

No. CCXXXIX.—"Manus autem ita introrsum flexae inveniuntur, ut margines eorum radiales antibrachia spectent—radius prorsus deesse vidi, quocum etiam omnes musculi rotatores, abductores et bini extensores pollicis desiderantur."

The dislocation or flexed condition of the hand appears to depend, in those cases in which the radius and thumb are absent, upon the want of the support of the radius, and upon the contractions of the flexors of the fingers not being opposed by the absent extensors of the thumb.
Thirdly. Dislocation of the hand, of which the radius is excessive in length.

Cases of this kind seem to be very rare. Mr. Adams mentions (in his article "Wrist-joint," before referred to) having seen such a case of congenital luxation of the wrist. He says: "The radius has, at its lower extremity, passed half an inch lower down on the back of the carpus than the ulna."

11. The deformity in the right arm does not conform to the rule laid down by Rokitansky: "When the radius is wanting, the thumb and fore finger, with so much of the carpus as belongs to them, are wanting too."

12. Although both upper extremities are deformed, the malformation is not precisely the same on each side.

In this paper I have purposely avoided entering upon the general subject of monstrosities, and have endeavoured to keep closely to the facts and inferences suggested by the particular instance before us.

Such observations as naturally presented themselves from consideration of the dissection have been noted, and one object of these has been to point out that a full grown monster is no such nature, but that its form is the result of certain definite laws, the tendency of which is to compel the tissues to rectify as far as possible the original defect in the germ, by means of a principle of vital accommodation and compensation; moreover, if, as Mr. Lawrence (in the 'Transactions,' vol. v, p. 213) proves, the malformation of the germ "owes its origin to something connected with our peculiar mode of existence"—and if, as I have endeavoured to show, the tendency of the laws regulating the formation of the body is to render the parts as nearly normal and the body as capable of life and existence as possible—it can scarcely be said that monstrosities "offer an apparent exception to the inferences which have been drawn from the animal kingdom in general concerning some attributes of the creating power." (Ibid.)
ON SOME POINTS

IN THE

PATHOLOGY AND MORBID ANATOMY

OF

GLAUCOMA.

BY

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The practice of excising the eyeball in hard and painful conditions of the globe, when all sight is lost, and when the diseased state of one eye places the other in jeopardy, was, I believe, first brought into practice in the Royal London Ophthalmic Hospital, in the winter of 1854-5.

Since then this operation has come into more general use; and I have had frequent opportunities of examining and dissecting eyes affected with glaucoma, directly after extirpation and whilst they were in a perfectly fresh state.

I wish to offer to the Royal Medical and Chirurgical Society a short record of the symptoms, the ophthalmoscopic phenomena, and structural changes, which have presented themselves in a large number of cases of this disease. Many have occurred in the Moorfields Ophthalmic Hospital, and I am glad to embrace this early opportunity of expressing how greatly I am indebted to the hospital staff for the courtesy and liberality which have enabled me to prosecute these investigations. I feel also great pleasure in expressing my deep obligations to Mr. Bowman, who has kindly permitted me to examine many cases which have occurred in his private practice, and given me many valuable suggestions.
Authorities have long recognised two varieties of glaucoma— the acute and the chronic, and this division has been adopted by Gräfe, in an excellent memoir on the 'Treatment of Glaucoma,' addressed to the French Institute.¹

Acute Glaucoma.—The symptoms of acute glaucoma have been so often and so graphically described, that I shall only briefly allude to them. They are, in short, violent pain in the eyeball; rapid blindness; a widely dilated, fixed pupil; a peculiar hazy greenish appearance of the lens, called glaucomatous; and great hardness of the globe.

The ophthalmoscopic phenomena are: a dilated state of the retinal veins, often tortuous and turgid with blood; small ecchymoses scattered over the surface of the retina; occasionally small blood-clots in the vitreous humour; pulsation in the central artery of the retina; and an excavated state of the optic papilla.

The pulsation of the central artery in glaucoma, and the excavated state of the optic papilla, were, I believe, first noticed by Gräfe, who insists upon their being pathognomonic symptoms of this disease. The pulsation in the arteria centralis is synchronous with the pulse at the wrist; I have counted it, beat for beat, with my finger on the radial artery. Gräfe points out that, when not present spontaneously, it may usually be produced by pressing on the eyeball with the finger. The cupped, excavated state of the optic papilla, persists for some time after removal of the eyeball. I found it well marked in an eye I dissected on 24th March, 1857 (in this case also the turgid retinal veins seemed to stop short at the margin of the optic papilla, whilst the arteries could be traced across the margin converging to a single trunk at the usual point of entrance).

In acute glaucoma the urgent symptoms often set in most suddenly, but a careful inquiry shows that, in a large number of cases, the sudden outbreak of the disease has

¹ 'Note sur la guérison de Glaucome au moyen d'un procédé opéra- toire, adressé à l’Institut de France,' par le Docteur A. de Gräfe.
been foreshadowed by a set of premonitory symptoms, which may, however, have been so slight as not to have excited the serious fears of the patient. These symptoms are:—occasional dimness, usually towards evening; shooting pains in the eyeball; muscae, and sparks of light. Gräfe also calls attention to the diminished size of the field of vision. He thinks these prodromic symptoms occur in about three fourths of the cases. There are, however, cases in which these premonitory symptoms have not been observed. After some time, the sudden outbreak may be followed by a remission; the pain abates, and the patient may be able to see large objects, though indistinctly. We must not be deceived by this improvement; it is only a lull in the disease, and is sure to be followed by fresh paroxysms, which will sooner or later end inevitably in blindness. As the disease advances, large turgid veins appear upon the sclerotic, emerging in the ciliary region, and running backwards in a tortuous course. The iris loses its brightness, (in a case under Mr. Bowman's care in the Moorfields Hospital, March, 1857, a large dilated vein was noticed upon the iris,) and the aqueous humour has a yellow tint. The lens becomes opaque and swollen. The cornea loses its lustre, and becomes dim; its epithelium is raised in minute vesicles, and sloughing may take place: (this mode of termination was well exemplified by a case treated by Mr. Bowman at Moorfields, in June, 1847,) Gräfe has drawn attention to the insensibility of the cornea in glaucoma.

At a further stage, in many instances, sclerotic staphylomata occur; and these take place precisely in the situations where anatomical considerations would lead us to expect them; they are most apt to occur just behind the insertions of the tendons of the recti muscles, a part where the sclerotic is naturally thinnest. In one case I examined, (19th September, 1856,) there was so large a staphyloma beneath the internal rectus, that, at this point, the diameter of the globe from side to side exceeded that from back to front by nearly one third.
In another class of cases, after a long time, the hardness of the globe diminishes; the pain lessens, and then occurs only occasionally; the eye becomes quiet, and finally undergoes atrophy.

**Chronic Glaucoma.**—This differs from the acute chiefly by the insidiousness of its course, which is unmarked by those violent symptoms which characterise the outbreak of the acute form. It is equally intractable as the acute affection, and, like it, is sure to end in blindness. Whilst the transparent media are still clear, small hemorrhagic spots may be seen with the ophthalmoscope upon the retina, just as in acute glaucoma, and the retinal veins are equally turgid. Another argument for supposing chronic and acute glaucoma to be merely different forms of the same disease, is the occasional occurrence of both in the same individual. An instance of this is now under treatment at the Moorfields Hospital.

Gräfe describes a form of amaurosis in which the optic papilla has an excavated appearance, but the other symptoms differ from those of glaucoma. The two affections are not identical, and must be carefully distinguished.

**Morbid Anatomy of Acute Glaucoma.**—In a large number of dissections of eyes affected with acute glaucoma, I have found the following morbid appearances uniformly present in a more or less marked degree.

**Vitreous humour.**—The vitreous humour is transparent and colourless, or it has a yellow tinge. According to Gräfe, it is, at first, yellow, and, subsequently, becomes colourless. It often contains blood-discs entangled in a delicate web of coagulated fibrine. The blood-corpuscles may be observed in all stages of withering. I have sometimes seen small filmy blood-clots imbedded in the vitreous humour, and tied by a slender point to the retina at the spot where the ruptured membrana limitans has allowed the blood to pass from the retina into the corpus vitreum. The vitreous humour has a much greater consistence than it naturally possesses; it does not readily flow away when the eyeball is
cut across, and it offers remarkable resistance when cut. With a view to relieve the tension of the globe, I have seen the sclerotic freely punctured with an extraction knife, after which, firm counter-pressure with the finger upon the opposite side of the globe only caused the protrusion of a very small bead of yellowish vitreous humour: such great firmness had it. The yellowish colour of the vitreous humour is derived from the colouring matter of the effused blood which soaks through it and stains it.

Retinal Tissues.—The veins are dilated, tortuous, and filled with blood. The inner surface of the retina is sprinkled with small ecchymoses, which, in point of fact, are small spots of capillary haemorrhage. I believe this condition was first recognised by actual dissection, in glaucoma by Mr. Bowman, in a case which occurred in his practice in May, 1856. The haemorrhage comes from the capillaries in the inner layers of the retina, and the effused blood either spreads laterally among the elementary structures of the retina, or bursts through the hyaloid membrane into the vitreous humour. The retinal capillaries are irregu-

larly dilated, and studded with small fasiform and globular enlargements, miniature aneurismatic pouches. I have, now and
then, seen them on small vessels one remove from the capillaries; but never, in a single instance, have the larger vessels and great trunks been affected in this way. The dilatations sometimes occur at the branchings of the vessels, and sometimes at intermediate points of their course.

These pouches, and the vessels communicating with them, are usually crammed full of blood-corpuscles. In the hemorrhagic spots, the tissues immediately around the dilatations are infiltrated with blood-discs, which have escaped from some of the pouches which have burst, and they are stained with blood-pigment to a still greater distance.

Excepting these dilatations, the retinal capillaries have a healthy aspect, and they do not present any traces of fatty or atheromatous change. The coats of the small arteries are hypertrophied, and the nuclei in their walls are highly developed. The other retinal structures retain a natural appearance during the early stages of this disease.
ANATOMY OF GLAUCOMA.

Lens.—The lens during life had a greenish glaucomatous appearance, but it now has a yellow tint when viewed by transmitted light. The bedding of the lens in the hyaloid fossa of the vitreous humour, and the thinness of the posterior half of the capsule, are circumstances favorable to the existence of osmotic currents between these two structures, and it is probable that they both derive their colour from the same source, viz., the hæmatine of the blood.

Choroid.—Besides fulness of its vessels, I have not found any morbid changes in the choroid, excepting when staphylomata have occurred beneath the recti muscles. In an eye which I examined in September, 1856, there was a very large staphyloma beneath the internal rectus, and a smaller one under the outer rectus. In this instance, the choroid corresponding to the staphylomata was pale; the pigment was irregularly distributed; and the tissues were opened out, if I may so express myself. The choroid preserved its natural connection with the sclerotic and retina respectively; and these membranes were not separated by any collections of fluid. The vitreous humour was firmer than natural, and more abundant; it contained small blood-clots, and had a yellow colour. The retina was marked with numerous ecchymoses. In this instance, then, in which large staphylomata were present, the choroid and retina were not separated by any serous effusions. The examination of several subsequent cases has given similar results; and I believe that in glaucoma, in the acute stages at least, serous effusions between the choroid and retina never occur.

The subsequent changes in the choroid and retina in the advanced stages of the disease have an atrophic character. In some cases I minutely examined, in the summer of 1856, I found the dilated capillaries of the retina had become dark and granular; they were charged with minute oil-globules and often contained a few grains and little masses of red pigment. In short, these vessels were in a state of fatty degeneration, and the neighbouring parts of the retina supplied by them participated in the atrophy, and formed small spots of a dull, stone-gray colour.
The structural changes which take place during the early stages of the disease all point to a state of great vascular excitement in the retina. An increased flow of blood augments the internal pressure on its walls. In practice, the degree of tension of the globe is measured by its hardness. Every one who has injected an eyeball knows how very hard it becomes when fluid is forced into it with an injecting syringe, and the hardness varies directly with the pressure. The cupped state of the optic papilla is also produced by the internal pressure, and Gräfe says the degree of excavation gradually increases as the disease advances. Probably, in consequence of the altered state of the optic papilla, the exit of the blood through the vena centralis is embarrassed, and a strain is thrown backwards upon the capillaries and arteries. The delicate capillary wall cannot sustain this strain, and hence the dilatations, the ruptures, the hemorrhages. The arteries, from their efforts to overcome the impediment, become hypertrophied. With every beat of the heart the tension of the globe is momentarily augmented, and simultaneously the return circulation through the central vein is stopped: the column of blood is checked and a pulse appears in the artery.

The violent pain, and the paralysed state of the iris, depend on the great pressure upon the ciliary nerves. When this pressure is relieved artificially, these symptoms are diminished. I have already mentioned a case in which some of the vitreous humour was let out to diminish the internal pressure. In this instance the operation was directly followed by contraction of the pupil; the iris acting on the side corresponding to the puncture in the sclerotic. Gräfe, in his memoir above cited, attributes the blindness which occurs early in acute glaucoma to the great pressure upon the retina and optic nerve, rather than to changes in the retinal tissues. In this way he explains the excellent results he has obtained in the treatment of acute glaucoma by the operation which he has originated, and which he has submitted to extensive trial.
ON THE

ANALYSIS AND IMMEDIATE PRINCIPLES

OF

HUMAN EXCREMENTS

IN THE DISEASED STATE.

BY

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I propose in this communication to begin by showing that there exists an easy and very practical method of analysis applicable to pieces in the diseased condition, and which will, I trust, afford considerable assistance in the diagnosis of disorders of the digestive system. I shall afterwards proceed to give an account of the results derived from this mode of analysis, as applied to the fecal evacuations of a few of my patients at the Westminster Hospital.

For the purpose of examining human excrements in a physiological and pathological point of view, I found it necessary to abandon the usual methods of chemical analysis, and adopt a process by which fecal evacuations might be mechanically divided into their immediate principles, instead of being chemically decomposed. M. Chevrel.
the first who understood the importance of this kind of analysis, and adopted it in his valuable researches on fatty matters; since then, Messrs. Robin and Verdeil, convinced of the weight of the objections against the usual methods of chemical analysis in physiological and pathological investigations, have written their elaborate treatise on anatomical and pathological chemistry, in which M. Chevreuil’s views are advocated throughout.

It is evident that an attempt at the extraction and study of the immediate principles of human feces in a state of disease could not have been made, with any certainty of obtaining accurate results, without a method of analysis having been previously adopted, and shown to yield certain fixed results in regard to healthy excreta. I therefore began by establishing a series of operations which enabled me to analyse the evacuations of men and animals, and then proceeded to a close examination of the immediate principles of human excrements in health.

These investigations\textsuperscript{1} were followed by an inquiry into the chemical composition of the evacuations of a patient suffering from malignant disease of the pancreas;\textsuperscript{2} and as these researches presented some interest in a pathological as well as a chemical point of view, I shall allude to the medical history of this case in my present communication.

In the communications referred to I described the mode of analysis I had first adopted for my researches on the composition of human feces; since then, and after further investigations, I have simplified and added to these operations, and trust I can now venture to offer to the medical profession a method for the analysis of excrements in a state of disease calculated to yield results at least as important as those we have already derived from the analysis of urine. The successive stages of the analysis may be easily understood, by a glance at the accompanying synoptic table.

\textsuperscript{1} ‘Philosophical Transactions’ for 1854 and 1857.
\textsuperscript{2} ‘Quarterly Journal of the Chemical Society’ for July, 1857.
OF HUMAN EXCREMENTS.

After having observed the physical characters, such as colour, consistency, odour, and reaction of the evacuations, the pathologist must next proceed to their microscopical examination; and then, if fluid or semifluid, they are to be concentrated on a water-bath until reduced to the degree of solidity of healthy faeces. The mass is now exhausted with boiling commercial alcohol, sp. gr. 850; the mixture being boiled for one minute, repeatedly shaken, and then strained through a double fold of fine muslin; the same operation is to be repeated until the insoluble residue has completely lost its pasty nature. I have been lately in the habit of making use, in this operation, of a strainer, in which the alcoholic extract is forced through the cloth by means of atmospheric pressure. This mode of proceeding is very effectual and convenient in every respect. I shall not dwell on the constituents of excrements insoluble in boiling alcohol; they consist, in health more especially, of muscular fibre, woody fibrous tissue, and other bodies which may be distinguished in the faeces by an ocular or microscopical examination. The alcoholic solution, which has become turbid on cooling, is to be left undisturbed for twelve or eighteen hours; the clear supernatant fluid is then decanted, the remaining mixture thrown on a filter, and the filtrate added afterwards to the decanted portion. The deposit is next to be washed with cold alcohol, and the washings added to the clear liquor; finally, the clear alcoholic fluid must be mixed with milk of lime, prepared when required from caustic lime. I am in the habit of adding about half a fluid ounce of thick milk of lime to the alcoholic extract of one evacuation, and then diluting the mixture with a bulk of distilled water equal to that of the alcoholic extract.

The whole being briskly stirred up with a glass rod, a distinct precipitate will appear, subsiding to the bottom of the vessel, and the fluid may be noticed to have acquired a pale yellow colour; the mixture is to be allowed to stand undisturbed for twelve hours. The residue on the filter which has subsided in the original extract, and is insoluble in cold alcohol, will be found to consist, in the healthy state,
of an amorphous olive-coloured mass, showing no crystals under the microscope. The quantity of this deposit does not appear to vary much in the healthy individual, but in disease it may be greatly increased or diminished, and assume a different colour. This deposit, after having been removed from the filter into a flask, is to be treated with small successive quantities of boiling alcohol, the extract being filtered through paper or calico after each operation, and the process repeated until the filtrate passes colourless or nearly so. There remains undissolved a proportion of the mass, which, when dry, will be found completely devoid of the fatty nature it previously possessed.

The pathologist is now to put aside the alcoholic solution for five or six days in order to allow the substances it may contain to crystallize, and proceed to the examination of the substance insoluble in boiling alcohol left on the filter in the previous operation. It is a singular circumstance that this mass should have become insoluble, when the same substance has previously been dissolved in the alcoholic extract of feaces, and this is undoubtedly an example of one of those curious reactions which organic substances may exert on each other when present in the same solution. The substance in question had been dissolved in the extract of feaces, probably on account of the free acids existing in the liquid; these acids having now been removed, accounts for the deposit becoming insoluble in pure alcohol.

To determine the composition of the deposit, it is to be dried and weighed, and then boiled in a solution of potash, when it will be noticed to dissolve nearly entirely. Next enough hydrochloric acid is added to impart to the fluid a strong acid reaction, and a bulky precipitate of fatty acid will appear. The fluid having cooled, the whole is to be thrown on a filter, and this filter thoroughly washed with distilled water, until the filtrate has completely lost its acid reaction, and fails to produce a precipitate with a solution of nitrate of silver. Then dry the filter containing the fatty acid, under the air-pump over sulphuric acid; finally wash the filter thoroughly with ether, in order to remove from it every
trace of fatty acid; collect the ethereal solution in a weighed capsule, and by evaporating the fluid to dryness in a water-bath or distilling it in a weighed flask, the weight of the fatty acid may be obtained. Its nature can be afterwards ascertained by obtaining it crystallized in alcohol, and taking the fusing point of the crystals. The filtrate from the fatty acid and the washings may be concentrated and submitted to chemical analysis for the detection and estimation of phosphoric acid, lime, and magnesia. I have observed, however, that the fatty acid and phosphoric acid of the deposit in question, are combined with the earthy base; margarate of lime, phosphate of lime, and margarate of magnesia may therefore be considered as constituting three immediate principles of healthy feces.

The analyst must now return to the alcoholic extract of feces, which has been mixed with milk of lime, according to the directions given above, after decanting the supernatant liquid, the deposit is to be collected on a filter and washed two or three times with distilled water; the filter and the precipitate are then transferred to a capsule and dried on the water-bath. It is perhaps more convenient to perforate the filter and wash out the lime-precipitate into a capsule, to be subsequently dried. In the dry state the lime-deposit is easily scraped off the paper filter. It is next to be placed in a glass flask and covered to the depth of about half an inch with a mixture of equal parts of cold alcohol and ether, with which it is freely agitated; the flask is to be closed and allowed to stand for twelve hours, now and then shaking the mixture; finally it must remain undisturbed for a short time in order to enable the precipitate to subside completely; and then the solution is decanted into a paper filter, taking care to remove as little as possible of the deposit. The clear filtrate is to be received in a glass capsule, and left in as cold a spot as the temperature of the weather will permit. This operation must be repeated by adding another quantity of alcohol and ether to the lime-residue in the flask, and filtering off some hours later, after which the whole of the lime-precipitate is thrown on the filter. After a period varying
from twelve hours to five days, according to the temperature of the air, the solution deposits in healthy cases an abundant mass of crystals of excretine.

Let the mother liquor be now decanted, or the whole filtered into another glass capsule. Dissolve the crystals in a mixture of alcohol and ether, add some animal charcoal to the solution, and throw it on a filter containing charcoal. This operation having been repeated three or four times, and the charcoal washed with pure ether, a perfectly colourless solution is obtained, which, by spontaneous evaporation or by concentration under the air-pump, yields in the healthy state beautiful colourless crystals of excretine. In the diseased condition, however, as I shall have an opportunity of showing, this result is not always obtained.

In order to proceed with the analysis, lay aside, for the present, the lime-precipitate free from excretine, and resume the examination of the alcoholic extract obtained from the first deposit which occurred in the alcoholic solution of feces. If after having been exposed to the atmosphere in an open capsule for four or five days, impure crystals have formed in it, collect these crystals on a filter and submit them to chemical examination. They may consist of stearic or margaric acids, or a mixture of the two with or without oleic acid, or finally of an acid or neutral soda soap. In healthy feces this alcoholic solution, on standing, yields a small amount of an amorphous substance, but I have noticed in diseased excrements a large quantity of a crystallized deposit to occur, under the above circumstances.

The filtrate from the above crystallized or amorphous deposit is to be evaporated to dryness on the water-bath, and the residue to be treated with ether, which dissolves the remaining fatty acids, and leaves undissolved the soap that had not been previously deposited in the alcoholic solution. The soap and fatty acids may now be submitted to chemical examination. In order to simplify the operations, it will be advisable to add the ethereal solution of the residue to an ethereal extract prepared from the above mentioned crystalline deposit and to consider the insoluble residue as the
same substance. I here beg leave to observe that with regard to the fusing points and chemical composition of the fatty compounds obtained by analyses of this description, it is very difficult, if not impossible, to aim at perfectly accurate results; for the quantities to be operated upon are always very minute, and consequently do not admit of being submitted to a complete process of purification; thus, for example, it will not be possible to purify so little as 0.08 gramme (1.235 grains) or even 0.1 gramme (1.538 grains) of fatty acid or of soap; the product must therefore be analysed at once, allowing a small error for impurities.

The lime-precipitate free from excretine is now to be submitted to analysis; it may yield fatty acids and colouring matter. Let this precipitate be dried on the water-bath, pounded and digested in a flask for twelve hours with four or five times its bulk of commercial hydrochloric acid diluted with an equal volume of water. If the lime-precipitate should contain any fatty matters, they will rise to the surface of the solution, and on filtering the fluid will remain on the filter. In case some of the lime-precipitate has remained undecomposed at the bottom of the flask the residue is to be again digested for twelve hours with hydrochloric acid, and the supernatant fatty acid collected afterwards on the filter. The filter is next to be washed with distilled water until the washings no longer exhibit an acid reaction and fail to produce a haziness with a solution of nitrate of silver; the filter is dried under the air-pump over sulphuric acid, and treated with ether in order to obtain a solution of the fatty matter. The fluid, decolorized by means of animal charcoal and allowed to evaporate spontaneously, or concentrated under the air-pump, may deposit white crystals of fatty acids which are to be submitted to chemical examination.

There now remains but one more stage to complete the analysis of fecal evacuations; it consists in the detection of the immediate principles soluble in water. I have not as yet succeeded in deriving from this part of the investigation results so conclusive as those obtained in the preceding operations, because the acids which, in healthy faces, might be
expected to be found in this stage of the analysis do not appear to form crystallizable salts. Still I have no doubt that some means will soon be discovered for effecting their extraction. There might very possibly occur in human evacuations, in certain pathological conditions, an organic acid of the acetic acid series, forming crystallizable salts, which suggestion is supported by the circumstance that I have found the castings of certain carnivorous animals, such as those of the tiger, to contain butyric acid; consequently this part of the analysis of excrements in disease is not to be neglected. These acids must occur dissolved in the filtrate from the lime-precipitate under the form of a soluble lime-salt. Proceed, therefore, to concentrate the filtrate from the lime-precipitate, (I have seen butyrate of lime crystallizing on the surface of the solution during this process of concentration), and, when nearly dry, decompose it with dilute sulphuric acid; finally digest the whole mass for twelve hours in a closed flask, with a mixture of alcohol and ether; afterwards decant the solution, mix it with lime, and endeavour to obtain a crystallized lime-salt.¹

Having described the method to be employed for the examination of feces, I shall next proceed to give an account of the results obtained by this means in three different pathological cases.

**Case 1.**—John H——, âgé 65. Admitted as out-patient at the Westminster Hospital, on the 22d October, 1856. Has been in bad health since Christmas, 1855. Complains principally of dyspeptic symptoms; for the last two months has been weaker than previously, and feels pain in the epigastric, umbilical, and lumbar regions. Tongue remarkably thickened, red patches and slight ulcerations near the apex; the back of the tongue is covered with a thick pale brown fur, which affection began fifteen years ago. In the region of the thyroid gland, and on the left side, is a tumour as large as a small hen’s egg, and quite circumscribed, moveable under pressure, and giving no pain; urine scanty:

¹ Liebig’s method for the extraction of lactic acid from flesh.
Notwithstanding the treatment adopted, the severity of these symptoms gradually increased. On November the 1st, the lumbar pain which he refers to the kidneys is become most acute; his urine has assumed a brown opaque appearance, and is occasionally tinged with blood; much pain in the epigastric and umbilical regions.

On the 22d November the pain across the loins is increased; the patient is sick at night and vomits a bitter substance; bowels moved twice a day; motions very light coloured. No appetite, no sleep; he now passes a great quantity of urine at night, which, according to his statement, is as red as blood.

On January the 3d, Hague became an in-patient. Symptoms the same as previously; is troubled with vertigo, and complains of sickness after breakfast; no sleep at night; acute pain, especially in the left lumbar region. At this period I ceased attending the patient regularly, but saw him every now and then in the ward, and afterwards at home; no change occurred in the symptoms, except that he had become emaciated to such a degree that when he died, on the 30th of March, there was hardly any flesh left on his body. Towards the last stage of the disease he suffered from continual sickness, and vomited most of the food he took. His medical attendant treated him entirely with opiates.

I undertook the post-mortem examination on the 2d of April. Body considerably emaciated, legs oedematous; the abdominal cavity was found to contain three quarts of a yellow muddy serum; the small intestines were tinged with bile, and exhibited a few red patches; cellular tissue distended with gas, probably from decomposition; the smell was but slight; kidneys both hypertrophied, and about half as large again as in the normal state; the pyramids were remarkably distinct and pale; pelvis of kidneys filled with a gelatinous transparent deposit, very adherent to the cavity, especially in the right organ. Liver enlarged, hard, and of a blue colour externally; when cutting into its tissue a fluid escaped, containing hard, dark-coloured, sandy particles,
which were easily crushed with the fingers, yielding a stain of an intense yellow colour. The fluid holding the sandy particles in suspension appeared to flow out of a number of minute cysts; the gall-bladder was full of bile and enormously distended; no biliary calculi; pancreas considerably indurated, slightly crepitating on pressure. On making a section of this organ a sensation was perceived, such as would be caused by cutting into a gritty substance; the tissue was found entirely disorganized, and apparently converted into a hard tubercular mass.

Previous to the patient's death, and with the view of throwing some additional light on the nature of the disease, I had examined the composition of his evacuations. This was done on three occasions: twice when he was my patient, and once a few days before he died. He had never taken cod-liver oil at the time of his passing the motions which I analysed. The above evacuations and urine were, at my request, voided in separate vessels, in order to ensure the purity of the former. The patient's motions had the consistence of putty, of a yellow-gray colour, and an acid reaction. The deposit obtained in the alcoholic extract yielded to boiling alcohol a substance which crystallized on cooling; these crystals were found to be partly soluble in ether, the insoluble portion dissolved in hot alcohol exhibiting an acid reaction.

The analysis of 0.275 gramme (4.245 grains) of this substance showed that it consisted of bistearate, or acid stearate of soda, which must be considered as constituting a new immediate principle of the human body in the diseased state. Its presence in this case results in all probability from the action of abnormally large quantities of free acid in the intestinal canal.

Free fatty acids do not exist in healthy human feces unless a very large amount of vegetable food has been taken. The excrements in the present instance yielded not only bistearate of soda, but a considerable quantity of free fatty acids; these were obtained in the filtrate from the bistearate of soda, which, after standing undisturbed for some hours,
OF HUMAN EXCREMENT.

129

deposited colourless crystals, consisting of a mixture of bistearate of soda and fatty acids. This deposit yielded to ether the pure fatty acid, which was found to be a mixture of stearic and margaric acids, fusing at 60° C. (140° Fah.), and, according to Gottlieb, corresponding to 18 parts of stearic acid and 10 parts margaric acid. The clear mother-liquor afforded, twenty-four hours afterwards, another crop of beautifully white glistening crystals, which, submitted to microscopical and chemical examination, proved to be margaric acid apparently quite free from stearic acid. Scirrhous cancer of the pancreas is a disease seldom met with. Cl. Bernard reports a case of this kind in his work 'Leçons de Physiologie Expérimentale,' vol. ii, p. 292, extracted from the 'Medico-Chirurgical Transactions,' vol. xviii; in which the pancreas was affected with scirrhous cancer, the passage of bile into the intestines being at the same time arrested.

In the case of Hague, the state of excessive emaciation was an obvious sign of defective assimilation, and this symptom is interesting when coupled with the absence of bile and pancreatic juice in the intestines, and with the presence of large quantities of fatty matters in the faeces. Although it be not possible from a single case of this kind to draw any inference as to the physiological properties of the bile and pancreatic juice in health, still it is remarkable that the absence of the pancreatic juice, which secretion, according to Cl. Bernard, acidifies the fatty matters of food, should have been attended by the excretion of fatty acids; at all events, the excessive quantities of fats excreted are strongly calculated to support Cl. Bernard's views respecting the property of the pancreatic juice to cause the digestion of the fatty parts of food. From the history of the present case we might infer that the existence of a large amount of fatty acids in the evacuations, is to be considered as a symptom of deficiency of bile or of pancreatic juice, or of both, in the intestines; my researches, however, have not as yet been sufficiently extensive to prove the correctness of this view; still, in the two following cases of jaundice, which
are the only instances of evident deficiency of bile where I have had the opportunity of analysing the excrements, they were ascertained to contain a considerable proportion of free fatty acids.¹

It may be interesting to notice that this view is supported by the experiments of Tiedeman and Gmelin, who, after having tied the bile-duct in a living dog, and thus induced symptoms of jaundice, discovered afterwards in the dead animal that the first half of the intestinal canal, after the duodenum and the rectum, contained a considerable proportion of acid fats, which yielded a substance resembling margaric acid. Tiedeman and Gmelin's 'Recherches sur la Digestion,' part ii, p. 38.

CASE 2.—John H—, set. 42, admitted as out-patient at the Westminster Hospital on the 18th of March, 1857. Skin of a yellow colour, and has suffered from jaundice for the last two years. Motions occasionally green, and at other times white, like putty; water usually very high coloured and nearly black.

This patient was first treated with calomel and rhubarb, and then with a mixture of iron and quassia; finally he began taking, on the 11th of April, one grain of protioide of mercury twice a day. On the 17th of April the yellow colour of the skin, though still conspicuous, had diminished, and he expressed himself better; that day he brought me some of his excrements to examine, which, at my request, he had taken care to avoid mixing with his urine; his usual diet for this last week has been as follows: beefsteak, bread, potatoes, butter, and tea; he had a few watercresses yesterday; takes no beer or spirits.

The excrements had a grayish colour, of a putty consistency, and acid reaction; when exhausted with alcohol in the usual way they yielded an acid solution; a precipitate gradually subsided to the bottom of the vessel, which, being

¹ Since writing this paper, I have examined the motions passed in another case of jaundice, and found them to contain a large proportion of fatty acids.
collected on a filter and treated with boiling alcohol, yielded a fluid in which an abundant deposit occurred on cooling. The greater part of this deposit was found to be soluble in ether, and after twenty-four hours I noticed a crystalline substance in the ethereal solution; it was collected on a filter and dissolved in hot alcohol, when on cooling it again crystallized. The crystals were perfectly soluble in hot alcohol and in ether; their fusing point was found to be 67° C. (152·6° Fahr.) After dissolving this substance in hot alcohol, and filtering the solution through animal charcoal, the first crystals which appeared were found to fuse at 69° C. (147·2° Fahr.) There could be no doubt left as to this substance being stearic acid; it had the same crystalline appearance, and when fused resumed the crystalline form on cooling.

The mother-liquor from the first deposit of crystals of stearic acid was next boiled with hydrated oxide of lead, in order to separate the oleic acid, if any should be present. The lead-soap, dried and treated with ether, yielded a solution from which oleic acid was obtained by means of acetic acid. The property of this fatty acid to form a lead-soap soluble in ether, and its fatty consistency, were considered sufficient indications of the presence of oleic acid. Milk of lime being now added to the original alcoholic extract of faeces, a precipitate appeared, which was washed and dried and subsequently treated with a mixture of alcohol and ether. This process, which in the case of healthy faeces yields excretine, failed in the present instance in separating the smallest appreciable quantity of the immediate principle in question; it must, therefore, have been absent. I finally submitted to a quantitative analysis the portion of the residue which had subsided in the original alcoholic extract of faeces on cooling, and had been found insoluble in boiling alcohol. This substance, dried on the water-bath, weighed 0·410 gramme (6·329 grains); it was boiled with liquor potasse, when it dissolved almost entirely; and, finally, the addition of hydrochloric acid to the fluid gave rise to a precipitate of fatty acids. This precipitate was collected on a filter, thoroughly washed with water, dissolved in ether,
and this solution finally evaporated to dryness. The residue weighed 0.223 gramme (3.442 grains). The amount of lime in the acid filtrate was also determined and found to be 0.021 gramme (0.324 grains). Consequently, 100 parts of the deposit contained of fatty acid 90.6 parts; of lime, 9.4 parts. According to Chevreuil, margarate or stearate of lime consists, in 100 parts, of 90.038 fatty acid, and 9.967 of lime.

The substance was therefore stearate or margarate of lime, or more probably a mixture of the two. It occurred under that form in the faeces, and therefore constituted one of its immediate principles. The presence of lime-soaps in this instance must not be considered as abnormal, for I have always found these compounds in healthy faeces.

Case 3.—John R.—has been suffering from symptoms of jaundice for the last few days. He is now better; skin is of a light yellow. Took since yesterday morning three doses of Barberry bark, which I had not prescribed, and has been purged considerably. The motion he brought me was passed this morning (the 15th October); it has a fluid consistence and a yellow canary colour. His usual food is as follows: for breakfast, eggs and bacon; for dinner, none yesterday, but usually a beef-steak or pork-chop; at tea time, bread and butter and tea; and for supper, bacon, potatoes, and scarlet beans.

Examination of faeces.—I much regret that the evacuation submitted to analysis was passed under the influence of a purgative medicine, a circumstance which must greatly diminish its value in a pathological point of view. Nevertheless, it is remarkable that in this case the same excretion of fatty acids occurred as in the previous instances.

The faeces were observed to be quite free from the usual smell of excrements in health. I concentrated the fluid evacuation in the water bath to about one quarter of its previous bulk and then exhausted it with alcohol in the usual way. The alcoholic extract on cooling yielded an abundant
deposit, which was treated with boiling alcohol, and the solution again deposited a white substance on cooling. This substance treated with ether was partly dissolved. The portion of it insoluble in ether was dissolved in boiling alcohol, and the alcoholic solution evaporated to dryness on the water-bath. The residue weighed 0·169 gramme (2·609 grains). I dissolved it in hot water, when, by the addition of hydrochloric acid, a precipitate appeared. This precipitate being collected on a filter, thoroughly washed with distilled water and subsequently treated with ether, yielded a solution which contained 0·148 gramme (2·284 grains) of a dry substance, having all the properties of a fatty acid. Its fusing point was between 64° and 65° C. (between 147° and 149° Fahr.) Therefore, according to Gottlieb, it consisted of 22 parts of stearic acid and 10 parts of margaric acid.

The acid solution obtained by decomposing the residue with hydrochloric acid was found to consist of chloride of sodium; 100 parts of the compound consisted therefore of fatty acids, 87·57; soda, 12·43; but, according to Chevreuil, a neutral soda-soap of stearic or margaric acids contains in 100 parts, fatty acids, 89·02; soda, 10·98. Consequently, the compound in question was a soda-soap, consisting in 100 parts of 31 parts of margarate of soda and 69 parts of stearate of soda.

The nature of the substance soluble in ether was next examined; it had an acid reaction, and being allowed to evaporate spontaneously for twenty-four hours, yielded a mass of white crystals of fatty acid, containing a considerable proportion of oleic acid, and fusing at 38° C. (100·4° Fahr.) It was a remarkable fact that the first crystals deposited did not fuse even at 100° C. (212° Fahr.) They appeared to consist of a small quantity of soap which had been dissolved in the ethereal solution. The dried mass of fatty acids weighed 0·437 gramme (6·746 grains).

I now proceeded to evaporate to dryness the mother-liquor which had deposited the fatty acids and soap. The yellow residue obtained from this operation was treated with ether,
which dissolved a quantity of impure oleic acid, fusing at 36° C. (100·4° Fahr.) This substance, previously de-
colorized by filtering its solution in ether through animal
charcoal, was ascertained to weigh, when perfectly dry,
0·393 gramme (6·067 grains). I now purified the colourless
but impure oleic acid by transforming it into a lead-plaster,
which was treated with ether, and this solution was subse-
sequently decomposed with tartaric acid; the oleic acid thus
obtained fused at 23° C. (73·4° Fahr.) It was ascertained
by means of the microscope to contain crystals of fatty
acids, which accounted for its high fusing point. Pure oleic
acid fuses at 15° C. (59° Fahr.) The small proportion of
the yellow residue insoluble in ether was found to be soluble
in hot water. The addition of hydrochloric acid to this
solution inducing a white precipitate, the residue consisted
evidently of a soap.

The clear alcoholic extract of feces filtered from the
deposit which had occurred on cooling was mixed with milk
of lime, and the precipitate treated in the usual way for the
extraction of excretine; but not a particle of this substance
could be obtained. It evidently did not exist in these
evacuations.

Finally, a fresh quantity of fatty acid was extracted from
the lime precipitate by decomposing it with hydrochloric
acid. After thirty-six hours a green substance was noticed
floating on the surface of the solution; it was collected on a
filter, and dissolved in boiling alcohol. Another week
having elapsed, the solution was found to have lost its
alcohol by spontaneous evaporation, and yielded a deposit of
crystallized fatty acids. This deposit, after having been
thoroughly washed with water, was dried, and ascertained
to weigh as much as 1·440 grammes (22·230 grains); its
fusing point was 37° to 38° C. (98° to 100° Fahr.), showing
that it contained a large proportion of oleic acid.

It was not possible for me to determine with any degree
of precision the form under which these 1·440 grammes of
fatty acids existed in the feces; they may have been com-
bined with soda or potash, which soap the lime-water might
have decomposed. I am, however, inclined to consider their presence in these excrements as occurring under the form of free fatty acids.

This case, independently of its pathological interest, will contribute, I trust, to illustrate the mode by which feaces are to be analysed in disease.

In conclusion, I beg to acknowledge the valuable aid of my assistant, Mr. Frederick Dupré, P.H.D., in these and other investigations.
**Synoptic Table, showing the method to be adopted for the Analysis of Human Excrements.**

<table>
<thead>
<tr>
<th>Excrements exhausted with alcohol, and strained through a cloth.</th>
<th>An insoluble residue left on the cloth.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Excrements exhausted with alcohol, and strained through a cloth.</td>
<td>A deposit which, exhausted with alcohol, yields An insoluble residue.</td>
</tr>
<tr>
<td>A solution yielding on cooling</td>
<td>A crystalline deposit on standing (<em>fatty acids and soaps</em>).</td>
</tr>
<tr>
<td>A solution which is mixed with milk of lime. The lime precipitate, treated with ether, yields</td>
<td>A residue, when evaporated to dryness, which, when treated with ether, yields An ethereal solution (<em>fatty acids</em>).</td>
</tr>
<tr>
<td>A solution.</td>
<td>An ethereal solution (<em>excretina, colouring matter</em>).</td>
</tr>
<tr>
<td>An insoluble residue, which, decomposed with hydrochloric acid, yields</td>
<td>A solution of chlorides of calcium.</td>
</tr>
<tr>
<td>A solution. It is to be evaporated to dryness, decomposed with sulphuric acid, exhausted with a mixture of alcohol and ether, and this solution tested for organic acids.</td>
<td>A solid substance, soluble in ether (<em>fatty acids</em>).</td>
</tr>
</tbody>
</table>
ON THE
MEMBRANA DECIDUA WHICH SURROUNDS
THE OVUM
IN CASES OF
TUBAL GESTATION.

BY
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In the seventy-ninth volume of the 'Philosophical Transactions,' Dr. Baillie states, that Dr. William Hunter had a preparation of tubal pregnancy, in which the uterus was found enlarged to double its natural size, and contained a decidua. From this appearance Dr. Hunter inferred that the decidua, or outer surface of the secundines, belongs to the uterus, and not to the ovary, or that part of the conception which is brought from the ovarium.

A case of tubal gestation was published by Dr. John Clarke, in which the embryo, amnion, and chorion, were contained in the Fallopian tube, and in the history of which it is stated, that "the uterus, little, if at all enlarged, was lined with a decidua." ¹

¹ 'Transactions of a Society for the Improvement of Medical and Chirurgical Knowledge,' vol. i, 1793.
"Though the foetus be extra-uterine," observes Dr. Denman, "the uterus becomes considerably enlarged, and performs its proper office, by providing the efflorescent or deciduous membrane for the reception of the ovum."

"Although it be extremely probable, that the decidua begins to be formed at the time that the ovum passes into the cavity of the uterus," observes Dr. Baillie, "yet it is not absolutely necessary for the formation of the decidua that the ovum should reach the cavity. Where an ovum grows in the ovarium, or Fallopian tube, the decidua is both formed in the uterus, and the uterus is considerably enlarged, so as to undergo, to a certain degree, changes exactly similar to those which take place in a natural pregnancy."

"It is curious to observe," says Professor Burns, "that invariably the uterus enlarges considerably, and, in every instance, decidua is formed."

The seventh volume of the 'Medico-Chirurgical Transactions,' published in 1816, contains a description of an extra-uterine foetus contained in the Fallopian tube, by George Langstaff, Esq. "The uterus was considerably larger than we generally observe that organ to be in the unimpregnated state, even in women who have borne several children. On laying it open, the uterine vessels were observed to be very large, but empty; and there was a great quantity of gelatinous matter in the cavity and neck of the uterus. When this was washed off, the internal surface of the viscus looked very vascular, having been highly injected, but there was not the least appearance of a decidua.

"The spermatic artery on the same side, and those arteries ramifying between the laminae of the peritoneum which form the ligamentum latum, and supplying the Fallopian tube, had been previously injected.

"The lacerations were in the posterior surface of the tubal enlargement, and in the longitudinal direction. The Fallopian contents were next minutely examined; and after carefully washing away the coagulated blood from beneath the peritoneal covering, I discovered," says Mr. Langstaff,
"a chorion and amnion, with a foetus of about eight weeks floating in the liquor amnii."

This preparation is now in the museum of the Royal College of Surgeons of England, and in the printed catalogue it is stated that "no decidua was found in the uterus," and no trace of this membrane can be perceived, coating its inner vascular surface.

In the thirteenth volume of the 'Medico-Chirurgical Transactions' published in 1824, there is a case of Fallopian-tube pregnancy, recorded by Dr. Elliotson. "The uterus was four inches and a quarter in length, and three inches at the fundus in breadth. The cervix and os uteri were filled with a colourless and translucent jelly-like matter, which also projected a little way from the latter into the vagina, in the form of a very large drop. In the cavity of the uterus was a beautiful decidua." This preparation is in the museum of the Royal College of Physicians.

In the paper on "Double Uterus," and on "The structure and formation of the Membranes of the Human Ovum," published in 1832, in the seventeenth volume of the 'Medico-Chirurgical Transactions,' I observed that "the difficulty of determining the precise period of impregnation must render all observations on the human ovum, before the middle or near the end of the second month, more or less vague and uncertain. After this time the organization of the ovum is so far advanced, that the membranous layers which envelope the embryo, and the form of the embryo itself, can be clearly perceived with the naked eye. The amnion is then a transparent sac, which contains the embryo, and the fluid in which it floats. The chorion, covered with villosities on the external surface, surrounds the amnion, but is separated from it a short distance by the interposition of a gelatinous fluid, which is deposited in a very delicate reticular texture. There is a third membranous layer, viz., the decidua, which completely surrounds the chorion, and connects the ovum with the inner surface of the uterus. This, as is well known, appertains not properly to the ovum itself, but is a production of the lining mem-
brane of the uterus; for, in cases of extra-uterine conception, the chorion and amnion alone envelope the embryo, and a deciduous membrane has been found, lining the cavity of the uterus.

"In the accompanying preparation, however, of Fallopian-tube conception, which I assisted in removing from the body of a lady who died about the eight or ninth week of pregnancy from rupture of the tube and internal hemorrhage, no organized deciduous membrane lined the inner surface of the uterus, but the whole of it was coated with a thin layer of albumen."

This case occurred in 1829, and the preparation was placed upon the table of this Society in 1832, but, until 1836, when another example of tubal gestation came under my observation, it does not appear that I made any attempt to determine whether, in these two preparations, the ova in the Fallopian tubes were surrounded by decidua.

In a paper entitled "On the Situation of the Deciduous Membrane in cases of Extra-Uterine Conception," published in the twelfth volume of the 'Medical Gazette' for 1839 and 1840, I gave the following description of the appearances observed in these two preparations, which are now placed upon the table of the Society, with an accurate drawing of the second by West.

Case 1.—A lady died suddenly in 1829 from internal hemorrhage produced by rupture of the right Fallopian tube, which contained an ovum. On opening the tube and examining the different parts of the ovum, I found a deciduous membrane everywhere surrounding the chorion and closely adhering to the inner surface of the tube, as the decidua usually does to the lining membrane of the uterus in ordinary gestation. Within the decidua the chorion, placenta, amnion, and embryo were distinctly seen. The uterus was larger than natural, and there was no appearance of decidua lining its internal membrane. The decidua and other parts of the ovum in the right Fallopian tube are all distinctly seen in the preparation of the uterus and its
appendages, which is now in the museum of St. George's Hospital.

Case 2.—On the 18th July, 1838, Mrs. K—, after suffering for some time with symptoms of inflammation and retroversion of the uterus, was seized with great faintness and soon expired. A large quantity of fluid blood was found in the abdominal cavity, and the right Fallopian tube which contained an ovum of ten or twelve weeks, was extensivelyacerated near the fimbriated extremity. On removing the uterus and its appendages from the body and carefully examining the ovum contained in the right Fallopian tube, it was evident that a deciduous membrane everywhere surrounded the chorion and adhered to the inner surface of the tube. The placenta, which was situated at the extremity of the ovum nearest the uterus, was seen covered with the decidua, and coagula of the fibrine of the blood were traced from the interstices of the placenta through the decidua into veins in the thickened muscular coat of the tube. At the part where the placenta was situated, the muscular coat of the tube was a quarter of an inch in thickness and could readily be separated into layers like the muscular coat of the gravid uterus. In this coat of the tube the veins were also readily traced from the inner surface outward, opening obliquely into one another and enlarging as they reached a great vein near the uterus.

The interstices of the villosities of the chorion, filled partially with clots of blood, were seen around the whole ovum, and presented nothing different in their appearance from those of ova which have been developed within the uterus.

Between the chorion and amnion, near the placenta, was the vesicula umbilicalis, with its slender peduncle proceeding to the umbilical cord.

The appearance of the amnion, cord and embryo was perfectly natural.

The uterus was considerably enlarged and its inner sur-
face was coated with a very thick layer of a yellowish-white soft substance. There was no trace of any arterial or venous canal in this coating; the orifice and neck of the uterus were closed with the usual viscid substance formed by the Nabothean glands. There was a corpus luteum in each ovarium. Both layers of the Graafian vesicle were enclosed within the yellow matter, and this was in immediate contact with the stroma of the ovary.

In the preparation of the parts, the decidua placenta, chorion, vesicula umbilicalis, amnion, umbilical cord, and embryo are all distinctly seen, and likewise the layer of the muscular coat of the Fallopian tube, with the veins proceeding from its internal to its external surface. The vesicula umbilicalis has become greatly diminished in size since the parts were immersed in spirit. With the exception of the coagula of blood in the interstices of the placenta and villi of the chorion, the constituent parts of the ovum are the same as in all cases of intra-uterine gestation and are in a healthy condition. The preparation of the parts is likewise in the museum of St. George's Hospital.

In the history of a case of Fallopian-tube gestation which occurred to M. Chaussier in 1814, it is stated that the walls of the tube were thin and vascular, that the placenta attached to the inner surface was broad and thin, and that when detached the membrana decidua surrounded the ovum. I have met with no case except this in which the deciduous membrane is distinctly described as surrounding the ovum in the Fallopian tube. Yet I am certain that this must be the fact in all cases of extra-uterine gestation, the circulation of the maternal blood being carried on chiefly by the blood-vessels of the deciduous membrane.

All the minute dissections since made confirm the correctness of this view of the structure of the ovum in cases of tubal gestation.

Case 3.—A woman, aged 35, who had been married thirteen years and had never been pregnant, ceased to
menstruate in July, 1840, and soon after began to have all the symptoms of pregnancy. In November the abdomen, which had previously enlarged, diminished somewhat in size, and, at the end of March, 1841, she had violent pains like those of labour, with a thin red-coloured discharge from the vagina. On the 24th April, 1841, the areolaæ were broad and dark, and milk could readily be pressed out of both nipples; but the glands were not enlarged. A hard irregular tumour occupied the hypogastrium; but on the left side it was soft, and an obscure fluctuation was felt in this portion of it. The umbilicus was very little protruded, and there were no white lines on the sides of the abdomen. No movements of a fetus were felt, nor any sound heard. The cavity of the pelvis was occupied by a hard immovable mass, and the os uteri was forced up by this behind the symphysis pubis so high that it could scarcely be touched. She died in December, 1841; and, on opening the abdomen, a large mass like the gravid uterus in the seventh month was found occupying the whole of the hypogastrium. The intestines and omentum firmly adhered to the upper part of the tumour, which felt hard in some parts and fluctuated in others. On opening this cyst a great quantity of thin fluid like pus escaped, and a perfect fetus of six months, with its umbilical cord and placenta. The cyst adhered to the whole of the posterior and upper surface of the uterus, which was larger than natural, and the cervix was considerably lengthened. The walls of the uterus were healthy, and the cavity empty. There was no decidua or substance of any kind coating its inner surface; the placental decidua covered in the usual manner the whole of the uterine surface of the placenta.

Case 4.—In 1841 I saw another case of tubal gestation, which had proved fatal at an early period of pregnancy. The preparation of the parts is now placed upon the table of the Society. The uterus is seen enlarged, and the inner surface coated with a substance of considerable thickness, of a yellowish-white colour, in which no arteries nor veins
could be traced. The ovum has been almost or completely removed from the tube which it had occupied; but the decidua reflexa is seen very distinctly covering a considerable portion of the villi of the chorion. That the decidua surrounded the entire ovum in this case it is impossible to doubt.

Case 5.—On the 26th May, 1850, Dr. Blakeley Brown presented to me a Fallopian tube which had contained an ovum of two months. The following is the history of the case furnished to me by Dr. Blakeley Brown:

"May 24th.—Mrs. B—, age 26. Saw her with Mr. C—, who has been attending her, and considered her to be labouring under morbus uteri. She has taken Hydr. Chlorid. gr. i, Op. i. She was lying on her back in bed with her eyes shut, and suffering from "spasms." Complains of intense pain over the abdomen, which is not constant. Pulse 50; soft. Tongue clean. Skin cool. Bowels rather confined. Cataractia absent between two and three months. Oe and cervix uteri soft, puffy, large, and healthy. Has had morning sickness; breasts have got rather larger and painful. I prescribed Haust. Cascarillae c. Sod. Bicarb. and Tinct. Card. co., thinking it a case of sickness from pregnancy.

"May 25th.—9 a.m. Has passed a restless night. Is sick and faint; vomited the mixture, and has taken two pills on her own account. I gave her some brandy and Seltzer water immediately, and prescribed a draught with Æther. Sulph. co. —1 p.m. Much the same; is very faint.—4 p.m. Saw her with Dr. Nairne, who prescribed Hs. Sp. Ammon. Acet. c. Liq. Op. Sed.—8 p.m. I found she had been dead about an hour, having kept the draught and some port wine without vomiting.

"May 26th.—Post-mortem examination made by Mr. Tutume; Dr. Nairne and myself present.—Body much blanched, otherwise healthy. On opening the abdomen a large quantity of blood came out, and there was in the pelvic cavity upwards of three quarts. The hemorrhage came
from the right Fallopian tube, which had burst from the ovum being lodged there. There was a fair corpus luteum. The uterus was healthy, with decidua, and had all the appearance of a healthy gravid uterus of two months, as certified by Dr. Lee.”

I have not succeeded in finding any account in my journal of the state of the uterus in this case; and, if my recollection does not fail me, the Fallopian tube had been separated from the uterus before it was kindly presented to me by Dr. Blakeley Brown. The preparation is now placed upon the table of the Society. The embryo and amnion have escaped; but the chorion and decidua remain in the tube, adhering to its surface.

Case 6.—Mr. Jackson, of Wimpole Street, presented to me, in 1856, the uterus and appendages of a woman who had died suddenly from internal haemorrhage at an early period of pregnancy. The preparation of the parts is now placed upon the table of the Society. It will be seen that the uterus is enlarged, and the whole lining membrane coated with a thick irregular layer of a substance, resembling the fibrine of the blood, of a red colour, in the upper part. This substance has been partially detached from the lining membrane of the uterus, which presents a natural appearance. When the preparation came into my possession the right Fallopian tube about the middle was as large as a walnut, or larger where its coats had burst and a coagulum of blood was hanging through the irregular aperture. The tube was pervious from the corpus fimbriatum to the dilated part. On cutting open this expanded portion, a small embryo enclosed in the amnion was observed, and the vesicula umbilicata, remarkably large, with its peduncle, came into view. All the cells of the placenta and villi of the chorion were seen distended with coagulated blood and surrounded with a deciduous membrane, a great part of which has been separated from the inner surface of the tube. By a careful dissection subsequently made, the decidua was found to consist of placental decidua, decidua vera, and
reflexa, with a decidual cavity. The ovum in this case of
tubal gestation was, therefore, perfect in all its structures,
and similar in every respect to ova which had reached
the cavity of the uterus and been developed there until the
end of the second month.

On the 30th October, 1857, I was informed by a gentle-
man who professes to be well acquainted with the Dutch
language, of which I am wholly ignorant, that Professor
Schröder Van der Kolk had published an account of a case
of tubal gestation in which a decidua surrounded the ovum,
and which was described as fulfilling the function of a
placenta. Professor Schröder Van der Kolk, I am informed,
has represented in fig. 47 a portion of a very soft spongy
mucous membrane of the uterus with blood-vessels, in a
Fallopian-tube gestation.

"The figure is magnified fifty times. It represents a
small portion of the mucous membrane itself of the uterus,
as seen under the microscope. The hollows in the part
of the figure are openings of utricular glands. In the lower
part the same are laid open by the section at the part where
the fragment was cut from the uterus. The uterus had
been injected. The pink are the arteries and the blue the
veins ramifying through the part."\(^1\)

There are five preparations of Fallopian-tube conception
in the museum of St. Bartholomew's Hospital. The
following is a description of them from the museum
catalogue:

"Series XXXIII, Nos. 13—18.

"No. 13. An uterus with the ovaries and the Fallopian
tubes. The middle of the left Fallopian tube was dilated
by an ovum. The dilatation is laid open and the chorion
and other parts of the ovum are shown. The uterus is
slightly enlarged, and its cavity is lined by a substance like

\(^{1}\) "Waarnemingen over het Maaksel van de Menschelijke Placenta,"
&c. 'Verhand. der Eerste Klasse van het Koninklijk-Nederlandsche
Instituut,' Vierde Deel, 4to, pp. 156-164, tab. vi, figs. 47 and 49.
decidua. There is a simple cyst in the right ovary. Rupture of the dilated portion of the Fallopian tube took place in the seventh week of gestation, and the patient died of hæmorrhage.

"No. 14. A similar specimen, in which, as in the preceding case, death was the result of hæmorrhage from the ruptured Fallopian tube in the seventh week of gestation. The middle of the right Fallopian tube is dilated into a sac, which contains the fœtus and its membranes. In one side of this sac is a small lacerated opening, through which the flocculent chorion protrudes. From this opening a gallon of blood was discharged into the cavity of the abdomen. On its other side a large portion of the sac has been removed to display the fœtus and membranes. The outermost membrane enclosing the fœtus has all the characters of decidua. Besides this membrane, the amnion and chorion are distinct. The fœtus and umbilical cord are also perfect. The right ovary contains a large corpus luteum, distinguishable by its circular form and yellowish colour. A bristle is passed through the aperture of the ovary, through which the ovum escaped. There is also a large cyst in the ovary, which contained a watery fluid. The cavity of the uterus is lined throughout by a perfect and thick decidua. Bristles are passed through it into the uterus. A bristle is also passed through the Fallopian tube into the dilated portion of it, which contains the fœtus and its membranes.

"No. 15. A portion of the broad ligament of a uterus, with the Fallopian tube and ovary. In the middle of its course the Fallopian tube is distended by the development of an embryo within it. On the surface of this part there is a small irregular aperture, through which fatal hæmorrhage into the abdomen took place. The ovary is large; at its lower part is a very large corpus luteum, with a central cavity.

"The patient, in the seventh week of the tenth pregnancy, was suddenly seized with pain in the situation of the
Fallopian tube and signs of internal haemorrhage, and died in ten hours.

"No. 16. A uterus with the Fallopian tubes and ovaries. A foetus has been developed in the right Fallopian tube close to the uterus, till it has attained a length of between three and four inches. The placenta and the several membranes of the ovum appear to be well formed. The uterus is covered by coagulated blood, effused probably from the ruptured Fallopian tube.

"No. 17. A similar specimen, in which the foetus contained in the left Fallopian tube is yet further developed, and measures between five and six inches in length. Both it and its membranes are well formed.

"No. 18. A similar specimen, in which death occurred in the third month of gestation. The middle of the left Fallopian tube, which contained the foetus, is dilated into a large sac. On one side of this sac is a lacerated opening, through which the foetus escaped into the cavity of the abdomen, and to the edges of which the membranes of the foetus remain attached. The left ovary contains a corpus luteum; the cavity of the uterus is lined by decidua."

In the description of Preparation No. 14, it is stated that "the outermost membrane enclosing the foetus has all the characters of decidua. Besides this membrane the amnion and chorion are distinct. The foetus and umbilical cord are also perfect." I have carefully examined this preparation, and there can be no doubt that a decidua surrounds the ovum in the tube. Though none of the other preparations have been dissected with the view of discovering a decidua surrounding the ovum in the tube, yet in all, the separation of the ovum from the inner surface of the tube has been carried to an extent sufficient to enable us to demonstrate the fact.

The following is a description of the preparations of extra-uterine gestation, from the museum catalogue of
Guy's Hospital. For this and for permission to examine
the preparations, I am indebted to Dr. Oldham, Dr. Wilks,
and John Bowes, Esq.

2516. Foetus of about three months old, with part of the
membranes, cord, and placenta developed in the extra-uterine
sac, seen in the next specimen.

2517. The uterus and parts concerned in forming the
cavity from which the preceding specimen was taken. The
remains of the sac in which the foetus was developed are to
be seen a little to the right of the fundus of the uterus,
which is very small and contains no decidua.

251780. Extra-uterine foetation. The ovum has been
arrested in the left Fallopian tube, which is expanded into
a tumour the size of a billiard ball. The surface is smooth,
with the exception of two small rents, neither larger than
sixpence; the edges of the opening are very thin. On the
opposite side the sac has been opened, and the walls are seen
to be thin. The outer membrane of the ovum was covered
with blood, and the villi were with some trouble separated
from it. Nothing like a deciduous membrane was found
between the chorion and the lining membrane of the tube.
A delicate amnion contains the foetus; the latter is nearly
an inch long. Umbilical vesicle not discovered. In the
opposite ovary the corpus luteum is seen. The uterus is
twice the size of a virgin one. The walls are thickened and
lined by a slightly raised efflorescent membrane, which had
several superficial alveolar depressions on the interior surface.
On examination with a lens, no true deciduous membrane
could be detected; it appeared to be more like the half
organized membrane of dysmenorrhœa.

251786. Extra-uterine pregnancy of about the third month.
The foetus was developed in the right Fallopian tube. The
ovum is contained in its membrane, surrounded by chorion,
and external to this is coagulable lymph or a coagulum of
blood. The uterus is enlarged and occupied by a slight
flocculent exudation.
2517\textsuperscript{80}. Extra-uterine fœtation. The right Fallopian tube is expanded at its distal extremity into a sac the size of an orange. The seat of rupture is at the lower part, from which originally protruded a large coagulum. On opening it the ovum was found within, and containing a fœtus of between three and four months' development, enveloped by membranes, and around these the tufts of chorion. Between the walls of the tube and the placenta was a coherent membrane, which seemed to act as a medium of connexion between the two; its structure could not be made out. The right ovary was found to contain a corpus luteum. The uterus was as large as an orange, or at a four months' pregnancy. A plug of mucus filled the os. The mucous membrane was raised into a very rich deciduous membrane and thrown into folds, and was tubular, the openings of which presented a cribiform appearance. The openings of the Fallopian tube were closed.

2517\textsuperscript{80}. Uterus with left Fallopian tube natural. The right communicates with a vicarious uterine cavity, containing a full-grown fœtus. The sac appears to have been originally formed in the Fallopian tube of the right side. The walls are thick and lined with a flocculent membrane.

2517\textsuperscript{80}. Extra-uterine pregnancy, about the fourth month. This preparation has been injected. The sac is found in the right Fallopian tube, the amnion is seen within, and the umbilical cord attached to the placenta, and immediately within the tube itself is a thick material, like coagulated blood. The uterus is enlarged, and its mucous surface is covered with shreds of membrane which are cribiform and porous like the decidua.

2517\textsuperscript{81}. Extra-uterine fœtation, between two and three months, in left Fallopian tube. The sac is partially open, showing the chorion; but the ovum is still in the sac of the amnion. The uterus is enlarged, and is filled with a thick exudation resembling decidua, and this is slightly cribiform. The corpus luteum is in the right ovary.
2517. Extra-uterine foetation of about the sixth week, developed in right Fallopinian tube. The corpus luteum is in the left ovary. The uterus is enlarged and contains some coherent shreds of exudation.

2517. Interstitial extra-uterine foetation. The ovum was imbedded in the left horn of the uterus, as seen in the upper part of the divided organ. The cavity is about the size of a horse-chestnut, and is quite closed. The uterus is much increased in size, and its cavity is filled with an exuberant growth of deciduous membrane closing the Fallopinian tube. This membrane is perforated throughout by small openings, varies in thickness, and is not easily separable from the uterus, and does not present those cup-like sacs which mark the decidua vera thrown off with an aborted ovum. Its prevailing character is tubular. The right ovary contains the corpus luteum, which is large. The ovum was lost in the abdomen.

2517. Extra-uterine foetation, seventh or eighth week of pregnancy. Development has taken place in the left Fallopinian tube. The foetus is contained in a delicate amnion, surrounded by its chorion. The uterus is enlarged, and its surface is covered with a cribiform deciduous membrane.

2517. Extra-uterine foetation, at about the sixth month. A large sac exists at the extremity of the left Fallopinian tube, being formed in part by the tube and in part by the ovary. The greater part of the tube can be traced entire, leading to the belief that the sac is formed partly in the ovary. It contains placenta, membranes, and foetus. The uterus is much enlarged, and covered with soft masses of lymph-like exudation, resembling deciduous membrane. The corpus luteum is in the left ovary.

2517. Extra-uterine foetation, about the sixth or seventh week, developed in the extremity of the left Fallopinian tube, whose end is adherent to the ovary. The ovum, which had escaped, is enclosed in the chorion, and the latter has on its
outer surface a flocculent exudation. The uterus is enlarged
and a cribiform deciduous membrane is seen on its interior.
The corpus luteum is in the ovary on the same side.

It is stated in the description of this preparation that
"the ovum which had escaped is enclosed in the chorion,
and the latter has on its outer surface a flocculent exudation."
I was permitted by Dr. Wilks, conservator of the museum,
to make a minute examination of this ovum, which had
escaped entire through the rent in the Fallopian tube, and
I had the great satisfaction, not only to discover the vesicula
umbilicalis, but to see the chorion completely surrounded
by a decidua, as in the preparation of tubal gestation
described in this communication. If the other preparations
in the museum of Guy's Hospital were subjected to a
minute anatomical investigation, there can be no doubt that
in all a decidua would be found surrounding the ovum in
the Fallopian tube.

Respecting the nature of the membrane or substance
found coating the inner surface of the uterus in the greater
number of these preparations, and which has been almost
universally considered to be decidua since the time of Dr.
William Hunter, although no blood-vessels in it have been
discovered, I shall at present refrain from expressing any
positive opinion, the great object of this communication
being to demonstrate the existence of a decidua around the
ovum in cases of tubal gestation.
SUPPLEMENT

TO A PAPER ON THE

MEMBRANA DECIDUA WHICH SURROUNDS THE OVUM

IN CASES OF

TUBAL GESTATION.

BY

ROBERT LEE, M.D., F.R.S.

Received Feb. 30th.—Read June 23rd, 1858.

The following case of extra-uterine fætation, by W. F. Favell, Esq., Sheffield, was published in the 'British Medical Journal,' Saturday, February 30th, 1858. "On Friday evening, January 15th, 1858, I was summoned to attend Mrs. —, aged 37, the mother of three children, who was stated to be suffering from sickness, with considerable pain in the belly. I found on my arrival that she had been in her usual health all day up to 4 o'clock p.m., when she began to complain of pain, which was speedily followed by vomiting. She had been engaged during the day in assisting in the removal of the furniture to another house, and, according to her husband's account, being a somewhat delicate woman, it was thought that the exertion consequent on their removal had induced this attack. She was not then in a condition to excite any serious apprehension; her pulse was good in volume, somewhat accelerated; the
abdomen was slightly tender on pressure; and the pain she described as being intermittent and very severe. She had also occasional vomiting. On inquiry I found she had not menstruated for nine weeks. The treatment consisted in the exhibition of opium, with small doses of calomel, a mixture containing hydrocyanic acid, with hot fomentations to the body.

"When seen by my father about 11 o'clock on the following morning, she said she was decidedly better; the pain was considerably relieved, but sickness had continued at intervals during the night. The pulse continued tolerably good; and she expressed an opinion that if she had something to allay the sickness, she could be removed in a cab. Shortly before 2 o'clock, her husband came down in great haste to say that she had been taken much worse about an hour previously, and her friends thought she was dying. On my arrival I found that it was so. The sickness had ceased and the pain become greatly aggravated about 12 o'clock; she was alarmingly faint; her extremities were cold; her pulse scarcely perceptible, and though brandy and other stimulants were freely administered, she died in about an hour after my arrival. The impression on our minds was that she had died from perforation of the stomach or bowels.

"A post-mortem examination was made the following day. On opening the abdomen several pints of extravasated blood were found in the cavity of the peritoneum, an immense clot filling the pelvis and extending into the abdomen on the right side. The stomach and bowels were distended with flatus, and healthy. After removing the clots, the uterus and appendages were examined, when the cause of the haemorrhage became at once apparent, viz., the existence of extra-uterine foetation in the right Fallopian tube. The cyst had been developed in the tube close to the fimbriated extremity which was dilated sufficiently to allow the passage of the end of the little finger, and plugged with coagulum. A rent existed in the cyst just within the tube, and the membranes (entire, with a small foetus floating in them) still
remained in the cyst, surrounded with coagulated blood. The uterus was considerably larger than in the usual unim-pregnated condition. The other organs were healthy, with the exception of the right kidney, which had undergone thorough scrofulous degeneration, consisting of nothing but cavities filled with scrofulous pus, the substance of the organ having been totally destroyed. The ureter was large and thickened, and was blocked up in its whole length with cheesy matter."

Being anxious to have another opportunity of examining the ovum in the tube, to ascertain whether there existed a membrana decidua around the placenta and chorion, I wrote to Mr. Favell, asking him to send the preparation for minute investigation, which he most courteously complied with. The following is his letter, which was quickly followed by the preparation itself, in a jar filled up with alcohol.

Sheffield, Feb. 17th.

DEAR SIR,—I shall have very great pleasure in forwarding the specimen of extra-uterine fœtation for your examination. I would have done so ere this, but have not had an opportunity since I received your note; but you shall have it to-morrow. The specimen is just in the state in which I took it from the body, for, being wishful to exhibit it to our medical society, I did not disturb it at all. I should perhaps mention to you that I found no trace of a membrane in the uterus, but its cavity contained a quantity of glairy mucus. I am, dear sir, yours truly,

W. F. Favell.

The preparation consisted of the right half of the uterus, right Fallopian tube, and ovarium. Having placed the parts in a shallow vessel and covered them with rectified spirits, I proceeded with fine forceps and needles, and the dissecting
lens magnifying ten diameters to determine the nature of the connexion between the inner surface of the Fallopian tube and the outer surface of the ovum. Little difficulty was experienced in separating the tube from the ovum and demonstrating that the placenta and villi of the chorion, as in all the other preparations of tubal gestation already exhibited to the Society, were everywhere completely invested with a deciduous membrane. In consequence of this membrane being much hardened by the alcohol, it was impossible to separate completely the two layers from one another and show the decidual cavity. The cells of the placenta and chorion were all found distended with coagula of the maternal blood. The appearance of the amnion was natural. In the cervix and fundus of the uterus there were several soft membranous shreds adhering slightly to the lining membrane. On cutting open the ovarium the corpus luteum was seen, with the yellow matter surrounding the remains of the Graafian vesicle.

The preparation of the parts is now placed upon the table of the Society.

Note.—I have examined two portions of the thick, yellowish-white substance found coating the inner surface of the uterus in two specimens of tubal gestation. No blood-vessels could be discovered ramifying through this substance, which did not present any of the anatomical characters of healthy decidua, and, when examined by an eminent microscopical observer, with a magnifying power of 250 diameters, was declared to be a granular fibrous tissue, resembling coagulated fibrine, and to be wholly destitute of arteries and veins. In none of the preparations of tubal gestation which I have examined has any injection passed from the vessels of the uterus into this substance adhering to its inner surface.

July 14th, 1858.
ON THE
ACTION OF GALVANISM UPON THE
CONTRACTILE STRUCTURE
OF THE
GRAVID UTERUS,
AND ITS REMEDIAL POWERS IN OBSTETRIC PRACTICE.

BY
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Received Dec. 29th, 1857.—Read Feb. 25th, 1858.

The object of the following communication is to submit to the Society some investigations which were undertaken for the purpose of determining the exact influence of galvanism upon the contractile structure of the gravid uterus, the best mode of applying it, and the results which have followed its employment in certain cases attended with difficulty and danger. I need scarcely observe, in doing so, that notwithstanding the length of time which has elapsed since this agent was first proposed as an excitor of uterine action, and the many trials which have been made of it, much difference of opinion still exists in the profession as to its value and remedial powers in obstetric practice, and hence it has happened that whilst its utility has been strongly
advocated by some, it has been denied altogether by others. In this divided state of professional opinion, it has occurred to me that some further investigations might be usefully undertaken for the purpose of determining the accuracy of these opinions respectively, and, if possible, reconciling the discrepancy which exists between them. I have accordingly been led to institute such inquiry, and the present paper contains a brief record of some experiments which were planned and undertaken for the purpose of determining the effective power and modus operandi of galvanism upon the gravid uterus, and the principles which should regulate its employment in obstetric practice.

On looking over the several cases which have been published during the last ten years in support of the value of galvanism as an excitor of uterine action during labour, no doubt would at first arise upon the subject; so strongly is this insisted upon by the reporters, and so apparently successful were the results of its employment. But on closer scrutiny it will be found that in many of them the facts stated do not justify the conclusions which have been drawn from them; that in many a proper distinction has not been made between the specific action of galvanism upon the uterus and that of other agencies collaterally in operation; and it is certain that in some the "post hoc" has been mistaken for the "propter hoc." When, for instance, in a case in which the membranes had been punctured for the induction of premature labour, and labour supervened upon the seventh day afterwards, we may reasonably doubt the conclusion of the writer that this was altogether due to the action of galvanism, although it had been used for half an hour daily after the operation; and when, in a case of uterine inertia occurring during labour, uterine action was restored by merely passing a few shocks through the uterus, we may assume that this effect was as much due to an influence exercised generally upon the nervous system, as to one specifically exerted upon the uterus itself; and this view is supported by the fact that, just as is the case with other powerful impressions made upon the nervous
system, the action of the uterus has been suspended as well as excited during labour by the application of galvanism.

But, on the other hand, many cases have been reported in which galvanism applied to the uterus during labour has been found to be either powerless or useless. Dr. Simpson has more especially recorded such, and he gives the following summary of the results of its employment in eight cases in which it was so applied under the superintendence of Dr. Martin Barry. In one the pains were more frequent in their recurrence, but shorter in their duration, during the application of galvanism. In five its employment neither increased the average frequency of the pains nor their average duration. In one the pains ceased whilst the galvanism was applied, and returned upon its removal; and in the eighth uterine action ceased while the galvanism was applied, and did not return upon its withdrawal, nor for twenty-four hours subsequently. It is impossible not to be struck with the discrepancy existing between the results of galvanism in these cases and in those reported by other observers, and the inevitable conclusion must be, either that the effects of galvanism were greatly misapprehended in the one case, or its proper mode of application disregarded in the other.

Two questions of a preliminary nature require to be decided before this agent can ever be satisfactorily employed in obstetric practice. First, the exact nature of the influence exercised by it upon the contractile structure of the gravid uterus; and, second, the best mode of applying it, so as to obtain the full benefit of such influence. Now, I need scarcely observe that these questions cannot be satisfactorily solved by experiments performed exclusively upon the human female, for in her a variety of circumstances are liable to vitiate their results. In her, for instance, uterine action is liable to be simultaneously affected—to be increased, retarded, or diminished by other agencies collaterally in operation—by emotion of mind, mechanical irritation of the cervix uteri, and the exposure or application of cold to the surface of the abdomen. Other causes
of a less appreciable character are known to affect power-
fully the action of the uterus during labour, and may thus
occasion so many sources of fallacy in our estimate of the
effective powers of any agent, such as galvanism, upon the
action of the uterus during labour.

To obviate these sources of error, and obtain more exact
information as to the power and modus operandi of gal-
vanism upon the gravid uterus of the human female than
could be otherwise gained, I some time ago planned and in-
stituted some experiments for the purpose of determining the
power of galvanism upon the gravid uterus of some of the
lower animals, and as in these the organ was exposed, and
the effects of galvanism upon it was ascertained both by
visual and tactile examination, they were of course exempt
from those sources of fallacy which vitiate similar experi-
ments upon the human female. In this way I was enabled
to obtain decisive information, both as to the modus ope-
randi of galvanism upon the pregnant uterus, and the best
mode of employing it; and believing that they suggest the
true principle by which the employment of galvanism should
be regulated in obstetric practice, I subjoin a brief record
of the principal facts which were observed.

Experiment I.—Does galvanism exercise any, and what,
influence upon the contractile function of the gravid uterus?
To obtain an answer to this question the following exper-
iment was performed at University College, with the assist-
ance of Dr. Boon Hayes, then teacher of practical physi-
ology, in the presence of Mr. Statham, assistant-surgeon to
University College Hospital, and other medical men, with
the following results.

1. On exposing the gravid uterus of a pregnant bitch
which had nearly completed the full period of gestation, and
applying the poles of a common electro-magnetic machine
to the exposed organ (the current being of moderate in-
tensity), no uterine contraction was immediately perceptible,
but in less than ten minutes a slight vermicular or peris-
taltic action was observed to take place in the included
portion of the uterus. This occurred slowly, was of a tonic and sustained character, and it gradually subsided on removing the poles of the machine.

2. On the subsidence of this contraction, and when the uterus had returned to its normal condition, Dr. Boon Hayes ascertained by touch the state of firmness and tension of the uterine parietes, and, having done so, the poles were again applied, his hand being continued in apposition with the organ. Without looking at the uterus he reported, after a few minutes had elapsed, that it felt firmer and tenser than it had been prior to the application of the galvanism, and the correctness of this observation was rendered evident to sight as well as to touch, for the part included between the poles was observed to become more concave and contracted, both as compared with other parts of the organ, and with what it had been prior to the application of the galvanism.

3. Having thus ascertained, both by sight and touch, that the uterus contracted under the stimulus of galvanism, when applied directly to its parietes, I proposed to ascertain in the next place how far the organ contracted when the galvanism was applied to it indirectly, through the medium of the nervous system. For this purpose one pole of the machine was applied to the upper part of the spine, and the other to the uterus, and it was found that uterine action was more powerfully and generally excited by this means than when both poles were applied directly to the uterus. This fact was repeatedly verified by alternately placing both poles to the uterus, and one pole to this organ and the other to the upper part of the spine.

The first question I had proposed to investigate was thus answered in the affirmative. The structure of the gravid uterus not only responds to the stimulus of galvanism, but it does so in a peculiar and remarkable manner, the action thus excited consisting in a slow, vermicular-like contraction of the organ, which is limited to the portion included in the current when the galvanism is applied locally, and affecting...
it more generally and powerfully when it is applied through the medium of the nervous system. I need scarcely observe that this contraction of the uterus under the stimulus of galvanism differs widely from that of the voluntary and other involuntary muscles when acted upon by the same agent. It is more slow in its development, more sustained in its duration, and more gradual in its subsidence, approximating in this respect to the contractions of the uterus during labour. In no instance were those violent and sudden contractions observed to take place which are so remarkable in the case of the voluntary and other involuntary muscles when stimulated by galvanism. On every application of the current the contraction of the uterus was gradual and sustained, some minutes often elapsing before it was discerned; and hence it follows that the mere application of galvanic shocks to the uterus during labour (as recommended by some writers) can be of little or no avail in promoting uterine contraction. It is evident that it is to the sustained action of galvanism upon the uterus, and the consequent production of a sustained tonic contraction of the organ, that we must mainly look for beneficial results in the practical employment of this agent.

To confirm or correct the observations made in this experiment, and more particularly with the view of obtaining an answer to the second question I had proposed, viz., what is the best means of applying the effective power of galvanism to the uterus, I performed a second experiment in the spring of the present year, and on this occasion had the advantage of the assistance of Dr. Harley, of University College, a gentleman who has been extensively engaged in physiological researches both in this country and on the continent.

Experiment II.—What is the best means of applying galvanism so as to obtain its most effective influence upon the uterus? To this question the following notes of
observations made at the time of the experiment afford the most accurate answer we were enabled to obtain.

1. Mere individual galvanic shocks of moderate intensity, passed through the uterus from the fundus to the cervix, did not produce any immediate uterine contractions.

2. After a continuous current had been passed longitudinally through the uterus, that is to say, from the fundus to the cervix uteri, or rather the vagina directly over it, a gentle vermicular-like contraction of the organ was observed to take place in the course of about sixty seconds; and, during the five subsequent minutes that the current was continued, this vermicular-like contraction was observed to propagate itself slowly over the whole of the uterine parieties.

3. A current directed transversely through the uterus for two and a half minutes caused slight contraction in that portion of the organ which was included in the current, but not in any other.

4. On directing the current longitudinally through the uterus, through the medium of the spinal cord—that is to say, by applying the positive pole of the machine to the nape of the neck of the animal, and the negative to the vagina, immediately over the os uteri—very powerful uterine contractions were produced in the course of two minutes. They commenced, as it were, in bands of the uterine fibres, and were of a much more general and powerful character than when the poles of the machine were applied locally to the uterus. The current was continued for five minutes with increasing effect, and, on discontinuing it, the contraction of the uterus continued for about five minutes longer, its intensity being apparently as great as during the application of the galvanism.

5. On repeating this experiment when the animal was somewhat exhausted, it was observed that uterine contraction was greater when the current was occasionally intermitted.

6. As the animal became still more exhausted the action of galvanism upon the uterus became much less apparent,
and as the vital powers declined it became powerless in exciting uterine contraction.

Before proceeding to consider the practical bearing of these facts, it may be convenient to recapitulate briefly the chief conclusions which are to be drawn from these experiments, viewed in relation to each other.

First. In both the power of galvanism to excite the contractile structure of the gravid uterus was unequivocally made manifest.

Secondly. In both it was rendered evident that the influence thus exercised by galvanism upon the organ is of a peculiar and specific character, differing greatly from the effects of the same agent upon the voluntary and other involuntary muscles.

Thirdly. In both it was shown that the influence of galvanism upon the gravid uterus is much more powerful and general when the current is applied to it through the medium of the nervous system—or rather its spinal portion—than when it is applied locally to the organ itself.

Fourthly. That galvanic currents directed longitudinally through the uterus, i.e., from fundus to cervix, promote powerful and general uterine contraction; whereas currents passed transversely through the organ excite partial contractions only in the direction of the current, and hence must exercise an unfavorable influence upon the parturient process.

Fifthly. It follows, therefore, from the last two observations, that the most efficacious mode of employing galvanism, so as to obtain its most effective influence upon the gravid uterus, is to direct the current in a longitudinal direction through the organ from the upper portion of the spinal cord, that is to say, by applying the positive pole of the machine to the upper part of the spine, and the negative to the cervix uteri.

Sixthly. That, so applied, the force of the current may be increased or intensified by occasionally breaking or
interrupting its continuity, that is to say, by momentarily removing, from time to time, one of the poles of the machine from contact with the body.

Seventhly. That mere individual galvanic shocks, passed through the uterus, exercise little or no influence in exciting uterine contraction, and that, to obtain the specific influence of galvanism upon the uterus, it is necessary that the current should be sustained until the desired object is attained.

Eighthly. That in proportion as the constitutional powers fail is the influence of galvanism less powerfully exercised upon the uterus, until at length, with increasing exhaustion, it ceases to exert any influence whatever.

The foregoing observations appear to me to point out with greater precision than has hitherto been done, the nature of the influence which is exercised by galvanism upon the gravid uterus, the best mode of applying it, and the kind of benefit we are likely to derive from it when so employed. Had this inquiry, however, terminated here, I should not have troubled the Society with any communication upon the subject. The facts now stated had indeed been long known to me, but they appeared to be suggestive rather than conclusive as to the best mode of employing galvanism, and the advantages to be derived from it in obstetric practice, and it was not until I had had an opportunity of testing at the bedside the value of the practical inferences I had ventured to draw from them, that I deemed them of any positive value. Now, however, that I have had that opportunity, and have found that the results of clinical experience support their general correctness, I have ventured to submit them to professional notice, and I will proceed in the next place to lay before the Society the clinical evidence I have obtained of the utility of galvanism so employed in obstetric practice.

The advocates of galvanism as an obstetric agent claim for it a wide range of utility, and recommend its employment in all cases in which it is deemed desirable to excite or promote uterine contraction. I need scarcely observe, however,
upon the inexpediency of resorting to the use of so formidable and in some respects so inconvenient an agent in cases in which simpler and less powerful means may suffice for the attainment of the object in view. It appears to me, indeed, whatever may be the value or remedial powers of galvanism in midwifery, that its use will be materially restricted by this consideration; and that it will always be regarded as an agent to be used in exceptional cases only where the better recognised and more established rules of practice either fail altogether or offer little prospect of doing good. Regarded, however, in this point of view, there are yet many in which it may be usefully resorted to and some in which, if I may judge from my own experience, it affords the best security to life. Of such I would instance more particularly certain cases of placental presentation in which profuse haemorrhages continue to recur, notwithstanding the employment of the plug and other means, before the os uteri is sufficiently dilated to admit of manual assistance; and cases of haemorrhage in the early months of pregnancy which resist the usual means employed for their suppression, and which, from the constricted state of the os and cervix uteri do not admit either of mechanical or manual interference. In both these cases I have employed galvanism, in the manner suggested by the foregoing experiments, with the best results—in the latter case when all other means had proved abortive, and the patient’s life was in the greatest peril—in the former so favorably as almost to justify the belief that it establishes a new principle of treating some of the worst cases of placenta praevia; and believing that they afford the most conclusive proofs I could adduce of the efficacy of galvanism so applied in obstetric practice, and furnish at the same time the best text I could select for the further observations I have to offer upon the subject, I will briefly submit to the Society the chief points in the history of each which bear more especially upon the question under consideration.
Case of profuse hæmorrhage occurring in the early months of pregnancy successfully treated by galvanism after other means had failed.

A young married lady, of a highly nervous temperament, consulted me on the 11th December, 1856, on account of uterine hæmorrhage which had commenced on the preceding day, and which she attributed to a long and fatiguing drive. She was apparently in the third month of pregnancy, and gave the following history in connection with the attack. She was married on the 9th September, 1856—three months before the date of my visit—and on the 15th of the same month she menstruated as usual. During October menstruation did not take place, and other circumstances pointed to the probable supervision of pregnancy. About the middle of November—six weeks from the date of the last menstruation—she had a slight hæmorrhagic discharge, which disappeared in the course of the same day, and was apparently brought on by over fatigue and travelling.

On the 15th of the same month—a period which corresponded with that of the second menstruation dated from the time of its cessation—hæmorrhage again occurred, which lasted two days, and on the 10th December—nearly a month afterwards—it again occurred and continued profusely until the following day, when I was sent for.

Nothing like an abortion had as yet taken place, although many large coagula had passed, and on examination I found the body of the uterus somewhat enlarged, the cervix rigid and undeveloped, and the os uteri small and almost closed. She was requested to remain in the horizontal position, to keep her apartment cool, and to drink freely of iced lemonade. Saline aperients, strongly acidulated with dilute vitriolic acid, were at first prescribed, and afterwards, gallic acid, matiko, and other astringents, alternated with aperients, and opiates when in pain. By these means, aided by the occasional application of cold, the hæmorrhage was moderated though not entirely restrained until the 24th of December,
when she experienced a good deal of backache and forcing-pain in the uterus, and in the course of the night a substance was expelled which she described as very fetid and unlike anything that had previously passed. This was followed by much relief, and the following day she expressed herself as feeling quite a different person, the haemorrhage and pain having almost entirely ceased. I had no opportunity of examining what had passed, but concluded, from the patient's account of it, and her subsequent feelings, that it was either the entire ovum or a portion of it. From this time up to the 10th of January, there was little or no haemorrhage or pain, but the patient continued very weak and anaemic, and had been obliged to be almost always recumbent. At this time haemorrhage again occurred, and continued for four days; but as it was not excessive and corresponded with her supposed menstrual period, it was attributed to that cause. On the 28th of the same month, however, it again occurred, and lasted two days, and as this did not admit of a similar explanation, I was again asked to see her. I found her excessively weak and depressed, she had frequent backache, and now and then forcing-pains in the uterine region. These symptoms, together with the return of haemorrhage at so unusual a period, led to the surmise that the entire ovum had not passed, and that some cause of irritation connected with it still existed in the uterus, and she was accordingly treated upon this view of the case. On the 10th of February haemorrhage again returned, and lasted four or five days, but it was not excessive, and it apparently coincided with the usual menstrual period. On the 27th of the same month it again occurred very suddenly and profusely, and its effects when I saw her were strongly marked upon the constitution. On again examining the uterus, I found the body still larger than it should be, but the cervix was undeveloped and the os uteri small and undilated. Feeling quite certain that the uterus still retained some portion of the ovum, the retention of which was the effective cause of uterine irritation and consequent haemorrhage, or, in other words, believing that the
repeated attacks of haemorrhage depended upon so many abortive attempts on the part of the uterus to get rid of such cause of irritation, I resorted to a variety of means to excite uterine action. Turpentine enemata were administered, and drachm-doses of the aethereal tincture of ergot, with gallic acid and decoction of bark, were given every four hours. These remedies, combined with occasional aperients, were continued until the 5th of March, when, as matters still remained the same, and no organized structure had as yet been expelled from the uterus, she was visited by Mr. Stone, who had formerly attended her. He concurred generally in the view which had been taken of the case, the cause of the floodings, and the practice which had been adopted, and merely suggested, as she was excessively exhausted, that some of Battley's concentrated preparation of bark should be added to the medicine she was taking. No material change occurred until the 8th of March, when another very profuse and alarming haemorrhage took place which left her almost lifeless, and on visiting her I found her in a state of alarming syncope. Her breathing was hurried; she complained of distressing faintness, throbbing of the temples, knockings in the head, &c.; and her pulse was so feeble as to be scarcely felt. In this emergency she was again visited by Mr. Stone, who observed that in the whole of his experience he had only once seen an instance of such excessive haemorrhage in the early months of pregnancy, and that if it were not the fact that such cases rarely prove fatal he should have little or no hope of her recovery. It should be observed that before his arrival I had firmly plugged the vagina by means of an oiled silk handkerchief, having previously found that the os and cervix uteri were still undilated, and not such as to admit of more than the point of the finger—a fact afterwards confirmed on examination by Mr. Stone—and feeling certain that any further haemorrhage would almost inevitably prove fatal. This treatment was continued throughout the whole of that and the following day, but without any apparent result, and on the 10th, the condition of the patient was extremely critical.
She was still suffering severely from the effects of loss of blood, notwithstanding the free administration of brandy, cordials, beef-tea, jelly, &c.; the os and cervix uteri were still too constricted to admit of manual interference; the plug was now becoming a source of excessive pain and discomfort, and the past history of the case had shown that astringents could not be relied upon, on the one hand, to prevent haemorrhage, or ergot and turpentine enemata to produce sufficient uterine action to expel its presumable cause, on the other. It was under these circumstances that I determined to have recourse to galvanism, having, as I conceived, fairly tried all the more established plans of treatment without success. I accordingly proceeded to apply it, in the course of the afternoon of this day, in the manner which I had found to be most effective in my experiments upon the lower animals, that is to say, by applying the positive pole to the nape of the neck, and the negative, by means of a vaginal conductor, to the cervix uteri. In this way a continuous current, occasionally interrupted, was applied and continued in action for half an hour, and during its continuance the patient felt occasional pains in the uterine region, and stated that it appeared to revive her and give her strength. I had examined the state of the os and cervix uteri before applying the galvanism, and at the end of half an hour I again did so, and was much struck with the change that had taken place in the interval. The cervix was more expanded, and the os uteri was more open and relaxed, so that the examining finger could pass more easily and further into its interior. So striking was this change, that I continued the current half an hour longer, anticipating some speedy result from its action; but in this I was disappointed, and as the patient was now somewhat fatigued, it was discontinued for the remainder of the day. The next morning, encouraged by these favorable effects, I again had recourse to it, and applied it as before, but on this occasion it was used for two hours instead of one, that is to say, for one hour in the morning, and another in the afternoon. As before, the os and cervix uteri became
softened and expanded during its employment, and occa-
sional uterine pains were felt; no hæmorrhage, however,
occuring, and the patient, on the whole, feeling stronger
and better for its use. I had intended applying it for
a still longer period the next day, but, on visiting her
for the purpose, found that it had become unnecessary,
and that, in fact, the object of the treatment had already
been attained. It appeared that, after I had left her the
preceding day, she had a good deal of uterine uneasiness,
that this recurred at intervals during the night, and that,
in the early part of the morning, an organized structure
was expelled from the uterus, portions of which I have
preserved, and beg to submit to the Society. On examina-
tion, this proved to be the decidua or uterine constituent
of the ovum; and with its expulsion, I felt that all
further anxiety about the case, as well as fear of hæmor-
rhage, was at an end. And so it proved, for from this
time neither hæmorrhage nor any other unfavorable symp-
tom occurred, and the patient’s recovery was as favorable
as could be wished. No other treatment was adopted than
that necessary for the restoration of her strength, and she
soon afterwards left London for the sea-side.

As a sequel to this history, I may add that this patient
has again become pregnant, has reached her seventh month,
and is, apparently, likely to do well.

I trust I shall not be deemed hasty or illogical in
venturing to connect the final termination of this case and
the safety and security of the patient with the ultimate
employment of galvanism; for, although the course of events
may not have been such as to render this conclusion
inevitable, yet it was such as to render it highly probable.
Let it be remembered that an interval of nearly four months
had elapsed between the first threatening of abortion in
November, and the final expulsion of the deciduous remains
of the ovum in March; that during this period many attempts
had been made by the uterus to effect their separation and
dislodgment, with no other result than that of giving rise to profuse and alarming hæmorrhages; that these were but little restrained by the various styptic and astringent remedies which were resorted to on the one hand, and that the action of the uterus was not more successfully excited by the administration of large and frequent doses of ergot and turpentine enemata on the other; that the plug had been assiduously applied for three successive days and nights without exciting any effective uterine action; and that its continued employment had at last become a source of intense suffering and distress to the patient. Now, bearing in mind these several circumstances, and the positive evidence which was obtained by tactile examination of the effect of galvanism in relaxing and opening out the os and cervix uteri; and that almost immediately after its second application uterine action set in, which in less than twelve hours effected the separation and expulsion of the decidua and secured the safety and well-doing of the patient. It appears to me that if it is not proved that galvanism was the effective cause of this result, yet that the facts, taken together and viewed in relation to each other, constitute a mass of demonstration in favour of this conclusion, than which nothing in the nature of medical evidence can be more decisive. And if such a conclusion is allowable, does it not follow as a necessary corollary that the case furnishes a precedent for the treatment of others of a similar nature? That is to say, that when dangerous hemorrhages occur in the early months of pregnancy, as a probable effect of the retention of portions of the ovum, and in which mechanical aid is impracticable, and other recognised means of treatment unavailable, that galvanism may be usefully and successfully resorted to. Such, I confess, is the impression left upon my mind after a careful consideration of the principal facts of this case; and looking to the inefficacy of the means adopted prior to the employment of this agent, I can only regret that it was not resorted to earlier in its treatment.

I proceed, in the next place, to submit to the Society the particulars of a case of placental presentation, in which
galvanism was successfully resorted to for the purpose of arresting hæmorrhage and accelerating the dilatation of the os and cervix uteri, so as to admit of the introduction of the hand and the delivery of the patient by turning.

CASE II.—Placental Presentation treated by Galvanism.

A lady, whom I had been engaged to attend in her approaching confinement, sent for me on the 30th October, 1857, on account of a slight attack of uterine hæmorrhage, which had occurred in the early part of the morning. She was apparently in the seventh month of her fifth pregnancy, and as the hæmorrhage had occurred when she was perfectly quiet and recumbent, and had not been occasioned by any obvious cause, it suggested the probability that it might be of the unavoidable character. As, however, it had been slight, and had entirely ceased before I saw her, I simply advised her to remain quiet, to take a saline aperient and some nitrate of potassa dissolved in cold water, in the manner recommended by the late Dr. Gooch, should there be any return. The next day I found that no further hæmorrhage had occurred, and I did not hear more of the case until the 23d of November, when another slight hæmorrhage occurred in the early part of the morning; but, as in the former instance, it had entirely ceased at the time of my visit, and it did not again recur throughout the day. On the 26th, I was again sent for, and now found that between this and my previous visit she had sustained a considerable loss of blood. It had returned very profusely early in the morning of the 24th, and, after continuing slightly throughout that and the following day, had again become very profuse on the morning of the 26th, so much so, indeed, that it was estimated that she had lost three pints of blood during these days. On examination, I found the cervix high up, and the os uteri small and undilated, forming a nipple-like projection into the vagina. I passed my finger within it, but could not determine with accuracy the nature of the presentation on
account of its height, and on endeavouring to pass my hand into the vagina for the purpose of making a more accurate examination; the patient experienced so much pain that I was obliged to desist. On examining the cervix externally, however, I could distinctly feel a soft cushiony body lying within it, and I could even detect, as I thought, the convex outline of the placenta. Undoubtedly some such body was interposed between the child and the cervix; and this fact, together with the history of the case, left no doubt on my mind that it was the placenta.

Having been long anxious to try the efficacy of galvanism in these cases, from a conviction, as I have elsewhere expressed it, "that it would simultaneously tend to the arrest of hæmorrhage and the dilatation of the os uteri," by exciting and maintaining a tonic contractile action of the organ, I communicated my views upon the subject to my friend, Mr. Forbes, and begged that he would visit the case with me, with the view of being favoured with his opinion both as to its nature and the course of treatment I proposed to adopt. He found much difficulty in determining accurately the character of the presentation, on account of the height of the cervix uteri; but, after a lengthened examination, stated that he thought he could feel the placenta, and on passing his finger over the exterior of the cervix, he further recognised the existence of the spongy convex body I have described. With his concurrence, I applied the galvanic current, in the manner I have already described. It was now 4 p.m., and it was intended that the current should be continuously kept applied until 8 p.m., when we agreed to meet again, and note the further progress of the case; but, by some accident, the current was interrupted shortly after we left, and it was not until 5 p.m. that its action was fairly commenced. At 8 p.m. we again visited the case, and found that no hæmorrhage had occurred during the three hours that it had been applied; and, on examination, both Mr. Forbes and myself recognised the fact that the os uteri was more open and the cervix more relaxed than when we had previously seen the patient. As, however, it was now
late, it was determined to discontinue the galvanism for that
day, but to reapply it the next, and then to keep it con-
tinuously in action until the state of the os uteri admitted
of delivery. At twelve at night, however, I received an
urgent summons from the patient on account of a fresh
attack of hæmorrhage which had just occurred, and as she
was now evidently suffering severely from the effects of loss
of blood, and the state of the os uteri did not admit of her
delivery, I determined to reapply the current and keep it
continuously in action until it would do so. This was
accordingly done, and it was continued for nearly six hours.
During this time little or no hæmorrhage took place, and
little or no pain was experienced by the patient, and, at the
end of it, I found that there would be no difficulty in turning
the child. I accordingly proceeded to do so, and easily
succeeded in passing my hand into the vagina and thence
through the os uteri; and now the nature of the case was
rendered evident, for the placenta was found stretching
equally over the whole of the uterine orifice. So centrally,
indeed, was it attached, that it was difficult at first to know
in what direction to seek for the membranes. Having, how-
ever, reached them, I was enabled to grasp and bring down
a foot with the greatest ease, and the delivery was effected
without any difficulty or hæmorrhage. The placenta was
afterwards removed with equal facility and without hæmorr-
hage; and, although the patient showed evident signs of the
loss of blood she had previously sustained, yet she felt
cheerful and comfortable; and her recovery was so favorable
that, on being visited by Mr. Forbes, on the 6th day after-
wards, he remarked that she appeared to be as well as if she
had merely gone through a natural labour.

I have remarked in the course of this narrative that
little or no hæmorrhage occurred during the employment
of the galvanism, and that the patient experienced little
or no pain or discomfort from it. It is right, however, to
observe that these points were carefully investigated
throughout the whole management of the case. An arrange-
ment was specially made for noting the amount of blood that
was lost during the use of the galvanism, and I can safely assert that it did not exceed, or even amount to, a small teacupful; and, with regard to the amount of pain experienced by the patient, I may remark that, although frequently questioned upon this point, she almost invariably denied having any, although, on external examination, the uterus felt tense, rigid, and hard, whenever the hand was applied for the purpose of ascertaining its condition. We may therefore, I submit, without overstepping the limits of legitimate induction, draw three conclusions from the facts of this case in reference to the practice pursued in it.

1st. That a continuous current of electricity directed longitudinally through the uterus from the upper part of the spinal cord may be certainly relied on to effect the dilatation and development of the os and cervix uteri in cases of placenta previa.

2d. That such dilatation and development may be effected under its influence without any serious hemorrhage taking place during the necessary separation of the placenta which is contingent upon this process.

3d. That it may also be effected without any appreciable amount of pain being felt by the patient, and certainly without the recurrence of those acute rhythmical pains which are so constantly experienced in ordinary labour.

I have ventured to observe, in the introductory remarks to this paper, that the facts of this case, fairly considered, point to a new principle in the treatment of certain cases of placenta previa, and I would now wish to submit to the Society the grounds upon which such opinion is based.

I need scarcely observe that the treatment of cases of placental presentation is, under ordinary circumstances, one of great difficulty; so much so, indeed, that it has been calculated that the average mortality to the mother is as one to three, and this difficulty and fatality would appear mainly to depend upon the insufficiency of the means hitherto employed to restrain hemorrhage whilst the os uteri is undergoing the necessary dilatation, either for the descent of
the head or the introduction of the hand. To attain this object two modes of treatment have been hitherto resorted to—puncturing the membranes, and the employment of the plug; but neither can be always relied upon for the fulfilment of this object. The former, indeed, can scarcely be trusted to for the suppression of hemorrhage in these cases, and the latter is painful, tedious, and uncertain. Now if it can be shown that by passing a current of electricity through the uterus for a few hours in the manner recommended, both these objects can be accomplished, that is to say, the dilatation of the os uteri, and the simultaneous control of hemorrhage—then it appears to me that we possess in galvanism an agency which is far more effectual in the treatment of these cases than any which has hitherto been proposed. As compared with the action of the plug it has this advantage, that the end proposed can be attained more expeditiously, more safely, and less painfully. Thus the pressure of the plug requires sometimes to be maintained for many days before its object is gained, and it sometimes fails altogether to excite adequate uterine action, as in the first case I have reported; it can exercise no influence upon the tonicity of the uterus whereby internal hemorrhage can be prevented, whilst its employment is attended with much suffering and inconvenience to the patient. On the other hand the facts of the case I have related go far to show that the galvanic current may be relied upon to effect full dilatation of the os uteri in a few hours; it moreover excites a sustained tonic contraction of the uterine parietes, which is favorable to the prevention and suppression of hemorrhage, and, as I have shown, it may be used in such a manner as to give little or no pain or discomfort. Such were its results in the particular case I have laid before the Society, and such are the results I think we may venture to anticipate from its employment in others.

I am unwilling to trouble the Society with the details of any additional cases treated by galvanism, not only because in doing so I should extend the limits of this paper to an inconvenient length, and also because I have met with none
which testify so decisively to its value in obstetric practice. Moreover, it appears to me that individual cases like individual facts are only really useful when they tend to establish some new inference, or some new principle, either in theory or practice. I will therefore only observe that I have employed galvanism successfully in various other obstetric emergencies: for exciting uterine action in cases of uterine inertia, for the expulsion of coagula after labour, and for the suppression of passive haemorrhages after the expulsion of the placenta. The utility of it in these cases has, however, been insisted upon by others, and the particulars of several that I have so treated have already been made public.

How comes it, then, it may be asked in conclusion, if such are the facts, that different observers have arrived at such different conclusions as to the value and remedial powers of galvanism in obstetric practice, and how in particular are we to reconcile with them the negative results which were obtained and reported by Dr. Simpson? The answer, I apprehend, is to be sought for in the fact that the observations of Dr. Simpson are open to at least two sources of fallacy. First, in not having employed galvanism in the most effective manner; and, secondly, in having made the frequency and severity of the labour-pains the sole test of uterine action. Thus he informs us that in all his cases Dr. Radford's directions for the employment of galvanism were strictly adhered to, viz., "When the remedy is applied, the brass bar of the vaginal conductor is to be passed up to the os uteri, and moved about at intervals on to various parts of this organ. At the same time the other conductor must be applied to the abdominal parietes, over the fundus uteri. Shocks may also be passed transversely through the uterus, by simultaneously applying the conductor on each side of the belly." Now in my experiments it was distinctly noted: first, that the local application of galvanism is not the most effective means of employing it; secondly, that mere individual shocks passed through the uterus exercise no influence in promoting or exciting uterine action; and,
thirdly, that currents directed transversely through the uterus, by inducing partial contractions of the organ in the line of the current, actually tend to arrest the parturient process. And again with regard to the intensity and frequency of the labour-pains as the test of uterine action, I have only to observe that the last case I have reported tends directly to negative this conclusion; for although in this a firm tonic contraction of the uterus was distinctly felt on applying the hand to the abdomen; and the dilatation of the os uteri went on so speedily as to admit in a few hours of delivery, yet the patient, on being frequently questioned, scarcely ever admitted that she felt any distinct pain, and certainly not such as she had been accustomed to regard as labour-pains. It would appear, then, that on these several grounds we may venture to dissent from the conclusions which have been arrived at by Dr. Simpson, respecting the effective power and value of galvanism in obstetric practice, and in disposing of his objections to its utility we dispose of the greatest objections which have been raised to its employment in this department of medical practice.

I have only further to add that the apparatus I have used for the administration of galvanism has been the electro-galvanic machines, invented and sold by Messrs. Horne, Thornwaite, and Wood, of Newgate Street. They have been especially constructed with reference to the production of a large amount of electricity, and its transmission in one uniform direction, and as such they are preferable to the double-current machines, in which the quantity generated is not only smaller but is continually returning upon itself. In the first case I have reported the machine marked No. 2 was used, and in the second that marked No. 3.

Postscript, February, 1858.—Within the last few days I have had another opportunity of testing the remedial powers of a sustained current of electricity, in the treatment of a case of uterine hæmorrhage, which occurred in the last month of pregnancy, before the accession of labour, and was presumably dependent upon the attachment of the placenta.
to the cervix uteri. The application was perfectly successful, and, as the facts of the case in every respect support the views enunciated in the preceding paper, I beg to append the following notes of it.

Case.—A. B.—, an extremely diminutive young woman, who had nearly completed the full term of her first pregnancy, but as yet had had no symptoms of labour, was suddenly attacked with profuse uterine hemorrhage late in the evening of the 18th of February. It occurred when she was perfectly quiescent, and without any apparent cause. The blood gushed from her in a full stream; was of a bright arterial colour, and escaped so profusely, that before I saw her at 10 p.m., less than two hours after it had commenced, she was computed to have lost nearly two pints. At this time she was pallid and faint, and the hemorrhage was still going on. On examination, the vagina was found to be very contracted, the os uteri high up, and so undilated, that I could with difficulty insert the point of the index finger within it. I could, however, distinctly feel the head presenting, without any portion of the placenta intervening, but on pressing my finger around the cervix externally, a convex, thick, spongy body could be felt, anteriorly interposed between it and the fetal head. This was subsequently recognised by Mr. Norway, who observed, that he had no doubt whatever as to its being the placenta. Looking to the dangerous and uncertain character of these cases, to the loss of blood which the patient had already sustained, its suddenness and profuseness, and the effect it had produced upon her, I lost no time in applying the electric current, in the manner I have already described. It was applied at 10 p.m., and was kept almost continuously in action for three hours, during the whole of which period scarcely any hemorrhage occurred, and the labour was found, on occasional examination, to be rapidly progressing. Active parturient pains now set in in a regular and rhythmical manner, and as the os uteri was now considerably dilated, and the head was rapidly descending, without
any hemorrhage having occurred, the electric current was withdrawn, but so arranged that it could be again applied in the event of its returning. Such, however, was not the case, and the further progress of the labour was so favorable, that at 5 a.m. the next morning, seven hours only from its first application, the patient was safely delivered of a living child; the placenta was thrown off almost immediately afterwards, and the uterus contracted firmly, without any further hemorrhage.

With reference to the facts of this case, I will only observe that they harmonise in every respect with those of the one I have previously related, and that together they go far to show:—

1st. That a sustained current of electricity of moderate intensity, passed through the gravid uterus, in the manner described, exercises a remarkable influence in increasing the tonicity or contractility of the uterine fibre.

2d. That in such increased tonicity or contractility of the uterine fibre, so excited and sustained, we have a powerful and reliable means of moderating and controlling uterine hemorrhage, whether of the accidental or unavoidable variety, and of simultaneously accelerating the dilatation of the os uteri and the general progress of the labour.

3d. That such sustained current of electricity may be continued for a lengthened period, when the object to be attained requires it, without any appreciable pain or inconvenience to the mother, or any danger or detriment to the child.
A CASE
OF COMPLETE
INVERSION OF THE UTERUS,
of nearly twelve years' duration, successfully treated.

BY

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Received Dec. 31st, 1857.—Read April 13th, 1860.

The tenth and thirty-fifth volumes of the 'Medico-Chirurgical Transactions' contain the histories of two cases of inversion of the uterus occurring after parturition; the first by Mr. John Windsor, of Manchester, and the second by Mr. Gregory Forbes. In one of these cases extirpation was effected, and the patient recovered; in the other, palliative measures were resorted to, and the patient died of exhaustion eighteen months from the date of delivery. These cases are scarcely more interesting or remarkable than that which is the subject of the present paper, and as the latter illustrates a new method of treating this formidable affection, I have ventured to bring it before the notice of the Society.

Hitherto, inversion of the uterus has generally been
treated either by styptics and astringents, or the inverted organ has been removed by ligature or excision. The instances in which reinversion has been accomplished have been few in number, and chiefly limited to cases of recent origin. Mr. Forbes, who gives a very excellent account of current opinions on this subject, states that "with regard to the possibility of reducing the inverted organ, it may be admitted, on the testimony of the most competent observers, that unless this desirable end can be effected within a few hours after the accident has occurred, there is little or no hope of its being afterwards accomplished." He observes that, in a few rare and exceptional cases, reduction has been effected at a later period, and of the cases he himself collected, one had been repoised fifteen months after the inversion had occurred. In still more rare instances, it has been found that the accident has led to no great haemorrhage or other serious symptom; or that spontaneous reinversion has taken place. But the result, in the vast majority of cases, where the inverted uterus remains unrelieved, is undoubtedly a fatal one. Mr. Forbes refers to fourteen cases in which death followed at periods varying from eight months to five years after the occurrence of inversion. As regards the serious operation of extirpating the inverted organ, the paper of Mr. Forbes also contains some important statistical information. Of twenty-six cases treated by ligature, the operation was successful in twenty-one. In five cases it was unsuccessful, and of these three died. In the other two the ligature had to be removed soon after its application. Of two cases treated by excision, one recovered, and one died. In eight cases treated by the ligature and excision combined, five were successful, and three were unsuccessful. Thus, of thirty-four cases of extirpation, twenty-seven recovered, and seven died; but probably in this, as in other formidable operations, the results appear more favorable than they really are, from the non-publication of unsuccessful cases. To show the difficulty of diagnosis in inversion of the uterus, it may be mentioned that in no less than nine of the preceding cases, the uterus was mistaken
for polypus. With these remarks, I proceed to relate the case which fell under my own observation.

Case.—Mrs. J, aged 18, was delivered of a first child on the 6th of October, 1845. Immediately after the completion of labour her medical attendant left the house, having received a summons to another midwifery case, at a considerable distance in the country. Mrs. J lost a very large quantity of blood after his departure, and was said to have remained insensible for ten or twelve days. At the end of this time there was no swelling externally, and no suspicion of inversion was excited. She continued, however, to lose blood, which at first was considered to be the lochial discharge in unusual quantity, but none of the means used were sufficient to arrest the haemorrhage. The flooding continued to a greater or less extent for nearly twelve years. During this long time she was never for a single day free from sanguineous discharge. Before her pregnancy, the catamenia had been regular and moderate. From the time of her labour, the loss of blood generally increased at the monthly dates, and remained profuse for upwards of a fortnight, after which the discharge would become paler for about ten days, until the date of the next monthly return; but it never ceased entirely. The extent of the haemorrhage may be gathered from the fact that during the fortnight of excessive loss, from fifteen to twenty thick napkins would become saturated every twenty-four hours. When this state of things had continued for some time, she was examined, and the existence of a tumour in the vagina was detected. The opinions of the many medical men who saw her were divided as to whether the case was one of inverted uterus, or of polypus descending after labour, as sometimes occurs. The existence of this doubt prevented any operative means for the removal of the tumour, beyond abortive attempts to replace the uterus, by those who considered it a case of inversion. Astringents given internally, and used externally, with every other means that could be devised, failed of affording any relief.
In July, 1856, Mrs. J—— was sent to me from Port Madoc, North Wales, where she resided, by Mr. Griffith, under whose care she had been for a short time only, and from that gentleman, and the patient herself, I learnt the foregoing particulars of her case. When I first saw her, she was in a state of extreme anaemia. The skin was exceedingly pale and wax-like; the conjunctivæ were without any visible trace of blood-vessels, the tongue and gums were ash-coloured, as was also the mucous membrane of the vulva and vagina. She was not particularly thin, but flabby, and the lower extremities and the body generally, were œdematous. The pulse was scarcely perceptible at the wrist. When she was at rest the sounds of the heart were regular but weak, and loud venous murmurs were audible in the neck. Her appetite was habitually bad, and a sense of nausea, often proceeding to vomiting, was very distressing. She suffered severely from cephalalgia. Dimness of vision, sometimes amounting to blindness, tinnitus aurium, palpitation increased to a most alarming extent by slight exertion, and great breathlessness. On many occasions she had fainted and remained insensible for several hours. She had also been subject to convulsions of an epileptic character. Sleeplessness was a very marked feature in her case. She rarely slept soundly, and on falling off to sleep was constantly roused by a feeling of faintness and dissolution. This was increased if she lay on her left side. The sleeplessness and breathlessness mentioned are, I believe, constant attendants upon excessive and long-continued loss of blood. It appears as though, in such extreme cases, respiration could not go on without the assistance of volition, and as if the system could scarcely afford that amount of cerebral congestion necessary to sleep.

The effects of the constant haemorrhage on the secretions were very remarkable. She described herself as never having been in a perspiration since the loss of blood commenced. The action of the skin seemed to be entirely suspended. The bowels were obstinately constipated. She made very little water, and constantly went twenty-four
hours or more, without passing any urine at all. It appeared as if the loss of blood took the place, to a considerable extent, of the several secretions, and as if in this way the system had become, to some extent, accustomed to the drain from the uterus. Every succeeding year she had, however, become sensibly weaker, and the fits of fainting and insensibility had grown more frequent and lasting.

On making a digital examination, a pyriform tumour could be felt within the vagina, hanging from the os uteri. It was difficult to make out by the finger whether the os and cervix were inverted, or whether the tumour hung as a polypus from the interior of the uterus. The os uteri was small and rigid, and the neck of the tumour was of corresponding size. No amount of sudden pressure could have forced the mass of the tumour upwards, without rupturing the os and cervix uteri. A probe, or the uterine sound, could not be passed more than half an inch within the os, showing that no uterine cavity existed. The structure of the tumour did not appear harder or more elastic than in some cases of firm fibrous polypi, in which degeneration has not commenced. I have felt some cases of polypi which could hardly have been distinguished by the touch alone, from the tumour in the present case. The size was somewhat larger than the natural size of the unimpregnated uterus. The sensation communicated to the finger was very much like that of a mass of India-rubber, softened at the surface. Examined by the speculum, and with a fine probe, nothing like the openings of the Fallopian tubes could be detected. The mucous membrane, like the mucous covering of polypi, was seen to be shreddy and ulcerated in patches. Some parts of the tumour were smeared with pus, and at others blood freely exuded from its surface. The mass was comparatively insensible to the touch, and the prick of a needle or the pinch of a pair of forceps gave little or no pain; but, on one occasion, when a ligature was passed round the neck of the tumour, and held tightly for a short time, most acute suffering was produced.

Mr. Griffith, when he sent the patient to me, considered
the case to be one of inversion, and after several examinations, I arrived at the same conclusion, though some experienced accoucheurs to whom I showed the case, among whom I may mention the late Dr. Ashwell, inclined to the opinion that it was polypus. Acting, however, on the belief that the uterus was inverted, I resolved to attempt its reduction by continuous pressure. As the tumour was not painful, and as the circulation and respiration were feeble and distressed, I did not consider it prudent to use chloroform, or to effect any sudden replacement, for which chloroform is so well adapted in comparatively recent cases, but depended on the effects of moderate and sustained pressure, with the view of dilating or developing the os and cervix uteri, so as to admit of the return of the inverted organ. With this intention I passed the right hand into the vagina night and morning for several days, and endeavoured by squeezing and moulding the uterus with the fingers for about ten minutes at a time, to press the tumour upwards. At first no impression could be made in this way, but after repeated trials, I found the cervix uteri yield a little, and the tumour could be sunk slightly in the os. On each occasion, after removing the hand, I passed one of M. Gareil’s large air-pressaries into the vagina, and inflated it to as great extent as the patient could bear. By this means a very considerable force was constantly exerted upon the tumour. The air-pressary was worn day and night, with few exceptions; or when it was removed on account of pain, or for the purpose of relieving the bladder and bowels. From the time of the first introduction of the pessary, the haemorrhage ceased entirely, and the tumour became somewhat less in size. On each succeeding day, it could be passed a little higher within the os uteri. After more than a week of these proceedings, the patient felt a good deal of pain through the whole of one night, and in the morning, when an examination was made, it was discovered that complete reversion had taken place.

The os uteri was now found to be considerably dilated, and readily admitted the end of the finger. The sound could be passed fully three inches into the uterine cavity. There
was a considerable amount of muco-purulent discharge, and
on examining with the speculum, the os was found to be
abraded and patulous, but otherwise healthy. A small-sized
air-pessary was worn for a few days, and the recumbent
position preserved; but no further hæmorrhage, nor any
attempt at inversion occurred. The patient began to eat
and sleep well, and in the course of a fortnight returned to
Carnarvonshire, greatly improved in health. The leucor-
rhea soon ceased, by the use of simple remedies, under the
care of Mr. Griffith. During the greater part of the mani-
pulations which were necessary, I was assisted by Dr. Vernon,
at that time the resident obstetric officer at St. Mary's
Hospital.

Since the return of Mrs. J—to her home, I have frequently
heard of her through Mr. Griffith, who describes her as
having lost the appearance of anaemia, and become stout and
healthy. Menstruation has continued regularly, and in
moderate quantity.

The important questions for consideration are, first,
whether the foregoing is a rare and exceptional case of re-
inversion, such as those which have been recorded from time
to time; and, secondly, whether the means by which the
replacement was effected, are likely to prove useful in other
cases, and to supply a principle of treatment in the cure of in-
version. In the former point of view, the case would be of
little or no value, except as an obstetric curiosity, though I be-
lieve there is no other case on record in which an inversion of
such long standing has been reduced; if the latter question
should be answered in the affirmative, it of course becomes
of considerable importance. I proceed to state my reasons
for believing that the use of air or fluid pressure may be
made of general application in the reduction of all chronic
cases of inverted uterus.

An idea has always been entertained that the uterus
which has been inverted for a considerable time is in a hope-
less state of immobility. As soon as the accident has
happened, the powerful contractions of the os and cervix
upon the inverted portion, and the great size of the latter, render its return a matter of great difficulty. This difficulty is further increased, when the involution of the uterus or its return to the unimpregnated size, has taken place. Instead of mere contraction, it is now the structural condition of the organ which opposes its re-inversion. The comparatively bulky body and fundus, of almost cartilaginous hardness, cannot be made to pass through the contracted and rigid os uteri, by any mechanical force short of the rupture or laceration of the parts concerned. This is undoubtedly true, when the inverted uterus is treated as a mechanical displacement, and sought to be remedied by simply mechanical means.

But there is in the uterus a principle of growth, diminution, and alteration of form, under appropriate stimuli, or their withdrawal, surpassing that of any other part of the body, which I believe may be used in the reduction of inversion, and upon which I depended in the foregoing case. The presence of the ovum, excites the enormous development of the gravid uterus. Its return to the unimpregnated condition after the removal of this stimulus by delivery, is equally remarkable. . The organ enlarges in the same manner, but to a smaller extent, under the stimulus of a contained polypus, or of a fibrous tumour within its walls. In cases of ovarian disease, also, that side of the uterus in proximity to the tumour, often enlarges to a considerable extent. In polypus, when the tumour has attained a large size, the virgin os and cervix will become developed to such an extent as to permit the passage of the polypus. Under the influence of continuous irritation, the virgin uterus has even been known to invert itself completely, a much more difficult matter, it might be supposed, than the reposition of the uterus when inverted after parturition. We already make use of the principle referred to, therapeutically, in promoting the growth of the imperfectly developed uterus, when this condition is combined with sterility and amenorrhoea; and in dilating or developing a constricted cervix, in dysmenorrhoea, or in enlarging the cervical canal
when we desire a polypus to pass, by the use of sponge-tents, and other means. In the same way, from *à priori* reasoning, and from what I observed in the preceding case, I believe that the necessary development may be gradually imparted to the os and cervix, in cases of inversion, so as certainly to admit the return of the organ. In the case under consideration, nothing could have been more rigid and unpromising than the os and cervix uteri; but in obedience to the force and stimulus exerted, chiefly by the air-pessary, the fundus and body of the uterus was converted into a wedge or tent, which by constant pressure dilated and developed the narrow ring which had so long fixed the uterus in its inverted position. At the same time, the pressure diminished the size of the body and fundus, and in this way promoted the re-inversion, and the arrest of the hemorrhage. I should entertain little doubt of the possibility of replacing the uterus in any case of inversion, by similar means, and so far from the uterus being considered one of the most difficult and obstinate, it ought to be held as one of the most manageable organs of the body, in any matter relating to growth or form. The plan of treatment by air or fluid pressure must at least deserve a trial in all future cases, when we consider that the alternatives are, death from loss of blood, or the extirpation of the uterus, with its attendant risks.

Besides the employment of the air-pessary in the vagina, to effect reinversion of the uterus in the case described, I have it with good effect for the arrest of hemorrhage, in cases of menorrhagia resisting ordinary remedies, and to keep the uterus replaced in cases of retroversion. I have no doubt the same means would prove serviceable, in cases of flooding from fibrous tumour of the uterus; or from polypus, when the state of the os uteri does not admit of operation. It offers a better kind of tampon than any hitherto used, in cases of flooding during abortion, and in placenta praevia, before the dilatation of the os uteri. I have also used the air-pessary before the time of labour, in a case of high deformity of the pelvis from molilitles ossium, with con-
siderable effect, in separating the contracted bones. The case I refer to, I saw with Dr. S. W. J. Merriman, and before the use of the pessary, the tuberosities of the ischia were so close as scarcely to admit the passage of one finger. After the separation of the bones, delivery was effected by turning. Professor Carl Braun, of Vienna, Kiwisch, and others, have likewise used the air-pessary with great success as a means of inducing premature labour, and of hastening delivery, by promoting the dilatation of the os uteri, in puerperal convulsions, the new method being termed colpeurus.

Postscript.—October 19th, 1858. A few days after the reading of the preceding paper, I received a note from Mr. Griffith, to whom I was indebted for this interesting case, informing me of the pregnancy of Mrs. J—. Since then she has been delivered of a living child under his care. Some amount of flooding occurred after labour, but no tendency to inversion was manifested.
ON

EXCISION OF THE KNEE.

BY

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Received March 8th.—Read March 9th, 1855.

I propose in the following paper to give a brief account of thirteen cases in which I have excised the knee-joint; and to offer a few remarks upon the selection of cases which are best suited to the operation, as well as upon the mode of performing it.

Case 1.—Eliza H—, æt. 20, admitted in October, 1854, a light-complexioned, not very healthy-looking person, had suffered from disease of the left knee more than six years. The synovial membrane was first affected, and subsequently the other structures of the joint. She had been under my care in the hospital three years previously, when the disease was so severe that we feared amputation would be necessary; however, it gradually subsided, and she went to her home to wait the effects of time. The limb had become useless, indeed burthensome; for, though the disease

xli.
in the knee had passed away, the joint was left in so mutilated a state that she could not put the foot to the ground, or move the leg upon the thigh, or even lift it from the bed. Passive movements could be effected in a limited range; but they gave pain. The patella was fixed; the whole extremity was flaccid, though not much smaller than the other. The internal condyle of the femur projected on the inner side of the joint, and could be felt to be knotty from bony deposit upon it. The knee was a little bent; and the leg was slightly rotated outwards, and inclined outwards from the knee, so as to form an obtuse angle with the thigh. There was, therefore, no prospect of the limb being brought into a useful state by any ordinary treatment; and the girl, anxious to avoid amputation, readily acceded to the proposal of excision, which was made to her after consultation between my colleagues and myself.

On October 27th, I made H-shaped incisions in front of the joint, found it necessary to remove the patella; and, having divided the fibrous bands which connected the articular surfaces, sawed off about three quarters of an inch of the condyles of the femur and a thinner slice from the tibia. When the cut surfaces of the bones were placed in contact, I found that the leg was inclined a little outwards. This was rectified by the removal of a thin oblique slice from the femur. Some of the articular arteries required ligature. The limb was bandaged upon a straight splint, extending from the hip to the heel, with a pad under the head of the tibia to raise it; and proper apposition was further secured by broad long lateral splints. No unfavorable symptom followed; not the slightest fever, and scarcely any inflammation at the part. The discharge found its way through the bandages, which I seldom disturbed, not more than once in a fortnight or three weeks. In January, the wound being healed all but one small orifice, and the union of the bones being tolerably firm, the limb was encased in a gum-chalk bandage, and the patient went home. In September, she returned, in consequence of the part being painful and inflamed. There was a sinus ex-
tending from the wound three or four inches up the fore part of the thigh. This I laid open in its whole length; but could discover no diseased or bare bone. The wound soon healed up, and she went home. When she came again, after an interval of a few months, she was able to walk well, and I learn that she can still do so, there being very little lameness.

Case 2.—Edward W, æt. 47, admitted April 19th, 1855; a native of Barbadoes, and a sailor. He broke his right patella transversely by a fall upon the deck, the knee coming in contact with an iron ring, six months previously. He was taken ashore, and treated in the Hospital of Rio Janeiro. In three weeks, as his vessel was about to sail, he got up with a crutch and stick, and went on board. No medical treatment could be there obtained, and he continued to go about with a crutch. The broken fragments became consequently separated by an interval of four inches, and the limb almost useless. When he came to England he sought relief in various places, and, finding none, came into Addenbrooke's Hospital for the purpose of undergoing amputation. The upper fragment of the patella was above the condyles of the femur, and, as well as the neighbouring part of the thigh, was very tender. There appeared to be no connecting medium at all between the fragments, the articular surface of the femur lying immediately beneath the skin. He could bend the knee, but had no power whatever to extend it; and, in walking with his crutch and stick, made scarcely any use of the limb. I found that the left knee-cap also had been broken, and that the fragments were moveable upon one another, though in close contact. This had been caused by a fall upon the slippery deck fourteen years previously. The knee was cupped and bound up for about a fortnight, after which he went about. He had since felt little inconvenience from it, and was not aware that the knee-cap had ever been broken.

I had an apparatus constructed for the purpose of fixing the limb in a straight position, thinking that if he could
thus be enabled to walk about with the knee, the part might gradually acquire more strength. However, the upper fragment was so tender that no efficient pressure could be borne; and, as he was very impatient for more decided treatment, I excised the joint May 4th.

It being clearly desirable to remove the patella in this case, I made a large crucial incision on the front of the joint, and, having reflected the flaps, dissected the fragments away. In doing this I found that there was no tissue at all constituting a direct medium of communication between them, and that the upper one was connected by tough fibrous tissue to the fore part of the femur above the condyles. The joint was in other respects quite sound. I sawed off nearly three quarters of an inch of the articular end of the femur and a thin slice of the upper end of the tibia, and secured the limb in good position by splints as in the former case.

No unfavorable symptoms followed. Suppuration took place; and, after a time, the discharge gradually diminished. On June 19th, the wound was nearly healed, and there was pretty firm union between the bones. On September 21st, the wound was soundly healed, and the bones were firmly united. He could move the limb about freely, and bear some weight upon it. He left the hospital on this day, and I have not seen him since; but learn from friends who saw him in the ensuing autumn that he could walk very well.\[1\]

Case 3.—William C—, set. 12, a pale, but not unhealthy lad, was admitted June 1st, 1855, with the left knee bent to a right angle. Indented cicatrizes of sinuses, which had evidently extended deeply, told of former serious disease; but all acute symptoms had long passed away, and there was no swelling or pain. Still the joint had been destroyed, no movement could be effected, and the limb was quite useless. I tried extension under the influence of chloroform, found that the joint yielded without much difficulty, and, having straightened it, fixed it upon a splint.

\[1\] I saw him at Yarmouth last August. The part was quite firm and sound. He told me that he "could walk twenty miles like flying."
After it had been kept thus for a few weeks the limb was encased in gum-chalk bandage, and the lad allowed to bear upon it a little. The progress was not, however, satisfactory. The joint gained no strength; no movement could be effected in it; there was some swelling and tenderness, and the contraction began to recur. I, therefore, performed excision, August 31st, 1855. The external incisions were crucial as in the preceding case. The patella was firmly ankylosed to the femur, and was accordingly left. The articular surfaces of the tibia and femur were devoid of cartilage, uneven, and united by firm fibrous tissue, which was partly divided by the knife and partly torn through in bending the joint for the purpose of using the saw. A thin slice was removed from each bone, and the limb placed in splints; but not quite so straight as in the former cases. The operation was not followed by any febrile disturbance or other bad symptom. Suppuration took place, and subsided as the healing of the wound went on. The latter process was completed in little more than a month; and in two months, there being firm union between the bones, a gum-chalk bandage was applied, and the lad went home. Three months afterwards the bandage was left off, and he could walk very fairly. In January of this year I found him running about with other boys. The limb was quite strong, as strong as the other, and had grown in due proportion to it. He is, of course, somewhat lame; and I think the lameness is rather increased by the slight angle at which the bones have united. It would have been better if they had been placed, as in the other cases, quite straight.

Case 4.—John C—, aged 20, spare, sallowish, unhealthy, but without febrile symptoms, admitted July 15th, 1856, on account of severe disease of the left knee of three years' duration. There was great swelling of the joint, with starting pains at night, and wasting of the thigh and leg. He could not raise the foot from the bed, or flex or extend the knee. I excised the joint July 18th, making crucial incisions, and removing the patella with the articular
surfaces of the tibia and femur. The cartilages were partially destroyed, and the remaining portions of them were easily separable from the bones; the latter were superficially ulcerated, but appeared healthy where cut through. The synovial membrane was much thickened, pulpy, of grayish colour, with white spots in it. I removed as much of it as I could. Two or three small vessels were tied; the edges of the skin were united by a few sutures, and the limb was placed in splints as usual. The bones were not brought into such satisfactory position as in the former cases, the femur projecting rather too much in front.

There was some pain and a little fever during the first two days; not enough, however, to require any treatment; and it subsided when suppuration took place. The splints were changed on the 22d, when the discharge was free. After this the case went on quietly, without much inflammation or constitutional disturbance, and I hoped union between the bones was taking place. Too much swelling, however, continued; suppuration was maintained from several orifices; every now and then there was an accession of inflammation and suppuration, and the health began to fail. Amputation was, therefore, proposed in the early part of October, but not assented to till November 4th, when I removed the limb a little below the middle of the thigh. He recovered quickly, and left the hospital in December.

On dissection, we found that some portions of the thickened and diseased synovial membrane remained around the extremities of the bones. Indeed, the sinuses in each direction ran to these; and it appeared that its presence had been the cause of the continuance of the suppuration and of the unfavorable progress. The edges of the bones were united by tough fibrous structure; at one part only was there any ossific formation. The opposed surfaces of the bones were covered by softish granulation-like structure, which filled up the small interval between them; the middle part of that of the tibia had undergone slight absorption and rarefaction of its cancellous texture. At one point near
to the diseased synovial membrane the edge of the femur was bare and rough.

We did not from the first regard this to be a favorable case for excision, on account of the extent of the synovial disease and the indifferent state of the patient's health; but we were induced to resort to it in consequence of the unwillingness of the patient to submit to amputation, and I hoped that, if the joint were destroyed and the bones became united, any remaining portions of the synovial membrane would fall into a quiescent state and be ultimately absorbed.

Case 5.—Thomas H—, æt. 29, a thin, pale, unhealthy-looking man, was admitted May 21st, 1856, with disease of the left knee of three years' duration. It had commenced, apparently, in the synovial membrane, and extended to the cartilages and bones. The limb was wasted, and he could not lift it from the bed; the knee was swollen, painful, and not likely to be restored to a useful condition by treatment. Excision was determined upon soon after his admission; but deferred in consequence of the indifferent state of his health. In the meantime an abscess, originating in the joint, burst on the outside of the knee, confirming our suspicion that ulceration had extended into the bones. A foul ulcer resulted, which subsequently assumed a healthier aspect. As the man was anxious for the removal of the joint, but unwilling to submit to amputation of the limb, and as his health had somewhat improved, I performed excision on July 18th. The synovial membrane was thickened, the cartilages in part destroyed, and the ulceration had extended into the bones. There was a deep ulcer in the tibia; but the saw had passed beneath it through healthy bone. No unfavorable symptom followed. Suppuration took place, and the splints were changed on the 22d. Subsequently it was necessary to make incisions on the inner side of the thigh and leg, to give vent to matter. These were followed by marked improvement in the health and condition of the limb. The wound healed, and the
bones became firmly united, so that he was able to leave the hospital in the early part of October. He returned in a month to leave his crutches, being able to walk very well without them. I learn that, in November, 1857, he was seized with some internal complaint, and died in December. The attack had no relation to the state of the limb, which was quite satisfactory.

**Case 6.**—Harriet S—, aged 23, a delicate person, with quick, feeble pulse, was admitted in March, 1857, with considerable swelling and tenderness of the right knee. She was unable to move the joint at all, or to lift the limb from the bed. The thigh and leg were shrunken; the ligaments were relaxed, permitting lateral movement of the tibia upon the femur. The affection was of seven years' duration, and an operation had been recommended eight months previously, when she was in the hospital. At that time the pain was very severe, keeping her awake at night. Since then the severity of the pain had been mitigated, but the other symptoms remained unaltered. She left the decision between excision and amputation with us. Accordingly it was determined that I should commence the former operation, and should the synovial disease prove too extensive for its removal, that amputation should be performed.

March 27th.—I found a good deal of pus in the joint, mingled with flakes of lymph; and this constituted a considerable part of the swelling. There were sinuses about the ligamentum patellæ, and on the inner side of the head of the tibia. The articular surface of the head of the tibia was devoid of cartilage, and rough; and the hinder parts of the condyles of the femur were in a similar condition. The synovial membrane was not so much thickened as had been expected. Finding that I could dissect away the greater part of it, I did so, and sawed off the articular ends of the tibia and femur. Two vessels required ligature. Some bleeding took place after the patient was in bed, but subsided spontaneously. No unfavorable symptom followed,
and not much suppuration. On April 23d, when the
bandages were removed for the first time, the wound was
quite healed on the inner side, and soft union had taken
place between the bones. From this period, however, the
progress was not favorable. Discharge continued rather
profuse from the inner side, and it was occasionally mingled
with blood. I was anxious to get her from her bed, in con-
sequence of the delicate state of her health, and perhaps
hurried a little too much in this, for when the limb was
hung down she experienced pain and a pricking in the part,
and some bleeding took place. I was suspicious, therefore,
of some ulceration of the bones; but as moderately firm
union took place I hoped the disease would subside. In the
beginning of September she returned to her home. In the
middle of December I went to see her, as it was proposed to
remove the limb. I found both her health and the state of the
limb better than when she left the hospital. There was little
or no swelling; firm union had taken place between the bones
on the inner side, so that she could move the limb about
freely, though she could not bear upon it; and the inner
half of the wound was soundly healed. There were, how-
ever, sinuses on the outer side, which discharged matter and
communicated with diseased bone there. I advised further
delay, thinking that there was still hope of a useful limb
being preserved. No decided change taking place, she
wished for the removal of the limb; and amputation was
accordingly performed, January 22d, 1858, by Mr. Harris, of
Mildenhall, who had been in attendance upon the patient
since she left the hospital, and who informs me that she has
recovered from the operation.

We found very firm bony union of the inner halves of
the cut surfaces of the tibia and femur, so firm that it was
difficult to break it. The outer halves were separated by a
narrow interval which was occupied by soft lymph and pus.
There was an ulcerated hole, half an inch deep, in the outer
condyle, and the immediately adjacent cancellated tissue was
devoid of medullary membrane, and infiltrated with pus.
The margins of the cut surfaces of the bones were united by
strong fibrous tissue, and were quite sound. There were no discoverable remnants of diseased synovial membrane.

I had made up my mind, if the patient had been willing to submit to such a procedure, to lay open the sinuses or some of them, to explore the condition of the bones, and remove the diseased parts if possible. It is not improbable that this would have been successful; at any rate, where such firm union between the bones has taken place, I should be unwilling to give up the hope of saving the limb.

Case 7.—Charles L—, st. 10, a sickly lad, with cough, flushed face, white tongue, and indifferent appetite; had for several months been under treatment, as in- and out-patient, on account of synovial disease of the knee, which was of long duration. There was great swelling and indistinct fluctuation, not much pain; the limb was useless. The indifferent state of the health appeared to be dependent upon the local disease. I performed excision April 24th, 1857. The disease proved to be entirely synovial. That membrane was greatly thickened and altered in structure. There was some purulent fluid in the joint, and abscesses in the tissues external to it in contact with and originating in the thickened synovial membrane. One large abscess extended through the ham into the calf. The cartilages and bones were unaltered. I removed a thin layer from the articular surfaces, and carefully cut away all the diseased synovial membrane that I could. When this had been done the flexor muscles were so contracted that I could not bring the bones into proper position till an additional thin slice was removed from the lower end of the femur. Several vessels required ligature.

At first he went on well, and his health rather improved. But, after a time, abscesses formed in the middle of the leg, and discharged a good deal. The wound also discharged freely, was covered with large flabby granulations, and showed little disposition to heal. The lad's health began again to fail; his appetite was indifferent, and diarrhoea set
in. So, on July 5th, I removed the limb in the middle of the thigh, and he soon got well.

The condition of the part was not so bad as I had expected. There was no ulceration or necrosis of the bony surfaces; on the contrary they were united with considerable strength by tough areolar tissue, in which, doubtless, a firmer medium would soon have been developed. There was suppuration at some points around the ends of the bones, excited probably by remaining fragments of the synovial membrane; and there were large sinusae and cavities in the calf, containing thick creamy pus; these did not all extend up to the knee, or communicate with one another.

Case 8.—Amy E—, æt. 5, had, for three or four weeks, been attending as out-patient, on account of some swelling of the left knee. There was a small orifice of a sinus near the edge of the patella; but it was not certain that this communicated with the joint. The child was in good health; she suffered very little from the disease; and mild treatment only was adopted. On May 9th, 1857, she was brought to the hospital in a state of high fever, looking very ill, with great swelling of the knee, which was caused partly by edema of the subcutaneous tissues, and partly by effusion into the joint. The sudden aggravation of the symptoms was attributed to cold caught a few days previously. She was put to bed and a poultice applied. She passed a miserable, sleepless night; the limb jumping almost incessantly. Next morning she looked worse; pulse very quick; no appetite. The case was urgent. It was evident that acute suppuration had set in in the joint, and that unless relief were afforded the child would soon die. Indeed, so severe was the constitutional disturbance, that it was to be feared the termination would be unfavorable whatever measures were taken. I thought amputation and excision afforded about an equal prospect of saving life; and as by the latter the limb would also be saved, I determined to adopt it, and at once performed the operation, cutting into
the suppurated joint, removing the slightly thickened and inflamed synovial membrane, and sawing off a thin slice from the articular ends of the bones, the cartilages of which were not much altered. The child slept soundly all that afternoon and night, and was more comfortable next morning. In the evening she was again more feverish, and moaning with pain. There was dirty bloody discharge from the wound. I removed the splints and bandages, and applied a poultice all over the part. Four days after this healthy suppuration was established, and the wound looked well; but the child was feverish, with dry hot skin, red tongue, glazed lips, and patch of flush on the cheeks; and she refused food. Subsequently there was some improvement. Healthy suppuration and granulation were established over the whole extent of the wound, and cicatrization commenced at the edges; in short the wound was doing well. But the child did not regain a healthy aspect in a corresponding degree. Though her appetite improved a little, the lips remained dry, the tongue was aphthous, and the face was puffy. On May 24th there was pain and tenderness of the right ankle: this was relieved by a few leeches and fomentation, but did not get well. There was no cough or diarrhœa. In spite of such nutritious diet and remedies as we could administer, the child made no progress; on the contrary, she became more listless and drowsy, and died June 1st.

Case 9.—William K—, set. 25, a moderately healthy man, with firm swelling of the right knee. The joint was nearly straight, and admitted of but little movement, during which the bones could, now and then, be felt to grate upon one another; the integuments natural; the thigh rather wasted; not much pain, but inability to bear any weight upon the limb. The disease was attributed to a kick from a horse three years previously; it had progressed steadily though slowly in spite of treatment, in and out of the hospital, perseveringly conducted at several times, and for considerable periods. He was, therefore, anxious for more decided treatment, and willingly embraced the offer of excision.
The operation was performed May 15th, 1857. The articular surfaces were, to a considerable extent, devoid of cartilage and rough; and a piece of loose bone was lodged in a cavity larger than a nutshell in the inner part of the head of the tibia. An angular portion, including this cavity, was sawn out of the tibia after the articular surfaces had been removed. The synovial membrane was thickened and indurated, and I dissected away as much of it as I could. Several vessels required ligature.

No unfavorable symptoms followed. On July 8th the wound had nearly healed, and there was union between the bones. There remained, however, a thickened indurated state of the tissues around; and small abscesses from time to time formed, and burst or were opened, which retarded the progress of the case. In September the swelling had nearly subsided, though small orifices of sinuses remained, which appeared not to run deeply, and which did not discharge much. He could get about with crutches, and went home. On December 29th he could walk very well without crutch or stick. On March 6th he could walk firmly, and with very little lameness, and was in good health, though the sinuses were not quite closed.

Case 10.—Samuel B—, aet. 35, a healthy man, but of irritable temperament, admitted May, 1857, on account of swelling of the right knee, with some crepitus perceptible during movement. The joint was stiff, weak, and ached, but the disease was not severe, so that we were, at first, unwilling to concede to his request that amputation or excision should be performed. However, considering that he had been kept from his work for three years, and, during that time, had been subjected to a variety of treatment without benefit, and that, whatever might be the result of palliative measures adopted while he remained in the hospital, the disease would again make progress when he left, and would induce him again to press for the removal of the limb; considering it also to be a very favorable case for excision, we yielded to his solicitations, and I performed the operation
on May 15th. The synovial membrane was thickened, and firm white growths, many of them polyposis, covered its interior and hung into the joint; some of them were of large size. The cartilages were generally thinner than natural; in some places they were quite removed, and in others they were perforated by small smooth circular ulcers. The bones were healthy. I removed all, or nearly all, the diseased synovial membrane, as well as the ends of the bones.

The case did not go on well, owing apparently in great measure to the irritable nature of the patient’s temperament. He was restless after the operation, and complained of much pain at the part, lost appetite, could not sleep, and began to waste and look haggard, so that in the beginning of June I feared it would be necessary to amputate the limb. However, he gained some relief after the formation and opening of abscesses on the inner and outer side of the lower part of the thigh. The wound nearly healed, and union began between the bones. Every now and then, however, a recurrence of pain took place, the severity of which was not explained by anything that we could discover in the part. Some gatherings, it is true, formed and burst or were opened; but these were not large, and did not discharge much. Occasionally, also, he had attacks of diarrhoea, which prostrated him a good deal, and made me fear the result. Accordingly, although the limb was in an improving state, and there was nothing apparent in its condition to forbid the hope of its being a useful member, yet as he remained so weak, and as another attack of diarrhoea was threatening, I amputated on August 29th. After this he had severe diarrhoea and was very low, the bowels continuing irritable for a month. However, at the expiration of that time, as the stump was nearly healed, he went home, and returned in a month quite well.

Examining the part after amputation, we found the cut ends of the bones covered with granulation structure, which formed a soft uniting medium between them. There was slight superficial ulceration at one or two points, with sinuses running from them; but no disease, either of the
bones or soft parts, which appeared sufficient to have prevented recovery.

Case 11.—Elizabeth M—, æt. 18, delicate, puffy, not very healthy-looking person, with great swelling, pain, and tenderness of the left knee. The swelling extended between two and three inches above the joint. No movement possible without the assistance of the hand, and then very limited; and she could not raise the heel from the bed without the help of the hand. The affection was of seven years' duration and gradually progressing; the more severe symptoms lately superadded had confined her to her bed and made her weak. Pulse quick and feeble; appetite good.

I excised the joint July 24th, 1857. The synovial membrane was thickened; but the swelling was in great measure due to infiltration of the surrounding tissues. The articular surfaces were devoid of cartilage in great part of their extent, and were more or less deeply ulcerated in several places. One ulcer on the fore part of the femur was sawn out after the removal of the ends of the bones. The edges of the wound were stitched together more closely than in the former instances, and a fold of rag was left across the hinder part, projecting on either side to afford a drain for discharges. I do not think this was of any service.

There was very little constitutional disturbance after the operation; but an unusual amount of discharge, requiring poultices and frequent removal of the bandages. The limb was kept upon the back splint for two months, when, there being moderately firm union between the bones, and her health being but indifferent, she was allowed to get up and move about with crutches. The swelling then diminished a little, the unifying medium acquired strength, so that she was able to bear upon the limb, and her health improved. Still the discharge was profuse, and flowed from deep and extensive sinuses, traversing more particularly the lower third of the fore part of the thigh. Some of them were connected with bare rough bone at the fore and inner side of the adjacent edges of the tibia and femur. Accordingly,
on January 29th, 1858, I made free incisions, opening the various sinuses in nearly their whole length; and exposing the ulcers upon the bones so as to enable me to cut them out with saw and bone-pliers. This was done, I believe, effectually, and strips of lint were inserted to prevent the union of the integuments. The discharges, at first foul, soon improved; granulations sprang up and cicatrization began. The uniting medium was not interfered with by this second operation, and the patient is now (March 6th) able to bear the weight of her body upon the limb, and moves about the ward with crutches. The discharge is very much diminished. There are no evidences of deep or extensive sinuses remaining, and there is every reason to hope that a useful limb will be preserved.¹

The great drawback in this case was the diseased and infiltrated condition of the tissues around the joint, which led to such extensive and protracted suppuration, necessitating the second operation, and probably inducing also the ulceration in the edges of the bones.

Case 12.—Sarah D—, et. 21, a healthy person, with firm swelling of the left knee, extending over the lower fourth of the femur and the upper two inches of the tibia. Movements of the joint limited, and pressure upon the patella caused pain. Disease was excited by a kick seven years previously, and had continued slowly increasing ever since. Two years after the accident there was an accession of acute inflammation, attended with severe starting pains, which kept her awake at night; and abscesses then formed, burst, and healed—after which she was easier. This left the limb in an impaired state. Still she was able to walk upon it. Latterly, however, the lameness had increased, was attended with more pain, and interfered so much with her comfort and usefulness that she came to the hospital for the purpose of having the joint excised. The operation was performed October 25th, 1857. I could not with certainty distinguish the thickened and diseased synovial membrane

¹ She writes (August, 1858) that the limb is healed nicely, and that she can walk well with the help of a stick.
from the surrounding tissue, which was infiltrated, indurated, and adherent to it. I removed, however, the chief part of it, with the ends of the bones and the patella. The cartilage had been ulcerated away from the latter and from the fore part of the tibia and femur; but the bones were very little involved. The cut surfaces of the bones having been placed in apposition, the edges of the integument were closely united by sutures, and the splints applied as usual. Union by first intention took place through the whole extent of the wound. After six weeks the splints were discontinued, and she was allowed to get up; and after ten weeks, as she could move the limb about freely and bear some weight upon it, she was discharged.1

Case 13.—Henry S—, æt. 13, a pale, rather strumous lad, with disease of right knee of a year’s duration. It was swollen, tender, and slightly bent. He could not extend it, or lift the limb from the bed, or bear any weight upon it; but did not suffer much pain. Judging that a useful joint was not likely to be preserved, I excised it January 1st, 1858. The cartilages were in several places ulcerated, and their connexion with the bones was extensively loosened; and on the inner articular surfaces both of the femur and tibia the ulceration had extended into the bones. A thin slice of each bone only was removed, that from the tibia being only just thick enough to include the whole depth of the ulcerated tract. The synovial membrane was thickened, pulpy, and of gray colour; it was all, or nearly all, removed. The edges of the skin were closely adjusted by sutures. No constitutional disturbance followed, and very little suppuration. Indeed, the wound healed up almost entirely by first intention. On March 5th, there being pretty firm union between the bones, a gum-elastic bandage was applied, and the lad got up.2

1 She came to the hospital in July, 1858. The part was quite firm and sound; and she could walk well.

2 He came to the hospital October 23d, 1858, able to walk well without crutch or stick. Says that he has walked six miles this morning—the part is quite sound.
Of the thirteen cases just related, one died; in four amputation was necessary; these all finally recovered; and in the remaining eight a useful limb was preserved, or there is every prospect of that result.

The operation of excision of the knee-joint appears, from the narrative of these cases, not to be attended with much danger to life. This may be inferred, not merely from the fact that death followed in only one case, and in that case, which was a peculiar one, was attributable rather to the shock of the severe previous disease than to the effects of the operation, but still more from the fact that in no one of the cases was the operation productive of much constitutional disturbance. In the greater number there was scarcely any constitutional disturbance to be observed. Scarcely any acceleration of the pulse or other febrile symptom is noted to have occurred in the few days succeeding the operation; and none of the alarming sequences, such as erysipelas, inflammation of internal organs, suppuration in distant parts, or diffuse suppuration near the wound, which are dreaded as the probable effects of the more severe operations, were even threatened in any one of these cases.

This is to be attributed, doubtless, in part to the circumstances that none of the patients were in either a febrile or hectic state at the time of the operation, that in few was the disease in a very acute form, and that the greater number of the patients belonged to the class of agricultural labourers. The latter is, I think, a very important point to take into account in forming an estimate of the value of any particular mode of treatment, operative or other. The lower diet and the less excitable temperament of the agricultural labourer, as compared with the artisan in our larger towns, imparts a more chronic character to his maladies, and renders him a better subject for operations, which is probably a chief cause of the difference in the result between operations performed in the provincial and those in the metropolitan hospitals.¹

¹ It was recently stated, in the 'Medical Times and Gazette,' that one half of the adults operated on for the stone in London die; and the
EXCISION OF THE KNEE.

The limb in each case after recovery has been a very strong and useful member. The femur and tibia became firmly united into one bone, and the slight shortening caused by the loss of their articular portions is an advantage in walking, rendering the gait of the patient easier than when a mere ankylosis of the knee exists. There is no liability to recurrence of disease; and I think patients who have recovered from excision of the knee must be very thankful that they have not undergone amputation. My own prejudices, like those of many other surgeons, were decidedly against this operation; and though I had observed the good effects of the excision of joints in the upper extremity, I conceived that the instances in which the leg and thigh-bones would unite so as to form a useful limb, after the removal of their articular ends, must be exceptional. Moreover, I feared the dangers of interfering with so large a joint. I was first induced to perform the operation by the representations of my cousin, Mr. F. Humphry, of Brighton, who had seen some cases after recovery; and I was encouraged to repeat the procedure by the good results which followed in Cases 1, 2, and 3.

The success of such an operation must be very greatly dependent upon the judicious selection of cases for it. Upon this point I will say a few words.

The results of excision of the knee are likely to be satisfactory in proportion as the disease for which it is performed is slight, and not in an acute form. Thus, in Case 2, where there was no disease at all, the recovery was quick and complete; and Cases 1, 3, and 12, in which the more severe stages of the disease had subsided, leaving the joint maimed and useless, also did remarkably well. The first of mortality after amputation in the thigh is believed, by a writer in the 'Association Medical Journal,' Jan. 30th, 1858, to amount to 50 per cent. or more. Whereas in the Norfolk and Norwich Hospital the number of adults who died after lithotomy was about 1 in 6½ (Crosse on 'Uriney Calculus,' p. 162), and of 29 adults operated on by myself in Addenbrooke's Hospital, three only have died; of 92 cases of amputation in the thigh, performed in the latter hospital by Mr. Lestourgeon, Mr. Hammond, and myself, 17 only have terminated fatally.
these (Case 2) is quite exceptional; it merely proves that a healthy knee-joint, should occasion require it, may be excised with good result; and, with this remark, it may be dismissed from our present consideration. The other cases (1, 3, 12), however, are members of a large and important class; for the instances in which the knee has been so maimed by disease that it is unable to perform its movements and bear the weight of the body are very numerous; they form a considerable proportion of the cripples who are obliged to resort to crutches, or who limp along with a stick; and if it is found that, under such circumstances, excision of the partially destroyed joint can be performed without much danger to life, and with a good prospect of the restoration of usefulness to the limb, an additional class of cases will have been brought under the beneficial influences of surgery; and we shall have an additional reason to be sparing of amputation in the various stages of disease of the joint. Unless the constitutional symptoms demand the removal of the limb, we shall be more than ever disposed to wait the progress of events, and that subsidence of the disease which usually takes place after a time, knowing that, even if the joint have been spoiled, we have still the prospect of preserving a useful limb to our patient.

My own experience of the practice of extension and fixing the limb in a straight position, which is recommended in some of these cases where the knee is much contracted, is too limited to form the ground of a decided opinion; but so far as it goes it leads me unquestionably to give the preference to excision of the joint, particularly when the patella has become fixed; and it will have been observed that in Case 3 the latter procedure was adopted with success after the former had failed.

Let us turn from the cases in which the disease has subsided to those in which it is still progressing. In by far the greater number of instances of affection of the knee-joint, the disease commences in the synovial membrane. In one class of cases it is, in a comparative degree, confined to that membrane, and induces great thickening of it, with a
EXCISION OF THE KNEE.

variety of structural alterations, such as the transformation of
it into a grayish, more or less opaque, substance, with, per-
haps, the deposit in it of numerous white spots, like tu-
bercles, which have a tendency to soften, suppurate, and burst.
The cartilages and bones do, it is true, generally become, to
a certain extent, involved; but I have known the disease to
endure great length of time, to cause enormous thickening of
the synovial membrane, and to necessitate amputation,
without these structures being in the least degree altered.
In such cases the results of excision are not likely to be very favorable. First, because the object especially con-
templated, and most easily attained, in that operation, is the
removal of the articular ends of the bones, with their carti-
lages; some of the morbid synovial membrane, which has
extended beyond its proper limits, is almost sure to be left,
and is likely to remain as a source of suppuration and irri-
tation, inducing, it may be, ulceration of the bones, the
continuance of sinuses, and other evils. Secondly, because
the subjects of this form of disease are generally sickly and
of strumous temperament, the reparative processes are con-
ducted languidly in them, and the constitution is likely to
give way under the protracted discharge and irritation
attendant on a wound so large, and involving so much
exposure of bone. Three of the cases (4, 6, and 7) in which
amputation was required belonged to this class. Never-
thless, although these cases are among the least favorable
for excision, I would not altogether exclude them from the
chances it offers; because the remnants of the diseased synovial
membrane may break up and disappear in the discharge, or
they may shrink up and be absorbed after the bones have
become united together; and because amputation may, at a
subsequent period, be resorted to, if the wound does not go
on favorably, or if the health begins to fail.

In a second class of cases, in which the cartilages and
bones are invaded at an earlier period and to a greater
extent, which are characterised by greater amount of pain in
proportion to the swelling, and in which the constitution
and reparative powers of the patients are more vigorous, the
prospects afforded by excision are more promising; and I think that in most of these cases the operation should be performed as soon as it appears that the articular surfaces are so far involved as to preclude the hope of the restoration of a useful joint. It must rest with the discretion of the surgeon to decide in each particular case when this point has arrived. My own impression is that a really serviceable joint is very rarely preserved when the duration, the severity, and the starting character of the pain, the wasting of the limb, and the inability of the patient to lift the heel from the bed, indicate that the cartilages have been ulcerated, and more or less detached from the bone, still less where the bursting of the joint indicates, as it commonly does, that the ulceration has extended into the bones; and I am of opinion that in the greater number of such cases excision should be practised. There is this difference to be borne in mind between considering the questions of excision and amputation: that in the former we are to be guided chiefly by the condition of the joint and the probability of its restoration to usefulness, whereas in the latter we should be influenced rather by the impression the disease is making upon the constitution, and the probability of its leading to a fatal termination unless the limb be removed.

It has just been hinted that the eruption of matter from a joint usually indicates that ulceration has extended through the cartilages into the bones. The extent to which the latter are involved may often be estimated with tolerable accuracy, by the number and locality of the sinuses which have resulted from a succession of such eruptions, as well as by the enlargement of the articular ends or adjacent parts of the shafts of the bones. Where either or both of these signs render it probable that ulceration has extended far into the osseous structure, it need scarcely be said that excision is not to be recommended.

I conclude, then, that of the cases which are of common occurrence, excision of the knee may be performed with best prospects of success; first, in those where the acute stages of inflammation have been passed through, and the joint is
left crippled, contracted, and useless; and, secondly, in those where the disease, originating in the synovial membrane, has invaded the cartilages and bones, and has caused such alteration in all or any of these structures as to preclude the hope of the preservation of a useful joint. Where there is great thickening of the synovial membrane, with the probability of tuberculous or scrofulous transformations of its substance, or where there is much disease of the bones, the results of the operation are less likely to be favorable. There are other cases of less frequent occurrence, in which excision may be recommended, such as the more severe varieties of chronic rheumatic arthritis and knock-knee in the adult, unreduced dislocation,\(^1\) and badly united fracture of the patella (as in Case 2), &c. In some cases of wound into the knee-joint, more particularly where there is compound fracture of the patella, or injury of the articular surfaces of the tibia and femur, I should be disposed to recommend excision of the joint, without waiting for the onset of inflammation, which, in such instances, is so often destructive to the limb or the life of the patient.

It should be borne in mind that, as a general rule, the work of recovery is slower and more difficult after excision than after amputation; and that, therefore, when the patient is greatly reduced, or of bad constitution, it is better to remove the limb than to take out the joint.

In performing the operation of excision of the knee, I prefer the crucial to the H-shaped external incision, because it is more easily and quickly made, because it affords an equal facility in the performance of the other stages of the operation, with a less amount of division of skin, and because the edges of the wound are more readily brought again into apposition. I think it better to remove the patella, because, if left, it can serve no good purpose when the tibia and femur have become united together, and it must

\(^1\) I remember an example of unreduced partial dislocation of the tibia outwards, in which amputation of the limb was recommended by Sir Astley Cooper, and Mr. Crosse, of Norwich; it was a good case for excision.
offer some impediment to the escape of purulent and other
collections that may take place between the cut ends of the
bones; moreover, it is very often diseased, and its removal
facilitates the performance of the operation. The ends of
the bones should not be uncovered more than is absolutely
necessary for the purpose of removing the requisite
amount of their articular surfaces. It is better to divide
some of the soft parts with the saw than to dissect them too
closely to the bones. I find the common amputating saw
the most convenient instrument, and divide the bones from
before backwards. In young persons, care should be
taken to make the section through the epiphyses of the
tibia and femur, so that a thin layer of the epiphysis,
with the cartilaginous medium which unites it with the
shaft, is left upon each bone. If this precaution be taken,
there is every reason to believe that the limb will keep
pace in its growth with the opposite member. It is most
probable that, in the instances in which a want of proper
growth has been observed in the limb after recovery from
excision, the sections were made through the shafts, and
the entire epiphyses, with the thin cartilaginous matrix
lying between them and the shafts, were removed. Hence
the shafts of the two bones became directly united
together, and, the provision for elongation at the one end
of each having been taken away, the limb was thenceforth
unable to grow at the same rate as its fellow. It is the
more necessary to be very careful about this point, because
the epiphyses, not increasing in depth in anything like the
same proportion as the shafts during growth, are after
a few years relatively very shallow, especially that of the
upper end of the tibia; and unless the surgeon bear this
fact in mind, he is very likely to make the section through the
shafts (of one or both bones), when he imagines that he is
dividing the epiphyses.\footnote{See my 'Treatise on the Human Skeleton,' page 44.}

It is important that the cut surfaces of the tibia and
femur should be well adapted to each other. If they are
not, interspaces are left at some points, and undue pressure
is made at others; both which are unfavorable to union, and are likely to induce ulceration. Should it be found, therefore, when the limb is placed straight, that the surfaces of the bones do not fall into good relation, owing to the section of one or both having been made a little too obliquely, it is better to remedy the defect by removing an additional slice, cut in such a manner as to secure the object desired. I suspect that the failure in Case 6 was partly due to inattention to this particular; for ulceration was found to have taken place in the narrow interval that was left between the inner halves of the cut surfaces of the tibia and femur, whereas firm union was established between the outer halves, where closer apposition had existed.

Care should be taken to secure the bleeding vessels, as the effusion of much blood into the wound, after the limb has been done up, preysents immediate healing, and leads to foul collections and discharges, which, resting upon the cut surfaces of the bones, retard the healing processes, if they do no further mischief.

In the later cases I was more particular than in the early ones to maintain the edges of the skin in close union by sutures; and they have, on the whole, done better. In the last two cases the wound healed throughout its whole extent by the first intention. No dressings were employed in any of the cases; the plan of leaving the wound quite uncovered, which is usually adopted in our hospital, and which has proved more successful and more comfortable to the patient than any other, was followed in all these cases. The proper position and complete quiescence of the limb was in each instance secured by a back splint, and a long splint on either side. These were changed no oftener than was necessary for cleanliness. In some of the cases they were allowed to remain three weeks or a month without alteration. The splints were not discontinued till the union between the bones was sufficiently firm to enable the patient to lift the limb from the bed by its own muscles.
The constitutional treatment was exceedingly simple. Opiates were given only in Case 10, and sparingly in it. I am not in the habit of giving opiates after operations, unless the pain, irritation, or restlessness is producing a decidedly injurious effect upon the constitution. I think that they do harm by enervating the patient, by constipating the bowels, and by disordering the various functions, and that therefore they should not be given unless there be some distinct indication for their use. The diet was regulated in each case according to the requirements. Stimulants were not often needed, and were therefore seldom given, but such moderately nutritious food as the patients desired and were able to bear.

The operation of excision of the knee is, at present, on its trial. Some surgeons are prejudiced against it, as I confess myself once to have been; some, disappointed by their first cases, are unwilling to repeat the procedure; and we can well understand that those who have had better success, and have enjoyed the gratification of witnessing how strong and valuable a limb may become after the knee has been excised, are likely to form too high an estimate of the value of the operation. The foregoing cases are adduced in the hope of assisting to a fair judgment. They are related briefly; but each was the subject of much consideration and care. The conclusion I form with regard to them is, that I should not have attained an equally good result by any other mode of treatment.
CONTRIBUTIONS

TO THE

ETIOLOGY OF CONTINUED FEVER:

OR AN INVESTIGATION OF VARIOUS CAUSES WHICH
INFLUENCE THE PREVALENCE AND MORTALITY
OF ITS DIFFERENT FORMS.

BY

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Received March 30th.—Read April 27th, 1868.

Among the greatest benefits which medicine has conferred upon the human race are, undoubtedly, those which have reference to the prevention of disease. The truth of the adage, "Prevention is better than cure," is now fully recognised, as shown by the increased attention bestowed upon the subject of hygiene. At the present day, then, I need offer no arguments in proof of the importance of an investigation into the causes influencing the prevalence and mortality of a disease, which, during the last twenty years, has destroyed upwards of 350,000 of the population of England and Wales, and the annual mortality from which, in London alone, averages about 2500.

I purpose, in this essay, not only to investigate the causes which influence the prevalence and mortality of continued
fever, regarded as a single disease, but also to examine these causes in reference to the different forms of fever, and thereby, if possible, to throw some light upon a question which many regard to be still sub judice, viz., the specific identity or non-identity of these different forms. Much has of late years been written on the symptomatology and pathology of the various fevers; but probably the subject will ultimately receive the greatest elucidation from an investigation of their etiology. It would be well, however, if some, who are in the habit of asserting the identity of the different fevers, had, in the first place, studied more carefully their symptoms and morbid appearances. The vagueness upon these points, which is exhibited in their descriptions, neutralizes the value of the facts which they adduce, and invalidates the opinions which they advocate.

I do not pretend that the facts which I am about to bring forward are all of them novel, or sufficient to decide the disputed question. It is hoped, however, that they may be regarded as a contribution in the right direction towards the solution of the problem. Most of them are derived from statistics which I have collected from the records of the London Fever Hospital; and I have endeavoured, as often as possible, to compare the results thus obtained with those arrived at by observers in other quarters.

During the last ten years careful records of the diagnosis of all the cases of fever admitted into this hospital have been kept. Many cases of symptomatic fever are also constantly sent into the hospital; but all these have been excluded from the numerical statements which follow. The fevers have been divided into the four following: typhus, typhoid, relapsing, and febricula. These terms I shall, from their convenience, adopt, without pledging myself to the belief that I regard them all as indicating distinct species of disease. Even the most sceptical on this matter must allow that the first three, at all events, constitute well-marked varieties of fever; and it is obviously no less important to investigate their etiology, whether we consider them as varieties or species. A short definition of what is
ETIOLOGY OF CONTINUED FEVER.

meant by each of these terms may not be here out of place.

1. Typhus.—A disease often commencing more or less suddenly with rigors or chilliness, and attended by frequent, soft pulse; dry, brown tongue, and in most cases by constipation; headache; delirium, often appearing early, and generally low and wandering; contracted pupils; great prostration; in most cases a mulberry rash, showing itself between the fifth and eighth days, and continuing till death or recovery; the disease generally terminating by the fourteenth, and seldom protracted beyond the twenty-first day. No characteristic lesion found after death; but great congestion of all the internal organs.

2. Typhoid.¹—A disease often commencing insidiously, or ushered in with rigors, chilliness, or profuse diarrhoea, and characterised by frequent, but variable, soft pulse; red and fissured tongue, ultimately becoming dry and brown; tympanites and abdominal tenderness; gurgling in the iliac fossae; diarrhoea and frequently melæna; increased splenic

¹ Although I have employed the term “typhoid” throughout this essay, it is not without a deep conviction that it is a most unfortunate one, and that it is one which has been productive of much confusion on the whole subject of fever, among the great body of the profession. At the same time, none of the numerous synonyms appear to me to be free from objection. For example, it would not be desirable to have any name derived from the abdominal lesion, tending, as this would do, to revive, in the minds of many, the exploded doctrines of Broussais. An appropriate distinctive name for the disease remains a desideratum; and, after having devoted much thought and attention to the question, I would venture, with some diffidence, to propose one derived from what I believe to be the cause of the fever. In the course of this essay I shall bring forward what I consider positive proofs that this fever is produced by emanations from decaying organic matter; and I would therefore suggest for it the appellation of “pyrogenic fever”—πυρογενής, from πῦρ (πῦρ, putresco) and γεννᾶω; under which I would include the so-called “gastric fever,” which I believe to be merely a variety of the same affection. (‘Edin. Med. Journ.,’ Oct., 1858.)
dulness; epistaxis; headache; delirium, active, or often absent; dilated pupils; prostration coming on late, and often slight; an eruption of successive crops of elevated rose-coloured papules, appearing from the seventh to the fourteenth day, each crop lasting only for two or three days: the disease often protracted to the thirtieth day or beyond, and sometimes followed by a relapse of all the symptoms, including the eruption. After death, enlargement of the mesenteric glands and ulceration of Peyer's patches.¹

3. *Relapsing fever.*—A disease commencing very abruptly with coldness and rigors, and attended by quick and often incompressible pulse; white tongue; tenderness at epigastrium; vomiting; enlarged liver and spleen; occasionally jaundice; constipation; high-coloured urine; great heat of skin, but no eruption of any sort; severe headache and pains in the back and limbs; restlessness; rarely slight delirium. An abrupt cessation of all these symptoms, with free sweating, between the fourth and seventh days, usually on the fifth. After a complete apyretic interval, during which the patient may get up and walk about, an abrupt relapse on the fourteenth day from the first commencement, running a similar course to the first attack, and terminating on the third day of the relapse. Rarely, sudden syncope and death. After death no specific lesion; but, in most cases, enlargement of the liver and spleen.

4. *Febricula or Ephemerall fever.*—Febrile symptoms, with no eruption, lasting for a few days (one to ten) and subsiding with some critical discharge. It is not contended by many that this is a specific fever. Many of the cases entered under this head have been the result of fatigue, exposure to cold, or intemperance. Others again have been associated

¹ During ten years, no case has been observed at the London Fever Hospital in which there were rose-coloured spots during life without ulceration of Peyer's patches after death. On the other hand, in no case in which there has been a mulberry rash has the intestine been found diseased.
with herpes labialis, or derangement of the stomach and bowels, while not a few have undoubtedly been mild cases of some of the first three forms, in which the symptoms have not been sufficiently characteristic to establish their diagnosis. An accurate discrimination of the real nature of the complaint is, in some of these cases, difficult; and hence the term “febricula” is a convenient one, although the cases composing this class will not require much attention in the following remarks.

A. PREVALENCE OF CONTINUED FEVER.

I. ON THE PREVALENCE OF CONTINUED FEVER IN GREAT BRITAIN AND IRELAND DURING THE PRESENT CENTURY.

Some idea of this may be obtained from Table I, in which I have collected the number of admissions for fever into several of the principal hospitals in the United Kingdom during the last forty-one years. I could have wished that this table had been more complete; but I find that the records of few hospitals have been kept with sufficient accuracy to render them available for the purpose. To supply in some measure this deficiency, I have introduced two columns, showing the number of deaths from fever in England and Wales, and in London, during the last eighteen years, as furnished by the Registrar-General, and multiplied these by ten, which, as will afterwards appear, gives a fair approximation to the total prevalence of the disease. The last column gives the number of fever admissions into the principal hospital at Stockholm during twelve years.

A glance at this table shows that fever is never absent from any of the large towns specified; but that its prevalence varies very greatly in different years. Some authors have endeavoured to demonstrate a periodicity in the occurrence of these epidemics. Among others, Dr. Orr, of Glasgow, thought that they recurred about every ten years.¹ This regular periodicity is hardly borne out by the table—

Table I, showing the prevalence of Fever at different places during the last forty-one years.

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<th>Edinburgh</th>
<th>Glasgow</th>
<th>Aberdeen</th>
<th>Cork Hospital</th>
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</tr>
<tr>
<td>1847</td>
<td>303,200</td>
<td>31,840</td>
<td>1239</td>
<td>3689</td>
<td>5244</td>
<td>683</td>
<td>3875</td>
<td>5693</td>
<td>132</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1848</td>
<td>214,060</td>
<td>35,840</td>
<td>907</td>
<td>4693</td>
<td>1515</td>
<td>1648</td>
<td>2472</td>
<td>1249</td>
<td>104</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1849</td>
<td>179,020</td>
<td>24,820</td>
<td>401</td>
<td>726</td>
<td>570</td>
<td>584</td>
<td>2977</td>
<td>2565</td>
<td>65</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1850</td>
<td>142,960</td>
<td>19,290</td>
<td>361</td>
<td>520</td>
<td>597</td>
<td>255</td>
<td>2096</td>
<td>1756</td>
<td>284</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1851</td>
<td>171,210</td>
<td>21,400</td>
<td>614</td>
<td>959</td>
<td>1385</td>
<td>218</td>
<td>2133</td>
<td>2307</td>
<td>443</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1852</td>
<td>178,450</td>
<td>20,200</td>
<td>561</td>
<td>691</td>
<td>1721</td>
<td>148</td>
<td>2354</td>
<td>1731</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1853</td>
<td>180,130</td>
<td>24,830</td>
<td>787</td>
<td>574</td>
<td>1938</td>
<td>121</td>
<td>1388</td>
<td>1643</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1854</td>
<td>183,320</td>
<td>26,940</td>
<td>714</td>
<td>169</td>
<td>1058</td>
<td>304</td>
<td>2069</td>
<td>1906</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1855</td>
<td>160,320</td>
<td>23,420</td>
<td>622</td>
<td>201</td>
<td>656</td>
<td>345</td>
<td>2204</td>
<td>907</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1856</td>
<td>...</td>
<td>1300</td>
<td>180</td>
<td>591</td>
<td>225</td>
<td>1606</td>
<td>1067</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1857</td>
<td>...</td>
<td>561</td>
<td>126</td>
<td>543</td>
<td>145</td>
<td>...</td>
<td>827</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
</tbody>
</table>

1 The first two columns contain the number of deaths from fever, as reported by the Registrar-General, multiplied by 10.
2 From a paper by Dr. Christie son in the 'Edinburgh Medical Journal.'
Etiology of Continued Fever. 225

before us. Thus, although there have been great epidemics in 1827, 1837, and 1847, yet, in Scotland there was an epidemic in 1843, and in Glasgow in 1853. Again, in conformity with such an opinion, we should expect to have an epidemic at present in Edinburgh; but that city has never, within the memory of its oldest physicians, been so free from fever as for the last four years. Another circumstance exhibited by the Table is, that the epidemics in the various towns have, to a certain extent, been simultaneous. This remark applies especially to the great epidemics of 1817, 1827, 1837, and 1847. A closer scrutiny of the figures shows that the epidemics apparently commenced and reached their acme in Dublin before Glasgow, in Glasgow before Edinburgh, and in Edinburgh before Aberdeen. There is no reason to believe that this circumstance is due to the spread of an epidemic influence travelling from West to East, and, indeed, such an opinion is negatived by the fact, that the greatest epidemic at Stockholm occurred in 1846 before that of Great Britain. On the contrary, it is more probable, as will subsequently appear, that the disease originating in Ireland is propagated to Britain by contagion.

Which of the forms of fever now is it of which these great epidemics are composed? In order to answer this question, I proceed to consider:

_for January, 1858. Previous to 1826, the annual report was made up to December 31st, afterwards to the end of September, so that the number for 1826 represents only nine months. The number for 1857 represents the admissions from January 1st to December 31st._


4 From the Annual Reports of the Infirmary.

5 Communicated by the Registrar of the Hospital. Up to the end of 1831, the Hospital year terminated on January 4th of the following year; afterwards on March 31st of the following year, so that the entry for 1852 includes five quarters. The numbers for the first two years (1817-18) include the admissions into the "House of Industry."

6 Communicated by Dr. McEvers, of Cork.

7 Professor Magnus Huss, 'Statistique du Typhus,' &c., p. 29.

xlii.
II. The annual prevalence of the different forms of Continued Fever in London, as compared with other towns.

Whatever objections may be raised to the returns of the London Fever Hospital, as indicating the total prevalence of fever, the same will not apply to them as a fair test of the relative prevalence of the various forms of fever at different periods.

Table II contains the numbers of each form of fever admitted into the London Fever Hospital during the last ten years.

**Table II.**

<table>
<thead>
<tr>
<th>Years</th>
<th>1848</th>
<th>1849</th>
<th>1850</th>
<th>1851</th>
<th>1852</th>
<th>1853</th>
<th>1854</th>
<th>1855</th>
<th>1856</th>
<th>1857</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Typhus</td>
<td>526</td>
<td>155</td>
<td>130</td>
<td>68</td>
<td>204</td>
<td>406</td>
<td>337</td>
<td>342</td>
<td>1062</td>
<td>274</td>
<td>3506</td>
</tr>
<tr>
<td>Typhoid</td>
<td>152</td>
<td>138</td>
<td>157</td>
<td>324</td>
<td>140</td>
<td>211</td>
<td>228</td>
<td>217</td>
<td>149</td>
<td>214</td>
<td>1820</td>
</tr>
<tr>
<td>Relapsing</td>
<td>13</td>
<td>29</td>
<td>32</td>
<td>226</td>
<td>88</td>
<td>16</td>
<td>5</td>
<td>1</td>
<td>1</td>
<td>44</td>
<td>441</td>
</tr>
<tr>
<td>Febricula</td>
<td>16</td>
<td>79</td>
<td>62</td>
<td>129</td>
<td>132</td>
<td>144</td>
<td>62</td>
<td>89</td>
<td>72</td>
<td>861</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>707</td>
<td>401</td>
<td>614</td>
<td>561</td>
<td>787</td>
<td>714</td>
<td>622</td>
<td>1300</td>
<td>561</td>
<td>6628</td>
<td></td>
</tr>
</tbody>
</table>

With this I would contrast Table III, showing the corresponding admissions into the Glasgow Royal Infirmary during twelve years, for which I am indebted to Dr. McGhie, the Medical Superintendent. It is the more valuable, as I believe the Glasgow Infirmary is the only other institution from which such a return could be obtained.

**Table III.**

<table>
<thead>
<tr>
<th>Years</th>
<th>1846</th>
<th>1847</th>
<th>1848</th>
<th>1849</th>
<th>1850</th>
<th>1851</th>
<th>1852</th>
<th>1853</th>
<th>1854</th>
<th>1855</th>
<th>1856</th>
<th>1857</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Typhus</td>
<td>509</td>
<td>2399</td>
<td>980</td>
<td>342</td>
<td>382</td>
<td>919</td>
<td>1293</td>
<td>1551</td>
<td>760</td>
<td>385</td>
<td>385</td>
<td>314</td>
<td>10,210</td>
</tr>
<tr>
<td>Typhoid</td>
<td>?</td>
<td>127</td>
<td>7</td>
<td>?</td>
<td>?</td>
<td>44</td>
<td>134</td>
<td>45</td>
<td>92</td>
<td>145</td>
<td>163</td>
<td>157</td>
<td>914</td>
</tr>
<tr>
<td>Relapsing</td>
<td>777</td>
<td>2333</td>
<td>513</td>
<td>168</td>
<td>174</td>
<td>258</td>
<td>192</td>
<td>72</td>
<td>68</td>
<td>42</td>
<td>458</td>
<td>1425</td>
<td></td>
</tr>
<tr>
<td>Febricula</td>
<td>?</td>
<td>385</td>
<td>15</td>
<td>60</td>
<td>41</td>
<td>167</td>
<td>102</td>
<td>270</td>
<td>167</td>
<td>104</td>
<td>43</td>
<td>72</td>
<td>1425</td>
</tr>
<tr>
<td>Total</td>
<td>1565</td>
<td>5244</td>
<td>1515</td>
<td>570</td>
<td>597</td>
<td>1383</td>
<td>1721</td>
<td>1938</td>
<td>1087</td>
<td>656</td>
<td>591</td>
<td>543</td>
<td>17,124</td>
</tr>
</tbody>
</table>

1 About 200 additional cases, admitted during this year, and marked "doubtful," have been omitted. They were probably, for the most part, cases of "fabricula," and mild cases of typhus.
ETIOLOGY OF CONTINUED FEVER.

One of the most remarkable circumstances to be observed in these tables, is the extreme variation in the prevalence of relapsing fever in different years. In 1846, no fewer than 2333 cases were admitted into the Glasgow Infirmary, and in 1851, it constituted the largest number of the admissions into the London Fever Hospital. Since 1851 it has gradually disappeared, and for the last few years, none, except one or two doubtful cases, have been observed either in London, Glasgow, or Edinburgh. There is also a synchronism in the prevalence of this fever at different places. In 1843, it was epidemic in London, Edinburgh, Aberdeen, and Glasgow, and greatly preponderated over all the other forms of fever. Thus in the Glasgow infirmary there were 2871 cases of relapsing to 142 of typhus. In 1847, it was again epidemic at all of these places, but with a preponderance of typhus. A careful study of the history of fever in Great Britain and Ireland, will show that relapsing fever, for upwards of a century, has been almost constantly observed during the great epidemics.

The prevalence of typhus appears, from its entire history, to be also of an epidemic character. Thus, in the London Fever Hospital, the number of cases gradually decreased from 1848 to 1851, in which year there were only 68; after this, the number continued to rise till 1856, when it amounted to 1062. Since then it has almost entirely disappeared. During the last six months (October, 1858), not a single case of spotted typhus has been admitted; and I believe this fact accords with the experience of medical

1 'Report of London Fever Hospital,' for 1843, p. 15.
2 Cormack on the Epidemic Fever of 1843.
men all over London. Again, in Edinburgh, while typhus constituted the great proportion of the 8881 cases admitted into the Infirmary in 1847 and 1848,\(^1\) during the year 1857 Dr. W. T. Gairdner writes me, only 56 cases were admitted, and in January of the present year, I am informed by Dr. Haldane, that the building did not contain a single male fever-patient.\(^2\) In Glasgow, also, the diminution of typhus has been obvious although not so great. Here, as in Ireland, it appears to be constantly lurking, every now and then, apparently under the influence of external circumstances, breaking out into an epidemic. It is also to be noted, that relapsing and typhus fevers occur as epidemics simultaneously.\(^3\) The relative prevalence, however, of the two varies greatly at different times; and we may have epidemics of typhus without the coexistence of relapsing, but not, as far as I am aware, of relapsing without typhus. Thus, in the Glasgow Infirmary, the number of cases of relapsing fever in 1848 was 2871, of typhus only 142; and in 1853 the proportion was reversed, or there were of typhus 1551 cases, and of relapsing 72. Again, in the London Fever Hospital, the greatest number of relapsing cases in one year corresponded with the smallest number of typhus, and the largest number of typhus with a complete absence of relapsing. Another circumstance worth observing, is, that in an epidemic of both forms, relapsing seems to constitute the greater proportion of the cases at the commencement, and typhus towards the close. This is obvious by referring to the numbers of each in the Glasgow epidemic of 1846-49; and in the London Fever Hospital, the numbers of relapsing and typhus, for three successive years, were, respectively, 256 and 68; 88 and 204; 16 and 408. A similar observation was made in Dublin in 1826.

\(^1\) Christison, loc. cit., p. 592.

\(^2\) Since writing the above, I have myself had an opportunity of visiting the Edinburgh Infirmary (May, 1858). There were several cases of typhoid fever in the building; not a single case of typhus.

\(^3\) This circumstance was also noted in the epidemic of fever in Silesia, in 1848. See 'Brit. and For. Med.-Chir. Rev.,' vol. viii, p. 86, 1851.
Etiology of Continued Fever.

Typhoid fever, as regards its annual prevalence in London, presents a remarkable antithesis to the two forms already considered. The number of cases from year to year has varied no more than is the case with most diseases, or than might be attributed to the influence of local causes. In Glasgow, it will be seen that it formed no constituent of the great epidemic of 1847, there being only 127 cases out of the 244 fever-patients treated in the infirmary. In that city also, as in London, the numbers of late years have varied slightly as compared with those of the other two fevers. Before typhoid attracted much notice in this country, there can be little doubt that many cases of it were overlooked. It has been asserted that typhoid was unknown in Glasgow previous to 1856; but Dr. McGhie has shown that, for many years before, cases were recorded in the hospital books, under the name of "Muco-enterite," corresponding in all their symptoms to this disease.¹ There are no grounds whatever for the belief, that typhoid fever has only of late years appeared in this country;² and it seems not improbable that many of the cases described by Cullen and his contemporaries, as enteritis erythematica, were examples of this disease.³ From the circumstances above mentioned, it is reasonable to conclude that typhoid is quite independent of those epidemic agencies which appear to influence so greatly the prevalence of typhus and relapsing fever, and that in some places it is constantly endemic. I do not mean to deny, that, in places where typhoid is endemic, it may prevail more at one time than another, or even that outbreaks of it may occur at any place, and then disappear entirely. Many such outbreaks will be afterwards alluded to; but in all the instances with which I am acquainted, the disease, from the very isolated sphere of its prevalence, has appeared to be attributable to causes of a

³ See the description of this affection given by Dr. Alison, "Outlines of Path. and Pract. of Phys.," p. 322.
local nature. In addition to the examples of this nature, which I shall have occasion to bring forward in a subsequent part of this paper, I may here mention that many such outbreaks, appearing in limited localities of the United States, have been described by Dr. Bartlett. 1 One of the most remarkable instances of this nature, also, has been recorded by the late Dr. John Reid, of St. Andrews. 2 During the years 1838 and 1839, while typhus was very prevalent in Edinburgh, Dr. Reid, then pathologist to the infirmary, found that in scarcely a single case which proved fatal was there any morbid appearance in the intestines; while, at the same time, at Anstruther, a village on the opposite coast of the Frith of Forth, Mr. John Goodsir found ulceration of Peyer's patches, and of the solitary glands of the ileum, in every fatal case of fever which he examined.

Again, while epidemics of typhus and relapsing fever are for the most part limited to the United Kingdom, and the North of Europe, typhoid may occur in any part of the globe. It is the ordinary fever of the Continent and of America, and is even met with in the East Indies, 3 and in Burmah. 4

Typhus, then, and relapsing fever appear to be essentially epidemic diseases; but typhoid, to depend upon causes of a more constant, although more circumscribed and local, character.

The contrast between the annual prevalence of typhoid fever on the one hand, and of typhus and relapsing on the other, is well illustrated by diagram No. I.

III. The Prevalence of the different forms of Continued Fever according to the Months and Seasons.

Table IV gives the numbers of typhus and typhoid fever admitted during each month of ten successive years, into the London Fever Hospital.

1 'Fevers of the United States,' fourth edit., pp. 99 and 106.
4 Mr. Scriven, 'Med. Times and Gaz.,' vol. xix, p. 70.
Diagram I. shows the annual number of admissions of each form of Fever into the London hospitals during ten years.
Diagram II shows the number of admissions of Typhus into the London Fever Hospital, during each season of ten years.
### Table IV.

<table>
<thead>
<tr>
<th></th>
<th>Typhus</th>
<th>Typhoid</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1848</td>
<td>1849</td>
</tr>
<tr>
<td></td>
<td>1848</td>
<td>1849</td>
</tr>
<tr>
<td>January</td>
<td>43</td>
<td>27</td>
</tr>
<tr>
<td>February</td>
<td>37</td>
<td>19</td>
</tr>
<tr>
<td>March</td>
<td>66</td>
<td>25</td>
</tr>
<tr>
<td>April</td>
<td>65</td>
<td>14</td>
</tr>
<tr>
<td>May</td>
<td>66</td>
<td>17</td>
</tr>
<tr>
<td>June</td>
<td>43</td>
<td>13</td>
</tr>
<tr>
<td>July</td>
<td>48</td>
<td>22</td>
</tr>
<tr>
<td>August</td>
<td>32</td>
<td>9</td>
</tr>
<tr>
<td>September</td>
<td>39</td>
<td>11</td>
</tr>
<tr>
<td>October</td>
<td>22</td>
<td>6</td>
</tr>
<tr>
<td>November</td>
<td>44</td>
<td>4</td>
</tr>
<tr>
<td>December</td>
<td>21</td>
<td>3</td>
</tr>
</tbody>
</table>
Etiology of continued fever.

Table V gives the number of admissions of all the four forms for the months and seasons\(^1\) of the ten years taken collectively.

**Table V.**

<table>
<thead>
<tr>
<th></th>
<th>Typhus</th>
<th>Relapsing</th>
<th>Typhoid</th>
<th>Febricula</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>January</td>
<td>385</td>
<td>22</td>
<td>113</td>
<td>68</td>
<td>588</td>
</tr>
<tr>
<td>February</td>
<td>300</td>
<td>27</td>
<td>85</td>
<td>67</td>
<td>479</td>
</tr>
<tr>
<td>March</td>
<td>389</td>
<td>17</td>
<td>77</td>
<td>69</td>
<td>555</td>
</tr>
<tr>
<td>April</td>
<td>380</td>
<td>46</td>
<td>60</td>
<td>69</td>
<td>555</td>
</tr>
<tr>
<td>May</td>
<td>396</td>
<td>46</td>
<td>79</td>
<td>57</td>
<td>578</td>
</tr>
<tr>
<td>June</td>
<td>312</td>
<td>43</td>
<td>119</td>
<td>75</td>
<td>549</td>
</tr>
<tr>
<td>July</td>
<td>280</td>
<td>31</td>
<td>157</td>
<td>75</td>
<td>545</td>
</tr>
<tr>
<td>August</td>
<td>239</td>
<td>44</td>
<td>235</td>
<td>81</td>
<td>557</td>
</tr>
<tr>
<td>September</td>
<td>206</td>
<td>23</td>
<td>250</td>
<td>75</td>
<td>555</td>
</tr>
<tr>
<td>October</td>
<td>214</td>
<td>56</td>
<td>253</td>
<td>77</td>
<td>600</td>
</tr>
<tr>
<td>November</td>
<td>211</td>
<td>49</td>
<td>223</td>
<td>80</td>
<td>563</td>
</tr>
<tr>
<td>December</td>
<td>194</td>
<td>37</td>
<td>161</td>
<td>67</td>
<td>459</td>
</tr>
</tbody>
</table>

<p>| | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Spring</td>
<td>1069</td>
<td>90</td>
<td>222</td>
<td>205</td>
<td>1586</td>
</tr>
<tr>
<td>Summer</td>
<td>988</td>
<td>120</td>
<td>355</td>
<td>207</td>
<td>1670</td>
</tr>
<tr>
<td>Autumn</td>
<td>659</td>
<td>123</td>
<td>746</td>
<td>234</td>
<td>1762</td>
</tr>
<tr>
<td>Winter</td>
<td>790</td>
<td>108</td>
<td>497</td>
<td>215</td>
<td>1610</td>
</tr>
</tbody>
</table>

|        | 3506   | 441       | 1820    | 861       | 6628   |

From the fact that relapsing and typhus prevail for the most part as epidemics, continuing for one or two years, and then almost disappearing for several years, it was scarcely to have been expected that month or season of the year would have much influence over their numbers, and this anticipation is verified by the results before us. Out of the total 441 cases of relapsing fever, 328 were admitted between the beginning of February, 1851, and the end of March, 1852. From February, 1851, the numbers gradually increased up to the end of the year, when they began to decline. If this epidemic be excluded, the remaining

\(^1\) Under "Winter," I have included the three months, January, November, and December.
cases occurred without any reference to the period of the year. With regard to typhus, taking the ten years collectively, May, March, and January were the months in which there was the greatest number of admissions; September and December, those in which there was the smallest; and the largest number was in the spring season, the smallest in autumn. This distribution, however, was far from constant in the different years. Thus, in 1853, the number of cases in spring only exceeded that of autumn by 1, and in 1850 and 1855, there were most cases in autumn, and fewest in spring; again, in 1851, there were 5 cases in summer, and 22 in winter, while in 1854, there were 155 cases in the former season, 46 in the latter. (See Diagram II.) This non-dependence of typhus upon the season of the year, I find to be borne out by a comparison of all the published records of its various epidemics to which I have had access.

On glancing at the tables, one must be at once struck with the great contrast which typhoid presents to the two forms of fever just considered. By far the largest numbers have been admitted during the autumn months, September, October, and August, in the order here given; and the fewest in spring; those in the former season amounting to 41 per cent. of the whole; of the latter, to only 12 per cent. Moreover, this remark holds good not only with the ten years taken collectively, but also with every individual year. The contrast in this respect will be at once apparent by an examination of Table IV and of Diagram III. I may mention also, that, although the numbers of each form of fever have only been recorded at the Fever Hospital for ten years, I find on referring to the printed reports of the last 25 years, that ulceration of the bowel is invariably stated to have been found in the fatal cases oftenest in autumn.

This greater autumnal prevalence of typhoid is not limited to London. Dr. Gairdner writes me that, during September, October, and November, of 1857, there were admitted into the Royal Infirmary 18 cases, but during the three spring months, only 6. Speaking of Glasgow, in 1886-87, Dr. A. P. Stewart observes, that the cases of typhoid
admitted into the Infirmary were very numerous in the
ever part of summer and in autumn, very few in winter
and spring.1 Almost all the great outbreaks of typhoid
which have been reported in the various journals, as occurring
in the provincial towns and villages of England, have been
during the autumn. References will be found to a number
of these in a footnote.2 These outbreaks were particularly
numeros during the autumn of 1846, the summer of which
year had been remarkable for its unusually high temperature.
In the Report of the Fever Hospital for this year, I find
the following statement. "In the unusually hot weather
that prevailed in the summer and autumn months of this
year, diarrhoea occurred in almost every case of fever; and
the intestines were found, in a very large proportion of the
fatal cases, extensively diseased."

Again of 183 cases at Strasbourg, reported by Forget,3
60 were in autumn, 49 in summer, 38 in spring, and 36
in winter. In the United States, Dr. Bartlett informs
us, that his impression is, that typhoid is most prevalent in autumn: of 645 cases admitted into the Lowell
Hospital during seven years, 250 were in autumn, only 104
in spring.4 Again in New England, Dr. Austin Flint
observes that typhoid exhibits such a manifest predilection
for the autumn, that it is there known by the name of the
"Autumnal" or "Fall Fever."5 Lastly, from the descriptions of the "Autumnal Fever" in this country during the

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Journ.,' 1853, p. 793. Clergy Orphan Asylum, St. John's Wood, 1856,
4 'Lancet,' November 15th, 1856.
5 'Traité de l'Enterite Follic.,' p. 409.
7 'Clinical Reports on Continued Fever,' 1852, p. 20.
Diagram III shows the number of admis.
London Fever Hospital, during
3 years.

[Diagram with bars representing different years and seasons]
Diagram VII. Each column shows the number of cases with the number of deaths in each quinquennial period of life.

The right hand columns show the number of cases with the number of deaths of Typhoid Fever in each quinquennial period of life.
Diagram II shows the annual admissions of Typhoid Fever into the London I Hospital during each season of ten years.
Diagram IV. Left hand columns show the number of cases with the number of deaths of Typhus in each quinquennial period of life. The right hand columns show the number of cases with the number of deaths of Typhoid Fever in each quinquennial period of life.

In text: Page 238.
last century, given by Sir John Pringle and Rutty, it seems highly probable that this was Typhoid.¹

On the whole, then, without denying that an outbreak of typhoid may occasionally occur at other seasons, I think there are ample grounds for concluding, that the autumn is peculiarly favorable to its development.

IV. Influence of Sex upon the prevalence of Fever.

Table VI indicates the sexes of the cases of the different forms of continued fever, admitted into the London Fever Hospital during ten years.

<table>
<thead>
<tr>
<th></th>
<th>Typhus</th>
<th>Relapsing</th>
<th>Typhoid</th>
<th>Febricula</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>1737</td>
<td>233</td>
<td>905</td>
<td>449</td>
<td>3324</td>
</tr>
<tr>
<td>Females</td>
<td>1769</td>
<td>298</td>
<td>915</td>
<td>412</td>
<td>3304</td>
</tr>
<tr>
<td>Total</td>
<td>3506</td>
<td>441</td>
<td>1820</td>
<td>861</td>
<td>6628</td>
</tr>
</tbody>
</table>

From this Table it would appear that sex exercises but little influence over the prevalence of continued fever in London. In some years, the males exceeded the females; and in others, the females, the males; while taking all the ten years together, out of 6628 cases, there was an excess of 20 only in favour of the males. This slight excess was due to relapsing fever, and to the cases entered as febricula; for in both typhus and typhoid, the females were somewhat more numerous than the males. In all the four classes, however, the relative proportion of males and females varied in different years, so that one sex cannot be said to predispose more than another, even to any of the forms of fever.

Etiology of continued fever.

Some observers have thought that continued fever was more prevalent among females. Dr. Harty, in his statistical account of the Irish epidemic of 1817-20, says, that there were 34,398 females, to 32,144 males;¹ and Dr. Orr ascertained that of the cases admitted into the Glasgow Infirmary, between the years 1831 and 1845, inclusive, there were 16,834 females, to 15,863 males. Dr. Orr, however, showed that this difference was more than accounted for by the excess of females in the population of Glasgow.² On the other hand, of the cases of fever treated in the Edinburgh Infirmary, during the two years 1846-7, there were (excluding febricula) 4798 males, to 3061 females;³ and, out of 3186 cases, in the Seraphim Hospital, at Stockholm, more than one half, or 2181 were males.⁴ Dr. Huss, however, ascribes this minority of females at Stockholm to a larger proportion of them being treated at their own homes. With respect to typhoid individually, Louis⁵ found only 32 females out of 138 cases observed by him; but he accounts for this circumstance in the same way as Dr. Huss does at Stockholm: and out of a number of cases collected by Dr. Bartlett, from several sources, there were 1345 males, to 1229 females;⁶ while the same author mentions several instances in which the females suffered more extensively than the males.

There are always circumstances which at different places influence the admission into hospitals of one sex more than the other; but, from what has been stated, sex appears to be of little or no importance as a predisposing cause of fever, or of one form of it more than another.

V. Influence of Age upon the prevalence of Fever.

The influence of age upon the prevalence of fever, and of

¹ † Historical Sketch of the Fever-Epidemic in Ireland, Dublin, 1820.
ETIOLOGY OF CONTINUED FEVER.

its different forms, has been arrived at in two ways; by taking the average age of all the cases, and by ascertaining the numbers in each period of life.

Table VII gives the average age of each sex, of all the cases of each form of fever, admitted into the London Fever Hospital during ten years.

### Table VII.

<table>
<thead>
<tr>
<th></th>
<th>Males.</th>
<th>Females.</th>
<th>Males and Females.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. in which age was noted.</td>
<td>Average Age.</td>
<td>No. in which age was noted.</td>
</tr>
<tr>
<td>Typhus.........</td>
<td>1714</td>
<td>28:38</td>
<td>1742</td>
</tr>
<tr>
<td>Relapsing......</td>
<td>231</td>
<td>22:98</td>
<td>206</td>
</tr>
<tr>
<td>Typhoid .......</td>
<td>878</td>
<td>21:45</td>
<td>894</td>
</tr>
<tr>
<td>Relapsing......</td>
<td>440</td>
<td>21:56</td>
<td>405</td>
</tr>
<tr>
<td>Total ........</td>
<td>3263</td>
<td>25:22</td>
<td>3247</td>
</tr>
</tbody>
</table>

Thus, the average age of all the cases of continued fever being not quite 26, that of typhus was 29½; of relapsing 24½; and of typhoid only 21½.

Table VIII (see next page) shows the number of each fever in each quinquennial period of life for either sex. (See also Diagram IV.)

In continued fever, taken as a whole, and also in each of its individual forms, the three most common lustra of life appear, from Table VIII, to be 15 to 20 years, 20 to 25, and 10 to 15, in the order here given. The relative excess of cases, however, belonging to these lustra, varies greatly, being greatest in typhoid, and least in typhus. Thus, more than one half (52 per cent.) of the cases of typhoid are included in the two most common lustra, or between 15 and 25 years of age; but only 38½ per cent. of the cases of relapsing fever, and less than one third (30 per cent.) of the cases of typhus. Again, one fifth of the cases of typhoid,
### Table VIII. 1

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Typhus</th>
<th>Relapsing</th>
<th>Typhoid</th>
<th>Fibrilla</th>
<th>Total Cont. Fever</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M.</td>
<td>F.</td>
<td>M.&amp;F.</td>
<td>M.</td>
<td>F.</td>
</tr>
<tr>
<td>Under 5 years</td>
<td>9</td>
<td>8</td>
<td>17</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>From 5 to 10</td>
<td>88</td>
<td>95</td>
<td>183</td>
<td>18</td>
<td>14</td>
</tr>
<tr>
<td>10 to 15</td>
<td>175</td>
<td>188</td>
<td>363</td>
<td>41</td>
<td>22</td>
</tr>
<tr>
<td>15 to 20</td>
<td>295</td>
<td>251</td>
<td>546</td>
<td>54</td>
<td>38</td>
</tr>
<tr>
<td>20 to 25</td>
<td>287</td>
<td>208</td>
<td>495</td>
<td>40</td>
<td>35</td>
</tr>
<tr>
<td>25 to 30</td>
<td>185</td>
<td>158</td>
<td>343</td>
<td>16</td>
<td>21</td>
</tr>
<tr>
<td>30 to 35</td>
<td>157</td>
<td>166</td>
<td>323</td>
<td>18</td>
<td>19</td>
</tr>
<tr>
<td>35 to 40</td>
<td>121</td>
<td>129</td>
<td>270</td>
<td>8</td>
<td>11</td>
</tr>
<tr>
<td>40 to 45</td>
<td>109</td>
<td>183</td>
<td>292</td>
<td>16</td>
<td>24</td>
</tr>
<tr>
<td>45 to 50</td>
<td>98</td>
<td>114</td>
<td>212</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>50 to 55</td>
<td>71</td>
<td>79</td>
<td>150</td>
<td>8</td>
<td>7</td>
</tr>
<tr>
<td>55 to 60</td>
<td>48</td>
<td>52</td>
<td>100</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>60 to 65</td>
<td>40</td>
<td>48</td>
<td>88</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>65 to 70</td>
<td>17</td>
<td>25</td>
<td>42</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>70 to 75</td>
<td>9</td>
<td>15</td>
<td>24</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>75 to 80</td>
<td>4</td>
<td>2</td>
<td>6</td>
<td>12</td>
<td>1</td>
</tr>
<tr>
<td>Above 80 years</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Age doubtful</td>
<td>23</td>
<td>27</td>
<td>50</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Total, omitting doubtful cases</td>
<td>1714</td>
<td>1742</td>
<td>3456</td>
<td>231</td>
<td>206</td>
</tr>
</tbody>
</table>

1 In this Table, a patient who had completed his fifth or tenth year was reckoned as being between 5 and 10, and 10 and 15 years, respectively; and so on, for all the other periods of life.
a little more than one fifth of the relapsing cases, but less than one sixth of the cases of typhus, are below 15. As we advance in life the most of the cases are typhus or relapsing, and those of an extreme age are almost all typhus. Thus, nearly one half of the cases of typhus (43.6 p. c.), and one third of the relapsing cases are 30 years or upwards; but less than one seventh of the typhoid. Lastly, nearly one eighth of the typhus cases are 50 or upwards; of the relapsing one fifteenth; and of the typhoid, only one sixty-eighth.

The contrast between the ages of the different fevers will be more apparent from the following tabular comparison.

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Per cent. of Typhus cases</th>
<th>Per cent. of Relapsing cases</th>
<th>Per cent. of Typhoid cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 10 years</td>
<td>5.73</td>
<td>8.23</td>
<td>6.04</td>
</tr>
<tr>
<td>15</td>
<td>16.3</td>
<td>22.65</td>
<td>20.14</td>
</tr>
<tr>
<td>From 15 to 25 years</td>
<td>30.12</td>
<td>38.44</td>
<td>52.08</td>
</tr>
<tr>
<td>25 years and upwards</td>
<td>53.58</td>
<td>38.9</td>
<td>27.76</td>
</tr>
<tr>
<td>30</td>
<td>43.66</td>
<td>80.43</td>
<td>14.22</td>
</tr>
<tr>
<td>40</td>
<td>26.47</td>
<td>17.62</td>
<td>5.19</td>
</tr>
<tr>
<td>50</td>
<td>11.92</td>
<td>6.63</td>
<td>1.46</td>
</tr>
<tr>
<td>60</td>
<td>4.68</td>
<td>1.6</td>
<td>0.5</td>
</tr>
</tbody>
</table>

Judging, then, from the cases in the London Fever Hospital, at every period of life under 30, the susceptibility to typhoid is very much greater than to typhus; but under 15 years, the tendency would appear to be slightly greater to relapsing, than even to typhoid. Above 30, the tendency to typhus is almost as great as below that age, (or much greater if we take into account the proportion of the whole population above and below that age); but the tendency to typhoid is infinitely less.

With regard to febricula, the only circumstance deserving notice, is the large proportion of cases occurring in extreme youth, almost one ninth being under 10. This was probably

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1 The proportion of typhoid cases in early life would be still greater, were it not that many children labouring under this disease are treated at dispensaries, and at their own homes, as cases of "Infantile Remittent Fever."
owing to some of the milder cases of the other forms not being recognised at that early age, and hence included under this head.

The youngest case of typhus was a male infant under twelve months, the oldest, a man aged 84. The youngest case of relapsing fever was a female aged 2; the oldest, a man aged 74. The youngest cases of typhoid were three children aged 4; two cases are noted as above 65. One of these, a man, had rose-spots, and called himself 76, but he did not look more than sixty. The other was a man aged 65, who had no eruption. (M. Lombard, of Geneva, relates a case of typhoid in a woman aged 73; and Gendron, four cases between 60 and 75.)

As regards the ages of either sex, there are one or two points worth mentioning. Advancing age would appear to lessen the susceptibility to typhus and relapsing fever to a greater extent in males than in females. Thus, in every one of the ten years, the average age of the females exceeded that of the males, in both these forms. Again, of typhus,

Out of a total of 3506 cases, 1737 were males; 1769 females.
Below 15 years of age . 272 " 291 "
Between 15 and 30 . 767 " 617 "
Above 30 . . . . 675 " 834 "

In relapsing fever,

Out of a total of 441 cases, 233 were males; 208 females.
Of cases below 25 years . 155 " 112 "
Of cases above 25 . . 76 " 94 "

In typhus also, but not in relapsing, the excess of males was limited to between 15 and 30.

As to the period of life most susceptible to typhoid, there is little difference between males and females. In some years the average age of the males was greatest; in others, that of the females; and for the whole ten years, the mean age of the two sexes was almost equal. Again, of 1620 cases

1 Bartlett, op. cit., p. 108.
ETIOLOGY OF CONTINUED FEVER.

below 30 the females exceeded the males by 62. and in 173 cases above 30 the males exceeded the females by 14 so that on the whole we have a result somewhat contrary to what occurs in typhus and relapsing fever.

The various statements just made, under the head of age confirm, on a great scale, most previous observations: the only exception being the greater prevalence of typhus and relapsing fever, above 30, in females than in males. Dr. Cowan, of Glasgow, found precisely the contrary; 1 so that perhaps much stress must not be laid upon this circumstance.

VI. Predisposition to Fever from Occupation and Science in Life.

In Table IX the occupations of the patients belonging to each form of fever, admitted into the London Fever Hospital, during ten years, have been classified, and the percentage of each occupation upon the total number in which it was known, calculated. See next page.

It is not probable that many of the occupations specified in this table in themselves predispose to fever, or to one form of it more than another. I would, however, call attention to the circumstance, as important in connexion with facts to be subsequently added, that out of 19 cow-keepers admitted with fever, in 9 the fever was typhoid. I have also ascertained that, in several instances, patients entered as "labourers" had been employed in the drains; in every such case the fever was typhoid.

But the chief point which this table illustrates is the fact, that most of those who may be supposed to have occupied the better conditions of life, have been admitted with typhoid fever, whereas typhus and relapsing have been rare among them, and most common amongst the lowest classes. It would occupy too much time to enter minutely into the details of the table: a few of the most striking circumstances illustrative of the statement just made will

1 'Vital Statistics of Glasgow,' 1888.
<table>
<thead>
<tr>
<th></th>
<th>Typhus</th>
<th>Relapsing</th>
<th>Typhoid</th>
<th>Fibrricula</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total</td>
<td>No.</td>
<td>Per cent.</td>
<td>No.</td>
</tr>
<tr>
<td>Female Servants</td>
<td>958</td>
<td>321</td>
<td>11·82</td>
<td>38</td>
</tr>
<tr>
<td>Male ditto</td>
<td>36</td>
<td>10</td>
<td>3·6</td>
<td>...</td>
</tr>
<tr>
<td>Artizans</td>
<td>662</td>
<td>380</td>
<td>13·13</td>
<td>23</td>
</tr>
<tr>
<td>Shoemakers</td>
<td>204</td>
<td>138</td>
<td>6·80</td>
<td>10</td>
</tr>
<tr>
<td>Tailors</td>
<td>62</td>
<td>37</td>
<td>1·36</td>
<td>6</td>
</tr>
<tr>
<td>Sempatresses</td>
<td>372</td>
<td>242</td>
<td>6·92</td>
<td>10</td>
</tr>
<tr>
<td>Bakers</td>
<td>39</td>
<td>12</td>
<td>3·1</td>
<td>1</td>
</tr>
<tr>
<td>Householders</td>
<td>14</td>
<td>10</td>
<td>0·36</td>
<td>...</td>
</tr>
<tr>
<td>Shopmen</td>
<td>145</td>
<td>60</td>
<td>2·21</td>
<td>6</td>
</tr>
<tr>
<td>Schoolmasters</td>
<td>10</td>
<td>8</td>
<td>0·29</td>
<td>...</td>
</tr>
<tr>
<td>Scholars</td>
<td>90</td>
<td>31</td>
<td>1·14</td>
<td>2</td>
</tr>
<tr>
<td>Clerks</td>
<td>32</td>
<td>11</td>
<td>0·40</td>
<td>...</td>
</tr>
<tr>
<td>Butchers</td>
<td>33</td>
<td>18</td>
<td>0·66</td>
<td>1</td>
</tr>
<tr>
<td>Barmen</td>
<td>32</td>
<td>12</td>
<td>0·44</td>
<td>2</td>
</tr>
<tr>
<td>Policemen</td>
<td>43</td>
<td>10</td>
<td>0·36</td>
<td>...</td>
</tr>
<tr>
<td>Soldiers and Sailors</td>
<td>51</td>
<td>22</td>
<td>0·81</td>
<td>6</td>
</tr>
<tr>
<td>Milkmen and Women</td>
<td>21</td>
<td>12</td>
<td>0·44</td>
<td>1</td>
</tr>
<tr>
<td>Travellers</td>
<td>4</td>
<td>3</td>
<td>0·11</td>
<td>...</td>
</tr>
<tr>
<td>Porters and Errand Boys</td>
<td>146</td>
<td>63</td>
<td>2·32</td>
<td>13</td>
</tr>
<tr>
<td>Hawkers and Street Musicians</td>
<td>247</td>
<td>136</td>
<td>5·01</td>
<td>54</td>
</tr>
<tr>
<td>Shoeblacks</td>
<td>7</td>
<td>4</td>
<td>0·14</td>
<td>...</td>
</tr>
<tr>
<td>Cabmen and Outsiders</td>
<td>97</td>
<td>53</td>
<td>1·95</td>
<td>3</td>
</tr>
<tr>
<td>Cowkeepers</td>
<td>10</td>
<td>1</td>
<td>0·03</td>
<td>...</td>
</tr>
<tr>
<td>Labourers</td>
<td>637</td>
<td>436</td>
<td>16·07</td>
<td>87</td>
</tr>
<tr>
<td>Dustmen</td>
<td>12</td>
<td>9</td>
<td>0·33</td>
<td>1</td>
</tr>
<tr>
<td>Sweeps</td>
<td>6</td>
<td>4</td>
<td>0·14</td>
<td>1</td>
</tr>
<tr>
<td>Charwomen</td>
<td>171</td>
<td>125</td>
<td>4·61</td>
<td>9</td>
</tr>
<tr>
<td>Laundresses</td>
<td>208</td>
<td>154</td>
<td>5·67</td>
<td>9</td>
</tr>
<tr>
<td>Vagrants</td>
<td>64</td>
<td>44</td>
<td>1·62</td>
<td>12</td>
</tr>
<tr>
<td>&quot;Paupers&quot;²</td>
<td>106</td>
<td>83</td>
<td>3·05</td>
<td>3</td>
</tr>
<tr>
<td>Nurses in Workhouses</td>
<td>54</td>
<td>46</td>
<td>1·79</td>
<td>...</td>
</tr>
<tr>
<td>Bridewell and Ho. of Correction</td>
<td>14</td>
<td>13</td>
<td>0·47</td>
<td>...</td>
</tr>
<tr>
<td>Nurses, London Fever Hospital</td>
<td>40</td>
<td>25</td>
<td>0·92</td>
<td>1</td>
</tr>
<tr>
<td>Married Females *</td>
<td>206</td>
<td>180</td>
<td>7·33</td>
<td>7</td>
</tr>
<tr>
<td>Total of which occupation known</td>
<td>5095</td>
<td>2713</td>
<td>100</td>
<td>306</td>
</tr>
</tbody>
</table>

¹ These include all in-door workers, except those otherwise specified, such as smiths, carpenters, printers, &c.
² These include all out-door workers, such as masons, dock-labourers, gardeners, &c.
³ These have been entered in the register as "paupers." Many who were really so have been entered under the occupations which they previously followed. See Table XII.
⁴ Many other married females have been entered as following some occupation.
suffice. Thus, the proportion of female servants admitted with typhoid has been three times that of typhus; of policemen six times; and of shopmen more than double. On the other hand, the proportion of street hawkers affected with typhoid has been less than one third that of typhus, and scarcely more than one eleventh of that of relapsing. The number of cases of relapsing fever belonging to this class is remarkable. Of charwomen, the proportion with typhoid has been considerably less than one third that with typhus, or than one half that with relapsing. Among "paupers," typhus and relapsing fever, taken together, have been eight times as frequent as typhoid, and of 64 "vagrants" admitted with fever, in not a single instance was this typhoid.

There are one or two apparent exceptions to the general rule, as, for example, in the case of those entered as "schoolmasters," and "soldiers and sailors." The former, however, have been for the most part teachers in ragged schools, where they may have contracted the disease by contagion: the latter have been almost invariably discharged or out of employment; moreover, most of the individuals belonging to both classes have been in destitute circumstances.

The recorded occupation, however, of a patient, it must be remembered, is not always a certain index of the amount of worldly comfort which he has enjoyed. A female may be brought direct from one of the fashionable squares of the west end, or from one of the crowded courts of Holborn; and yet, in both instances, be entered as a "servant." Another test, therefore, becomes desirable for the accuracy of the statement which I have made; and, fortunately, such a test is available. The patients admitted into the London Fever Hospital may be divided into four classes, viz.—1. The servants of subscribers, and those who are able to pay (£2 2s.) for admission. 2. Persons in destitute circumstances, but not receiving parochial relief. This is a somewhat mixed class: some have been very destitute, but many have been in tolerably easy circum-
stances, previous to their illness. 3. Persons receiving parochial relief, but not actually inmates of a workhouse. And 4. Those who, for a greater or less period, have been inmates of a workhouse. Now, I have determined the number of cases of each form of fever, in each of these four classes. The results are given in the following tables.

Table X gives the numbers and proportion of each form of fever in the first class for ten years. I have included in this table the policemen, as they are paid for by the police authorities, and none of them can be said to be destitute.

**Table X.**

<table>
<thead>
<tr>
<th>Class</th>
<th>Paying Patients</th>
<th>Servants of Subscribers</th>
<th>Policemen</th>
<th>Total</th>
<th>Per centage of each Fever of paid cases</th>
<th>Per centage of each Fever of total paid cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Typhus</td>
<td>80</td>
<td>4</td>
<td>10</td>
<td>94</td>
<td>23.6%</td>
<td>2.6%</td>
</tr>
<tr>
<td>Relapsing</td>
<td>2</td>
<td>...</td>
<td>...</td>
<td>2</td>
<td>4.7%</td>
<td>0.4%</td>
</tr>
<tr>
<td>Typhoid</td>
<td>242</td>
<td>9</td>
<td>30</td>
<td>281</td>
<td>65.9%</td>
<td>15.4%</td>
</tr>
<tr>
<td>Febricula</td>
<td>42</td>
<td>2</td>
<td>5</td>
<td>49</td>
<td>11.5%</td>
<td>5.69%</td>
</tr>
<tr>
<td>Total</td>
<td>366</td>
<td>15</td>
<td>45</td>
<td>426</td>
<td>99.99%</td>
<td>6.43%</td>
</tr>
</tbody>
</table>

The proportion of typhoid cases in this class is thus seen to be six times that of typhus, and more than thirty-four times that of relapsing fever.

Table XI gives the fever-patients admitted during ten years, who were unable to pay, although not receiving parochial relief.
ETIOLOGY OF CONTINUED FEVER.

TABLE XI.

<table>
<thead>
<tr>
<th>Class II.</th>
<th>Total &quot;Fever&quot; cases.</th>
<th>Per centage of each Fever on total of each Fever.</th>
<th>Per centage of free cases on total of each Fever.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Typhus</td>
<td>130</td>
<td>29·75</td>
<td>3·7</td>
</tr>
<tr>
<td>Relapsing</td>
<td>9</td>
<td>2·06</td>
<td>2·04</td>
</tr>
<tr>
<td>Typhoid</td>
<td>245</td>
<td>56·06</td>
<td>13·46</td>
</tr>
<tr>
<td>Febricula</td>
<td>53</td>
<td>12·13</td>
<td>6·15</td>
</tr>
<tr>
<td>Total</td>
<td>437</td>
<td>100·</td>
<td>6·59</td>
</tr>
</tbody>
</table>

Here the proportion of typhoid cases is still predominant, although not to such an extent as in the former class; being only four times in excess of that of typhus, and six times of that of relapsing.

Lastly, in Table XII, we have the patients paid for by the various parishes; a distinction being made between those sent direct from their own homes, and the inmates of workhouses.

TABLE XII.

<table>
<thead>
<tr>
<th>Class III. Sent from own homes, by Parishes.</th>
<th>Class IV. Inmates of Workhouses.</th>
<th>Class III and IV. Total paid for by Parishes.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class</td>
<td>Number</td>
<td>Per centage of total of each Fever</td>
</tr>
<tr>
<td>-------</td>
<td>--------</td>
<td>----------------------------------</td>
</tr>
<tr>
<td>Typhus</td>
<td>2544</td>
<td>53·26</td>
</tr>
<tr>
<td>Relapsing</td>
<td>383</td>
<td>8·92</td>
</tr>
<tr>
<td>Typhoid</td>
<td>1299</td>
<td>25·31</td>
</tr>
<tr>
<td>Febricula</td>
<td>640</td>
<td>13·4</td>
</tr>
<tr>
<td>Total</td>
<td>4776</td>
<td>99·99</td>
</tr>
</tbody>
</table>
The relative proportion of the different fevers is in this table reversed; that of typhus and relapsing being in considerable excess of typhoid. The gradual change in the prevalent fever, as we pass from one class to another, is strikingly brought out by the following tabular comparison:

<table>
<thead>
<tr>
<th></th>
<th>Per cent. of Typhus and Relapsing</th>
<th>Per cent. of Typhoid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Of the paying patients</td>
<td>22.5</td>
<td>65.96</td>
</tr>
<tr>
<td>Of the &quot;free&quot; patients</td>
<td>31.8</td>
<td>56.06</td>
</tr>
<tr>
<td>Of those sent from homes by parishes</td>
<td>61.28</td>
<td>25.31</td>
</tr>
<tr>
<td>Of the inmates of workhouses</td>
<td>79.37</td>
<td>8.59</td>
</tr>
</tbody>
</table>

This contrast would be the more striking, if we had the means, which, unfortunately, the Fever Hospital does not afford, of comparing with the above the fevers which prevail in the upper classes of society. I believe, however, that there would not be much difficulty in establishing the fact, that such fevers are almost exclusively typhoid. Typhus, among the rich, except as the result of contagion, is excessively rare, while cases of typhoid, originating without any traceable contagion, are far from uncommon. I find, by inquiry, that this is in accordance with the experience of every one of the physicians in London, who, for many years, have had the best opportunities of judging.

Moreover, in the account of every outbreak of typhoid with which I am acquainted, all classes of the community are stated to have suffered alike. Two instances out of many with which I am familiar will suffice. In an account of an outbreak of typhoid, which occurred at Nottingham, in 1846, Dr. Sibson remarks that "very many were in good circumstances of those who were attacked."\(^1\) Again, in what was called the Croydon Fever, in 1852 (which, as we shall find, was unmistakably typhoid), we are told that the victims were "not, as usually the case, among the poor, but among the gentry and principal tradesmen of the town."\(^2\)

\(^2\) 'Med. Times and Gaz.,' Jan. 29th, 1853.
I think, then, it may be concluded that typhus and relapsing fever are, for the most part, confined to the poor; but that typhoid makes no distinction between one class and another.

VII. *Localities of London in which each form of Fever is most prevalent.*

In order to form some opinion upon this point, the returns of no hospital could be better suited than those of the London Fever Hospital, inasmuch as its patients are derived from every district of the metropolis. It is true that, from various circumstances, some districts send in a larger proportion of their fever cases into the hospital than others, so that the returns do not correctly indicate the *amount* of fever prevalent in each district; yet those cases which are admitted from each locality, furnish a very fair criterion for judging of the form of fever there most prevalent. In order, then, to render the returns of the Fever Hospital available for the purpose in question, I have constructed Table XIII, in which London is divided into districts, and the number and proportion of the different forms of fever admitted from each district are given. The area and population of each district are also shown in the table.¹

¹ These have been copied from the Weekly Returns of the General Board of Health.
### TABLE XIII.

<table>
<thead>
<tr>
<th>Divisions and Districts of London</th>
<th>Area in Statute Acres</th>
<th>Population in 1881</th>
<th>Total Fever Cases from each district</th>
<th>Number and per centage of each Fever in each District</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>typhus</td>
</tr>
<tr>
<td>I. (a) Kensington</td>
<td>1,942</td>
<td>44,053</td>
<td>74</td>
<td>16</td>
</tr>
<tr>
<td>&quot; (b) Paddington</td>
<td>1,277</td>
<td>46,305</td>
<td>14</td>
<td>12</td>
</tr>
<tr>
<td>&quot; (c) Fulham</td>
<td>4,155</td>
<td>29,646</td>
<td>29</td>
<td>8</td>
</tr>
<tr>
<td>II. Chelsea</td>
<td>865</td>
<td>56,533</td>
<td>26</td>
<td>10</td>
</tr>
<tr>
<td>III. (a) St. George's, Hanover Sq</td>
<td>881</td>
<td>33,196</td>
<td>9</td>
<td>7</td>
</tr>
<tr>
<td>&quot; (b) Belgravia</td>
<td>880</td>
<td>40,034</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>IV. Westminster</td>
<td>917</td>
<td>65,609</td>
<td>119</td>
<td>21</td>
</tr>
<tr>
<td>V. St. Martin's in Fields</td>
<td>303</td>
<td>24,640</td>
<td>180</td>
<td>27</td>
</tr>
<tr>
<td>VI. St. James's, Westminster</td>
<td>164</td>
<td>36,406</td>
<td>103</td>
<td>35</td>
</tr>
</tbody>
</table>

**West Division** ...

<table>
<thead>
<tr>
<th>Area in Statute Acres</th>
<th>Population in 1881</th>
<th>Total Fever Cases</th>
<th>Number and per centage of each Fever in each District</th>
</tr>
</thead>
<tbody>
<tr>
<td>10,786</td>
<td>376,427</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| VII. Marylebone        | 1,509              | 157,698           | 257                                 | 192   | 59·14     | 5       | 1·94     |
| VIII. Hampstead        | 2,252              | 11,986            | 16                                  | 16    | 62·36     | 2       | 12·5     |
| IX. St. Pancras        | 2,716              | 166,555           | 503                                 | 17    | 37·87     | 11      | 10·33    |
| X. Islington           | 3,127              | 95,329            | 596                                 | 232   | 28·67     | 15      | 15·25    |
| XI. Hackney            | 3,929              | 58,429            | 86                                  | 20    | 24·09     | 2       | 2·41     |

<table>
<thead>
<tr>
<th>Area in Statute Acres</th>
<th>Population in 1881</th>
<th>Total Fever Cases</th>
<th>Number and per centage of each Fever in each District</th>
</tr>
</thead>
<tbody>
<tr>
<td>13,533</td>
<td>490,396</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**North Division** ...

<table>
<thead>
<tr>
<th>Area in Statute Acres</th>
<th>Population in 1881</th>
<th>Total Fever Cases</th>
<th>Number and per centage of each Fever in each District</th>
</tr>
</thead>
<tbody>
<tr>
<td>1453</td>
<td>688</td>
<td>47·28</td>
<td></td>
</tr>
<tr>
<td>2·33</td>
<td>357</td>
<td>38·28</td>
<td></td>
</tr>
<tr>
<td>175</td>
<td>12·09</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Division</td>
<td>District</td>
<td>Area</td>
<td>Population</td>
</tr>
<tr>
<td>----------</td>
<td>----------------</td>
<td>------</td>
<td>------------</td>
</tr>
<tr>
<td>Central Division</td>
<td>St. Gile's</td>
<td>245</td>
<td>54,214</td>
</tr>
<tr>
<td></td>
<td>Strand</td>
<td>174</td>
<td>44,460</td>
</tr>
<tr>
<td></td>
<td>Holborn</td>
<td>196</td>
<td>46,621</td>
</tr>
<tr>
<td></td>
<td>Clerkenwell</td>
<td>380</td>
<td>64,778</td>
</tr>
<tr>
<td></td>
<td>St. Luke</td>
<td>220</td>
<td>54,055</td>
</tr>
<tr>
<td>City of London</td>
<td></td>
<td>723</td>
<td>129,128</td>
</tr>
<tr>
<td></td>
<td>Central Division</td>
<td>1,936</td>
<td>393,256</td>
</tr>
<tr>
<td></td>
<td>XX. Shoreditch</td>
<td>646</td>
<td>109,257</td>
</tr>
<tr>
<td></td>
<td>XXI. Bethnal Green</td>
<td>760</td>
<td>90,193</td>
</tr>
<tr>
<td></td>
<td>XXII. Whitechapel</td>
<td>406</td>
<td>72,759</td>
</tr>
<tr>
<td></td>
<td>XXIII. St. George's in the East</td>
<td>243</td>
<td>45,376</td>
</tr>
<tr>
<td></td>
<td>XXIV. Limehouse</td>
<td>576</td>
<td>54,173</td>
</tr>
<tr>
<td></td>
<td>&quot; (d) Mile-End</td>
<td>681</td>
<td>56,602</td>
</tr>
<tr>
<td></td>
<td>&quot; (a) Poplar</td>
<td>1,490</td>
<td>28,384</td>
</tr>
<tr>
<td></td>
<td>&quot; (b) Bow</td>
<td>1,428</td>
<td>18,778</td>
</tr>
<tr>
<td></td>
<td>East Division</td>
<td>6,230</td>
<td>485,522</td>
</tr>
<tr>
<td>Area of Statute and Division of London</td>
<td>Population in 1851</td>
<td>Total Fever in each District</td>
<td>Number and per cent of each Fever in each District</td>
</tr>
<tr>
<td>-------------------------------------</td>
<td>-------------------</td>
<td>-----------------------------</td>
<td>---------------------------------</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Trachoma</td>
</tr>
<tr>
<td></td>
<td>No.</td>
<td>Per cent</td>
<td>No.</td>
</tr>
<tr>
<td>St. Sepulchre</td>
<td>169</td>
<td>50</td>
<td>19,371</td>
</tr>
<tr>
<td>St. Olave</td>
<td>688</td>
<td>62.255</td>
<td>48,138</td>
</tr>
<tr>
<td>St. George's Southwark</td>
<td>662</td>
<td>107.845</td>
<td>41,826</td>
</tr>
<tr>
<td>St. Andrew's</td>
<td>622</td>
<td>102.981</td>
<td>13,923</td>
</tr>
<tr>
<td>Lambeth</td>
<td>4,715</td>
<td>139.285</td>
<td>43,792</td>
</tr>
<tr>
<td>Cripples and Woolwich</td>
<td>1,179</td>
<td>52.809</td>
<td>10,380</td>
</tr>
<tr>
<td>Battersea</td>
<td>2,434</td>
<td>52.809</td>
<td>14,694</td>
</tr>
<tr>
<td>Southwark</td>
<td>1,622</td>
<td>52.809</td>
<td>14,067</td>
</tr>
<tr>
<td>Tooting</td>
<td>231</td>
<td>52.809</td>
<td>4,434</td>
</tr>
<tr>
<td>Camberwell</td>
<td>3,542</td>
<td>52.809</td>
<td>24,467</td>
</tr>
<tr>
<td>Battersea</td>
<td>3,542</td>
<td>52.809</td>
<td>24,467</td>
</tr>
<tr>
<td>Wandsworth</td>
<td>1,530</td>
<td>52.809</td>
<td>6,166</td>
</tr>
<tr>
<td>Woolwich</td>
<td>1,724</td>
<td>52.809</td>
<td>34,835</td>
</tr>
<tr>
<td>Source Division</td>
<td>45,542</td>
<td>45.344</td>
<td>616,635</td>
</tr>
<tr>
<td>London Districts</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Doublet Districts</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Total</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Number and per cent of each Fever in each District</th>
<th>Tatal</th>
<th>Per cent</th>
<th>No.</th>
<th>Per cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trachoma</td>
<td>108</td>
<td>36.533</td>
<td>37</td>
<td>52.067</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>108</td>
<td>36.533</td>
<td>37</td>
<td>52.067</td>
</tr>
<tr>
<td>Typhus</td>
<td>108</td>
<td>36.533</td>
<td>37</td>
<td>52.067</td>
</tr>
<tr>
<td>Typhoid</td>
<td>108</td>
<td>36.533</td>
<td>37</td>
<td>52.067</td>
</tr>
<tr>
<td>Plague</td>
<td>108</td>
<td>36.533</td>
<td>37</td>
<td>52.067</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Number and per cent of each Fever in each District</th>
<th>Tatal</th>
<th>Per cent</th>
<th>No.</th>
<th>Per cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trachoma</td>
<td>277</td>
<td>55.556</td>
<td>50</td>
<td>15.515</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>277</td>
<td>55.556</td>
<td>50</td>
<td>15.515</td>
</tr>
<tr>
<td>Typhus</td>
<td>277</td>
<td>55.556</td>
<td>50</td>
<td>15.515</td>
</tr>
<tr>
<td>Typhoid</td>
<td>277</td>
<td>55.556</td>
<td>50</td>
<td>15.515</td>
</tr>
<tr>
<td>Plague</td>
<td>277</td>
<td>55.556</td>
<td>50</td>
<td>15.515</td>
</tr>
</tbody>
</table>
ETIOLOGY OF CONTINUED FEVER. 251

The districts, it will be seen, which I have selected are those into which London has been divided under the new "Metropolitan Local Management Act." To each of the districts specified a medical officer of health has been appointed; and I have thought that the information contained in the table might enable these officers to throw some light on the causes of each fever in the respective districts to which they belong, as none can be better acquainted than they with the sanitary condition of these districts. To the attention of these gentlemen, therefore, I shall submit the consideration of the prevailing fevers in each district; and shall confine myself at present to some remarks on one or two individual localities, and to a few general observations on the conclusions to which my investigations have brought me.

In the first place it is obvious that, in some districts, one form of fever is more prevalent than in others. Typhus and relapsing fever only prevail to any extent in certain districts, while typhoid appears to be confined to none. Hence it follows that, in some districts, typhoid is almost the only form of fever met with. The districts in which typhus and relapsing fever have prevailed most, are those which are the most overcrowded, and which are inhabited by the poorest classes of the population. Typhoid again is met with, not only in the districts just alluded to, but also in those which are considered the most favoured in the metropolis. As a general rule, the relative proportion between typhus (together with relapsing fever) and typhoid, diminishes as we proceed from the centre of London towards the suburbs, until at last the two former nearly disappear, and we have mostly the latter.1 Thus, of the cases admitted from the central district of Holborn, there were of typhus and relapsing fever 608 cases, and only 91 of typhoid; and from the whole

1 Kensington appears to be an exception to this rule, but the majority of the cases of typhus admitted into the London Fever Hospital from this district were from the workhouse, during the single year 1848.
central division of London there were 1,675 cases of the two former fevers, to 398 of the latter. The predilection of relapsing fever for this part of London was very remarkable. Of the total 441 cases, more than one half came from the central division; and considerably more than one third from the single district of Holborn.

There are certain small courts in the vicinity of Gray's Inn Lane, and other parts of the central division of London, which have long been justly regarded as the hot-beds of epidemic fever. They were noticed as such by Dr. Bateman more than forty years ago; and they still maintain their unenviable reputation. One or two of these may be mentioned. From Pheasant Court, Holborn, there were admitted into the Fever Hospital, during the years 1851 and 1852, sixty-six cases of relapsing fever and eight of typhus; from Tyndall's Buildings, during the same period, thirty-two cases of relapsing fever and ten of typhus; and from Field Lane, in the City of London, in a corresponding period, twenty-three cases of relapsing fever and thirty-nine of typhus. Among other localities for typhus and relapsing fever may be mentioned Gray's Inn Lane and Spread Eagle Court in Holborn; Plum Tree Court, in the city of London; Brill Place and Court, in St. Pancras; Devonshire Street, in Marylebone; Old Gravel Lane in St. George's in the East, and many courts and lanes in Lambeth.

This predominance of typhus and relapsing fever in the overcrowded dwellings of the poor has been a matter of universal observation in all places. In Edinburgh, where there is a greater separation between the dwellings of the rich and of the poor than in almost any city I know, typhus, even in the midst of the greatest epidemics, is for the most part confined to the most wretched closes of the "old town." Dr. Christison even informs us that out of about a hundred instances, in which he had known typhus introduced into the dwellings of the better classes by medical

1 On 'Contagious Fever,' Lond., 1818.
students who had contracted it by contagion, he was not acquainted with one in which the disease had been propagated.¹

Typhoid fever, on the other hand, exhibits no such predilection for overcrowded localities. It prevails in the less populous districts equally with the most crowded, and consequently, as we approach the suburban districts of London, the number of cases of typhoid equals or exceeds that of typhus. Thus, of 16 cases from Hampstead, 4 were typhus and 10 typhoid; of 83 cases from Hackney, 22 were typhus or relapsing, 53 typhoid; of 6 cases from Bow, 5 were typhoid and none typhus; and of 177 cases from Camberwell, 80 were typhus or relapsing, 75 typhoid; while of 51 cases admitted from beyond the London districts, 8 were typhus and 36 typhoid.

Of the London districts Paddington and Belgravia may be regarded as two of the least populous, and at the same time as inhabited by the better classes of the community. Now of 6 cases of fever from Belgravia, 4 were typhoid and 1 typhus; and of 14 cases from Paddington, 12 were typhoid and 1 typhus. That typhoid is the prevailing fever in each of these districts is also shown by the cases admitted into their local hospitals. By the published reports of St. George’s Hospital,² situated in Belgravia, I find that out of 44 fatal cases of fever dissected during three years, there was ulceration of Peyer’s patches in 29, and in 5 only were the intestines perfectly healthy. With regard to Paddington, the following table shows the forms of fever admitted into St. Mary’s Hospital during the last five years from that parish. From this table, for which I am indebted to the kindness of Dr. Sanderson, the medical registrar to the hospital, all the cases of fever admitted from other districts, except Paddington, have been excluded.

TABLE XIV.

<table>
<thead>
<tr>
<th></th>
<th>1853</th>
<th>1854</th>
<th>1855</th>
<th>1856</th>
<th>1857</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Typhus</td>
<td></td>
<td>1</td>
<td>1?</td>
<td>2</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Typhoid</td>
<td></td>
<td>15</td>
<td>20</td>
<td>12</td>
<td>14</td>
<td>14</td>
</tr>
<tr>
<td>Febrisola</td>
<td></td>
<td>1</td>
<td>6</td>
<td>5</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Doubtful cases</td>
<td>111</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>3</td>
<td>17</td>
</tr>
<tr>
<td>Total</td>
<td>27</td>
<td>28</td>
<td>19</td>
<td>20</td>
<td>23</td>
<td>117</td>
</tr>
</tbody>
</table>

It appears from this table, then, that during five years, 75 cases of typhoid fever have been admitted from Paddington into St. Mary's Hospital, and only 5 of typhus. Also, in the year 1856, only 2 cases of typhus were admitted, while in the same year, there were admitted into the London Fever Hospital, 1062 cases.

Shoreditch is another district where typhoid fever appears to be most prevalent; yet its population is considerable, and it is certainly not one of the most aristocratic quarters of the Metropolis. Of 33 cases of fever admitted from this district, 28 were typhoid, and only 4 typhus. Dr. Barnes, also, who has been medical officer of health of the district for two years, writes to me "the form of fever I have seen is always typhoid." I have reason to believe, however, that this district does not escape epidemics of typhus, and the explanation why such a small proportion of typhus cases should have been admitted into the Fever Hospital probably consists in the fact that the majority of the pauper fever cases have been treated in a fever hospital attached to the workhouse, and only those sent to the Fever Hospital who were able to pay for admission. Still the prevalence of typhoid fever in this district is remarkable, and, as will shortly be shown, there appear to be local causes sufficient to account for it.

1 Of the majority of the cases marked "doubtful," no records have been preserved.
In connection with the subject of locality, I may also mention, that a careful study of a great number of recorded outbreaks of fever in country towns and villages throughout England, has convinced me that these outbreaks are almost invariably typhoid. It is also a fact worthy of notice, that several instances have come under my own observation of typhoid fever making its appearance in an isolated house in the country, in a family living in easy circumstances without any traceable source of contagion; of its attacking several individuals, and then disappearing without spreading beyond that house. Dr. Bartlett, also makes similar observations, as the results of his experience in America.\(^1\) On the other hand, I am acquainted with no instances of typhus or relapsing fever originating in this way.

Such cases as those just alluded to point to the probability of some local cause being capable of generating typhoid. This probability is still further confirmed by such facts as the following. It not unfrequently happens, that isolated cases of typhoid fever originate in the same house year after year, quite independently of any prevalence or absence of epidemic typhoid in the neighbourhood. I have come across several curious instances of this sort, in going over the list of residences of the cases admitted during the ten years into the Fever Hospital. One of the most remarkable is the following. Six cases of typhoid fever have been admitted, during the period above mentioned, from a single house in High Street, Shoreditch; one, in June 1849; one in October, 1851; one in February, 1854; one in November, 1855; one in November, 1856; and a sixth in July, 1857. There are of course many instances of a number of cases of typhus, coming from the same house within a few days, weeks, or even months of each other;\(^2\) but I have met with no instance in which cases were

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1 Bartlett, op. cit., p. 90.
2 For example, in 1857, seven of one family were admitted with typhus on the same day. Their ages varied from 8 to 50, and all had well-marked mulberry rash.
admitted at lengthened intervals from the same house, in the manner just specified with regard to typhoid fever.

My investigations also, quite bear out the results arrived at by Dr. Jenner, in an able paper recorded in the thirty-third volume of the 'Transactions of the Royal Medical and Chirurgical Society,' as regards the origin of typhus and typhoid fever from different foci of infection. In no single instance during the ten years, have I met with a case of typhus and typhoid fever admitted from the same family or even from the same house, except (and the exceptions have been only one or two) after the lapse of many months or even years. I am aware that this is contrary to the asserted experience of some physicians, who would have us believe that they are frequently observing both these forms of fever simultaneously in the same family; but I am not acquainted with any observations of this nature, sufficiently complete or authentic in their details to warrant their being relied on.

On the other hand, I am convinced, that the same law does not hold good with regard to typhus and relapsing fever. We have already found that both these forms frequently coexist as epidemics; so also they may occur simultaneously in the same localities, houses, and even families. Instances of this nature I know have occurred to the Scotch physicians, who have had such ample experience in both fevers; and they

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1 To this statement there were two exceptions, but both of these tended to confirm the rule laid down rather than otherwise. One of these will be found in Dr. Jenner’s paper above alluded to. The circumstances of the other were as follows. In November and December, 1851, four servants were admitted from an hotel in the Haymarket, all with typhoid, and in the following January, a servant was admitted from the same house with typhus. This typhus patient, however, was one of the same four who had been admitted in the previous year with typhoid. She had only left the Fever Hospital about ten days previous to her re-admission; and she had no doubt contracted typhus there during her convalescence.

2 Dr. Allison, speaking of the Edinburgh Epidemic of 1843, remarks that he had seen two cases of typhus, “with the characteristic eruption,
are not wanting in the records of the London Fever Hospital.

Many of the cases entered as "Febricula" have come from the same houses as one of the other forms of fever; but more especially from the houses furnishing relapsing and typhus cases.

Perhaps I cannot better illustrate the remarks just made, than by the following tabular arrangement of the admissions from certain limited districts during the year 1852. The numbers are those of the houses in the different courts or streets; their frequency of repetition denotes the number of cases. (R.S. means Ragged School.)

<table>
<thead>
<tr>
<th></th>
<th>Typhus</th>
<th>Relapsing</th>
<th>Typhoid</th>
<th>Febricula</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pheasant Court,</td>
<td>March, 3, 3, 7</td>
<td>April, 3, 6, May</td>
<td></td>
<td>March, 2,</td>
</tr>
<tr>
<td>Holborn, 1852.</td>
<td>April, 7, 7</td>
<td>6, June, 7, 3,</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>July, 6, 7</td>
<td></td>
<td>April, 7, May,</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>6.</td>
</tr>
<tr>
<td>Tyndall's</td>
<td>Jan., 14, Feb, 9</td>
<td>Feb., 11, 17, 17</td>
<td></td>
<td>March, 14,</td>
</tr>
<tr>
<td>Buildings,</td>
<td>March, 1, 9, 5</td>
<td>March, 6, 6, 6,</td>
<td></td>
<td>April, 9, 9,</td>
</tr>
<tr>
<td>Holborn, 1852.</td>
<td>April, 9, 14, 9</td>
<td>17, 6, May, 12,</td>
<td></td>
<td>9, 6, 9, July,</td>
</tr>
<tr>
<td></td>
<td>June, 14</td>
<td>July, 6</td>
<td></td>
<td>1.</td>
</tr>
<tr>
<td>Field Lane,</td>
<td>March, R.S.,</td>
<td>Jan., R.S., R.S.</td>
<td></td>
<td>April, R.S.,</td>
</tr>
<tr>
<td>City, 1852.</td>
<td>R.S., R.S., R.S.</td>
<td>Feb., R.S., R.S.,</td>
<td></td>
<td>May, R.S.,</td>
</tr>
<tr>
<td></td>
<td>R.S., R.S., R.S.</td>
<td>R.S., R.S., R.S.</td>
<td></td>
<td>R.S., June, 26,</td>
</tr>
<tr>
<td></td>
<td>R.S., R.S., R.S.</td>
<td>R.S., R.S., R.S.</td>
<td></td>
<td>Dec., R.S.</td>
</tr>
<tr>
<td></td>
<td>R.S., R.S., 27</td>
<td>R.S., April, R.S.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>27, June, 27</td>
<td>May, R.S.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>27, July, R.S.,</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>R.S., Oct., 27</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Nov., R.S.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High Street,</td>
<td>May, 84</td>
<td></td>
<td>August, 61, 61,</td>
<td></td>
</tr>
<tr>
<td>Shoreditch, 1852.</td>
<td></td>
<td></td>
<td>152, 178,</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Sept., 152.</td>
<td></td>
</tr>
<tr>
<td>Paddington, 1851.</td>
<td></td>
<td></td>
<td>Six cases from</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>different</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>houses.</td>
<td></td>
</tr>
</tbody>
</table>

brought from the same room in which a succession of relapsing cases had occurred at the same time." (‘Edin. Monthly Journ. of Med. Sc.,’ vol. iv, p. 253.) Dr. Henderson's observations, however, of the same epidemic were of an opposite nature.
VIII. Overcrowding, with Deficient Ventilation and Destitution.

After what has already been said under the head of locality, it would be superfluous to add much in proof of the influence of overcrowding on the prevalence of typhus and relapsing fever.

It is a fact, which is universally admitted, that overcrowding of human beings has an immense influence over the propagation of typhus by contagion;¹ but there are some, who maintain that the specific poison must first be introduced from without, and that it cannot in this way be generated de novo; among whom I may mention more particularly the name of Bancroft,² whose opinions have more recently been endorsed by Dr. Watson.³ The arguments, however, which have been brought forward in support of this view, are for the most part of a negative character; and it is not a little curious to observe, that Bancroft has endeavoured to contort the same facts in his favour, which other writers have adduced to show that typhus was not contagious, but always originated from impure air. In such a question, a few positive facts are worth a thousand arguments. Now, there are many instances on record, of typhus originating in jails, hospitals, armies, and transport ships, in which every source of contagion would seem to have been next to impossible.

Any one who will take the trouble of studying the records of the "black assizes," held at Cambridge in 1522, at Oxford in 1577, at Exeter in 1586, at Taunton in 1780, at Launceston in 1742, and at the Old Bailey in 1750, must, I think, be convinced, that the "jail fever," so far from being introduced from without, was generated de novo by

¹ See Graves's 'Clinical Lectures,' vol. i, p. 92.
² 'Essay on the Yellow Fever, and on Febrile Contagion,' 1811.
the overcrowding of the prisoners, and by them was communicated to all who came near them.\textsuperscript{1}

The distinguished writers on camp and jail fevers, during the last century, show, by their descriptions, that this disease was what we now called typhus, while they were all of opinion that overcrowding of itself could generate it.

Sir John Pringle, in his work on 'The Diseases of the Army,' makes the following statement, as the result of his extensive and acute observations: "The hospitals of an army when crowded with sick, or at any time when the air is confined, produce a fever of a malignant kind, often mortal. I have observed the same sort to arise in full and crowded barracks, and in transport ships, when filled beyond a due number, and detained by contrary winds, or when the men have been long at sea, under close hatches, in stormy weather." Pringle's description of this fever, including that of the eruption, shows clearly that he meant typhus.

Lind, also, notwithstanding his belief that typhus might in most cases be traced to contagion, has given several instances in which, without any traceable contagion, it originated in transport ships during long voyages, owing to overcrowding, and to the hatchways being kept down;\textsuperscript{2} and the typhus which raged among the troops landed in England after the battle of Corunna, was attributed to a similar cause.\textsuperscript{3}

But again, what other cause can be assigned for that murderous epidemic of typhus, which broke out in the ships in which the unfortunate French prisoners were confined at Plymouth, in the spring of 1810? Here there were no sources of contagion. The seclusion of the prisoners could hardly have been more complete, and the disease did not appear among them until after they had been confined for a lengthened period, and crowded together to such a degree,

\textsuperscript{1} In a paper published in the current volume of the 'Edinburgh Medical Journal' (vol. iv), I have given an account of these six "black assizes," and of many other outbreaks of typhus.
\textsuperscript{2} 'Two Papers on Fevers and Infection,' Lond., 1763.
that each individual had barely a space measuring five feet by two to lie upon; while the air of the deck, where they remained for thirteen hours out of the twenty-four, was so thick that a lighted candle appeared in it as through a thick mist. Certainly there was some justice in the epithet of "floating tombs" bestowed on these ships by the French.¹

In the winter of 1829-30, true typhus raged with much severity on board the French convict hulks at Toulon. The disease, by no means a common one in France, was unknown at the time in the town of Toulon, there not being a single case even among the workmen in the part. Overcrowding and deficient ventilation were the causes to which it was attributed.²

For numerous other illustrations bearing upon this point, I would refer to the elaborate treatise of Gaultier de Claubry;³ to a paper by Dr. Peebles;⁴ and to Dr. A. P. Stewart's memoir already quoted. I shall conclude with one other, the circumstances of which are in the memory of all. After the capture of Sebastopol, typhus ravaged the French and Russian armies with a fury which is described as unknown since the great epidemics of the imperial wars. We are told that this fever was attributed to two causes, overcrowding and a scorbutive diathesis; and one celebrated Russian physician asserted that, "in all cases overcrowding must be recognised, if not as the unique, yet as the essential and most active cause, of the epidemic."⁵ Jacquot, also, after his extensive observations among the French troops, remarks: "Le typhus naît toujours de l'encombrement, de la concentration des masses."—"On peut faire naître le typhus à volonté, pour ainsi dire."⁶

¹ Gaultier de Claubry, ' Analogies et différences entre le Typhus et la Fievre typhoïde,' Paris, 1838.
² 'Archiv. gén. de Méd.,' sér. i, tom. xxii, p. 265.
If typhus can only arise from a specific contagion, how are we to account for its origin in troop-ships, prisons, and armies, under the circumstances above mentioned, unless we are to believe, what seems impossible, that the specific poison of the disease is always and everywhere present, ready to manifest itself whenever, through the unwitting instrumentality of man, the conditions favorable to its propagation are presented to it. I cannot support the view which I advocate by a greater authority than that of one, whose opinion on such a matter must always be regarded with veneration and respect. I allude to Dr. Alison, of Edinburgh, who, in speaking of foul air and destitution in regard to typhus, says that it is highly probable that these causes are adequate not only to the extensive diffusion, but even to the generation of the disease, which is afterwards propagated by contagion.

Destitution is a most powerful predisposing cause of typhus, as has already been shown. Dr. Peebles has collected a number of instances of epidemics of typhus in Italy, immediately following great famines; and it is well known that most of the great epidemics which have devastated Ireland, and spread to Britain, have supervened upon seasons of scarcity and want; such, for example, as the epidemics of 1741, 1803, 1817, 1827, and 1847. In Dublin, in 1847, it was observed that those first attacked with typhus were those who had been reduced by insufficient food; while in many instances the fever first set in immediately on recovery from the effects of starvation.

After a careful study of numerous records of fever, I feel perfectly convinced that typhus may be generated de novo.

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1 'Pathology and Practice of Medicine,' 1844, p. 429.
4 'Dublin Quarterly Journal,' new ser., vol. viii, p. 3.
through the contamination of a confined portion of air by the pulmonary and cutaneous exhalations of numerous individuals; that the influence of this contamination is most marked when the body has been debilitated by want or previous disease; and that, consequently, seasons of famine have generally been followed by great epidemics of typhus among the poor.

Mere overcrowding for a limited period will not generate the poison of typhus. All experience goes to show, that a considerable time is necessary for its production. Hence typhus was not generated in the black hole of Calcutta in 1756, or on board the Irish steamer 'Londonderry' in 1848.

Relapsing fever never occurs in the epidemic form, except as the companion of typhus, and appears to originate from similar causes.

I am not, however, acquainted with any authenticated facts, to prove the influence of the conditions just enumerated over the prevalence of typhoid fever. We have found that it prevails among the wealthy, as well as among the poor; in country villages, as well as in the heart of populous cities. Every one who has paid any attention to the subject is well cognisant of such facts. Louis, the great French authority on typhoid fever, observes, that densely inhabited places cannot be regarded as producing it. Only one in eighteen of the patients, whose cases are given in his work, had been so circumstanced. We must therefore seek for the causes of its origin elsewhere. What these causes are Louis does not attempt to explain. Dr. Stewart remarks, in reference to them, "all is vague and uncertain;" and Dr. Tweedie, in the Lumleian lectures, delivered at the Royal College of Physicians during the present month, spoke of them as obscure and unknown. (March, 1858.) I have already shown that the causes of typhoid, whatever they may be, differ from those of typhus, in being for the most part of a very limited and circumscribed character. What these causes really are, I shall endeavour to explain under the following head.
IX. *Putrid Emanations from decomposing organic matter in Drains, Cesspools, Churchyards, &c.; and Organic Impurities in Drinking-water.*

Many of the older medical writers have recorded cases of fever as originating from exposure to the effluvia from decomposing organic matter;¹ but, generally, the symptoms of the fever have not been detailed with sufficient clearness to enable us to decide on what was its nature. Within the last ten or twelve years, however, many outbreaks of fever attributed to such a cause have been reported, while the symptoms of the fever have been recorded with such minuteness as to render its nature unmistakable. I find, on careful examination, that in every instance where this has been the case the fever has been typhoid; in none is there the slightest indication that it was typhus or relapsing. A brief résumé of the evidence bearing upon this point may be of service.

Many years ago, MM. Gaspard and Majendie² showed that, by injecting putrid substances into the veins of animals, symptoms very similar to those of typhoid fever might be induced; and that, after death, the intestines were much congested; and more recently the same results have been obtained by M. D'Arçet, by injecting into the veins putrid pus.³ Louis,⁴ however, denies that in Gaspard's cases the characteristic lesions of typhoid fever were present; but he himself mentions the case of a man who, for six months, had been in the habit of drinking large quantities of a very putrid infusion of straw, for the cure of a gleet. At the end of this time he died, with symptoms of typhoid fever; and, after death, Peyer's patches were found enlarged and ulcerated. Louis seems to attach great importance to this

¹ A collection of such cases will be found in Sir John Pringle's 'Diseases of the Army,' pp. 324, 328.
² 'Journal de Physiologie,' vol. ii, p. 1, and vol. iii, p. 81.
case, although he is inclined to regard the disease and its alleged cause as mere coincidences.

In the first volume of the 'Transactions of the French Royal Academy of Medicine,' an outbreak of fever, which occurred in 1749 among the girls of the 'Maison d'Enfant Jesus' is recorded. It was generally admitted to have resulted from the disgusting effluvia which proceeded from an adjoining field, in which a number of cattle had been buried, scarcely beneath the surface of the earth. That the fever was typhoid there seems little doubt from the following symptoms which characterised it, viz., fever, with great prostration, tympanites, abdominal pain, diarrhoea, and melena. Thirty of the girls were seized all at once, shortly after the interment of the cattle.¹

In the 'Gazette Médicale' for 1834, M. Ruef gives the details of an epidemic of typhoid fever, which occurred at Bischofsheim, in the department of the Lower Rhine, during the autumn of 1832. The disease first showed itself without suspicion of being transmitted in the upper, best aired, and, as is expressly said, "the most healthy part of the village," which is situated partly on, and partly at the foot of, a rising ground, and it spread successively to the middle and lower quarters. The disease was attributed to two causes: first to the cemetery which was situated in the centre of the village, and in the immediate vicinity of which the greatest number of cases occurred; and secondly, to the pipes conveying the water to two fountains, one in the centre of the village, and the other at the bottom of the hill. These pipes were of wood, and passed at but little depth below the cesspools of the place.

Dr. Southwood Smith, in evidence given in 1843, before the Health of Towns Commission, stated: "I have been struck with the number of cases of fever in houses opposite gully holes." He adds, that servants were being continually admitted into the London Fever Hospital from houses of the upper class so situated; and that he had generally found that only they who had slept on the ground-

¹ M. de Lassone, 'Mém. de la Soc. Roy. de Méd.,' tom. i, p. 97.
floor were attacked. Although Dr. Smith says nothing as to the nature of the fever, yet, from what has already appeared in the course of this paper, there can be little doubt that the cases he alluded to were typhoid. Out of the total servants admitted from houses of the better class into the London Fever Hospital, during ten years, there were 118 cases of typhoid to 13 of typhus.

On the 14th of July, 1845, M. Ançelon, a French physician, communicated to the Academy of Sciences at Paris some remarkable facts connected with epidemics of typhoid fever observed in the commune of Guermange, in the duchy of Lorraine. Many years before, typhoid fever had been constantly endemic in this place, making its appearance every year during the hot season; but for twenty-five years, it had entirely disappeared from the northern part of the commune; and its disappearance had been simultaneous with the suppression of a stagnant pond in that locality. At the southern part of the commune, however, there had been epidemics of typhoid fever every third year, viz., in 1830-33-36-39 and 42, always in the hot weather. Now, at this part of the commune there was a large lake, called the "Indre-basse," which, every third year, was emptied and cultivated, and afterwards the water was allowed to collect again for two years more. The appearance of the epidemics coincided with the second year, in which the lake was full of water, and the author endeavoured to account for the phenomenon by the action of the heat and moisture upon an immense quantity of animal and vegetable débris, which, during the two years, had been collecting upon the banks of the lake. The houses also in the commune were not sufficiently elevated, damp, and badly drained.

Mr. Shearman has given an account of an outbreak of fever which occurred at Rotherham in 1845, and which, from the symptoms, was undoubtedly typhoid. The fever

2 'Compt. Rend. de l'Acad. des Sciences,' tom. xxi, p. 158, "Notes sur les maladies endémiques périodiquement développées par les émanations de l'étang de l'Indre Basse."
was attributed to the imperfect drainage and deficient supply of water. It commenced in July, and for several months before, the drains had been open for repair, and had emitted a suffocating stench. Moreover, in the overcrowded churchyard, situated in the centre of the village, bodies were often exposed half decayed, and frequently large quantities of a horridly-smelling liquid were taken out of a newly-made grave and poured down into the street-sewers. In July, 1841, an outbreak of the same fever had occurred in the vicinity of this churchyard, immediately after the opening of fifty graves. The fever occurred in the same localities where cholera had prevailed in 1832.\(^1\)

A remarkable instance of a limited outbreak of fever was recorded by Dr. Christison in 1846. It occurred in an isolated farm-house in the thinly-peopled county of Peebles, N.B. Every one of the fifteen residents of the house were seized with fever, and three died. Many also of the servants who worked during the day at the farm were also affected, but none of them communicated the disease to their families, who did not visit the farm. The only explanation of this outbreak—which, however, Dr. Christison considered satisfactory—was that "the drains and sewers were found all closed up, and obstructed with the accumulated filth proceeding from the privies and farm-yard," the effluvia from which were very offensive. In this fever there was no diarrhoea nor abdominal tenderness; but both these symptoms may be absent in typhoid fever, and Dr. Christison observes that its "want of resemblance to the habitudes of ordinary epidemic typhus struck the attention as something very remarkable;" and that "the leading symptoms were those of great gastro-intestinal derangement," so much so, that suspicions of poisoning were entertained. Moreover, the lengthened duration of the cases, the clearness of the intellect, and the marked absence of prostration, oppression, and delirium, seem to leave little doubt that the fever was typhoid. This affection, it should be noted, had not at that period attracted much attention in this country.\(^2\)

ETIOLOGY OF CONTINUED FEVER.

During the autumn of 1846, numerous outbreaks of undoubted typhoid fever were recorded as occurring in many small towns and villages throughout England. The summer and autumn of this year were remarkable for their excessive heat, which favoured the decomposition of organic matter in open drains and privies. In every one of the published accounts of these outbreaks distinct mention is made of this circumstance. Dr. Sibson, in his notice of the outbreak at Nottingham, says that "the infected districts were offensive to the smell." Mr. Taylor, in his account of the fever at Old Lenton, says that there were stagnant pools loaded with decaying organic matter, and sending up the most filthy emanations; and that at New Lenton, where the fever also prevailed, "there were nuisances innumerable—stagnant pools of water, privies pouring out their offensive and bad air." Mr. Alfred Turner writes that at Minchinhampton the privies at the back of the houses in which many of the fever cases occurred "exhaled an intolerable stench." With regard to this last place, also, another circumstance deserves to be mentioned. In the middle of the hot weather about a thousand cartloads of earth had been taken from the churchyard, and scattered as manure over the neighbouring fields and gardens. The fever first appeared immediately after this; and, among others, the rector's wife, daughter, and gardener died of it. Mr. Daniel Smith, who was then practising at Minchinhampton, informs me that he has no doubt in his own mind that this was the cause of the fever. Mr. Smith also tells me that, during the same year, he knew of several instances of typhoid fever, originating in isolated houses, in some of the most elevated districts of Gloucestershire, and that he invariably succeeded in accounting for them by some glaring defect in the drainage or cesspools. Towards the end of this same autumn, an unusual number of typhoid cases were

observed in Edinburgh and Glasgow, along with the typhus at that time epidemic. It is important to notice that typhoid was at the same time very prevalent in the country towns of England, without any typhus.

About Easter, 1848, a formidable outbreak of fever occurred in the Westminster School and Abbey Cloisters; and for some days there was a perfect panic in the neighbourhood respecting what was called the "Westminster Fever." Within a little more than eleven days it affected thirty-six persons, all of the better class; and in three instances it proved fatal. Shortly before its first appearance, "there occurred two or three days of peculiarly hot weather," and a disagreeable stench, so powerful as to induce nausea, was complained of in the houses in question. It was found that the disease followed very exactly in its course the line of a foul and neglected sewer, in which fecal matter had been accumulating for years without any exit, and which communicated by direct openings with the drains of all the houses in which it occurred. The only exception was that of several boys who lived in a house at a little distance, but who were in the habit of playing every day in a yard, in which there were gully-holes opening into the foul drain. The Metropolitan Sanitary Commission gave it as their decided opinion, that the epidemic "arose from the bad state of the sewers and drains of the precinct, and especially from the foul condition of the large sewer described."¹ Dr. Watson also expresses his belief, that the "Westminster Fever" was due to the effluvia from this drain; but he does not consider that the cases were continued fever at all.² Dr. Watson, however, only saw one of the cases; and it is to be remembered that he expressed the above opinion before he recognised typhoid fever as distinct from typhus. Dr. Todd, Dr. Fincham, and Mr. McCann were the other medical men consulted. Dr. Todd, who saw five or six of the cases, tells me that they were unquestionably examples

ETIOLOGY OF CONTINUED FEVER. 269

of typhoid. Dr. Fincham, who, by the way, also saw the case alluded to by Dr. Watson, writes to me that all the cases which he saw "were unquestionably examples of typhoid fever. In all, the bowel complication (the diarrhoea, &c.) was well marked. I believe that every case that occurred exhibited the same symptoms." The same opinion has been expressed to me by Mr. McCann.

Towards the end of the autumn of 1852, a fever broke out at Croydon, which attracted great attention, and was made the subject of various official reports by the Board of Health. That this fever was typhoid, is clearly shown by all the accounts of it which appeared; and also by the expressed opinion of a committee of the Epidemiological Society, consisting of Drs. A. P. Stewart, Jenner, and Sankey. Five cases, moreover, were admitted at this time into the London Fever Hospital, from Croydon: all were typhoid. Dr. Arnott and Mr. Page, C.E., in their report, stated, as their conviction, that the want of proper precautions, during the excavation of the new works connected with the sewerage, had been influential in producing the disease. Numerous instances were mentioned, in which the disease immediately followed exposure to the fetid emanations liberated during the opening and emptying of cesspools, the cleansing of old drains and open ditches, and to the foul gases which were forced into the houses when any obstruction took place in the drain-pipes. Mr. Carpenter, of Croydon, has adduced facts, which render it highly probable that, in many cases, the fever was owing to the contamination of the drinking-water in the wells, from the disturbance of the drains; and the same view has been ably advocated by Dr. Snow.1

Dr. Beadle has described a fever which broke out at Tewkesbury, in August, 1853. His description leaves no doubt that it was typhoid. Many of the houses in which

the cases occurred were carefully examined, and were invariably found to contain nuisances of the most gross description. A clergyman who was present at the inspection of one of them exclaimed, that it "breathed out typhus."¹

On April 2d, 1855, Dr. Camps communicated to the Epidemiological Society an account of a very remarkable outbreak of typhoid fever, which had occurred at Cowbridge, in Wales, in November, 1853. Two balls had been held at the hotel of this town, and had been attended by about 140 persons, from all parts of the surrounding country. Shortly after, many of these persons were seized with fever, presenting all the symptoms of typhoid, and about eight died. This fever was not prevalent at the time, and it only attacked those who had attended the balls, some of whom were not taken ill until after their return to their homes in Devon and Somerset. An inspection of the hotel was made by order of the local authorities; and it turned out, that the supper-room was merely a temporary transformation of a loft over a seven-stalled stable; and that the passage between it and the ball-room was built over a large tank, which collected the water from the roof of the house.²

In this case, also, there were suspicions of poisoning.

At the same meeting of the Epidemiological Society, Dr. Brown read a paper on the prevalence of typhoid fever at Rochester and Stroud, which he attributed to bad drainage. Typhoid, in fact, he considered as nothing else than "night-soil fever," and he thought that every instance of it might be traced to such an origin.³

Dr. Routh mentions an instance in which typhoid fever broke out in a house at Hastings, where at the time the disease was unknown. Six persons in the house were attacked. "The origin of the disease was traced to a direct communication between the cistern of the water

¹ 'Association Journal,' 1853, p. 793.
² 'Trans. Epid. Soc.,' in second vol. of 'Sanitary Review.'
³ 'Med. Times and Gazette, vol. xxxi, p. 447.'
drunk by the inmates and the pipe from the water-closet." The ventilation of the house was perfect.\textsuperscript{1}

In October, 1856, typhoid fever made its appearance in the Clergy Orphan School, at St. John's Wood. The school had only just reopened after the holidays. Nineteen of the pupils were taken ill within thirty-six hours. The drains of the building had been taken up for repairs during the holidays; and a "close, damp, oppressive" atmosphere was observed in the house at the time the outbreak commenced.\textsuperscript{2}

In the spring, 1857, a number of strangers came to reside at the National Hotel, Washington, in order to be present at the inauguration of Mr. Buchanan as President of the United States. A large number of them were seized almost at the same time with typhoid fever, including the President elect himself. Rumours were rife that they had all been poisoned; at first it was said with arsenic, for some political purpose; and then by copper, from the culinary utensils. A rigorous investigation ensued; and the result was, that both the committee appointed for this purpose and all the medical attendants coincided in the belief, that the disease was due to noxious exhalations from a sewer. At one part of the building there was a direct opening into this sewer, and through this a strong current of fetid air was distinctly perceptible. The fever first appeared after three very warm days, during one of which the rain fell in torrents. The sudden rise of the river Potomac, into which the sewer opened, was thought to have driven back the noxious vapours through the gully-hole.\textsuperscript{3}

In the 'Weekly Return of the Health of the Metropolis,' published by the Board of Health, for October 24th, 1857, the following fact is communicated by Dr. Todd: "One day last week, I saw in a suburban district four cases of

\textsuperscript{1} 'Assoc. Journ.,' 1856, p. 763.
\textsuperscript{2} 'Lancet,' Nov. 15th, 1856.
typhoid fever in one house; and of these, one had already ended fatally. Close by were the cuttings of a new railway, which had opened into a large drain." Dr. Todd also informs me, that not long ago he was called to see some cases of typhoid fever at Thames Ditton, a country village fourteen miles from London. Two servants and two young ladies had been seized with it, and one of the former had died. At the bottom of the garden, behind the house, was an open drain, and for some time the wind had been blowing from this in the direction of the house. On another occasion, Dr. Todd saw a gentleman with typhoid fever at Wareham, who afterwards died. At the time he was taken ill the drains in his house were undergoing repairs.

Out of 65 cases of fever, recorded by Dr. Jenner, in only two is there any mention made of the effluvia from drains as a possible cause. Both of these were typhoid. In Case 25 the patient had been taken ill a few hours after working in a cellar, where he had "observed a most offensive odour;″ and the house from which Case 26 came, Dr. Jenner found, from personal inspection, to be filthy and offensive, the inhabitants complaining bitterly of the "offensive sewer." Several other cases had occurred in this same house.1

According to Dr. Peacock's experience, typhoid fever generally arises "in persons living or working in low, ill-drained localities or houses, and especially in persons working in under-ground cellars, or in the sewers, or in damp situations, near the bank of rivers or canals."2

The cases admitted into the London Fever Hospital furnish many examples of typhoid fever, traceable to such causes as those we have been considering.

In November, 1851, a porter and three housemaids were admitted with typhoid fever from a hotel in the Haymarket. The proprietor of the hotel informs me, that the drains in the house were at that time very defective, so much so as to be often offensive. They were shortly after thoroughly

1 'Med. Times and Gaz.,' vol. xxi, p. 235.
repaired. The servants slept on the ground-floor, below the level of the street.

Several cases of typhoid fever have been admitted, at long intervals, both into St. Mary's and the Fever Hospitals, from Brooks Mews, Paddington. The houses are built over the course of a large drain, and several of them are cowsheds.

Some years ago, a number of servants were admitted with typhoid fever, both into St. Mary's and the Fever Hospitals, from several of the best houses in Oxford Square and Hyde Park Square. I find that there are gully holes, and that the sewers terminate in cul-de-sacs opposite those very houses which furnished the cases; and that, at the period in question, a quantity of sewage had accumulated in these cul-de-sacs, and that there was a "back vent" from the sewers into the houses.

During 1857, six policemen were admitted into the Fever Hospital from the Peckam Police Station, with typhoid fever: 3 in June, 1 in July, 1 in August, and 1 in September. On inquiry, it was stated that there was no defect in the drainage of the building. The men, however, affirmed that they had often complained of dreadful odours in the room in which they sat. I accordingly applied to Dr. Bristowe, the Officer of Health for the district, to have the building carefully examined. The result of this examination was the discovery that a water-closet on the ground-floor emptied itself, not into the main drain, but into an old well, immediately underneath the passage adjoining the room in question. Here an accumulation of upwards of ten feet of soil had taken place; and the top of the well was merely covered by the flagstones of the passage.

In the beginning of the present month (March, 1858), a mother, with her three children, was admitted with typhoid fever, into the Middlesex Hospital, under Dr. A. P. Stewart. Dr. Stewart and myself visited the house in Dudley Street, Soho, from which they had come. The family had resided in two moderately sized rooms on the street floor. On entering the back room in which they had slept, we were both struck with an overpowering smell; and on going down...
stairs, we found that immediately below the bedroom there was a large dust-bin, in which the refuse of all the residents in the house had been accumulating for months; 2d, a water-closet, of which the handle by which the water was let on had been broken a month before; 3d, the water-cistern of the house without any cover; and lastly, we ascertained that a fortnight before, the drain by which the water-closet communicated with the main sewer had been taken up for repairs.

The records of St. Mary's Hospital show that typhoid fever is very common in those streets which are in the immediate vicinity of the Paddington Canal Basin, concerning which extract the following from Dr. Sanderson's 'Sanitary Report:"

"The canal basin may be described as a stagnant fetid pool its water contains a large quantity of animal and other organic impurities, and from its surface every breeze carries noxious emanations. It receives the offensive drainage from the slop-yards, lay-stalls, and dust-wharves on the banks, and serves as a common cesspool to the numerous inhabitants of the barges. In the dust-wharves just mentioned the refuse of a large portion of the metropolis is collected.""

Lastly, we have found that many cases of typhoid fever have been admitted into the Fever Hospital from Shoreditch. Dr. Barnes, in his sanitary report for that district, informs us that the whole surface-soil is composed of foul earth, which he calls the "pest-stratum," and which varies in thickness from one to sixteen feet. "This soil is sodden with fecal matter—the soakage of cesspools; and more or less contamination from this and other sources must necessarily reach the wells;" while many parts of the district are badly drained.

The array of evidence which has been brought forward, in my opinion, demonstrates, as clearly as can be, that typhoid fever is often, if not always, generated by the putrid emanations from drains and other sources, or by decomposing organic matter in drinking-water. ¹ If this view be

¹ Since writing the above, I find that Dr. W. Budd, of Bristol, has long been of opinion that typhoid fever may be propagated by means of sewers,
correct, it at once explains why typhoid fever attacks the rich as well as the poor; why it occurs in solitary houses in the country, as well as in the centre of great cities; and why it should always be most prevalent in the autumn season. The reason why this cause has not hitherto been generally recognised lies in the circumstance, that typhus and typhoid fever are so very generally confounded together. Those who deny the possibility of "fever" originating from putrid emanations, appeal to thousands of typhus cases as overwhelming negative evidence; in the same way as there are not wanting a few, who bring forward typhoid cases to prove that "fever" is independent of overcrowding and destitution. One might as well argue that profound coma does not result from an overdose of opium, on the ground that in a host of cases a somewhat similar condition is totally independent of such a cause.

If, then, overcrowding with deficient ventilation and destitution may generate typhus, and typhoid fever may result from putrid emanations, it comes to be a question whether these several causes combined may not give rise to a disease partaking of the characters of both. I am inclined to think that such may be possible; but it is certainly very rarely the case. The grounds on which this supposition is based are as yet few, and I would submit them with all deference. At the same time, I consider them of sufficient importance to justify me in bringing them forward.

In the month of December last, a girl, aged 16, was admitted into the Fever Hospital from 17, Windmill Row, Lambeth; ill a week. Her body was covered with an unmistakeable mulberry rash, and she presented all the usual privies, and water-closets; yet he believes "that the effluvia from defective sewers have no power to communicate this specific fever, except when they are charged with the specific poison contained in the secretions from the diseased bowels of persons already affected." ('Association Journ.,' 1855, p. 208.) It seems difficult, however, on such a view, to account for the outbreak of the disease in isolated country houses, where there has been no traceable source of introduction, or for the influence of autumn over its prevalence.
symptoms of typhus—dry, brown tongue; confined, bowels; heavy confused expression; small pupils; and low, wandering delirium. The case attracted particular notice; as typhus was at that time very uncommon. Two days after, the symptoms underwent a complete change. The mulberry rash (which was certainly not the scarlet rash which occasionally precedes the eruption of typhoid) faded, and was succeeded by rose spots, which came out in successive crops for more than a week, and were accompanied by diarrhoea and abdominal tenderness. The tongue became moist and red; the pupils, dilated; and the drowsiness and wandering vanished. This girl was a hawker; for some weeks had been very destitute, and a fortnight before, she had slept for two or three nights at another house, in the same bed with a girl who had "fever." This second girl, with her mother and sisters, was admitted into the Lambeth Workhouse; but the father and brother were admitted into the Fever Hospital, with well-marked typhus. On the other hand, Dr. Odling, the officer of health for Lambeth, informs me that the courtway in front of No. 17, Windmill Row, is badly paved and badly drained; and that, although the cesspools in the house have been done away with, the habits of the inmates have rendered the privy arrangements as insalubrious as before. The girl was therefore exposed both to the contagion of typhus, and to the causes which there is reason to believe generate typhoid.

Again, an outbreak of fever occurred last autumn in Dudley Street, Paddington, in which I am assured by Dr. Sanderson\(^1\) that there were some cases which presented the characters of both typhus and typhoid, including the presence of the two eruptions. Now, I have shown that in Paddington there are various causes to account for the generation of typhoid, and that typhus is there extremely rare. It becomes interesting, then, to ascertain under what circumstances typhus, or something resembling it, may there originate. Now, in the houses in which these "mixed cases" occurred, the two causes which I have supposed to generate typhus

\(^1\) Dr. S. has had ample opportunities of studying the two fevers at the London Fever Hospital.
and typhoid were present in a marked degree. First, the residents were principally dustwomen, and the houses were daily stocked with selections from the street sweepings of the metropolis, such as old grease pots, &c., materials sufficiently prone to decomposition in hot weather. But, secondly, these two houses were overcrowded to such a degree, that compulsory measures had to be adopted to diminish the number of inmates. Cases of fever occurred at the same time in other houses of the same street, which were not overcrowded; but these were pure examples of typhoid.

Lastly, M. Landouzy\(^1\) has given an account of a remarkable fever which prevailed in the gaol at Rheims, in the autumn of 1840. Many of the symptoms during life, including the eruption, were those of typhus; but the intestines after death presented the lesions characteristic of typhoid. Now, from the locality and the season of the year, one would have expected typhoid fever; and in addition to these causes, we are informed that there was a most disagreeable odour in the gaol, proceeding from the grease of the woolen fabrics manufactured by the prisoners. That the fever was really typhoid is proved by the lesions found after death. On the other hand, a cause was not wanting to account for the symptoms of typhus during life; and, it must be remembered, that a copious mulberry rash would entirely mask a few rose coloured spots, even if these were present. The circumstance to which the fever was mainly attributed was the overcrowding of the prisoners. The number which the gaol was calculated to hold was from 130 to 150; but a month or two previous to the outbreak of the fever, this had been raised to 190.

Such cases as those just brought forward might be urged in support of the view, that typhus and typhoid are merely different manifestations of the same poison. They are, however, of such rarity, while at the same time all other evidence tends to establish the non-identity of the

\(^{1}\) 'Archiv. Gén. de Méd.,' third ser., vol. xiii, p 7.
poisons of the two diseases, that it seems more probable that they admit of another explanation. In the first place, on the supposition that the two diseases are distinct, there seems no reason why a person may not have both at the same time. It has recently been denied that two of the exanthemata can coexist in the same individual;¹ but the correctness of this assertion may be doubted. Several instances have occurred at the Fever Hospital of scarlet fever and typhoid attacking the same patient, the eruptions of the two diseases being present at one time; and Dr. Walshe has furnished me with the particulars of a case most carefully observed at University College Hospital, in which the eruptions of scarlet fever and smallpox existed at one and the same time.² Why, then, may not typhus and typhoid occasionally coexist? But, secondly, there is an important difference between these two fevers and the several exanthemata with which they are so frequently compared, which is this: the poisons of the former, whatever they are, can be produced by agencies which, to a certain extent, are under the control of the human will; but there is as yet no evidence to show that any agencies within our knowledge can generate the poison of scarlatina, measles, or smallpox. Now, if a certain poison can generate one group of symptoms, and another poison generate another, surely it is but reasonable to expect, that a combination of the two poisons may give rise to a morbid condition of an intermediate character, without its being necessary to conclude, from the existence of such a hybrid affection, that the first two morbid conditions have been merely different manifestations of the same poison.

X. Contagion.

Typhus and relapsing fever have been almost universally regarded as contagious, and some even go so far as to main-

¹ Dr. Chambers, 'Lancet,' Feb. 6th, 1858.
² Case of Caroline Lennell, 'Female Case Book, U. C. H.,' vol. ii, p. 217, June 23rd, 1847. Since the above was written, the records of several similar cases have been published. See 'Lancet,' Aug. 28th and Sep. 11th, 1858.
tain that they are invariably the product of contagion. On the other hand, much difference of opinion has existed with regard to typhoid. Many French observers, and amongst them Andral, have maintained that it was not contagious, whereas Dr. Nathan Smith, Bretonneau, Gendron, Louis, Gaultier de Claubry, Piedvache, and Bartlett, have considered it contagious. Dr. Peacock observes that he has never known typhoid fever communicated to the attendants in St. Thomas’s Hospital; and Chomel remarks, that during the nineteen years he had been connected with the Hotel Dieu, he had only known four cases of typhoid fever contracted in the wards.

What light do the statistics of the London Fever Hospital throw upon this point? During eight years and a quarter those cases of fever have been indicated in the register in which the disease was ascribed by the patients to contagion, or in which others were admitted from the same house with fever. On analysing these cases, I have obtained the following results:

<table>
<thead>
<tr>
<th>Disease</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Relapsing</td>
<td>440</td>
<td>17.1, or 39.7%</td>
</tr>
<tr>
<td>Typhoid</td>
<td>1576</td>
<td>20.4, or 33.7%</td>
</tr>
<tr>
<td>Febricula</td>
<td>736</td>
<td>8.4, or 13.4%</td>
</tr>
</tbody>
</table>

Out of 2811 cases of Typhus there were 729, or 25.13 per cent.

From this it would appear, that relapsing fever is nearly three times, and typhus more than twice, as contagious as typhoid. It is obvious, however, that but little dependence can be placed on a patient's own statement in such a matter. Many may be exposed to contagion without being aware of it; while, on the other hand, a number of patients being admitted from the same house or locality is

1 Bartlett, op. cit., p. 102.
3 Ibid.
4 Ibid.
6 'Mém. de l'Acad. de Méd.,' 1850.
9 'Med. Times,' vol. xxi, p. 20.
Etiology of continued fever.

No absolute proof of the contagiousness of the disease. A better criterion of the relative contagiousness of the different forms of fever will result from an examination of the cases which have originated in the hospital. With this object, I have constructed the following table, which shows the form of fever in those cases in which it has originated, during the ten years, among the nurses, medical attendants, or patients admitted with other diseases.

**Table XV.**

<table>
<thead>
<tr>
<th>Diseases contracted in Hospital in 10 years</th>
<th>Typhus</th>
<th>Relapsing</th>
<th>Typhoid</th>
<th>Febrile</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nurses</td>
<td>25</td>
<td>1</td>
<td>2</td>
<td>11</td>
</tr>
<tr>
<td>Porters and Servants</td>
<td>1</td>
<td>...</td>
<td>2</td>
<td>...</td>
</tr>
<tr>
<td>Medical Attendants</td>
<td>3?</td>
<td>...</td>
<td>1</td>
<td>...</td>
</tr>
<tr>
<td>Patients admitted with Typhus</td>
<td></td>
<td>...</td>
<td>...</td>
<td></td>
</tr>
<tr>
<td>&quot; &quot; Relapsing</td>
<td>1</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>&quot; &quot; Typhoid</td>
<td>6</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>&quot; &quot; Febricula</td>
<td>4</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>&quot; &quot; Scarletina</td>
<td>5</td>
<td>...</td>
<td>1</td>
<td>...</td>
</tr>
<tr>
<td>&quot; &quot; Pneumonia</td>
<td>1</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Total</td>
<td>48</td>
<td>1</td>
<td>8</td>
<td>11</td>
</tr>
</tbody>
</table>

The table shows, that during ten years forty-eight cases of typhus originated in the hospital, but only eight of typhoid fever. It might be difficult to account for the origin of most of these cases of typhoid, except by means of contagion; yet the infinitely (six times) more contagious nature of typhus is at once obvious. At the same time, when compared with other fever hospitals, the number of nurses attacked with typhus has been wonderfully small—a circumstance which is accounted for by the extreme airiness and perfect ventilation of the building. With regard to relapsing fever, the table is hardly a fair test, considering the small number of patients admitted. The records of various epidemics, however, testify to its being eminently contagious.

There can be little doubt, then, that typhoid fever is, in
a limited degree, contagious; and perhaps it may even be more so than the above table would indicate, for it must be remembered, that most of the nurses are beyond the age at which they would be likely to contract the disease. It would be an important question to decide, whether typhoid may not be propagated through the medium of the discharges of the bowels, in the same way, as there is good reason to believe is frequently the case, in cholera and dysentery. Dr. W. Budd, of Bristol, maintains that typhoid is generally propagated in this way; and I believe he has collected many facts (not published) which render the evidence as conclusive upon this point, as he has already shown it to be in the case of cholera. I would here call attention to the fact, that Dr. Austin Flint has recorded a remarkable instance, in which typhoid fever was apparently communicated to a number of families in a village of the United States through the medium of the water of a well, the only families who escaped being three, who derived their water from an independent source. The demonstration that typhoid might be communicated by the dejections, would constitute another marked distinction between it and typhus.

Table XV suggests many other points of interest in connexion with the identity or non-identity of typhus and typhoid fever. On these, time will not allow me to dilate. I would merely observe, that the fact of having passed through either form confers no immunity from the other. Several patients, after having become perfectly convalescent from typhoid, have contracted typhus while still in hospital, or have been re-admitted with it a week or ten days after dismissal. On the other hand, it is generally admitted that it is extremely rare for the same individual to have two attacks of either typhus or typhoid. I, myself, as far as I have been able to ascertain, am the only instance of an individual having had two attacks of undoubted typhus, on both occasions with a mulberry rash.

1 *Clinical Rep. on Cont. Fever;* Buffalo, 1862, p. 381.
XI. Recent Residence and Birth-place.

Louis strongly insists upon recent residence as a predisposing cause of typhoid fever. Out of 129 cases, which he gives in his work, 73 had not resided in Paris more than ten months, and 102 not more than twenty months.\(^1\) Again, of 92 cases of typhoid fever under Chomel, in the Hotel Dieu, one half had resided in Paris only one year, or less.\(^2\)

Table XVI shows the length of residence in London for each of the forms of fever admitted into the Fever Hospital, during the two years 1851 and 1857.

**Table XVI.**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>No.</td>
<td>No.</td>
<td>No.</td>
</tr>
<tr>
<td>Not exceeding 1 week</td>
<td>1 32</td>
<td>2 51</td>
<td>1 45</td>
<td>...</td>
</tr>
<tr>
<td>1 month</td>
<td>2 64</td>
<td>6 153</td>
<td>5 225</td>
<td>1 96</td>
</tr>
<tr>
<td>2 months</td>
<td>7 228</td>
<td>16 41</td>
<td>7 315</td>
<td>3 288</td>
</tr>
<tr>
<td>3 ditto</td>
<td>9 293</td>
<td>23 59</td>
<td>15 675</td>
<td>4 384</td>
</tr>
<tr>
<td>6 ditto</td>
<td>13 433</td>
<td>43 1102</td>
<td>29 1306</td>
<td>7 672</td>
</tr>
<tr>
<td>9 ditto</td>
<td>16 521</td>
<td>56 1436</td>
<td>38 1712</td>
<td>7 672</td>
</tr>
<tr>
<td>1 year</td>
<td>19 619</td>
<td>71 182</td>
<td>63 2858</td>
<td>12 1153</td>
</tr>
<tr>
<td>14 year</td>
<td>20 651</td>
<td>77 1974</td>
<td>72 3243</td>
<td>13 125</td>
</tr>
<tr>
<td>2 years</td>
<td>25 814</td>
<td>97 2487</td>
<td>107 492</td>
<td>16 1538</td>
</tr>
<tr>
<td>5 ditto</td>
<td>33 1075</td>
<td>141 3615</td>
<td>150 6757</td>
<td>22 2115</td>
</tr>
<tr>
<td>Above 10 years</td>
<td>49 1596</td>
<td>168 4307</td>
<td>164 7387</td>
<td>34 3269</td>
</tr>
<tr>
<td>Life</td>
<td>50 1628</td>
<td>28 716</td>
<td>25 1126</td>
<td>10 961</td>
</tr>
<tr>
<td></td>
<td>208 6775</td>
<td>194 4974</td>
<td>33 1486</td>
<td>60 5769</td>
</tr>
<tr>
<td>Total known</td>
<td>307 9999</td>
<td>390 9999</td>
<td>222 9999</td>
<td>104 9999</td>
</tr>
<tr>
<td>Doubtful</td>
<td>35 58</td>
<td>35 58</td>
<td>24 24</td>
<td></td>
</tr>
</tbody>
</table>

It would appear, then, that recent residence does predispose to typhoid fever in London; although not to such an extent as the results of Louis' and Chomel's obser-

---

\(^1\) Louis, op. cit., tom. ii, p. 357.

\(^2\) See Bartlett, op. cit., p. 110.
vation would render probable in Paris. Out of 390 cases, upwards of one ninth had not resided in London more than six months, and almost one fifth not more than a year. On the other hand, of 307 cases of typhus, only one sixteenth had resided less than a year. Of the typhus cases, 68 per cent. had resided in London all their lives; of the typhoid less than one half.

But recent residence would appear from the table to predispose more strongly to relapsing fever even than to typhoid. Thus, out of 222 cases, almost one eighth had not resided in London more than six months, and considerably upwards of one quarter not more than a year; only one seventh had resided in London all their lives. An explanation of this circumstance will be found by referring to the next table, which indicates the countries from which the subjects of each of the forms of fever came during the same two years—1851 and 1857.

**Table XVII.**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Natives of London</td>
<td>208</td>
<td>64·6</td>
<td>33</td>
<td>13·3·1</td>
</tr>
<tr>
<td>Ditto of rest of England</td>
<td>64</td>
<td>19·87</td>
<td>26</td>
<td>10·4·8</td>
</tr>
<tr>
<td>Ditto of Scotland.</td>
<td>2</td>
<td>0·62</td>
<td>1</td>
<td>0·4</td>
</tr>
<tr>
<td>Ditto of Ireland.</td>
<td>40</td>
<td>12·4·2</td>
<td>184</td>
<td>74·19</td>
</tr>
<tr>
<td>Foreigners</td>
<td>8</td>
<td>2·4·8</td>
<td>4</td>
<td>1·61</td>
</tr>
<tr>
<td>Total known</td>
<td>322</td>
<td>99·99</td>
<td>248</td>
<td>99·99</td>
</tr>
<tr>
<td>Doubtful</td>
<td>20</td>
<td>9</td>
<td>34</td>
<td>34</td>
</tr>
</tbody>
</table>

It is obvious from this table, that the great proportion of the cases of relapsing fever were Irish. Three fourths of the whole cases had come from Ireland; and many of the remainder, from their names, were evidently the children of Irish parents. On the other hand, only one eighth of the cases of typhus were Irish, and not one twenty-third of the
cases of typhoid. Moreover, of the twenty-six patients with relapsing fever who had come from the provinces of England, upwards of one half had lived in London for five years or more, so that it is clear that almost every one of the patients seized with relapsing fever soon after their arrival in London had come from Ireland. The Irish have long been the reputed introducers of epidemic fever into England and Scotland. Dr. Cowan informs us, that out of 2257 cases of fever admitted into the Glasgow Infirmary, about one third, or 715, were Irish. The question then comes to be —What fever is it, which the Irish chiefly import? The number of typhus cases among the Irish in the above table is small; and out of the forty, all but ten had resided upwards of a year in London; so that, as far as London is concerned, typhus does not appear to be imported from Ireland. On the other hand, it seems probable that the vast majority of the cases of relapsing fever which occur in London may be traced to an Irish origin. It will afterwards be shown that a mild form of fever, resembling relapsing fever in its small mortality (and perhaps corresponding to its first stage without the relapse), is at all times more prevalent in Ireland than in Britain. This suggests another question—Whether the same animal poison, which in Ireland more commonly produces a short, mild fever, followed during certain epidemics by one or more relapses, may not, in England, with a change in the external conditions and in the recipient body, generate typhus? I shall not, at present, attempt to give a definite answer to this question; but the following facts have an important bearing upon it. In the last six months of 1851, there were admitted, from Field Lane, in the City of London, into the Fever Hospital, nine cases of relapsing fever, but none of typhus; in 1852, there were, from the same locality, fourteen admissions of relapsing —the last in May, and twenty-four of typhus—the first in March; and in 1853, there were sixteen admissions from the same lane—all typhus. Again, from Tyndall’s Buildings,
Etiology of continued fever.

Holborn, there were admitted in 1851 twenty-two cases of relapsing and one of typhus; in 1852, ten of relapsing and nine of typhus. In several instances, a case of relapsing and of typhus were brought from the same house, within a few weeks or days of each other. Lastly, during an epidemic of relapsing fever at Newcastle, in 1848, which, from its novelty in the place, attracted much attention, and was shown to have been imported by the Irish, many of the clergy and medical men who attended upon the sick, were attacked with fever, but in not one of these cases did this assume the relapsing form, being more prolonged, and like the ordinary typhus.¹

The explanation which has been given of the great proportion of cases of relapsing fever being amongst the newly arrived in London, will not apply to typhoid fever. The most of the patients with typhoid fever, not born in London, came from the provinces of England; and it is worthy of notice, that the majority of those who come up to push their fortunes in the great metropolis, arrive just at that age which is most prone to the affection in question. This greater liability of the newly arrived to contract typhoid, points to the dependence of this disease upon some local cause, which they are less able to resist than those who have for long been habitually subjected to it. This is well known to be the case with ordinary diarrhœa. Mr. Carpenter, of Croydon, in writing some years ago of the bad water at that place, observed, that he knew several houses in which this was used by the ordinary inhabitants with impunity, but in which any visitors or new servants were invariably seized with diarrhœa, soon after their arrival.² Several instances of a similar circumstance, as regards typhoid fever, have come under my own notice. One of the most remarkable is the following. In the autumn of 1853, a lady and her two daughters arrived from the country, on a visit to some friends residing at Kennington, in the

¹ Dr. White's 'Report of the Newcastle Fever Hospital,' 1848.
district of Lambeth. Behind the house there was an open sewer, inundated from the Thames by every tide, and often emitting towards evening an intolerable stench. The drinking-water of the house was derived from a well, and required no microscope nor chemical tests to demonstrate its great impurity. All three suffered from diarrhoea, and in about a fortnight one of the daughters took typhoid fever and died. The following autumn, the mother and remaining daughter again came up from the country, and visited the same house. Within a fortnight, the former died from cholera. On both of these occasions, the ordinary dwellers in the house were quite exempt even from diarrhoea.

B.—Mortality from Continued Fever.

I. The Rate of Mortality from Fever in the London Fever Hospital, as compared with that of other hospitals.

At but few hospitals is any distinction recorded between the different forms of fever; and consequently a comparison can only be made between the mortality in each, by taking all the forms together. Any such comparison will certainly not be in favour of the London Fever Hospital, where the mortality is unquestionably great. Some, who consider all fever hospitals objectionable on principle, might be inclined to attribute this great mortality to the accumulation of a number of patients in one building, and a consequent concentration of the fever-poison. Before forming such a conclusion, however, it must be remembered, that there are other circumstances peculiar to the London Fever Hospital, which in a great measure account for the large number of cases which terminate fatally; and, if a due allowance be made for these, the mortality will probably not exceed that of other places. In the first place, a much larger proportion of the patients admitted are from the most indigent classes, and of an advanced age, than is the case with any of the other hospitals in London. A large number of them are the aged and decayed inmates of the various metropolitan workhouses. The influence of age and destitution in increasing
the mortality from fever will shortly be made apparent. Again, the mortality is greatly increased by the patients being often brought from the most distant parts of London, in an advanced stage of their complaint—as in the second or third week. The powerful operation of this cause is proved by the fact, that every year a considerable number of the patients are moribund on admission, and die within the first or second day after. Thus, in the year 1855, to quote from the 'Annual Report,' "out of the total number of 173 deaths from all causes, sixty-eight occurred before the close of the fourth day, and forty-two before the end of the second. Ten were in a dying state on reaching the hospital; one lived only three minutes after admission; and one was brought in dead." Lastly, the proportion of the more fatal forms of fever admitted into the London Fever Hospital is greater than in other hospitals; for, as will be shown, the rate of mortality from typhus and typhoid only is not greater than has been observed elsewhere.

Table XVIII gives the mortality from all the cases of fever admitted into the London Fever Hospital during ten years, including those which died from various complications. Thus, for the whole ten years, the mortality has been at

<table>
<thead>
<tr>
<th>Table XVIII.1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Years.</td>
</tr>
<tr>
<td>1848</td>
</tr>
<tr>
<td>1849</td>
</tr>
<tr>
<td>1850</td>
</tr>
<tr>
<td>1851</td>
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<td>1852</td>
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<td>1853</td>
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<td>1854</td>
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<tr>
<td>1855</td>
</tr>
<tr>
<td>1856</td>
</tr>
<tr>
<td>1857</td>
</tr>
<tr>
<td>Total</td>
</tr>
<tr>
<td>Deducting 61 who died within 24 hours after admission</td>
</tr>
<tr>
<td>And 146, within 48 hours</td>
</tr>
</tbody>
</table>

1 In this and the following tables, the deaths for each year have
the rate of almost 16 per cent., or about 1 in 6; but, deducting the cases which died within twenty-four hours after admission, the mortality falls to 15 per cent.; and, deducting those which died within forty-eight hours, it is only 14 per cent., or rather less than 1 in 7. It will also be observed, that the rate of mortality has varied considerably in different years. In one year, it was only 7 per cent., and in another under 9 per cent., while in a third it rose to

<table>
<thead>
<tr>
<th>Years</th>
<th>St. George's Hospital</th>
<th>Newcastle Fever Hospital</th>
<th>Nottingham General Hospital</th>
<th>Birmingham Queen's Hospital</th>
<th>Bristol Royal Infirmary</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of Cases</td>
<td>Mortality per cent.</td>
<td>No. of Cases</td>
<td>Mortality per cent.</td>
<td>No. of Cases</td>
</tr>
<tr>
<td>1840</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1841</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1842</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1843</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1844</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1845</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1846</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1847</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1848</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1849</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1850</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1851</td>
<td>125</td>
<td>16</td>
<td>117</td>
<td>5</td>
<td>98</td>
</tr>
<tr>
<td>1852</td>
<td>141</td>
<td>7</td>
<td>102</td>
<td>19</td>
<td>86</td>
</tr>
<tr>
<td>1853</td>
<td>108</td>
<td>13</td>
<td>88</td>
<td>20</td>
<td>83</td>
</tr>
<tr>
<td>1854</td>
<td>195</td>
<td>23</td>
<td>124</td>
<td>19</td>
<td>137</td>
</tr>
<tr>
<td>1855</td>
<td>188</td>
<td>9</td>
<td>94</td>
<td>10</td>
<td>86</td>
</tr>
<tr>
<td>1856</td>
<td>164</td>
<td>19</td>
<td>106</td>
<td>19</td>
<td>83</td>
</tr>
<tr>
<td>1857</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td></td>
<td>911</td>
<td>11</td>
<td>1481</td>
<td>11</td>
<td>845</td>
</tr>
</tbody>
</table>

| Total |                        |                         |                        |                         | 1890              |                         |
|       | 1 death in 8| 1 in 8| 1 in 7| 1 in 7| 1 in 10.|

reference only to the patients admitted in that year. A patient admitted in December, 1851, and dying in January, 1852, has been entered as a death in 1851.

2 Communicated by Dr. Robinson, of Newcastle.
3 Annual Reports of Hospital.
4 Communicated by the House Surgeon, Mr. Allis Smith.
5 Communicated by Mr. Crisp, the House Surgeon.
ETIOLOGY OF CONTINUED FEVER. 289

upwards of 20 per cent.1 This great variation, as will shortly be shown, is partly, but not entirely, due to a prevalence of different fevers in different years.

In Table XIX, the rate of mortality from fever in eleven other hospitals, during the last eighteen years, is compared. One of these hospitals is in London, four in the provinces of England, three in Scotland, two in Ireland, and one in Stockholm.

XIX.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of Cases.</td>
<td>Mortality per cent.</td>
<td>No. of Cases.</td>
<td>Mortality per cent.</td>
<td>No. of Cases.</td>
<td>Mortality per cent.</td>
<td>No. of Cases.</td>
</tr>
<tr>
<td>2188</td>
<td>13-75</td>
<td>437</td>
<td>7-09</td>
<td>4329</td>
<td>5-63</td>
<td>2441</td>
</tr>
<tr>
<td>846</td>
<td>13-59</td>
<td>282</td>
<td>5-32</td>
<td>2072</td>
<td>7-14</td>
<td>1467</td>
</tr>
<tr>
<td>817</td>
<td>6-85</td>
<td>1280</td>
<td>3-75</td>
<td>3467</td>
<td>4-53</td>
<td>2529</td>
</tr>
<tr>
<td>1135</td>
<td>7-77</td>
<td>780</td>
<td>5-25</td>
<td>1468</td>
<td>8-92</td>
<td>2375</td>
</tr>
<tr>
<td>679</td>
<td>11-34</td>
<td>378</td>
<td>6-61</td>
<td>535</td>
<td>11-03</td>
<td>2954</td>
</tr>
<tr>
<td>685</td>
<td>12-70</td>
<td>377</td>
<td>8-75</td>
<td>1365</td>
<td>9-71</td>
<td>4535</td>
</tr>
<tr>
<td>3771</td>
<td>13-26</td>
<td>683</td>
<td>11-86</td>
<td>5244</td>
<td>13-34</td>
<td>5873</td>
</tr>
<tr>
<td>4798</td>
<td>15-09</td>
<td>1648</td>
<td>12-68</td>
<td>1515</td>
<td>11-75</td>
<td>2472</td>
</tr>
<tr>
<td>726</td>
<td>12-12</td>
<td>584</td>
<td>8-56</td>
<td>570</td>
<td>13-86</td>
<td>2977</td>
</tr>
<tr>
<td>522</td>
<td>12-44</td>
<td>255</td>
<td>11-0</td>
<td>597</td>
<td>19-26</td>
<td>2096</td>
</tr>
<tr>
<td>959</td>
<td>3-34</td>
<td>218</td>
<td>15-13</td>
<td>1385</td>
<td>12-79</td>
<td>2133</td>
</tr>
<tr>
<td>691</td>
<td>4-84</td>
<td>146</td>
<td>17-9</td>
<td>1721</td>
<td>12-9</td>
<td>2354</td>
</tr>
<tr>
<td>574</td>
<td>7-49</td>
<td>121</td>
<td>6-61</td>
<td>1938</td>
<td>14-86</td>
<td>1388</td>
</tr>
<tr>
<td>168</td>
<td>15-47</td>
<td>304</td>
<td>11-18</td>
<td>2069</td>
<td>8-07</td>
<td>1096</td>
</tr>
<tr>
<td>201</td>
<td>20-39</td>
<td>345</td>
<td>10-43</td>
<td>2204</td>
<td>6-44</td>
<td>907</td>
</tr>
<tr>
<td>167</td>
<td>24-06</td>
<td>225</td>
<td>10-22</td>
<td>1606</td>
<td>6-1</td>
<td>1067</td>
</tr>
<tr>
<td>132</td>
<td>21-21</td>
<td>145</td>
<td>13-10</td>
<td>...</td>
<td>...</td>
<td>827</td>
</tr>
<tr>
<td>22,586</td>
<td>11-61</td>
<td>8783</td>
<td>9-14</td>
<td>20,091</td>
<td>11-28</td>
<td>47,651</td>
</tr>
<tr>
<td>1 in 8 ½.</td>
<td>1 in 11.</td>
<td>1 in 8 ½.</td>
<td>1 in 13 ½.</td>
<td>1 in 23 ½.</td>
<td>1 in 9 ½.</td>
<td>3186</td>
</tr>
</tbody>
</table>

1 In this year, however, but few cases of febricula were included. See note to Table II.
2 'Statistical Tables,' tenth ser., p. 20.; and private communication from Medical Superintendent.
3 'Annual Reports.'
5 Communicated by the Registrar of the Hospital.
6 Communicated by Dr. McEvers, of Cork.
7 Dr. Magnus Huss, 'Statist. du Typhus,' p. 48.
Speaking in general terms, it would appear from this table that the rate of mortality from fever during a series of years differs but little in the various hospitals of England and Scotland, being about 1 in 8; in some rather more, in others rather less. In the Aberdeen Infirmary, however, the mortality from 8783 cases during eighteen years has only been 1 in 11. This was due, however, to the small mortality of the four years, 1842-45, which will shortly be accounted for. Taking the cases only for the last ten years, the mortality in Aberdeen, as elsewhere, was 1 in 84. Again, in every instance it will be seen the mortality has varied greatly from year to year. In Aberdeen it was under 4 per cent. one year, and in another nearly 18 per cent.; at Nottingham it was one year 30 per cent., in another less than 7 per cent.

The mortality at Stockholm appears to be much the same as in England, or on the whole rather less.

To all of these results, the Irish hospitals present a marked antithesis. Out of 150,939 cases of fever admitted into the Dublin Fever Hospital, since the year 1817, only 10,632, or less than 1 in 14, have died;1 and during the last eighteen years, it will be seen from the table that the mortality has only been 1 in 183. Again, in the Cork Fever Hospital, the mortality has been even much less. Since the year 1817, out of 82,293 patients only 3222, or 1 in 25½, have died;2 and during the eighteen years contained in the table, the mortality has only been 4½ per cent., or 1 in 234. Moreover, the rate of mortality has varied much less in different years than it does in England and Scotland. Thus in Dublin, in no year during the last forty has it reached 10 per cent.; and in the Cork Hospital, in only one year of the last forty has it slightly exceeded 6 per cent. In the year 1838, Dr. Cowan, of Glasgow, drew attention to the striking discrepancy in the mortality from fever between the British and Irish hospitals;3 and I find on referring to

1 From data furnished to me by the Registrar of the Hospital.
2 From data furnished by Dr. McEvers, of Cork.
Barker and Cheyne's report of the Irish epidemic of 1817-19, that out of 100,737 patients in the hospitals of all Ireland, 4349 died, making a mortality of 4.3 per cent., or of only 1 in 23.1 No doubt the circumstance to which I have just called attention is partly accounted for by the greater facilities afforded to mild cases for entering the hospitals in Ireland; but whether this be the case or not, it plainly shows that there is a form of fever constantly present in Ireland, which is much milder, and the mortality from which is much less, than is the case with the fever we more generally meet with in this country. I shall endeavour further to explain this discrepancy, in my remarks under the following head:

II. Rate of Mortality in the different forms of "Continued Fever."

The mortality from the different forms of continued fever admitted into the London Fever Hospital, during ten years, is given in Table XX.

<table>
<thead>
<tr>
<th>Years</th>
<th>Typhus</th>
<th>Relapsing Fever</th>
<th>Typhoid</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Admissions</td>
<td>Deaths</td>
<td>Mortality per cent.</td>
</tr>
<tr>
<td>1848</td>
<td>526</td>
<td>106</td>
<td>20.15</td>
</tr>
<tr>
<td>1849</td>
<td>155</td>
<td>39</td>
<td>25.16</td>
</tr>
<tr>
<td>1850</td>
<td>130</td>
<td>24</td>
<td>18.46</td>
</tr>
<tr>
<td>1851</td>
<td>68</td>
<td>6</td>
<td>8.82</td>
</tr>
<tr>
<td>1852</td>
<td>204</td>
<td>24</td>
<td>11.75</td>
</tr>
<tr>
<td>1853</td>
<td>408</td>
<td>90</td>
<td>22.06</td>
</tr>
<tr>
<td>1854</td>
<td>337</td>
<td>68</td>
<td>20.18</td>
</tr>
<tr>
<td>1855</td>
<td>342</td>
<td>82</td>
<td>24.24</td>
</tr>
<tr>
<td>1856</td>
<td>1062</td>
<td>207</td>
<td>19.49</td>
</tr>
<tr>
<td>1857</td>
<td>274</td>
<td>69</td>
<td>25.18</td>
</tr>
<tr>
<td>Total</td>
<td>3506</td>
<td>715</td>
<td>20.39</td>
</tr>
<tr>
<td></td>
<td>Deducting 49 who died within 24 hours</td>
<td>3457</td>
<td>668</td>
</tr>
<tr>
<td></td>
<td>Deducting 115 who died within 48 hours</td>
<td>3391</td>
<td>600</td>
</tr>
</tbody>
</table>

1 Vol. ii, p. 190.
ETIOLOGY OF CONTINUED FEVER.

The exclusion of the cases of "febricula" from this table, of course raises the rate of mortality from the other forms of fever. This remark, as is obvious, applies more especially to typhus and typhoid.

Out of 3506 cases of typhus 715 died, making a mortality of 20·39 per cent., or of about 1 in 5. Deducting the cases which proved fatal within twenty-four hours after admission, the mortality falls to 19·3 per cent.; and deducting those fatal within forty-eight hours, it is only 17·7 per cent., or about 1 in 5½. In two years, the mortality exceeded 25 per cent., and in one year it was under 9 per cent. The year in which the rate of mortality was smallest, was also that in which there were the fewest cases. The mortality from typhus in the London Fever Hospital does not exceed what has been observed elsewhere. Thus, in the Edinburgh Infirmary, in the year 1848-49, out of 363 cases of typhus 80, or 22·3 per cent., died;¹ and in the Glasgow Infirmary, out of 9485 cases of typhus admitted during eleven years 18 per cent. died.²

The mortality from typhoid fever appears, on the whole, to be somewhat under that from typhus. Out of 1820 cases 333 died, or 18·29 per cent., or about 1 in 5½. Subtracting the cases fatal within twenty-four hours, it was only 17½ per cent.; and subtracting those fatal within forty-eight hours, it was under 17 per cent., or about 1 in 6. In one year, however, the mortality was greater than in any year from typhus, being 28 per cent., or about 1 in 3½; and in no year was it so low as what we have found it to have been in some years from typhus, the smallest mortality in any year being scarcely under 18 per cent. Moreover, the year in which the mortality was least, was also that in which there was the greatest number of cases; whereas the mortality from typhus appeared to be lowest when it was least prevalent. In the Glasgow Hospital the mortality from typhoid fever, taken separately, has been greater even

¹ 'Statistical Tables,' ninth ser., p. 14.
ETIOLOGY OF CONTINUED FEVER. 293

than in London. Out of 356 cases admitted into the infirmary during several years, 77 died, making a mortality of 21.6 per cent., or of almost 1 in 4î.¹

The mortality from relapsing fever, when compared with that of the two other forms, is strikingly small. Out of the 441 cases only 11 proved fatal, making 2î per cent., or about 1 in 40. This small mortality from relapsing fever has been a matter of general observation. Out of 203 cases in the Edinburgh Infirmary in 1848-49, only eight cases, or less than 4 per cent., died;² and out of 7804 cases in the Glasgow Infirmary, between the years 1843 and 1853, only 405, or 5 per cent., died.³

It is therefore evident, that the more predominant fever of the relapsing form is at any time, the less will be the mortality from continued fever, taken as a whole. For example, this was the real explanation of the wonderfully small mortality from fever observed in London in 1851, and in Edinburgh, Aberdeen, and Glasgow, in 1843. It must also be obvious, how important it is, in comparing the mortality from fever at different times and places, in order to judge of the merits of different plans of treatment, or for other purposes, that we take into account the form of fever which has prevailed. If this be not done, any such comparison can be of little worth. Thus, while the total mortality from fever in Glasgow was much below that of the London Fever Hospital, that in each of the individual forms was greater, the difference resulting from the much larger proportion of relapsing cases which have occurred at Glasgow. The same remarks obviously apply to “febrícula.” The greater the proportion of cases coming under this category, the less will be the rate of mortality for all the cases of fever taken together.

If, then, the very small mortality from fever occasionally observed in England and Scotland admits of the explanation just given, it seems not unreasonable to suppose that a

¹ Dr. McGhie, loc. cit.
² 'Statist. Tables,' ninth ser., p. 15.
³ Dr. McGhie, oc. cit.
similar explanation may account for the constant small mortality observed in Ireland. During the great relapsing epidemic of 1843, when the mortality from fever in Scotland was so small, that in Ireland remained at its usual standard. Secondly, the smallest mortality from typhus itself, in the London Fever Hospital, corresponded with the greatest prevalence of relapsing fever. Thirdly, we have already seen that all the great epidemics of fever have originated in Ireland; and it is a fact which has generally been admitted during these epidemics, that the fever has been imported from Ireland into Britain. Fourthly, we have also seen, that at the commencement of some epidemics in Britain, the proportion of relapsing cases to typhus is greater than towards the close; and that this remark applies also to limited localities in London. Lastly, I have shown that a large proportion of the cases of relapsing fever (much larger than that of typhus) admitted into the London Fever Hospital, have been Irish recently arrived in London. Putting all these facts together, there seem grounds for believing that a short mild fever (corresponding, perhaps, to the first paroxysm of relapsing fever) is at all times more common in Ireland; that under certain circumstances this assumes the relapsing form; and that it is this relapsing fever which in many great epidemics the Irish have chiefly imported into this country.

III. Influence of Months and Seasons on the Mortality of Fever.

Months and seasons of the year appear to exercise little or no influence on the rate of mortality of continued fever taken as a whole, nor on that of any of its forms. This is obvious from Table XXI, which gives the mortality for the aggregate seasons of ten years, among the cases admitted into the London Fever Hospital.
ETIOLOGY OF CONTINUED FEVER.

TABLE XXI.

<table>
<thead>
<tr>
<th>Seasons</th>
<th>Typhus.</th>
<th>Relapsing.</th>
<th>Typhoid.</th>
<th>Total, including Febricula.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spring</td>
<td>1069</td>
<td>218</td>
<td>20.39</td>
<td>90</td>
</tr>
<tr>
<td>Summer</td>
<td>988</td>
<td>206</td>
<td>20.85</td>
<td>120</td>
</tr>
<tr>
<td>Autumn</td>
<td>659</td>
<td>131</td>
<td>19.88</td>
<td>123</td>
</tr>
<tr>
<td>Winter</td>
<td>790</td>
<td>160</td>
<td>20.25</td>
<td>108</td>
</tr>
<tr>
<td>Total</td>
<td>3506</td>
<td>715</td>
<td>20.39</td>
<td>441</td>
</tr>
</tbody>
</table>

For the whole cases, it will be seen that the rate of mortality was least in autumn, and greatest in spring; but the difference was not great.

The mortality from typhus appears almost uniform for every season. As regards the individual years, the rate of mortality varied very greatly at different times, but without any reference to months or seasons.

The mortality from relapsing fever was greatest in autumn and in spring, but the cases were too few to draw any conclusions.

The mortality from typhoid was least in winter and considerably less in autumn than in spring. In five of the ten years, although the number of cases of typhoid admitted in autumn far exceeded that admitted in spring, the rate of mortality in the latter season was much greater than in the former. In two years only (1853 and 1854) was the mortality greatest in winter, but in these the winter mortality was double that of any other season (40 per cent.) Forget and Chomel have endeavoured to show that the mortality from typhoid fever in France is double in winter what it is in summer, but their conclusions are drawn from a very limited number of cases.  

1 See Bartlett, op. cit., p. 125.
IV. Influence of Sex on the Mortality of Fever.

Sex, like season, appears, on the whole, to exercise little or no influence on the mortality of fever, as shown in Table XXII, which gives the results of the London Fever Hospital on this point for ten years.

**Table XXII.**

<table>
<thead>
<tr>
<th></th>
<th>Typhus.</th>
<th>Relapsing Fever.</th>
<th>Typhoid.</th>
<th>Total, including Ferricula.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>1737</td>
<td>368</td>
<td>21.18</td>
<td>233</td>
</tr>
<tr>
<td>Females</td>
<td>1769</td>
<td>347</td>
<td>19.61</td>
<td>208</td>
</tr>
</tbody>
</table>

Taking all the cases together, the rate of mortality for the two sexes was almost identical. In typhus, it was slightly greater among the males, and in typhoid among the females. In five of the ten years, however, the mortality from typhoid was greater among the males; and in three, the mortality from typhus was greater among the females. Of the few relapsing cases which proved fatal, the majority were females.

It will be found, on referring to various statistical reports, that in some places the mortality from fever has been observed to be greater in the male; and in others, in the female sex. In Stockholm, out of 2181 males 252, or 20.7 per cent., died; whereas, out of 1005 females only 87, or 8.65 per cent., perished.

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2 Magnus Huss, op. cit., p. 58.
V. Influence of Age upon the Mortality of Fever.

The influence of age upon the mortality of fever may be ascertained by comparing the mean age of the fatal cases with that of those which recovered; or still better, by ascertaining the rate of mortality in each period of life. Both of these plans have been adopted; and the results for the ten years are given in the two following tables, and in Diagram IV. (See page 238.)

### Table XXIII.

<table>
<thead>
<tr>
<th></th>
<th>Typhus</th>
<th>Relapsing</th>
<th>Typhoid</th>
<th>Total, including Febricula</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
<td>Mean Age</td>
<td>Number</td>
<td>Mean Age</td>
</tr>
<tr>
<td>Total admissions in which age known.</td>
<td>3456</td>
<td>29.33</td>
<td>437</td>
<td>24.41</td>
</tr>
<tr>
<td>Cases which recovered</td>
<td>2753</td>
<td>26.15</td>
<td>426</td>
<td>24.14</td>
</tr>
<tr>
<td>Cases which died</td>
<td>703</td>
<td>41.78</td>
<td>11</td>
<td>35.09</td>
</tr>
</tbody>
</table>

It will be observed in Table XXIII, that the mean age of the fatal cases far exceeds that of those which recovered; or, in other words, that the mortality increases as life advances. Thus, the mean age, in round numbers, of all the fatal cases being 36, that of those which recovered is only 24. This rule, however, holds good more especially with typhus and relapsing fever; there being a far greater difference between the age of the fatal cases and of the recoveries in these than in typhoid. Thus, in typhus, the age of the cases which recovered being 26, that of the fatal cases is 42; and in relapsing, the age of the former being 24, that of the latter is 35; in typhoid, the age of the former is 27, and of the latter only 23.5. Moreover, as regards typhus, the difference of age between the fatal cases and recoveries given in the table held good for every one of the ten years, whereas in typhoid the difference for several years was much less than that in
<table>
<thead>
<tr>
<th>Age Group</th>
<th>Typhus</th>
<th>Relapsing Fever</th>
<th>Typhoid</th>
<th>Total, Including Fabricula</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 5 years</td>
<td>17</td>
<td>3</td>
<td>17-65</td>
<td>4</td>
</tr>
<tr>
<td>From 5 to 10 years</td>
<td>185</td>
<td>14</td>
<td>7-65</td>
<td>32</td>
</tr>
<tr>
<td>10 to 15</td>
<td>385</td>
<td>18</td>
<td>4-95</td>
<td>63</td>
</tr>
<tr>
<td>15 to 20</td>
<td>546</td>
<td>26</td>
<td>4-76</td>
<td>92</td>
</tr>
<tr>
<td>20 to 25</td>
<td>493</td>
<td>47</td>
<td>9-5</td>
<td>76</td>
</tr>
<tr>
<td>25 to 30</td>
<td>343</td>
<td>52</td>
<td>15-15</td>
<td>37</td>
</tr>
<tr>
<td>30 to 35</td>
<td>323</td>
<td>55</td>
<td>17-02</td>
<td>37</td>
</tr>
<tr>
<td>35 to 40</td>
<td>370</td>
<td>89</td>
<td>32-96</td>
<td>19</td>
</tr>
<tr>
<td>40 to 45</td>
<td>292</td>
<td>87</td>
<td>29-79</td>
<td>40</td>
</tr>
<tr>
<td>45 to 50</td>
<td>212</td>
<td>83</td>
<td>39-15</td>
<td>8</td>
</tr>
<tr>
<td>50 to 55</td>
<td>150</td>
<td>78</td>
<td>52</td>
<td>15</td>
</tr>
<tr>
<td>55 to 60</td>
<td>100</td>
<td>51</td>
<td>51</td>
<td>7</td>
</tr>
<tr>
<td>60 to 65</td>
<td>88</td>
<td>48</td>
<td>53-68</td>
<td>5</td>
</tr>
<tr>
<td>65 to 70</td>
<td>82</td>
<td>28</td>
<td>66-66</td>
<td>1</td>
</tr>
<tr>
<td>70 to 75</td>
<td>24</td>
<td>17</td>
<td>70-83</td>
<td>1</td>
</tr>
<tr>
<td>75 to 80</td>
<td>6</td>
<td>5</td>
<td>83-33</td>
<td>6</td>
</tr>
<tr>
<td>Above 80 years</td>
<td>2</td>
<td>2</td>
<td>100</td>
<td>...</td>
</tr>
<tr>
<td>Age not known</td>
<td>50</td>
<td>11</td>
<td>22</td>
<td>4</td>
</tr>
<tr>
<td>Total</td>
<td>3506</td>
<td>715</td>
<td>20-39</td>
<td>441</td>
</tr>
</tbody>
</table>

The table, and in one year the mean age of the cases which recovered slightly exceeded that of the fatal cases. This fact, with regard to typhoid, is of course partially explained by the circumstance that this fever is most prevalent in early life; but that it is not so entirely, we find on referring to the mortality in each quinquennial period of life. (Table XXIV.)

Taking all the cases together, it appears that the mortality is greater in the first lustrum of life than in the second; and greater in the second than in the third. Between the ages of ten and fifteen the rate of mortality is less than at any other period of life, and after this it rapidly increases, until of those above fifty years 48⅓ per cent. die. Here again, however, these remarks have reference more especially to typhus and relapsing fever.
Thus, in typhus the mortality during the first five years of life is upwards of 17 per cent., in the second lustrum it falls to 7.65, and between ten and twenty it is under 5 per cent. After twenty, it goes on increasing rapidly, until of those—

<table>
<thead>
<tr>
<th>Above 30 years of age</th>
<th>36-65 per cent. died.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>40</td>
</tr>
<tr>
<td></td>
<td>&quot; 50</td>
</tr>
<tr>
<td></td>
<td>&quot; 60</td>
</tr>
</tbody>
</table>

As regards relapsing fever, the number of deaths is rather small to draw any very accurate conclusions; but it appears to resemble typhus in the greatly increased mortality in advanced life. Thus of 304 cases under thirty years only 2 died, whereas of 133 cases above thirty 9 died.

In typhoid fever, on the other hand, there is, in the first place, not that remarkably small rate of mortality in early life observed in typhus, the lowest (also between the ages of ten and fifteen) being almost 13 per cent. (See Diagram IV, p. 238.) There is also a greater uniformity in the mortality at different periods of life than in typhus; and, although the mortality does certainly increase as life advances, it does so to a less extent. Thus, of those—

<table>
<thead>
<tr>
<th>Above 30 years of age</th>
<th>27.38 per cent. died.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>40</td>
</tr>
<tr>
<td></td>
<td>&quot; 50</td>
</tr>
<tr>
<td></td>
<td>&quot; 60</td>
</tr>
</tbody>
</table>

A curious circumstance observed both in typhus and typhoid is, that between forty and forty-five the mortality is considerably less than in the period of life immediately preceding. In typhus the diminished rate of mortality at this period of life was, in the female sex, 26.23 per cent.; in typhoid it was, among the males, 14.28 per cent.

The greater mortality of typhus and relapsing fever in advanced life has been a matter of universal observation. Thus, of 363 cases of typhus admitted into the Edinburgh Infirmary in 1849, only 9 per cent. of those under twenty years of age died; but of those above thirty years, 40 per cent. died; and of those above fifty, one-half. Again, out
of 203 cases of relapsing fever occurring during the same year, 3 only, or 2 per cent., died of those under thirty, but five cases, or 10 per cent., of those above thirty. \(^1\)

With regard to typhoid fever, Louis found that none perished out of six of his patients under seventeen years; and he observes that, during ten years' hospital experience, he had only known one case prove fatal under twenty. \(^2\) Probably, however, there are but few cases admitted at a very early age into the Hotel Dieu, for in another Parisian hospital Riliet and Barthez found that 29 out of 111 children attacked with typhoid fever died; \(^3\) and the experience of the London Fever Hospital shows that childhood by no means proves a barrier to a fatal termination, and certainly to a less extent than it does in the case of typhus.

**VI. Station in Life.**

When speaking of the influence of occupation and station of life upon the prevalence of the different forms of fever, I mentioned that the patients admitted into the London Fever Hospital were divisible into several well-marked classes. I shall now endeavour to show the rate of mortality in these classes during the ten years by the following table:

**Table XXV.**

<table>
<thead>
<tr>
<th></th>
<th>I. Paying Patients, Servants of Subscribers, and Policemen.</th>
<th>II. Total patients not included in Class I.</th>
<th>III. Inmates of Workhouse only.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Typhus</td>
<td>94 14 14.89</td>
<td>3412 701 20.54</td>
<td>738 204 27.64</td>
</tr>
<tr>
<td>Relapsing</td>
<td>2 ...</td>
<td>439 11 2.5</td>
<td>47 2 4.25</td>
</tr>
<tr>
<td>Typhoid</td>
<td>281 47 16.72</td>
<td>1539 286 18.58</td>
<td>85 13 15.3</td>
</tr>
<tr>
<td>Total, including Febricula</td>
<td>426 61 14.32</td>
<td>6202 998 16.09</td>
<td>989 219 22.14</td>
</tr>
</tbody>
</table>

\(^1\) Statist. Tab., Edin. Infirmary, ninth ser., pp. 14 and 15.
\(^3\) Bartlett, op. cit., p. 125.
It is evident from the above table that the mortality from all forms of fever taken together was least in the paying cases, and greatest of all among the inmates of workhouses; in the former being 14½ per cent., in the latter upwards of 22 per cent.

On examining, however, the mortality from each form of fever, it is found that this remark is, in a marked degree, applicable to typhus, and scarcely, if at all, to typhoid fever. Thus, of typhus less than 15 per cent. died among the paying cases; of all the remaining 20½ per cent., and of the inmates of workhouses alone 27·6 per cent. On the other hand, in typhoid fever, the rate of mortality in all three divisions differed but slightly, and was indeed least of all among those who had been the inmates of workhouses. Several practitioners, also, have informed me that they have found typhoid fever quite as fatal among the upper classes as among the poor. Destitution would thus seem to exercise little or no influence over either the prevalence or the mortality of typhoid fever, whereas typhus appears to be not only most prevalent but most mortal among the very poor.

This conclusion is opposed to the opinions of various Irish writers, that “fever” is most fatal among the rich;¹ but in none of the instances to which allusion is made, has any distinction been noted between typhus and relapsing fever; and the greater mortality among the “rich” (who appear to have been chiefly medical men) may have been owing to typhus having been the most prevalent form among them, whilst the more common form among the poor was relapsing fever.

VII. Influence of Recent Residence on the Mortality of Fever.

The influence of this circumstance over the mortality of fever is shown in Table XXVI, for the patients admitted into the London Fever Hospital during the two years 1851

and 1857. It is to be observed that these were the two years in which the total mortality from typhoid fever was smallest; in the former it was only 12.8, and in the latter, 14 per cent.

<table>
<thead>
<tr>
<th>Residence in London</th>
<th>Typhus</th>
<th>Relapsing Fever</th>
<th>Typhoid</th>
<th>Total, including Pneumonia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Admissions</td>
<td>Deaths</td>
<td>Mortality per cent.</td>
<td>Admissions</td>
</tr>
<tr>
<td>Not exceeding 6 months</td>
<td>13</td>
<td>2</td>
<td>15.38</td>
<td>29</td>
</tr>
<tr>
<td>More than 6 months</td>
<td>294</td>
<td>62</td>
<td>21.09</td>
<td>193</td>
</tr>
<tr>
<td>Total in which it was known</td>
<td>307</td>
<td>64</td>
<td>20.85</td>
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<table>
<thead>
<tr>
<th>Race</th>
<th>Typhus</th>
<th>Relapsing Fever</th>
<th>Typhoid</th>
<th>Total, including Pneumonia</th>
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<tr>
<td></td>
<td>Admissions</td>
<td>Deaths</td>
<td>Mortality per cent.</td>
<td>Admissions</td>
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<tr>
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<td>272</td>
<td>51</td>
<td>20.59</td>
<td>59</td>
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<tr>
<td>Irish</td>
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<td>5</td>
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<td>Total in which birthplace known</td>
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<td>66</td>
<td>20.5</td>
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Recent residence appears to have a marked influence over the rate of mortality, as well as over the prevalence of typhoid fever. Of 43 patients who had not resided in London more than six months 11 died, or rather more than 1 out of every 4; whereas of 347 patients who had resided in London upwards of six months 40 died, or only 1 out of every 8.5.

The same law does not hold good with typhus, in which, indeed, the rate of mortality was less among the newly arrived, although the number of cases is rather small to draw any very decided conclusions on the matter.

Another circumstance to be observed is, that the rate of mortality from typhus is almost equal among the English
and Irish patients; but in typhoid fever much greater among the English.

I shall now briefly recapitulate the principal conclusions which I think may be legitimately deduced from the facts which I have brought forward in this essay.

1. Typhus and relapsing fever occur at irregular intervals, and often simultaneously, as wide-spread epidemics. They then gradually disappear, and both of them, but especially the latter, may be absent for years from those places where, during the epidemics, they are usually most prevalent.

2. Typhoid fever does not occur in such wide-spread epidemics. In certain places it is never absent, and its prevalence varies but little from year to year. When outbreaks of it occur in other situations, these are always of the most local and circumscribed character.

3. Typhus and relapsing fever are quite independent of the season of the year, whereas typhoid fever is almost invariably most prevalent during autumn, at the time that diarrhoea is most common, and it has been observed to be especially prevalent in seasons remarkable for their high temperature.

4. Sex has no influence over the prevalence of continued fever, nor over that of any of its forms.

5. Typhoid fever is pre-eminently a disease of childhood and adolescence, at which periods of life we know that there is a marked proneness to enteric affections. Less than one seventh of the cases of typhoid are above thirty years of age. Typhus and relapsing fever exhibit no such predilection for youth; of typhus one half, and of relapsing fever one third, of the cases, are above thirty.

6. Typhus and relapsing fever appertain exclusively to poverty and destitution, and seldom or never occur among the wealthy, except from direct contagion. Typhoid fever attacks both poor and rich, without distinction.

7. In large cities typhus and relapsing fever are, for the most part, limited to those localities remarkable for the
overcrowding of their inhabitants; and in country districts they are seldom or never met with, except as the result of direct importation. Typhoid, on the other hand, occurs alike in the centre and suburbs of cities; in the crowded hovels of the poor, and in the spacious mansions of the great; and also in isolated houses and hamlets in the country, without any traceable sources of contagion.

8. When fever breaks out in a house or locality, it seldom or never happens that some of the cases are typhus and others typhoid; but typhus and relapsing fever occur not unfrequently together.

9. Cases of what has been called "febricula" may co-exist along with any of the three other forms, but especially with typhus and relapsing fever. Most of them are either mild varieties of some of these, or dependent upon some derangement of digestion or other non-specific causes.

10. Overcrowding, with deficient ventilation and destitution, appear to be the essential causes of typhus and relapsing fever, and to be capable of generating them de novo, while there is no evidence that they have any such influence over the production of typhoid fever.

11. There are many circumstances which tend to the belief that the emanations from decaying organic matter, or organic impurities in drinking-water, or both of these causes combined, are capable of generating typhoid fever; but there is no authenticated evidence whatever to prove that such causes can give rise to typhus or relapsing fever.

12. There are some grounds for believing that a combination of the causes mentioned in the two last paragraphs may occasionally, although rarely, generate a disease intermediate in its characters between typhus and typhoid, or may (to speak, perhaps, more correctly) cause typhoid fever to assume some of the characters of typhus; but such cases cannot be used as an argument in favour of the identity of the poisons of the two diseases.

13. Typhus is eminently contagious. Typhoid fever is also contagious, but in a more limited degree, and possibly through a different medium. Again, typhus has in no
instance been proved to communicate typhoid, nor typhoid
to communicate typhus. An attack of either confers an
immunity from a future attack of itself, but not of the
other.

14. Recent residence increases the liability to typhoid;
scarcely, if at all, that to typhus.

15. The great majority of the cases of relapsing fever
have been Irish, and of these a large proportion had but
recently arrived in London. There seems reason for be-
lieving it possible, that fever imported from Ireland as
"relapsing" may gradually pass into typhus.

16. Relapsing fever offers a marked contrast to typhus
and typhoid, in the small mortality which it occasions.

17. In comparing the mortality from continued fever at
different times and places, it is essential to take into con-
sideration the form of fever which has prevailed. If this be
not done, the comparison is valueless.

18. The small mortality from continued fever constantly
observed in Ireland, along with other circumstances, renders
it probable that in that country a fever, more or less allied
to the relapsing form, is more common than in Britain.

19. Season of the year has no influence over the mortality
of any of the forms of fever.

20. In all of the fevers there is not much difference in
the mortality of the two sexes.

21. Typhus is least fatal between the ages of ten and
twenty, the mortality at that period of life being under 5
per cent. Above twenty, the mortality increases with the
age, until of those above fifty considerably more than one
half die. The mortality from relapsing fever appears to be
influenced by age in a similar manner. In typhoid fever,
on the other hand, in no period of life is the mortality under
12½ per cent.; and although, as in typhus, the rate of
mortality increases with the age, it does so in a less
degree.

22. The mortality from typhus is greater among the very
poor than among those in better circumstances. Typhoid
fever appears to be equally mortal in all classes.
23. Recent residence increases the mortality from, as well as the liability to, typhoid fever; but does not appear to have any such influence over typhus.

24. Typhus and relapsing fever are strongly assimilated in the causes which give rise to them, if they be not mutually convertible diseases.¹ Typhoid fever, on the contrary, appears to be a perfectly distinct affection, dependent upon totally different causes.

25. The facts which have been adduced in reference to the mode of origin of the different forms of fever, deserve the serious attention of those entrusted with the care of the public health; for it is manifest that, should they be confirmed by subsequent experience, they must have an important bearing on the question of hygiene.

¹ A further study of the conditions under which epidemics of relapsing fever have appeared, has induced me to believe that it will yet be shown that this fever is the result of famine alone, and that the poison of typhus is generated by destitution, and over-crowding combined.
CASE

OF

HYDATIDS OF THE TIBIA.

BY

WILLIAM COULSON,

SENIOR SURGEON TO, AND LECTURER ON SURGERY AT, ST. MARY'S HOSPITAL.

Received Feb. 27th. — Read May 30th, 1858.

I am induced to lay the following case before the Fellows of the Royal Medical and Chirurgical Society, on account of the rarity of the affection to which it refers.

Sarah G—, æt. 25, married, was admitted into St. Mary's Hospital under my care, on the 20th October, 1857. Eight years ago the patient received on the front of the right tibia, a little below the insertion of the ligamentum patellæ, a kick, which was soon followed by a swelling, that increased in a gradual and steady manner, until it attained the size of a hen's egg. The development of the swelling was attended by no great inconvenience until four years afterwards, when the pain becoming very severe, the patient was admitted into a metropolitan hospital. The treatment there employed mainly consisted in the use of blisters. After this the pain was less than it had previously been, but the swelling remained much in the same condition until ten weeks before she came under my notice. At this time the swelling gave way spontaneously,
and matter containing acephalocysts was discharged. The patient was then advised by Dr. Daubeney, under whose care she was, to come into St. Mary's hospital.

On her admission, there was a swelling, of the size of an orange, in the front of the right leg, just below the tubercosity of the tibia; in the centre of the swelling there was a small ulcer, and the surrounding integuments were red and swollen. The discharge, which was not considerable, was found to contain some acephalocysts, and thus the nature of the affection was revealed.

No doubt being entertained of the existence of a cavity in the tibia, containing hydatids, I determined to lay it open and remove its contents. On the 4th of November, by means of a crucial incision, I reflected the integuments, and with the saw and bone-nippers removed a large thin piece of bone, forming the anterior wall of the swelling; in this was impacted a large hydatid, which had partially made its escape. The cavity—which extended within half an inch of the joint, and three inches down the shaft—was now exposed, and hydatids in considerable numbers were removed. The whole of the cavity was lined by a white, glistening membrane. After removing all the hydatids which I could find, I rubbed the lining membrane of the cavity with solid nitrate of silver, and filled the cavity with cotton wool.

The cysts were found to consist of a friable, translucent membrane, which could be readily separated into distinct layers. Under the microscope, this membrane presented the laminated appearance which is usually found in hydatid cysts, without any trace of structure. The cysts contained a clear watery fluid, in which granular matter, with a considerable quantity of oil-globules, was detected. No trace of ecchinococci could be found.

On the 7th, the cotton wool was removed, and the cavity washed out with a solution of chloride of soda, when several hydatids came away. From this time the wound was dressed daily in the same manner. On the 11th, two other hydatids came away.

On the 18th, healthy granulations were seen springing
up from a great portion of the cavity, but at the upper part the floor of the cavity presented a dark appearance, and here a piece of necrosed bone was detected. On the 80th, this sequestrum, which had become loose, was removed with the forceps. This portion of bone was about two inches long, and one inch and a half broad; it was thickly covered on both sides by small hydatids, not much larger than a good-sized pin's head; so numerous were they, that the bone looked as if it were covered by a layer of coagulable lymph. On a close inspection, however, the hydatids could be readily detected; some collected in grape-like clusters, others single and attached by fine particles to the bone.

The removal of this sequestrum was attended by immediate benefit; granulations began to spring up from the part from which it had been removed, the cavity rapidly filled up, and on the 5th of February the patient was discharged with the wound nearly healed.

Remarks.—Hydatids are very seldom met with in the osseous system. The 'Transactions of the Society' contain one case only, which was communicated by the late Mr. Keate. In that case the hydatids occupied the cranium; but it is worthy of remark, that in the majority of cases published the tibia has been the bone affected.

The osseous system is so seldom the seat of hydatids, that no general description can be drawn from the limited number of cases on record. Hydatids of bone are developed in a slow manner. In the case related by Mr. Keate, and in one by Mr. Wickham, the disease was of six years' standing, and in my own case of eight years, before an operation was had recourse to.

The tumour is evidently connected with bone; its development does not excite much pain, and for a considerable time the diagnosis is attended with great difficulty; in fact,

1 'Medico-Chirurg. Transactions,' vol. x, p. 278,
the case does not become clear until the thin wall of the bone gives way, and the foreign bodies are discharged.

The treatment consists in removing as many of the hydatids as can be done without danger, and then destroying with chemical agents any which remain in the cavity, as likewise the lining membrane of the cavity itself. If any cysts, or any portion of the main cyst, be allowed to remain, new hydatids may be developed with great rapidity. This was a source of much annoyance, and even danger, in the case published by Mr. Keate. In the case just related, I rubbed the whole of the cyst freely with nitrate of silver, and I think this a practice which should always be followed.
A CASE
OF
FIBROUS POLYPUS
OF THE
URINARY BLADDER,
WITH OBSERVATIONS, AND A TABLE OF THE RECORDED CASES.

BY
JOHN BIRKETT,
SURGEON TO GUY'S HOSPITAL; CORRESPONDING MEMBER OF THE SOCIETY
DE CHIRURGIE DE PARIS; FELLOW OF THE ROYAL MEDICAL
AND CHIRURGICAL SOCIETY.

Received March 4th.—Read May 30th, 1848.

If the rareness of a disease, its practical bearings, and
generally fatal results, afford good reasons for imposing a
claim upon the attention of the Fellows of this Society, the
case which I am about to relate requires no apology from
me for its introduction.

The new growths, or tumours, developed within the
urinary bladder may be thus described:

1. Papilloma.
2. Fibrous polypus.
3. Villous growths.
4. Epithelioma.
5. Carcinoma—
   a. Infiltrating.
   b. Tuberous—

Of these the classes 1, 2, 3 and 4 are developed in relation
with the mucous membrane and sub-mucous connective tissues. They may be sessile or pedunculated. Growths of the fourth class may either infiltrate all the tissues of the viscus, or form circumscribed masses in or around it.

It is upon the second class that I desire to concentrate our attention in this paper.

In order to identify this new growth, I would state that it resembles in every anatomical particular the succulent fibrous growth, so well known by the name of the "nasal polypus," this being the true type of the disease.

I wish to exclude from this class all those diseases described as "fungus" of the bladder, a very common one in comparison with true polypus.

Polypus of the bladder would seem to be a very uncommon disease, at least if we judge of its frequency by recorded cases and the statements of authors.

Dr. Baillie writes: "A polypus sometimes grows from the mucous surface of the bladder; but this morbid appearance occurs very rarely. I have only seen one example of it."¹

Dr. Gross "has not had an opportunity of observing an instance of polypi in the bladder."

Warner relates one case; Crosse another.²

M. Petit describes a case; M. Chopart two cases, although I am doubtful whether one of these was really polypus.

Walter relates a case very closely resembling the one to be detailed directly; there is one preparation in the museum of Guy's Hospital;³ and my friend, Mr. Prescott Hewett,

¹ This preparation is in the Hunterian Museum, at the Royal College of Surgeons of England (No. 1999), and is in very fine preservation. It was "from a young girl. The urethra is stretched by the growths projecting into it to nearly an inch diameter." (Catalogue.) The engraving in Dr. Baillie's work very imperfectly represents its character.

² This preparation is in the museum of the Royal College of Surgeons (No. 2000), and exhibits the character of the disease very well.

³ This was presented by Sir Astley Cooper, and among his drawings at the Royal College of Surgeons there is a delineation of it, with these words subscribed in his own handwriting: "Tumours of the mucous mem-
remembers seeing, at St. George's Hospital, the bladder of a male adult, in which a large polypus was attached to the side of the neck of the bladder by a long pedicle. This allowed the polypus to pass into the urethra which was thereby dilated.

Mr. W. S. Savory has given the details of a case of polypus of the urinary bladder, which occurred in the practice of Mr. Stanley, at St. Bartholomew's Hospital, in the museum of which establishment the preparation is preserved. In this instance there was a pedunculated growth, "stretching transversely across the fundus of the bladder, immediately behind the apertures of the ureters which were much dilated. This mass was attached at either side, but was free in the centre." The minute anatomy of this growth is described by Mr. Paget.¹

In chronological order, Mr. Warner, formerly surgeon to Guy's Hospital, is the first author who has recorded a case of this disease. He successfully applied a ligature around the pedicle of the growth, for it sloughed off, and he cured the patient.

Cystotomy has been performed upon some patients, in consequence of obscure symptoms of calculus vesicæ existing. This occurred in the practice of Mr. Crosse, of Norwich, and M. Petit, of Lyons. The patients both died. In two cases the patients were the subjects of stone in the bladder. The operator on one case, M. Deschamps, removed the stone, but did not interfere with the growth; the other surgeon, M. Dessault, after the extraction of the stone, twisted off the growth. Both these cases are reported cured.

With this brief outline of the literature of the subject, I shall proceed to the relation of the case.

brane of the bladder everted. Polypus excrescences producing symptoms of stone." ("Path. Drawings," vol. i, under heading "Urinary Bladder.") All these drawings have recently been arranged by Mr. Quackett.

THE CASE.—S. A. J., a female, 5th, was admitted into Guy's Hospital, under my care, December 26th, 1857. She was a strumous child, in a most cachectic and miserable condition.

The mother stated that about eight weeks since her child first complained of pain in the hypogastric region, and that she, the mother, observed a diminution in the quantity of the urine passed; and that about five weeks since complete retention of urine occurred. A catheter was employed by a surgeon to empty the bladder about this date, for the first time. Muco-purulent deposit was noticed in the urine about four weeks since. The child had suffered from a severe fall about six months before admission, but had quite recovered from the effects of the accident.

At the time of admission the child was unable to pass urine voluntarily; some dribbled away involuntarily, but unless the catheter was used, the bladder became greatly distended. It was difficult to institute a very rigid examination at this moment, from the irritability of the patient and her intolerance of the least restraint. Her constitutional powers were very low; she was emaciated, had little appetite, and appeared as if she had lost much blood. The mother, however, said she had never noticed any bleeding. The urine was turbid, dark coloured, ammoniacal, and deposited a muco-purulent precipitate.

Treatment.—The catheter to be introduced as frequently as required. The diet to be nutritious and light, and to take Syr. Ferri Iodidi, 3ss, three times a day, in a little water.

Day after day there seemed to be, at first, slight constitutional improvement, but the difficulty in micturition continued, and on the 7th January, a dark-red growth protruded between the vulva. This rather interfered with the passage of the catheter, unless the instrument was passed along its side or behind it. As the child would not permit a careful examination, I placed her under the influence of
chloroform on January 12th, and then, having emptied the urinary bladder, I ascertained, with the utmost facility, that the growth projected through the meatus urinarius, and was attached to the superior boundary of the neck of the bladder. The index finger could be passed into the bladder behind the growth without impediment, and the projecting portion could be partially returned into the cavity of the organ. A hard mass could be felt in the pubic region, immediately behind the symphysis, which was enveloped by the contracted bladder. The new growth was composed of lobes and lobules, was soft, but strong enough to resist ordinary manipulation; it was not very vascular, but resembled closely the firmer varieties of nasal polypus. The attachment of the growth to the superior region of the neck of the bladder was satisfactorily ascertained by the fact that the finger freely passed into the bladder behind it, as well as on its side, but, directly in the middle line of the body and above, the peduncle of the growth itself prevented the introduction of the finger into the cavity of the viscus.

I passed a strong silk ligature around the root of that portion of the growth which protruded, and tied a knot tightly. This ligature slightly cut into the substance of the growth, but there was not any bleeding.

This operation caused neither local nor constitutional disturbance, but the same amount of retention of urine continued, and the child became daily more weak. The piece of the growth around which the ligature was tied sloughed off, but more protruded.

A few days after this, considerable pyrexia supervened, and on the twenty-fifth day after her admission she died.

A portion of the growth which came off when I examined her under chloroform was at that time carefully dissected by Dr. Wilks, who described it to be composed of fibrous tissue, resembling the ordinary forms of polypus developed in relation with the mucous membranes, especially the nasal.

Necropsy and description of the Polypus, by Dr. Wilks.—
The body was emaciated. The viscera, with the exception of those to be mentioned, were free from disease and any trace of new growths.

Both the kidneys were pale, rather large, and firm. The right showed small points of suppuration in its cortical substance. The pelvis and upper part of the ureter was inflamed. The ureters were dilated and their coats thickened.

The urinary bladder was of large size for a child, its mucous coat injected, and the muscular tissue hypertrophied. The posterior border of the external orifice of the meatus was thick and everted, and projecting through it there was a growth about one inch and a half in diameter. Within the bladder were a number of other growths, which together formed a mass of equal size to that without.

To commence with a description of the growths within the bladder. They were growing from the internal surface of the anterior wall of the organ, reaching as low as the meatus, and extending on the sides to the junction of the anterior two thirds with the inferior third of the viscus. They consisted, for the most part, of distinct growths, approximated together and having, more or less, of the polypoid form; that is, their points of attachment were much narrower than their free extremities, although, with the exception of one or two, they could scarcely be called pedunculated. However, when taken together, they formed a conical or pyriform mass, the apex of which was attached to the vesical orifice of the urethra, and the base was free in the bladder. The organ being contracted, it was nearly filled by the growth, especially at its lower part. When the bladder was everted, the growths became separated from one another, and showed then their distinct points of attachment, or became so many polypi. The largest of them, about an inch in diameter, had its origin on the anterior wall of the bladder, just above the meatus, and it projected backwards into its cavity. At its sides and on the posterior wall were three or four other polypi, of similar shape, but smaller; one of these, fissured on the surface, assumed a warty as-
pect, and resembled very closely the papillary growths on
the labia pudendi. Between these larger polypi were some
smaller excrescences of the same character, and around them
all, forming a kind of collar, in which all the growths were
included, was a raised margin of new tissue. This sur-
rrounded the openings of the ureters, but did not occlude
them. Beyond this margin the mucous membrane was
healthy, and so continued through the fundus. All the
growths had their seat in the mucous membrane and sub-
mucous connective tissues, the former extending over their
surfaces.

The growth outside the bladder which protruded from
the meatus grew, principally, from the anterior surface and
sides of the canal, the posterior surface being free; and
thus it was along the under surface of the growth that the
catheter entered the bladder. The mucous membrane of
the urethra was slightly everted with the extruding mass.
This was composed of seven or eight lobes, all of which,
however, sprung from the same source—the superior sur-
face of the meatus; and these could not be so distinctly
separated into polypi as those within the bladder. One of
these was of a warty character, like that in the interior.
This extra-vesical tumour had evidently grown within the
bladder, but from having been extruded for some time, it
could not be returned. If it had been possible to replace
it, it would have become approximated to those within, and
thus together they would have formed a conical mass, filling
the lower half of the moderately distended bladder.

Microscopical Examination of the Structure of the New
Growth.—The growths, when cut, were found to be soft;
amost as soft and succulent as nasal polypi. They showed
none of the fibrous vascular characters of uterine polypi.
They were easily broken up, having no fibrous arrangement
visible to the naked eye, and when a small piece was torn
up on a plate of glass, such a structure was only just per-
ceptible. The blood-vessels could not be seen, when mag-
nified, owing to this breaking up into amorphous pieces.
These consisted of cells and nuclei, having a glistening appearance, but, at their edges, the matrix could be seen. This was composed of a network of most delicate fibres, which were short, curled, interwoven, and resembled somewhat elastic tissue, producing such an appearance as a number of short, curling hairs would, if mixed together. In this delicate interlacing reticulum the cells and nuclei were seen. These split up into a number of fine fibres, which joined with the fibres of the plexus. In some places, where the cells had been removed, the network and the cells themselves could be examined separately.

The surface of the growth was covered with epithelium, and on scraping it an abundance of nucleated cells, columnar, and some ciliated epithelium, were observed. Probably the last mentioned came from the vagina.

The progressive stages of this case appear to be as follows:

1. The development of a growth within the urinary bladder, causing, at first, an impediment to the free passage of the urine, and, at last, complete retention of that secretion.

2. The development of secondary inflammatory disease in the bladder, ureters, and kidneys, the result of irritation arising from a morbid state of the mucous membrane of the bladder itself.

The origin of that irritation must, then, be assigned to mechanical causes interfering with the escape of the urine, rather than to any influence exerted on the constitution of the child by the nature of the primary disease or growth itself. For this was simply composed of fibre-tissue; and if the child had come under treatment at an earlier period, there might have been a chance of effecting a permanent cure.

The incipient results of the secondary disease were manifested even on admission, by the cachetic state of the sufferer and the extreme constitutional depression; although, doubtless, the immediate cause of death—suppuration of the right kidney—was not developed until within three or four days of dissolution.
From the cases recorded, it appears that both the sexes may be afflicted with this disease; and excluding the two cases related by M. Chopard, on account of their doubtful nature, there are five males and four females.

The respective ages of these patients have varied between thirteen months and twenty-eight years of age. In five cases the disease was developed before puberty; in four, after that period.

Of these nine cases, eight terminated fatally.

In six only the treatment is described. In two of them cystotomy was performed; the patients surviving this operation, in one case, forty-four hours, in the other, about a year. The polypus could not be entirely removed from either. Mr. Warner applied a ligature around the peduncle of the growth in one case; it sloughed off, and this patient is the only one that was cured.

The duration of the disease, from the manifestation of the first symptoms to the death of the patients, has varied between about ten weeks and eighteen months. In Mr. Warner's case, however, the disease had existed three years before the patient came under his treatment.

In three cases death is stated to have been caused by disease generated in the kidneys, as the result of irritation in these secreting organs from retention of urine and the morbid state of the bladder and ureters.

The anatomical description of the morbid growths developed in these cases agrees very closely; the chief difference between them appearing to be the size of the new growth, the site of its attachment to the organ, its shape, and mode of connexion with the viscus, some of the growths being sessile, others having long peduncles.

**Differential Diagnosis.**—Polypus in the urinary bladder is developed, generally, at an earlier age than the other growths which are met with in this organ.

It is not accompanied with the same amount of hæmorrhage as the growths known by the name "vascular fun-
gus," "epithelioma," or "medullary cancer." If a little
blood should mingle with the urine, it is generally the re-
sult of abrasion of surface by the passage of an instrument
to relieve retention of urine.

Dysuria, at first, which is soon after followed by reten-
tion of urine, distinguishes this disease from other intra-
vesical growths, for, symptomatic of such growths, unless
they be developed very close to the vesical orifice of the
urethra, and have long peduncles—a circumstance of rare
occurrence—blood mixed with the urine is generally the
first indication which attracts the notice of the patient.
Especially, too, in some cases, the expulsion of a few drops
of blood, or of two or three small coagula, after the volun-
tary act of micturition has ceased. After the bleeding, in
these cases, dysuria next ensues. In polypus, dysuria first
occurs, and then, after the use of the catheter, perhaps a
little bleeding.

The anaemic state of the urine then forms a distinguishing
feature of the disease, of course, after having ascertained
that the dysuria is independent of prostatic or urethral
disease.

Polypus in the bladder would be distinguished from
calculus, generally, by sounding the stone—that is, by
hearing it ring against the metallic instrument with which
the operation is performed. A careful distinction being
made by the surgeon between hearing the sound produced
by the metal impinging against the stone, and detecting, by
the sense of touch, that some foreign body exists in the
viscus.

Some of the diseases of the prostate might give rise to
symptoms resembling those accompanying polypus in the
bladder, but a correct diagnosis might be formed by con-
sidering the ages of the patients in whom these diseases
occur, as well as by a careful examination of the region
per anum.

*Treatment.*—An examination of the morbid anatomy of the
bladders in which polypus growths are developed, affords but slight encouragement to hope that any operation would be successful in removing the whole of the disease. The application of a ligature around the growths which happen to protrude through the urethra in the female, might be again successful, as was the issue in Mr. Warner’s patient, who even dilated the urethra, and enlarged it by incision, in order to reach the peduncle of the growth. In that case, however, the fortunate circumstance of the growth having a long peduncle, and of its being, in all probability, solitary, must be regarded.

The case related by Mr. Crosse cannot, perhaps, be quoted in uncontrovertible opposition to the performance of an incision into the bladder of the male in the hope of removing the growth, since the case terminated successfully which is related by M. Chopart, of Dessault, who “twisted off a fungus” which he found growing from the bladder of a male, upon whom he had performed cystotomy to remove a stone.

Also, in the case of M. Petit, although he did not remove the polypus, the operation of opening the bladder was not attended with any bad result, and a year afterwards, on post-mortem examination, it was discovered that there was a polypus the size of the fist in the bladder, of a pyramidal figure, and attached by a very delicate pedicle.

Surely it would be practicable to apply a ligature around a growth similar to this!
TABLE OF CASES OF POLYPUS

<table>
<thead>
<tr>
<th>Case</th>
<th>By whom related. Date</th>
<th>Sex</th>
<th>Age</th>
<th>Treatment</th>
<th>Result</th>
<th>Cause of death</th>
</tr>
</thead>
<tbody>
<tr>
<td>I.</td>
<td>Warner, 1747</td>
<td>F.</td>
<td>23</td>
<td>Dilatation and incision of meatus; ligation of peduncle.</td>
<td>Cured.</td>
<td>......</td>
</tr>
<tr>
<td>II.</td>
<td>Baillie, 1799</td>
<td>F.</td>
<td>'young'</td>
<td>......</td>
<td>Death.</td>
<td>......</td>
</tr>
<tr>
<td>III.</td>
<td>Walter, F. A., 1800</td>
<td>F.</td>
<td>20</td>
<td>......</td>
<td>Death.</td>
<td>......</td>
</tr>
<tr>
<td>IV.</td>
<td>Crose, 1830 to 1835</td>
<td>M.</td>
<td>1 to 2</td>
<td>Cystotomy and removal of portions of the growth.</td>
<td>Death, 44 hours after.</td>
<td>Exhaustion.</td>
</tr>
<tr>
<td>V.</td>
<td>Stanley-Savory, 1832</td>
<td>M.</td>
<td>1</td>
<td>Introduction of sounds and catheters.</td>
<td>Death.</td>
<td>Disease of Kidney.</td>
</tr>
<tr>
<td>VI.</td>
<td>Prescott Hewett</td>
<td>M.</td>
<td>Adult</td>
<td>......</td>
<td>Death.</td>
<td>......</td>
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<tr>
<td>VII.</td>
<td>Cooper, Sir Astley</td>
<td>M.</td>
<td>10 to 12</td>
<td>......</td>
<td>Death.</td>
<td>......</td>
</tr>
<tr>
<td>IX.</td>
<td>Chopart, of Dessault.</td>
<td>M.</td>
<td>...</td>
<td>Cystotomy; stone extracted; &quot;fungus twisted off.&quot;</td>
<td>Cured.</td>
<td>......</td>
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OF THE URINARY BLADDER.

<table>
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<tr>
<td>About 3 years.</td>
<td>&quot;The size of the tumour was nearly equal to a turkey's egg, and something like to it in shape.&quot;</td>
<td>Cases in Surgery, &amp;c., 8vo., fourth ed., p. 303, Lond., 1784.</td>
</tr>
<tr>
<td>......</td>
<td>The growth filled the greater part of the cavity of the bladder, and consisted of various projecting masses, some of which, passing into the urethra, stretched it.</td>
<td>Morbid Anatomy, and Series of Engravings, 4to., fasc. vii, pl. iv, fig. 2, Lond., 1799.</td>
</tr>
<tr>
<td>About 6 months.</td>
<td>Bladder filled with polypi. Ureters dilated; renal pelvis also. Fuss in walls of bladder.</td>
<td>A treatise on the formation, &amp;c., of the Urinary Calculus, 4to., p. 44, pl. xx, Lond., 1835.</td>
</tr>
<tr>
<td>......</td>
<td>Growth projecting into urethra.</td>
<td>Personal information.</td>
</tr>
<tr>
<td>......</td>
<td>Numerous pedunculated growths in bladder.</td>
<td>Preparation in Guy’s Hospital Museum.</td>
</tr>
<tr>
<td>About 3 months.</td>
<td>Growth attached to anterior surface of neck of bladder, lobulated, and projecting through a very dilated meatus urinarius.</td>
<td>Preparation and drawings in Guy’s Hospital Museum.</td>
</tr>
<tr>
<td>......</td>
<td>Necropsy. A polypus the size of the fist in the bladder, of a pyramidal figure, and attached by a very delicate pedicle.</td>
<td>Méd. du cœur et discours, p. 349.</td>
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RESEARCHES ON GOUT.

PART I.—THE URINE IN THE DIFFERENT FORMS OF GOUT.

PART II.—THE INFLUENCE OF COLCHICUM UPON THE URINE.

BY

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Received June 7th.—Read June 8th, 1868.

PART I.—THE URINE IN THE DIFFERENT FORMS OF GOUT.

Considerable discrepancy of opinion at present exists with regard to the characters presented by the urine of patients labouring under the different forms of gout, and statements of a conflicting nature have been often put forward, in works devoted to the consideration of this disease. Such discrepancies have arisen, partly from the urine presenting great varieties of appearance in different subjects and at different periods, and partly, also, from the imperfect methods which have been adopted for the examination of this fluid. The errors, however, have not usually been caused by any defect in the methods of analysis,
but have arisen rather from the mode in which the estima-
tions have been afterwards made.

In the present examination, one of my chief objects has
been to give the actual results arrived at by the clinical
investigation of the urine in cases of this disease, carried
over a period of several years.

The points most dwelt upon are the amount of uric acid
eliminated in the twenty-four hours; in some cases, the
quantity of urea also; and I may here mention that the
following very simple methods of examination were adopted.

Every care was taken that the entire quantity of urine
passed in the twenty-four hours should be collected and mea-
sured. After the specific gravity, reaction, and other points
regarding the physical characters were ascertained, a portion,
usually from 10 to 20 fluid ounces, was acidulated with
hydrochloric acid, by the addition of about one twentieth of
its bulk of that acid, the urine having been previously
filtered, and any deposit, if present, made soluble; the
mixed fluids were then set aside for at least forty-eight hours,
and the deposited uric acid carefully collected, washed
with spirit, dried, and weighed.

The urea determinations were made either by Liebig’s or
by Dr. Davy’s process, the same method being always em-
ployed in the examinations throughout the investigation of
each case.

It should be observed that, in the uric acid determina-
tions, the weighed quantities are always under the real, as a
minute portion of uric acid is soluble in the acidulated
urine. This slight error, however, does not in any way
alter the deductions drawn from the results obtained.

On referring to some of the works on gout usually
looked upon as of authority, it will be seen that the uric
acid is often considered to be thrown out in great excess,
and the facts appealed to, in support of such opinion, are
the appearance of a copious deposit of urates, which not
unfrequently occurs during and after an acute gouty attack;
also the common phenomenon of the brick-dust sediments
in the urine of gouty individuals; and lastly, the occasional
alternation of nephritic gravel and gout, and the liability to
the occurrence of gout and calculus in the same patient.

In speaking of the constitutional symptoms of gout, the
late Sir C. Scudamore, in his treatise, remarks: "The urine
is of a deeper colour than natural, is secreted scantily with
relation to the quantity of the patient's drink, and, on
cooling, deposits a pink or brick-dust sediment, with much
mucus. Its specific gravity is much increased beyond the
healthy standard. During the most urgent symptoms of
the paroxysm it is usually passed with considerable irrita-
tion, both as to frequency and sense of heat. The pink or
lateritious sediment appears, more or less, in every portion
of the urine during the inflammatory symptoms." This
description of the urine is intended to apply to the genuine
acute affection, and in patients not suffering from the
chronic forms of the disease. In another part of the same
work, we find it remarked: "It is very commonly found,
upon inquiry, that the patient has noticed a very deficient
secretion of urine a short time before the occurrence of the
paroxysm, and consequently it has been of a deeper colour
than natural. It also now and then certainly occurs that,
for a day or two before the fit, the urine is passed copiously,
and of a pale colour; but this seldom happens, except in
persons of the nervous temperament, and whose constitu-
tions have been much weakened by gout." And again:
"A deposition of pink or brick-dust sediment, on the
cooling of the urine, is of such ordinary occurrence when
any active symptoms of gout are present, that its connexion
becomes forcibly impressed on the mind of the patient, and
he gives it the name of gouty urine." These statements
agree, in the main, with those of other writers who have
noticed the condition of this fluid; but, on the other hand,
it must be observed that the same author further asserts:
"In several examples of chalk stones, both in the hands and
feet, I have found, by repeated experiments, a deficiency,
and sometimes almost total absence of uric acid in the
urine. I must add, however, that even in patients of this
class, during the paroxysm, this principle has existed in the
urine in considerable quantity, and in some cases to the extent of a copious deposit of pink sediment."

These statements of Sir C. Scudamore, as to the physical appearances of the urine in different forms of gout, are, I believe, essentially correct; and it will be seen, from the present communication, that the results now arrived at in no way militate against them; they will, however, prove, as before stated, that the deductions which have usually been drawn are in a great measure erroneous.

I shall divide the cases of gout, in which the examinations have been made, into three classes.

1st Class.—Cases of acute gout occurring in patients, most of whom, in the intervals of the attacks, enjoyed pretty good health. I may here remark the great difficulty of procuring cases of this kind in hospital practice. In the first place, but few, suffering from a simple attack, enter such institutions; and, among the labouring classes, there is a greater tendency for the disease to assume, at an early period, a chronic and asthenic character, than in the higher walks of life—a fact doubtless much depending upon the different modes of living in the two classes. This renders the examinations of the urine, in the acute disease, less numerous and satisfactory than those made in chronic forms of the affection; and it still remains an interesting point to determine the exact condition of the urinary secretion in genuine acute and asthenic gout occurring among the higher classes of society. The difficulty of such an investigation, when the analysis of the twenty-four hours' urine is desired, is by no means small.

2d Class.—Cases of chronic gout occurring in patients, the majority of whom were not suffering from any very urgent symptoms, but many were afflicted with some of the sequelae of the affection, as shown by the concretions of urate of soda upon different parts of the body, and the stiffened condition of the joints.
3d Class.—Cases of individuals, hospital or private patients, who were free from all symptoms of the disease when the examinations were made, and who, at the same time, were not troubled with any of the sequelæ so commonly found after repeated attacks of gout.

Class I.—Urine of subjects suffering from Acute Gout, but in whom the general health had been pretty good during the intervals of the paroxysms.

Besides the great difficulty already alluded to in procuring the urine of the whole day in private practice, many little circumstances are likely to occur which interfere so much with the results as not to allow of the analysis being depended upon; and, as before observed, in hospital practice, where the investigations can be so much more readily carried out, the number of these cases is limited, patients seldom applying for admission to such institutions till the disease is advanced, and has assumed more or less of a chronic character.

The examinations of the urine in this class are thirty-two in number, and in seven different individuals.

Case 1.—W. R.—male, æt. 32; suffering from acute gout of right foot and ankle, likewise of some of the smaller joints of hands, and accompanied with much febrile disturbance of system, and redness, swelling, and pain of affected parts. In the first attack, seven years before, the balls of both great toes were specially affected. No deposit of urate of soda observed either on ears or any other part of the body.

March 24th, 1853.—Urine of twenty-four hours, 35 fluid ounces, clear, sp. gr. 1019; total quantity of uric acid in the day = 5.95 grains.

The blood of this patient was found to be rich in uric acid, and crystals were also obtained from the serum of a blister applied to the knee. No medicines administered at the time.
CASE 2.—W. B—, male, æt. 46; acute gout, with inflammation of ball of great toe; no deposits of urate of soda; taking no medicine at the time of examination of urine; had no meat, and fish only during the last two days.

February 20th, 1858.—Urine of twenty-four hours, 42 fluid ounces; sp. gr. 1012·5, acid; uric acid in twenty-four hours = 0·84 grain.

21st.—Urine of twenty-four hours, 46 fluid ounces; sp. gr. 1015, acid; uric acid = 1·84 grain.

22d.—Urine of twenty-four hours, 63 fluid ounces; sp. gr. 1012, acid; uric acid = 2·52 grains.

After this time, took some colchicum for a day or so, which purged somewhat. Had discontinued it before the next examination.

26th.—Urine of twenty-four hours, 18 fluid ounces; sp. gr. 1023, acid; uric acid = 2·30 grains.

27th.—Urine of twenty-four hours, 36 fluid ounces; sp. gr. 1015, acid; uric acid = 2·76 grains.

From the 20th till the 22d the symptoms gradually subsided; after that time little more than tenderness of the previously affected parts remained.

The blood of this patient contained uric acid in abundance.

CASE 3.—B. F—, male, æt. 38; had suffered, during the last two years, from five or six attacks of gout, commencing in ball of great toe. No deformity of joints or deposits of urate visible in any part; the serum of blood rich in uric acid. From the 15th of January to the 19th of the same month was suffering considerably from inflammation of many large joints, besides the great toe; the urine was unaffected by medicines during the days the examinations were made.

January 15th.—Urine, 25 fluid ounces; sp. gr. 1012 at 60° Fahr., acid. No albumen. Uric acid eliminated in twenty-four hours = 1·30 grain.

16th.—Urine, 30 fluid ounces; sp. gr. 1010, acid; uric acid in twenty-four hours = 1·95 grain.
RESEARCHES ON GOUT. 331

17th.—Urine, 26 fluid ounces; sp. gr. 1012, acid; uric acid in twenty-four hours = 2.73 grains.
18th.—Urine, 39 fluid ounces; sp. gr. 1011, acid; uric acid in twenty-four hours = 2.14 grains.
19th.—Urine, 47 fluid ounces; sp. gr. 1009, acid; uric acid in twenty-four hours = 3.05 grains.
The blood of this patient was rich in uric acid.

Case 4.—T. C,—male, æt. 57; gouty patient; had a few nodules of urate of soda in ear; suffering, when the urine was examined, from acute gout in the great toes and several other joints, both of upper and lower extremities; blood loaded with uric acid.
May 2d, 1856.—Urine, 20 fluid ounces; sp. gr. 1024 at 60° Fahr.; turbid from urates, but clearing with moderate heat, containing a distinct trace of albumen. Uric acid excreted in twenty-four hours = 3.76 grains. Urea excreted in twenty-four hours = 320 grains.

Case 5.—J. P,—male, æt. 46; has had several attacks of gout during last ten years; no deformities of joints produced by the disease; one small deposit of urate of soda seen on helix of one ear. Blood contains abundance of uric acid. Suffering from slight, but increasing, gouty inflammation of both ankles and some other parts. During the first three days no medicine was taken; on the fourth had commenced taking colchicum, which was continued during the fifth and sixth days; the inflammation rapidly subsided under its influence.

Feb. 15th, 1858.—58 fl. ounces, sp. gr. 1020, uric acid in 24 hours = 8.12 grs.
  " 16th, " 58 " not determined " 8.12 "
  " 17th, " 43 " not determined " 3.44 "
  " 18th, " 59 " 4.80 "
  " 19th, " 59 " 2.95 "
  " 20th, " 42 " 0.17 "

No albumen in urine, acid in reaction.

Case 6.—T. D. M,—a male patient, about 50 years of age; gout of more than twenty years' standing; tophi of urate
of soda in ears, and many joints affected with chalk-like deposits around them. When the urine was first examined, January 8th, 1853, many joints were red and swollen from acute gout. The note of that day was as follows: swelling, heat, redness and tenderness of dorsum of right hand, and also of smaller joints of fingers, also of left wrist; considerable pitting of dorsum of hand on pressure; right knee swollen, red, and tender; left knee less affected; pulse 100, rather full and hard; skin somewhat hot and moist. January 11th, the gouty pains were almost gone; and the patient could move his joints. Pulse 72.

January 15th.—Quite free from pain. Pulse 64.

18th.—Discharged cured.

This patient was ordered, on the 8th, to take, three times a day, 20 minim doses of Vinum Colchici, 3ss of Magnesia, usta, and m/s of Tinctura Opii; the opium was omitted on the 11th, after the subsidence of the pain.

The blood of this patient, examined during several attacks, always contained abundance of uric acid.

The urine dated January 9th was that passed from 10 a.m. January 8th, to 10 a.m. January 9th, and so on for the other days.

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<tbody>
<tr>
<td>1853.</td>
<td>Jan. 9th.—1/4 oz. 10 gr.</td>
<td>1016-5 at 60° F.</td>
<td>Acid</td>
<td>Dark claret red, no deposit.</td>
<td>0-5 gr.</td>
</tr>
<tr>
<td>10th.</td>
<td>26 gr.</td>
<td>1014</td>
<td>Acid</td>
<td>Dark red, no deposit.</td>
<td>1-7</td>
</tr>
<tr>
<td>11th.</td>
<td>32 gr.</td>
<td>1013</td>
<td>Acid</td>
<td>Lighter colour, clear, with uric acid deposit.</td>
<td>5-3</td>
</tr>
<tr>
<td>12th.</td>
<td>36 gr.</td>
<td>1013</td>
<td>Acid</td>
<td>Clear pale yellow, no deposit.</td>
<td>5-5</td>
</tr>
<tr>
<td>13th.</td>
<td>28 gr.</td>
<td>1016</td>
<td>Acid</td>
<td>Clear.</td>
<td>5-5</td>
</tr>
<tr>
<td>14th.</td>
<td>14 gr.</td>
<td>1016-5</td>
<td>Acid</td>
<td>Clear.</td>
<td>4-0</td>
</tr>
<tr>
<td>15th.</td>
<td>18 gr.</td>
<td>1019-5</td>
<td>Acid</td>
<td>Clear.</td>
<td>3-3</td>
</tr>
<tr>
<td>16th.</td>
<td>18 gr.</td>
<td>1019</td>
<td>Acid</td>
<td>Clear.</td>
<td>3-6</td>
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<tr>
<td>17th.</td>
<td>25 gr.</td>
<td>1016-5</td>
<td>Acid</td>
<td>Clear.</td>
<td>3-4</td>
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<tr>
<td>18th.</td>
<td>36 gr.</td>
<td>1015-3</td>
<td>Acid</td>
<td>Clear.</td>
<td>2-9</td>
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The same patient's urine, examined at the commence-
ment of another attack, before taking medicine, gave the following results:

Quantity, 17 fluid grains; sp. gr. 1021, acid; uric acid in twenty-four hours = 0.425 grain.

The patient’s urine, during acute attacks of gout, gave distinct evidence of albumen, but this principle could not be discovered in the intervals.

Case 7.—W. F—, a male patient, æt. 41; has had, during the last ten years, several attacks of gout; many small deposits of urate of soda seen on the palmar surface of left index finger, but none on ears. An acute attack now passing off.

March 27th, 1858.—Urine, 24 fluid ounces; sp. gr. 1024, acid in reaction, of an amber colour, and depositing a yellow urate sediment on cooling. Uric acid passed in the twenty-four hours = 5.20 grains. Urine gave an abundant crystallization of nitrate of urea on the addition of nitric acid.

April 8th.—Has been free from all acute symptoms for some days. Urine, 48 fluid ounces; sp. gr. 1017, acid, clear, no deposit. Uric acid in the twenty-four hours = 1.36 grain.

From these cases it will be seen that in acute gout, as far as hospital patients are concerned, the daily excretion of uric acid is by no means necessarily increased, nay, often notably diminished; for we can fairly take the normal average amount of uric acid at about eight grains in the twenty-four hours, although either much larger or considerably smaller quantities may be eliminated in conditions of perfect health.

In the seven cases given above the highest amount was only 3.12 grains; the lowest, 0.425 grain; the average of the analyses in each case being 5.95 grains, 2.05 grains, 2.58 grains, 3.76 grains, 4.46 grains, 3.28 grains, and 3.28 grains; and the average of the analyses in all the cases, 3.62 grains. In some the urine was of a high colour, in
others turbid from urates; and again, in certain specimens there was a deposition of crystalline uric acid; lastly, in many cases the urine was clear and free from all deposit.

It should be remembered that the appearance of any given specimen of urine is by no means a certain criterion of the quantity of uric acid contained in it; many urines which remain perfectly clear on cooling are richer in this acid than others which are exceedingly turbid, circumstances other than the mere amount of uric acid determining its precipitation, either in the free form or in that of a urate.

The cause of the very prevalent idea, that there is always an excess of uric acid thrown out of the system by gouty patients, and especially in the acute disease, may, I believe, be explained by the fact, that when the febrile disturbance is great, the renal secretion usually becomes scanty in quantity, its acidity is often increased, and almost the whole of the uric acid is thus rendered visible to the eye. The precipitation of the colouring matter with the uric acid increases greatly the appearance of excess.

Having now established the fact, that patients suffering from acute gout do not necessarily excrete an excessive amount of uric acid, but in fact that the reverse often occurs, it next becomes an interesting question to determine whether this defect depends on any diminution in the formation of this principle in the economy, or on its imperfect excretion by the kidneys. This point is readily settled by an examination of the blood, which was performed in every instance, and proof was afforded by such analyses that in all there existed a considerable quantity of uric acid in the serum of that fluid; at once proving that the renal organs were unable to excrete the whole of the uric acid formed in the system: and as the elimination of this acid was shown to be under the average amount, demonstration was afforded that the kidneys had lost, at least for a time, some of their usual power. In many diseases where an abnormal formation of uric acid takes place, such as in cases of diseased spleen, liver, &c., although the production of uric acid may be greatly increased, still the kidneys retain the power of
fully eliminating it, and the blood is kept, as far as this principle is concerned, in a pure condition; such, however, as we have now seen, and as I have demonstrated in former papers published in the 'Transactions' of this Society, is not the case in gouty subjects, and during the continuance of this affection the circulating fluid is always contaminated by the presence of a large quantity of uric acid, whatever may be the amount thrown out by the kidneys. This point is one of considerable importance, and not generally fully understood, for it is frequently assumed that the appearance of a large quantity of uric acid in the urine indicates that the blood is contaminated with the same body, although, on reflection, it will be seen that such an occurrence rather favours the opposite view, for if the kidneys excrete freely, the blood has a much greater chance of being kept pure.

It will be observed, in reviewing the results obtained from the above analyses, that in acute gout the quantity of uric acid eliminated in different days is liable to much variation; as a rule, when the examinations are made in the early stages of the attacks, the excretion is small, it then gradually becomes augmented, and afterwards returns to a lesser quantity. In Case 5 the quantity was largest during the two first days that the analyses were made; but in this instance the examination was not undertaken until the disease was about to become alleviated. My present experience on the subject appears to show that, in the earlier stages of acute gout, the urine, as a rule, is small in quantity, and the uric acid, measured by the twenty-four hours' excretion, likewise diminished; that this acid is thrown out in much larger quantities as the disease is becoming mitigated, and then amounts, even far above the patient's daily average, may be passed, forming frequently the so-called critical discharges; and lastly, the acid again becomes lessened, although not to the extent which occurs either just before or at the commencement of an attack.

In one case only (of patients in this class) was the amount of urea estimated, and then 320 grains, a fair
average quantity for a patient under low diet, was found. The elimination of urea was therefore shown not to be affected, in the same degree as that of the uric acid; although from repeated experiments I have ascertained, as I first pointed out in my communication to this Society in 1848, that the blood usually contains a slight abnormal amount of this latter principle in the acute forms of the disease, a circumstance which may possibly account for oedema being so frequently an accompaniment of this form of inflammation.

Sometimes a very distinct trace of albumen is found in the urine in acute attacks of gout; such was the case with the fourth and sixth patients. I may here remark that this phenomenon is not very frequent in early gout, but when the patient's disease has assumed a more chronic form, and especially when deposits of urate of soda are found, then, during the fit, albumen in traces is of very frequent occurrence, although in the intervals not the slightest evidence of the existence of this body may be obtained.

Class II.—Cases of Chronic Gout. The majority of the patients were not suffering from any very urgent symptoms at the time the analyses of their urine were made; but many were afflicted with some of the sequelæ of the affection, as shown by the concretions of urate of soda on different parts of the body, and the stiffened condition of the joints.

In this class the examinations were made upon fourteen different patients, and the results of more than sixty analyses are given.

Case 1.—J. H., male, aged 45; a sufferer from gout for fourteen years; has a deposit of urate of soda in one ear, but the joints are not deformed. At the time the urine was examined he was suffering in feet and ankles, also in right knee; no medicine was administered; the blood was rich in uric acid.

March 30th, 1853.—Urine, 56 fluid ounces; sp. gr. 1011;
clear, yellow; no deposit; very slight trace of albumen. Uric acid thrown out in the twenty-four hours = 5.78 grains.

**Case 2.**—J. M., æt. 85; a gentleman, having suffered from gout for fifty years, with concretions of urate of soda in many parts, and gouty abscesses of fingers, discharging chalk-like matter.

June, 1856.—Urine passed at 3 p.m., sp. gr. 1017, pale, clear; gives evidence of a small amount of albumen. Mere traces of uric acid thrown down by hydrochloric acid. Urine passed during the night, pale, sp. gr. 1018, acid; distinct traces of albumen. A few microscopic crystals of uric acid thrown down by hydrochloric acid.

**Case 3.**—W. L., æt. 61; a gentleman, much deformed in the joints of the hands and feet from large deposits of urate of soda.

May, 1857.—Urine three or four hours after dinner, sp. gr. 1015, pale yellow, acid in reaction; decidedly, though slightly, albuminous. Acidulated with hydrochloric acid, it gave only a few microscopic crystals of uric acid, not sufficient to collect and weigh.

**Case 4.**—A. H., æt. 52; gout for many years; deposits of urate of soda in both ears, but not noticed elsewhere. No active symptoms; blood rich in uric acid; urine always pale and clear.

October 22d, 1849.—Urine, 72 fluid ounces; sp. gr. 1010.

27th.—Urine, 56 fluid ounces; sp. gr. 1612 at 60° Fahr.; uric acid eliminated in twenty-four hours = 0.38 grain.

November 4th.—Urine, 52 fluid ounces; sp. gr. 1009 at 60° Fahr.; acid in reaction; uric acid in the twenty-four hours = 0.20 grain.

**Case 5.**—R. H., male, æt. 41; patient with chronic gout of several years' standing; some nodules of urate of xli.

22
soda in the ears and on the palmar surfaces of one or two fingers; no active gout present when the urine was examined; blood rich in uric acid.

June, 1847.—Urine, 42 fluid ounces; sp. gr. 1015, acid, pale in colour, slightly albuminous; uric acid in twenty-four hours, only a few microscopic crystals.

This patient's urine was often examined, and with similar results.

Case 6.—D. R., male patient, set, about 45, with deposits in ears and distorted hands, having suffered from gout during ten years. Urine, 65 fluid ounces; sp. gr. 1014, clear, acid; no trace of uric acid exhibited. A very small quantity of albumen contained in the urine.

Case 7.—W. B., set, 52; a patient having very extensive deposits of urate of soda, in the form of chalk stones, and with considerable deformity of the joints; often suffering more or less from pain and tenderness in some of the articulations. Several examinations of urine were made at different periods; blood rich in uric acid.

September, 1848.—Urine, 50 fluid ounces; pale, acid. Uric acid obtained from the urine of twenty-four hours = 0.50 grain.

December 9th.—Urine, 38 fluid ounces; sp. gr. 1014, acid. Uric acid in twenty-four hours, a few microscopic crystals.

1849.—Urine, 60 fluid ounces. Uric acid in twenty-four hours, not sufficient to weigh.

February, 1860.—Urine, 60 fluid ounces. Uric acid in twenty-four hours, a mere trace.

This patient's urine always gave indications of the presence of a minute quantity of albumen.

Case 8.—D. M., a female, set. 39, spare habit, extremely gouty, although the first attack came on but three years since. She is covered with chalk stones, which have caused the greatest deformity both of hands and feet.
1848.—Urine, quantity in twenty-hours not ascertained; pale; sp. gr. 1010; slightly albuminous; contained no trace of uric acid.

Case 9.—W. M—, æt. 52; a patient having suffered from gout many years; considerable stiffness of joints, and deposits of urate of soda in ears and elsewhere. No active symptoms present.

March 29th, 1853.—Urine, 35 fluid ounces; sp. gr. 1016, clear, yellow; no distinct evidence of albumen. Uric acid in twenty-four hours = 2·5 grains.

At various periods this patient's urine was examined, and often gave but a mere trace of uric acid; the albumen, however, was always present. The blood, examined several times at intervals, was always found charged with the acid.

Case 10.—W. F—, male, æt. 38; has had several attacks of gout, at first affecting ball of great toe, afterwards implicating other joints, both of lower and upper extremities. A few points of urate deposit seen on palmar surface of left index finger. At the time of examination of the urine no symptoms of active gout were present, and no medicine taken; blood rich in uric acid.

May 11th, 1854.—Urine, 33½ fluid ounces; sp. gr. 1024, acid; no deposit; no albumen. Uric acid eliminated in twenty-four hours = 1·34 grain. Urea eliminated in twenty-four hours = 306 grains.

12th.—Urine, 32 fluid ounces, acid. Uric acid in twenty-four hours = 0·76 grain. Urea in twenty-four hours = 333 grains.

13th.—Urine, 30 fluid ounces; sp. gr. 1025, acid. Uric acid in twenty-four hours, too small to weigh. Urea in twenty-four hours = 344 grains.

15th.—Urine, 39½ fluid ounces; sp. gr. 1018, acid. Uric acid in twenty-four hours, too small to weigh. Urea in twenty-four hours = 358 grains.

Nearly four years after the above date, namely, in March
and April, 1858, this patient’s urine was again analysed. At the time the first examination was made the attack was just passing off. When the second analysis was performed he was free from all active symptoms.

March 28th, 1858.—Urine, 24 fluid ounces; sp. gr. 1024, acid, of an amber colour, depositing yellow urates on cooling. Uric acid in the twenty-four hours = 5·20 grains.

April 8th.—Urine, 48 fluid ounces; sp. gr. 1017, acid, giving rise to no deposit. Uric acid in the twenty-four hours = 1·36 grain.

Case 11.—C. F.—male, æt. 38; a man subject to gout for ten years. Attacks very numerous, first few confined to foot. Has concretions of urate of soda in left ear, and also a semi-fluid collection of urate of soda in left foot; some deposits also on finger; blood rich in uric acid.

For fourteen days in January, 1854, the patient’s symptoms being very chronic in character, the urine, when examined, gave the following results:

<table>
<thead>
<tr>
<th>Date</th>
<th>Quantity</th>
<th>Sp. gr.</th>
<th>Reaction</th>
<th>Physical Appearance</th>
<th>Uric Acid in 24 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jan. 4th.—57 fl. oz.</td>
<td>1012</td>
<td>Acid</td>
<td>Clear</td>
<td>0·50 gr.</td>
<td></td>
</tr>
<tr>
<td>&quot; 5th.—44 &quot;</td>
<td>1013</td>
<td>&quot; &quot;</td>
<td>&quot; &quot;</td>
<td>0·05 &quot;</td>
<td></td>
</tr>
<tr>
<td>&quot; 6th.—44 &quot;</td>
<td>1013</td>
<td>&quot; &quot;</td>
<td>&quot; &quot;</td>
<td>0·00 &quot;</td>
<td></td>
</tr>
<tr>
<td>&quot; 7th.—37 &quot;</td>
<td>1012</td>
<td>&quot; &quot;</td>
<td>&quot; &quot;</td>
<td>0·14 &quot;</td>
<td></td>
</tr>
<tr>
<td>&quot; 8th.—46 &quot;</td>
<td>1011</td>
<td>&quot; &quot;</td>
<td>&quot; &quot;</td>
<td>0·00 &quot;</td>
<td></td>
</tr>
<tr>
<td>&quot; 9th.—44 &quot;</td>
<td>1011</td>
<td>&quot; &quot;</td>
<td>&quot; &quot;</td>
<td>0·00 &quot;</td>
<td></td>
</tr>
<tr>
<td>&quot; 10th.—44 &quot;</td>
<td>1012</td>
<td>&quot; &quot;</td>
<td>&quot; &quot;</td>
<td>0·71 &quot;</td>
<td></td>
</tr>
<tr>
<td>&quot; 11th.—54 &quot;</td>
<td>1014</td>
<td>&quot; &quot;</td>
<td>&quot; &quot;</td>
<td>0·09 &quot;</td>
<td></td>
</tr>
<tr>
<td>&quot; 12th.—61 &quot;</td>
<td>1013</td>
<td>&quot; &quot;</td>
<td>&quot; &quot;</td>
<td>2·40 &quot;</td>
<td></td>
</tr>
<tr>
<td>&quot; 13th.—55 &quot;</td>
<td>1012</td>
<td>&quot; &quot;</td>
<td>&quot; &quot;</td>
<td>0·00 &quot;</td>
<td></td>
</tr>
<tr>
<td>&quot; 14th.—87 &quot;</td>
<td>1009</td>
<td>&quot; &quot;</td>
<td>&quot; &quot;</td>
<td>0·00 &quot;</td>
<td></td>
</tr>
<tr>
<td>&quot; 15th.—81 &quot;</td>
<td>1011</td>
<td>&quot; &quot;</td>
<td>&quot; &quot;</td>
<td>0·00 &quot;</td>
<td></td>
</tr>
<tr>
<td>&quot; 16th.—73 &quot;</td>
<td>1015</td>
<td>&quot; &quot;</td>
<td>&quot; &quot;</td>
<td>0·00 &quot;</td>
<td></td>
</tr>
<tr>
<td>&quot; 17th.—84 &quot;</td>
<td>1014</td>
<td>&quot; &quot;</td>
<td>&quot; &quot;</td>
<td>0·00 &quot;</td>
<td></td>
</tr>
</tbody>
</table>

The experiments on the urine were again repeated in February, as will be seen below:

February 18th, 1854.—Urine, 57 fluid ounces; sp. gr. 1012·5 at 60° Fahr., acid; no albumen. Uric acid eliminated in twenty-four hours = 1·22 grain. Urea eliminated in twenty-four hours = 411 grains.
19th.—Urine, 51 fluid ounces; sp. gr. 1014 at 60° Fahr. Uric acid in twenty-four hours = 0·71 grain. Urea in twenty-four hours = 392 grains.

20th.—Urine, 74 fluid ounces; sp. gr. 1010 at 60° Fahr. Uric acid in twenty-four hours = 0·67 grain. Urea in twenty-four hours = 356 grains.

21st.—Urine, 50 fluid ounces; sp. gr. 1015 at 60° Fahr. Uric acid in twenty-four hours = 3·650 grains. Urea in twenty-four hours = 384 grains.

When taking colchicum the urea and uric acid was examined on one day.

January 26th.—Urine, 38 fluid ounces; sp. gr. 1017 at 60° Fahr. Uric acid in twenty-four hours, no trace. Urea in twenty-four hours = 288 grains.

Case 12.—C. F., male, æt. 35; a patient having had several attacks of gout, and having nodules urate of soda in the ears and also around some of the joints of the fingers. No acute gouty symptoms at the time of the urinary examination, and no medicine taken; blood rich in uric acid.

Jan. 21st, 1854.—57 fl. oz., sp. gr. 1012 at 60° Fahr. 

" 22d, " 51 " 1013 "
" 23d, " 59 " 1014 "
" 24th, " 59 " 1013·5 "

None of these specimens gave any deposit of uric acid crystals, when treated with hydrochloric acid and allowed to remain for above 48 hours.

Case 13.—F. P., male, æt. 43; subject to frequent attacks of gout for eleven years; two small urate of soda deposits in ears; no active symptoms at the time; no medicine; blood contains much uric acid.

Jan. 24th, 1854.—70 fl. oz., sp. gr. 1012

" 30th, " 52 " 1011 "
" 31st, " 58 " 1011 "

These three specimens, acidulated with hydrochloric acid, gave a mere trace of uric acid.

Case 14.—F. P., æt. 45; the same patient as Case 13, but the examination of the urine made at a different date,
and after recovery from a slight attack of gout; no active symptoms of the disease present, and no medicine administered.

The quantity of urine and the amount of urea were as follow:

<table>
<thead>
<tr>
<th>Date</th>
<th>Ounces</th>
<th>Urea in 24 hours, grs.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Feb. 23rd</td>
<td>4</td>
<td>213</td>
</tr>
<tr>
<td>&quot; 24th</td>
<td>2</td>
<td>364</td>
</tr>
<tr>
<td>&quot; 25th</td>
<td>4</td>
<td>299</td>
</tr>
<tr>
<td>&quot; 26th</td>
<td>2</td>
<td>359</td>
</tr>
<tr>
<td>&quot; 27th</td>
<td>4</td>
<td>315</td>
</tr>
<tr>
<td>&quot; 28th</td>
<td>2</td>
<td>302</td>
</tr>
<tr>
<td>March 2d</td>
<td>4</td>
<td>312</td>
</tr>
<tr>
<td>&quot; 3d</td>
<td>2</td>
<td>264</td>
</tr>
<tr>
<td>&quot; 4th</td>
<td>4</td>
<td>262</td>
</tr>
<tr>
<td>&quot; 5th</td>
<td>2</td>
<td>234</td>
</tr>
<tr>
<td>&quot; 6th</td>
<td>4</td>
<td>287</td>
</tr>
</tbody>
</table>

On several days the urine was tested to ascertain the amount of uric acid, but only a few microscopic crystals could be detected, too small in quantity to collect and weigh. The blood was rich in uric acid.

Case 15.—T. C,—, male, st. 57; gout of about twelve years' duration. Many large concretions of uric acid in the ears and other parts of his body; urine pale, gave a mere trace of uric acid, but a little albumen. The blood was rich in uric acid.

Case 16.—R. W,—, a male patient, st. 53; suffered from gout for fourteen years. The first and two subsequent attacks were confined to the ball of the left great toe; other joints subsequently became affected. Has noticed deposits in his ears for about seven years. These are now very large and numerous; spots of urate of soda are also seen on the left lower eyelid. Considerable chalk-like masses exist about the hands and elbows, which are thereby much deformed and crippled.

June, 1858.—Urine passed at noon; sp. gr. 1011, distinctly albuminous; when treated with hydrochloric acid it gave no trace of uric acid.
Case 17.—T. F., a gentleman, æt. 64; has had gout for twenty years, at first confined to the ball of one or other great toe, and, in course of years, gradually travelling upwards; is now suffering from chronic pains and slight enlargement of some of the fingers, and one of these, on being punctured, gave exit to a creamy fluid, consisting of crystallized urate of soda; no deposits upon the cartilages of ears. Urine 80 fluid ounces, of a pale yellow colour; sp. gr. 1013; acidulated with hydrochloric acid, gave no trace of uric acid. On another occasion the urine gave a moderate deposit of uric acid.

From a review of the foregoing analyses it will be at once observed, that the diminution of the uric acid in the urine of these patients is most marked. In no case did it amount to more than 5·78 grains, and this only on one day; the next highest number representing the elimination of uric acid in grains is only 3·67, and such appeared to be a very exceptional instance, the excretion being usually even under one grain. The total average of all the analyses in this second class of cases is exceedingly low, far under a single grain per diem. Several urea determinations were likewise made—four in Case 10, where the quantities were found to be 306 grains, 333 grains, 344 grains, and 358 grains, giving an average of 335 grains for the daily elimination of this principle; and it will be noticed, on looking at the details of these analyses, that, although the quantity of urea remains nearly fixed and of about the normal amount, the uric acid is exceedingly deficient, and subject to great fluctuations, varying, in the four days, from 1·34 grain, to a quantity too small to collect and weigh. Again, in Case 11, four determinations of the urea give the daily average of 385 grains, with but slight differences between the mean and extremes; the quantity of uric acid during the same time exhibiting the same violent fluctuations as in Case 10, varying from 3·65 grains to 0·71 grain and from 2·4 grains to 0·0 grain in the previous month. In Case 14 twelve urea determinations were performed, on twelve
successive days (with one exception), and the average quantity found to be 286 grains, the extremes being 213 grains and 359 grains. On several of the above days the uric acid was sought for, but in no analysis was a sufficient quantity found to collect and accurately weigh.

From these observations, therefore, it is evident, that the function of the kidney for excreting urea from the blood, often remains intact in cases of chronic gout, where the power of the same organ for eliminating uric acid has become most seriously impaired.

An examination of these cases will also show the extreme prevalence of a small amount of albumen in the urine in chronic gout. In ten out of the seventeen cases it is asserted to be present, and it is possible that in some of the remaining seven it might have existed, as it is not always specially noted that it was absent; as a rule, the quantity of this principle is not large, but still sufficient to give a marked haziness when the fluid is boiled with the addition of nitric acid, and also a notable precipitate after the tube is allowed to remain at rest for a short time.

I may remark here that I have observed this phenomenon, in very many cases of the disease; in the present communication notice is only taken of its occurrence where quantitative examinations of the urine have been made, and these have been performed only in a small per-cent age of the patients who have come under my care.

To sum up, the more common appearances and characters of the urine in chronic gout, as shown by these and other examinations, are as follow: It is usually rather light in colour, decidedly paler than the average tint of the healthy secretion, of lower density, and often increased in quantity. The amount of urea, except in extreme cases, about the same as in health (due account being taken of the diet of the patient at the time); the uric acid very much diminished, and subject to excretion in very varied quantities at different periods; and lastly, the presence of a small amount of albumen is exceedingly frequent. Deposits are not
of common occurrence in the urine of these subjects, but they occasionally occur on the cooling of the fluid, sometimes in the form of urate of soda or ammonia, at other times as the crystallized, and more or less coloured, varieties of uric acid.

Class III.—Urine of individuals who had suffered more or less frequently from attacks of Gout, of varying degrees of intensity, examined at the time of complete freedom from any symptoms of the disease.

Case 1.—The first three analyses were made on the urine of a gentleman, æt. 40, rather tall and stout, subject to occasional slight attacks of gout in the great toe, but having had no other joints affected; general health very good.

January 16th, 1852.—Urine, 41½ fluid ounces; sp. gr. 1024 at 60° Fahr.; clear, amber coloured, acid, contains no albumen, and gives rise to no deposit on cooling. Uric acid secreted in the twenty-four hours = 4·72 grains.

17th.—Urine, 43 fluid ounces; sp. gr. 1022 at 60° Fahr., and on cooling, a light pinkish-coloured deposit of urates is seen thrown down. Uric acid in twenty-four hours = 6·50 grains.

21st.—Urine, 47 fluid ounces; sp. gr. 1023, clear, amber coloured; no deposit. Uric acid in twenty-four hours = 3·85 grains.

Case 2.—A gentleman, æt. 56, having suffered from gout many years, but with no external deposits; has often latterly had the large joints affected as well as the great toe. No symptoms present when the examination of urine was made.

February 12th.—Urine, 60 fluid ounces; sp. gr. 1014 at 60° Fahr., acid; no albumen. Uric acid thrown out in the twenty-four hours = 4·26 grains.

In this case a small blister, applied to the ball of the
great toe, which had become stiffened, yielded a fluid containing a considerable quantity of uric acid.

Case 3.—A lady, wet. 38, of a gouty family, has suffered from an atonic form of gout for many years, but without any deformity of joints or external deposits of urate of soda. When the urine was examined no active symptoms were present, and no febrile disturbance. Urine, 42 fluid ounces; of a pale colour, no albumen; sp. gr. 1019 at 60° Fahr. Uric acid, no trace. Urea = 373 grains.

Case 4.—J. Z. R.—, a male, wet. 48; this patient has suffered from about three fits of gout, the great toe being in each attack especially affected; no deposit of urate visible in ears or any part of body. During last attack, the blood had been examined, and found rich in uric acid. At the time the examinations of the urine were made, the patient was convalescent, and free from all joint affection.

February 21st, 1854.—Urine, 58 fluid ounces; sp.gr.1015 at 60° Fahr. Uric acid in twenty-four hours' urine = 2.93 grains.

23d.—Urine, 88 fluid ounces; sp. gr. 1010 at 60° Fahr. Uric acid thrown out during twenty-four hours = 1.23 grain. Urea in twenty-four hours = 385 grains.

24th.—Urine, 60 fluid ounces; sp. gr. 1014 at 60° Fahr. Uric acid in twenty-four hours = 1.83 grain. Urea in twenty-four hours = 335 grains.

25th.—Urine, 77 fluid ounces; sp. gr. 1011 at 60° Fahr. Uric acid in twenty-four hours = 1.69 grain. Urea in twenty-four hours = 370 grains.

27th.—Urine 64 fluid ounces; sp. gr. 1013. Uric acid in twenty-four hours = 0.19 grain. Urea in twenty-four hours = 338 grains.

During the time this patient's urine was being examined quantitatively it was clear and free from deposits, but at the time of the attack, which was very severe in character, the urine became turbid on cooling, from the deposition
of pink and red urates. No albumen was present at any time.

**Case 5.**—T. B—, male, æt. 54, having no visible deposits of urate of soda in any part of body, but who has had numerous attacks of gout, with special great-toe affection; at the time of examination of the urine was free from all symptoms; had omitted medicines for some time, but had a return of symptoms a few days after the analyses were made; he was taking a moderate quantity of meat; the serum of blood was found, during the attack, to be rich in uric acid.

May 11th, 1854.—Urine, 60 fluid ounces; sp. gr. 1019, acid, clear. Uric acid in twenty-four hours, too small to collect and weigh. Urea in twenty-four hours = 372 grains.

12th.—Urine, 54 fluid ounces; sp. gr. 1017, acid. Uric acid in twenty-four hours, too small to collect and weigh. Urea in twenty-four hours = 441 grains.

13th.—Urine, 74 fluid ounces; sp. gr. 1015. Uric acid in twenty-four hours, too small to collect and weigh. Urea in twenty-four hours = 569 grains.

**Case 6.**—S. N—, male, æt. 68; has suffered for many years from attacks of gout, chiefly in great toe; other joints have also been occasionally affected; at the time of examination of urine was free from all symptoms; during attack the blood had been examined, and found to contain much uric acid.

February 21st.—Urine, 55 fluid ounces; sp. gr. 1013. Uric acid eliminated in twenty-four hours = 1·93 grain. Urea eliminated in twenty-four hours = 317 grains.

22d.—Urine, 50 fluid ounces; sp. gr. 1014. Uric acid eliminated in twenty-four hours = 2·28 grains. Urea eliminated in twenty-four hours = 312 grains.

23d.—Urine, 44 fluid ounces; sp. gr. 1014. Uric acid eliminated in twenty-four hours = 1·60 grain. Urea eliminated in twenty-four hours = 275 grains.

24th.—Urine, 29 fluid ounces; sp. gr. 1016. Uric acid
eliminated in twenty-four hours = 1.48 grain. Urea eliminated in twenty-four hours = 234 grains.

25th.—Urine, 40 fluid ounces; sp. gr. 1014. Uric acid eliminated in twenty-four hours = 2.72 grains. Urea eliminated in twenty-four hours = 269 grains.

This patient had taken small doses of the acetic extract of colchicum, in the form of pill, for several days previous to the 21st, and was ordered to omit it on the 22d, on account of slight diarrhoea coming on, which continued, more or less, to the 25th. From his own statement, there was no loss of urine during this period.

It will be seen, from these analyses, that in no one of the six patients' urine did the amount of uric acid excreted in the twenty-four hours reach the quantity usually considered to be the average in health; in most it was very far below; and, from these and many other partial examinations, I am inclined to think that in individuals who have suffered frequently from gout the kidneys lose, at least to some extent, their power of readily excreting uric acid; and moreover, I am convinced, from the result of several trials, that the blood is often kept in an impure state by the presence of this principle. This is probably one cause of the extreme liability of such patients to periodic visitations of the painful malady, and also of the great difficulty of effecting a radical cure of the disease.

It is interesting, again, to observe that in these cases, as in those patients suffering from acute and chronic gout, the urea-eliminating function of the kidneys appears to remain almost if not completely intact.

**PART II.—ON THE ACTION OF COLCHICUM UPON THE URINE.**

Upon this subject I believe there exist in the medical profession, at the present day, opinions which are extremely erroneous and without the slightest foundation; opinions
which have arisen from the wrong interpretation of phenomena occurring in disease, from mistaking, in fact, the natural effects of the progress of a malady for the results of the administration of remedies.

That colchicum, administered in its various forms, has a most powerful influence upon the progress of gouty inflammation is, I am sure, undeniable; whether for good or evil in its ultimate effects may perhaps, be still a subject for inquiry. The fact, however, of its powerful influence being admitted, it becomes an interesting and important question to ascertain in what manner the effects are produced; and one method will be to find out, if possible, its action upon the renal secretion, that is, upon the quantity and composition of the urine.

Before, however, giving the results I have arrived at from my examinations, it may be as well to notice, in a few words, the principal views which have been held by different observers upon the action of this medicine. Certain of the effects usually attributed to this drug are easily demonstrated. When given in large quantities or in moderate doses frequently repeated, it purges, and not uncommonly produces nausea and vomiting; its influence upon the liver is also usually very readily recognised in the appearance of the feces and of the vomited matters. It is also generally regarded as possessing a diuretic action, but this latter property, namely, to increase the renal secretion, will be found to be by no means constant or well marked.

Besides being supposed to act as a diuretic, colchicum has been thought to influence the composition of the urine. Dr. Christison found that after giving colchicum to a patient for two days, the quantity of urea was nearly doubled; that before taking the drug the urine had a specific gravity of 1020, and 2 per cent. of urea was contained in the fluid; it was also free from any urate deposit; but that during the first and second days after commencing the medicine the urines were turbid from urate of ammonia, of the densities 1033·5 and 1034, and the second specimen, on analysis, gave at least 3·5 per cent. of urea.
It appears, however, that Dr. Christison took specimens of urine for examination without regard to the quantity passed in the twenty-four hours, so that although, after the administration of the colchicum, a given sample might have been richer in uric acid and urea, still no proof was afforded that the total elimination of these principles was augmented; in fact, so far as these results indicate, they might have been decreased, as a notable diminution of the urinary secretion often occurs from the action of colchicum upon the alimentary canal. Dr. J. M'Grigor Maclagan seems to have made experiments in the same manner as Dr. Christison, and to have arrived at similar results, the analyses having been made only on specimens of urine passed at particular times, and no attempt made to show the real daily averages of these principles. Dr. Graves, on the other hand, regards the action of colchicum as tending to prevent the formation of uric acid, not as causing an increased elimination.

Professor Chelius, of Heidelberg, also made some observations, the results of which have been most extensively copied in different works; he considered that colchicum possessed the power of causing the kidneys to throw out uric acid in greatly increased quantities, so that in the course of a few days it was nearly doubled; but his observations were made upon a gouty subject and one recovering from an attack of the disease, and this, as we have already seen, must vitiate the whole of his experiments.

The following are some of the observations I have made on the action of the drug upon the urinary secretion:

Case 1.—The first patient, W. L., a man about 30 years of age, suffering from a slight chronic eczematous eruption, but whose general health was good, was put upon a regulated diet, and his urine carefully collected and examined.
RESEARCHES ON GOUT.

Before taking colchicum—

Oct. 30th.—urine 56 fl. ounces, sp. gr. 1016; uric acid 8·73 grs.

<table>
<thead>
<tr>
<th>Date</th>
<th>Sp. Gr.</th>
<th>Uric Acid</th>
<th>&quot;</th>
</tr>
</thead>
<tbody>
<tr>
<td>31st</td>
<td>64</td>
<td>1014</td>
<td>7·93</td>
</tr>
<tr>
<td>Nov. 1st</td>
<td>88</td>
<td>1012</td>
<td>8·09</td>
</tr>
<tr>
<td>3d.</td>
<td>65</td>
<td>1015</td>
<td>8·18</td>
</tr>
</tbody>
</table>

Mean...... 68·5 " 8·24 "

When taking vinum colchicum—

Nov. 4th.—urine 56 fl. ounces, sp. gr. 1015; uric acid 6·05 grs.

<table>
<thead>
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<th>&quot;</th>
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<tr>
<td>5th</td>
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<tr>
<td>6th</td>
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<tr>
<td>9th</td>
<td>54</td>
<td>1021</td>
<td>9·18</td>
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</table>

Mean. . . . . . 55·6 " 7·67 "

Case 2.—The next case, C. W,—a young man, aged 19, suffering from some chronic affection of the upper lip, but whose general health was pretty good. During the time the analyses were performed the diet remained fixed, and every precaution was taken to ensure accuracy of results.

Without medicine—

1854.

Jan. 13th.—Urine 55 fl. oz., sp. gr. 1017; uric acid in 24 hours 7·61 grs.

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<td>15th</td>
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Under colchicum—

Jan. 16th.—Urine 47 fl. oz., sp. gr. 1018; uric acid in 24 hours 1·34 grs.

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</tr>
<tr>
<td>22d</td>
<td>19</td>
<td>1027</td>
<td>5·34</td>
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The colchicum was continued three times a day for two days, when purging was produced; it was then repeated night and morning only. The action of the bowels continued to be kept up by the medicine, but the urine said to be carefully preserved.
The average results before and when taking the medicine are as follow:

*Without medicine.*—Quantity of urine, 37.7 fluid ounces. Uric acid in twenty-four hours = 5.03 grains.

*When taking colchicum.*—Quantity of urine, 25.2 fluid ounces. Uric acid in twenty-four hours = 5.29 grains.

**Case 3.**—B. F—, the same man as in Case 3 of Acute Gout, during an attack of acute gout, and without medicine, passed, during the five previous days, 1.30 grain, 1.95 grain, 2.73 grains, 2.14 grains, and 3.05 grains. The wine of colchicum was then administered in half-drachm doses, with calcined magnesia, three times a day, and an almost immediate and marked effect upon the joint affection was produced.

January 20th.—Urine, 43 fluid ounces; sp. gr. 1011, acid. Uric acid eliminated in twenty-four hours = 2.58 grains.

21st.—Diarrhoea from medicine caused loss of much urine.

22d.—Urine, 35 fluid ounces; sp. gr. 1012, acid. Uric acid in twenty-four hours = 3.32 grains.

23d.—Urine, 47 fluid ounces; sp. gr. 1011, acid. Uric acid in twenty-four hours = 3.99 grains.

On 21st took but two doses of medicine, and on the 22d but one, then discontinued it altogether.

26th.—Urine, 74 fluid ounces; sp. gr. 1011, acid. Uric acid in twenty-four hours = 4.07 grains.

27th.—Urine, 56 fluid ounces; sp. gr. 1011, acid. Uric acid in twenty-four hours = 3.08 grains.

Average of uric acid in twenty-four hours before taking colchicum = 2.23 grains.

Average of uric acid in twenty-fours when taking colchicum = 3.41 grains.

**Case 4.**—C. F—, a male patient, on half-extra diet, and taking Vinum Colchici in half-drachm doses, night and morning; patient not suffering from any very active symp-
toms, but has a considerable abscess, discharging urate of soda and some pus.

**Under colchicum—**

Jan. 4th.—Urine 57 fl. oz., sp. gr. 1012; uric acid in 24 hours 0·56 gr.
   " 5th. " 44 " 1013 " 0·06 "
   " 6th. " 44 " 1013 " 0·00 "
   " 7th. " 37 " 1012 " 0·14 "
   " 8th. " 46 " 1011 " 0·00 "
   " 9th. " 44 " 1011 " 0·00 "

The diet remaining the same, no medicine taken—

Jan. 10th.—Urine 44 fl. oz., sp. gr. 1012; uric acid in 24 hours 0·72 gr.
   " 11th. " 34 " 1014 " 0·85 "
   " 12th. " 61 " 1013 " 2·44 "
   " 13th. " 55 " 1012 " 0·00 "
   " 14th. " 87 " 1009 " 0·00 "

**Under colchicum.—**Average, 45·6 fluid ounces. Uric acid in twenty-four hours = 0·13 grain.

**Without medicine.—**Average, 56·2 fluid ounces. Uric acid in twenty-four hours = 0·80 grain.

**Case 5.**—J. L,—a male, set. 57; has had several attacks of gout, usually a year or more elapsing between each. For the first few years the balls of the great toes only were affected, the disease afterwards extending upwards. No visible deposits of urate of soda on any part of the body.

During the time the joints were severely affected the urine was scanty, high coloured, giving rise to a red sediment of urates. When the quantitative analyses were commenced, the patient was suffering only from slight affection of the joints, and there was an absence of febrile disturbance; no medicine had been taken, and the disease appeared to be still hovering about. Blood contained abundance of uric acid.

**No medicine—**

February 7th, 1856.—Urine, 26·5 fluid ounces; sp. gr. 1021 at 60° Fahr., clear. Urea in twenty-four hours = 213 grains.

**xli.**
8th.—Urine, 28 fluid ounces; sp. gr. 1020·5 at 60° Fahr. Urea in twenty-four hours = 199 grains. Uric acid crystals, too small in quantity to collect.

9th.—Urine, 37 fluid ounces; sp. gr. 1019 at 60° Fahr. Urea in twenty-four hours = 259 grains. Uric acid, only a few microscopic crystals.

**Under colchicum** (half a fluid drachm of Vinum Colchici three times a day)—

10th.—Urine, 38 fluid ounces; sp. gr. 1019 at 60° Fahr. Urea in twenty-four hours = 209 grains. Uric acid, not able to be collected, from the minuteness of its quantity.

11th.—Urine, 32 fluid ounces; sp. gr. 1021 at 60° Fahr. Urea in twenty-four hours = 244 grains. Uric acid in microscopic quantities only.

12th.—Urine, 41 fluid ounces; sp. gr. 1019 at 60° Fahr. Urea in twenty-four hours = 270 grains. Uric acid, a trace only.

13th.—Urine, 39 fluid ounces; sp. gr. 1017·5 at 60° Fahr. Urea in twenty-four hours = 241 grains. Uric acid, a very few crystals—too few to collect.

14th.—Urine, 33 fluid ounces; sp. gr. 1021·5 at 60° Fahr. Urea in twenty-four hours = 259 grains.

Omitted colchicum, as faintness was produced.

**No medicine taken**—

16th.—Urine, 30 fluid ounces; sp. gr. 1020 at 60° Fahr. Urea in twenty-four hours = 201 grains.

19th.—Urine, 30 fluid ounces; sp. gr. 1020 at 60° Fahr. Urea in twenty-four hours = 201 grains.

20th.—Urine, 42 fluid ounces; sp. gr. 1017·5 at 60° Fahr. Urea in twenty-four hours = 251 grains.

21st.—Urine, 51 fluid ounces; sp. gr. 1014·5 at 60° Fahr. Urea in twenty-four hours = 279 grains. No crystals of uric acid precipitated by the addition of hydrochloric acid.

22d.—Urine, 54 fluid ounces; sp. gr. 1013·5 at 60° Fahr. Urea in twenty-four hours = 250 grains.

**Average before medicine.**—Quantity of urine in twenty-
RESEARCHES ON GOUT.

four hours, 30·5 fluid ounces. Amount of urea, 223·6 grains.

*Average during administration of colchicum.*—Quantity of urine, 36·6 fluid ounces in the twenty-four hours. Amount of urea, 244·6 grains.

*Average after omitting colchicum.*—Quantity of urine in twenty-four hours, 41·4 fluid ounces. Amount of urea, 236·4 grains.

The quantity of uric acid, both before, during, and after the administration of the drug, was too small to collect and weigh.

**CASE 6.—H. C., a male, 25. 51;** has suffered from many attacks of gout, but at the time of the examination of the urine was not labouring under any acute affection. Four days before colchicum was administered the urine gave the following results:

Nov. 25th.—Quantity 98 fl. oz., sp. gr. 1008; uric acid in 24 hours 1·95 grs.

``
26th. " 72 " 1012 " 3·12 "
27th. " 68 " 1014 " 2·72 "
28th. " 60 " 1014 " 3·00 "
``

*Under colchicum (mxx ter in die)*—

Nov. 29th.—Quantity 68 fl. oz., sp. gr. 1011; uric acid in 24 hours 1·81 grs.

``
Dec. 1st. " 64 " 1012 " 2·29 "
2d. " 72 " 1008 " 1·36 "
``

*Average before medicine.*—Quantity, 74·5 fluid ounces. Uric acid, 2·70 grains.

*Average when taking colchicum.*—Quantity, 67·5 fluid ounces. Uric acid, 1·88 grain.

The results of these observations on the action of colchicum may be thus summed up:

In Case 1, where no gouty affection existed, and no febrile disturbance was present, colchicum appeared to have the effect of slightly diminishing the quantity of urine, and also of decreasing somewhat the excretion of uric acid.

In Case 2, where the circumstances were very
similar to those in Case 1, the influence of the medicine was notably to diminish the quantity of urine, the uric acid being very slightly augmented, the increase, however, was less than a quarter of a grain per diem.

In Case 3, a gouty patient recovering from an acute attack of the disease, the uric acid was somewhat increased, but certainly not more than often occurs in individuals under the same circumstances, when no medicine has been taken.

In Case 4 both the uric acid and the quantity of urine were much less when colchicum was administered.

In Case 5, where the examinations were made both before and after the drug was given, and also during the time of its administration, the uric acid remained about the same, but in such extremely small quantities as not to be capable of being weighed; the urea was also somewhat augmented. The increase, however, formed but a small fraction of the total elimination of that principle.

The amount of urine became greater when the patient was placed under the influence of the drug, but it continued to increase still further after its omission.

In Case 6, a gouty patient, the uric acid excretion became much less in amount, and the quantity of urine decidedly smaller. In this patient, however, considerable variations were observed in these respects, irrespective of any remedies, and therefore but little stress can be laid upon the figures obtained.

Besides the results detailed under this head, I have alluded, in other parts of the present communication, to analyses of urine which must have shown somewhat the influence of colchicum upon this secretion. Thus, among the cases of acute gout, we find that the patient in Case 5, on February 19th and February 20th, was under the marked influence of the remedy, and the peculiarity of the urine consisted in its then containing a very small quantity of uric acid, compared with the amount present during the time the inflammation was severe. In Case 6 the col-
chicum was continued during the whole course of the attack, but the dose was rather small, and the patient had been long accustomed to its administration; probably, therefore, the daily alterations in the uric acid seen in his urine depended rather on the different phases of the disease than the action of the medicine. Among the cases of chronic gout, it will be seen that patient No. 11 was put under the influence of colchicum, with the apparent effect of diminishing the excretion both of the uric acid and urea.

From a review of the results of these observations, we must, I think, draw the following conclusions:

First. That there is no evidence that colchicum produces any of its effects upon the system by causing the kidneys to eliminate more uric acid; in fact, I believe that when continued for any length of time, the contrary rather holds good.

Secondly. That from the observations in the present communication, we cannot assert that colchicum has any influence upon the excretion of the urea or other solids of the urine.

Thirdly. That colchicum is by no means a diuretic in all cases, but, on the contrary, it often diminishes the quantity of urine, especially when it produces a marked effect upon the secretions from the alimentary canal.

As yet I have made no experiments sufficiently decisive to enable me to say whether or not colchicum produces a diminished formation of uric acid in the system.

Dr. Christison’s observations would have agreed with many of my own, had I taken only a small portion of urine for examination. Thus, Case 2 might have been made to show that the uric acid was more than doubled by the remedy, although there was no increase in the daily amount excreted. I am inclined, therefore, to believe that where the uric acid has been stated to have been increased by the administration of colchicum, the circumstance has arisen from the disease having become altered in phase during the examination, as occurred probably in Chelius’s case, or else that erroneous deductions have been made, from analyses of portions only of the daily urine having been performed.
APPENDIX.

The subjoined analyses, seventy-one in number, were made on the urine of a male patient, fifty-one years of age, who was admitted into the hospital, suffering from chronic bronchitis, accompanied with much difficulty of breathing. These symptoms were suddenly relieved, on the occurrence of a fit of gout in the ball of the great toe, which afterwards extended to the side of the foot and the knee. The patient became relieved by treatment, and left the hospital, but in the course of a short time was readmitted, having bronchitis and flying pains in different joints, with some tenderness of the affected parts. The examinations of the urine were commenced on his readmission. The points attended to were the quantity of urine passed each day, its specific gravity, and also the amount of uric acid eliminated in the twenty-four hours. The results will be seen to illustrate, in a forcible manner, the very irregular and somewhat intermittent way in which the kidneys, in gout, secrete the uric acid from the blood; they will also show any influence which colchicum possesses of altering the amount of the acid, and are in entire accordance with those previously arrived at in the present communication.

<table>
<thead>
<tr>
<th>Date</th>
<th>Quantity</th>
<th>Sp. gr.</th>
<th>Uric Acid passed in 24 hours</th>
<th>Circumstances and state of Patient</th>
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### RESEARCHES ON GOUT.

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Uric Acid passed in the twenty-four hours.

Maximum of uric acid . 8·60 grs.
Minimum of uric acid . 0·18 "
Average . 1·96 "
Average without colchicum . 2·26 "
Average under colchicum . 1·65 "

Quantity and weight of the Urine.

Average quantity of urine passed without colchicum, 51 fl. oz.
Average specific gravity, 1012·5.
Average quantity of urine passed when colchicum was administered, 51 fl. oz.
Average specific gravity, 1011·4.
CONTRIBUTIONS
TO THE
PATHOLOGY
OF THE
GLANDULAR STRUCTURES OF THE
STOMACH.

BY
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Communicated by Dr. Garrod.

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The great frequency of derangements in the functions of the stomach, apparently unassociated with those corresponding changes in organic form which microscopic investigation has aided so largely in revealing to the pathologist, has long been a stumbling block to those whose attention has been occupied with the disorders of the digestive process; and it has become an urgent desideratum, for the progress of our knowledge of these complaints, that the symptoms which they exhibit should be connected as far as possible with anatomical conditions, an acquaintance with which—serving as they do, in the state of health, as a foundation for the laws of physiology—must also, in disease, be recognised as the basis and stepping-stone to the pathology of the changes induced by morbid action.
The medical profession is greatly indebted to Dr. Hanfield Jones, for having been the first to offer an elucidation of this problem; since, with the exception of a paper by Dr. F. Schäfer, published in vol. vii of Virchow’s ‘Archiv’, there is little to be found in the numerous treatises on disorders of digestion relative to the actual anatomical state present.

Dr. Habershon, in his recent admirable work on the alimentary canal, appears to have partially adopted Dr. H. Jones’s views, and I am indebted to him for a hint as to the mode of making preparations of the mucous membrane for the microscope, which (with a slight modification) I have found of great service, and have invariably adopted.

While at Berlin during the past winter, I resolved to avail myself of the unusually great opportunities for a research of this nature afforded in the Pathological Institute attached to the Charité Hospital, under the direction of Professor Virchow, to whom I shall ever feel myself gratefully indebted, not only for the liberality with which he placed the requisite materials at my disposal, but also for the invaluable advice and assistance with which he has aided me in the prosecution of this inquiry. The statements which I have here to bring forward are based upon the notes of observations made upon 100 stomachs, taken indiscriminately from the bodies of patients brought for post-mortem examination. I have examined many more, but rejected all those made at an earlier period, before I had fully satisfied myself of the exact anatomical structure, and, (what is of equal importance in structures which suffer so rapidly from cadaveric change), of the nature of the alterations induced by decomposition. To these I shall have occasion hereafter to refer, and I will here only premise, that it is only in rare instances that conclusions can be drawn with certainty from the condition of the fundus of the stomach after death, which, in a very large proportion of cases, is more or less acted on by the contents of the organ, and in which decomposition sets in at a very early period.

It has been a cause of regret to me, that the nature of my engagements prevented me from instituting any accurate
comparison between the post-mortem appearances here recorded and the symptoms of the patients during life; this was, however, impossible, and I can only hope that I may be found to have contributed, in some degree, in aiding to lay a foundation for further clinical and pathological inquiry. The mode of making preparations for the microscope which I have followed, has consisted in stretching the stomach over a large cork by means of pins, and then making sections in any part which I wished to examine by means of a Valentin's knife. These can then be removed by means of a fine forceps and scissors, and spread out under water (pressure with the covering-glass being carefully avoided). I have found this plan, proposed by Dr. Habershon, preferable to that adopted by Dr. H. Jones from the fact that it gives a thin and uniform section, and is also much less liable to crush and injure the natural arrangements of the parts.

The morbid condition to which I would first direct attention, as affecting the glandular structures of the stomach, is that known under the name of recent or acute catarrh. The alterations which the glands undergo in this state have been but little noticed by writers on this disease, who have principally studied the nature of the altered secretion and the appearances which the membrane presents to the naked eye.

The characters of the mucous secretion found post mortem on the surface on the stomach are very variable, and depend, I believe, in many cases—(1) on the physiological rather than on the pathological state preceding death; and (2) on chemical changes taking place in the contents, either as the result of spontaneous decomposition, or from this cause combined with the action on it of other decomposing substances which may be accidentally present.

During the act of digestion the whole surface of the stomach is covered with a tenacious "mucus," containing both spheroidal and columnar epithelium, free nuclei, and molecular matter, held together by a tough translucent substance, which becomes opaque on the addition of most acids, the histological elements forming, however, a large proportion of the whole. It is very frequently found in large quantities
where the stomach is almost empty, and has in these cases an alkaline, neutral, or faintly acid reaction to test paper, and is often unaccompanied by any morbid change in the finer structures. In other cases, where decomposing food is present, we have also a mucus which has still a high degree of tenacity, but much more fluid, and sharing with the other substances present in a highly acid reaction, while the histological elements are much less distinct. An alkaline or neutral mucus has been found by Donders in a considerable number of stomachs of animals killed while fasting.

I am inclined to the opinion that no important conclusion can be drawn, as to the pathological condition of the membrane, from the simple presence of this mucus. In some cases, where the microscopic appearances have convinced me that catarrh was present, this mucous layer was of a pale colour, though I believe such cases to be exceptional; and where a recent catarrh of any intensity affects the membrane the secretion is generally found post mortem tinged of a reddish hue, owing to the escape of hematin from the over-loaded vessels. The mucus, even when pale, has, however, in these instances a peculiar "glassy" translucent character. I am not prepared to say that I consider this as diagnostic of the condition in question, but it certainly has in many cases a look differing from the ordinary layer found on the surface of healthy stomachs.

The naked-eye appearances of recent catarrh have been so fully described by numerous observers, that I need not dwell upon them here. Injection, as shown by redness of the surface and fulness of the veins in the submucous tissue, indicating a state of hyperæmia, thickening of the membrane, and unusual prominence of the orifices of the glands—all contributing to produce a swollen appearance—are its most marked characteristics; but the latter alone is by no means distinctive, nor is the condition first observed and described by Louis (the état mamelonné) specially limited to this affection, but is met with in a great number of cases where no changes can be found in the glands. Dr. H. Jones be-
lieves that it is occasionally caused by a series of atrophied spots, produced by a condition which he describes as nuclear degeneration, occurring in limited patches of tubes, and giving an appearance of undue prominence to other parts, and he draws an analogy between this state and the granular kidney, where the irregularity of surface arises, not from hypertrophy of some convolutions, but from atrophy of others. I have made sections in various directions through stomachs found in this state, and have rarely seen either marked swellings, and increase of the size or amount of contents of some tubes, or atrophy of others; but in some cases (though I have only observed this in the slighter forms of the appearance in question) it does at times appear to be associated with some increase in the connective tissue intervening between the glandular structures. This latter state I shall hereafter discuss more fully; but I may mention here, that in some of the cases where it has been most fully developed, it has been unattended with the mamillated condition. With regard to the microscopic appearances in acute catarrh, they are of a well-marked kind, and the results of my observations have corresponded very closely with the description given of them by Dr. F. Schäffer, in the seventh volume of Virchow's 'Archiv.'

Viewed with a low power, the hyperemic condition of the finer vessels is well seen; the smallest capillaries being often found filled with blood, which in a vertical section occupy nearly the whole of the interspaces between the tubes, or regarded from the surface show the openings of the canals surrounded by a zone of vessels. The glands are enlarged, and have an unusually dark and granular look by transmitted, and a whiter appearance than normal by reflected, light, both of which are removed by the addition of caustic alkalies. Irregular swellings are also noticed in various parts of their course. When more highly magnified, they are found filled with granular matter and epithelium; the cells of the latter being much enlarged, and much more granular in appearance than normal. The nucleus also often undergoes a considerable increase in size, and is in some cases
darkly granular. The glands lie closely packed together, and often appear to be only separated by the swollen capillaries which fill the inter-tubular spaces. The irregularities and swellings in their course are caused by groups of enlarged cells, or by accumulations of smaller ones mingled with granular matter at the dilated parts.

Dr. Schäper remarks, that he has not been able in these cases to find entire cells, but only molecular débris in the tube. In some of the cases which I have observed, and of the appearances in which I have made drawings, (Pl. I, fig. 1), the cells were remarkably distinct; in other instances they were filled chiefly with the molecular matter described by Dr. Schäper. Of two cases dying in the typhoid stage of cholera—a mother and a child—in the former the epithelium had in a great measure broken down, and the membrana limitans of the glands had disappeared in many parts, while in the latter the cells were preserved, and on being pressed out from the tubes many assumed an elongated form. The cells in some of these cases were increased from one third more to twice their normal size, and their appearance when pressed out from the glands was very peculiar and characteristic.

Many of the cells which were still entire contained numerous fat-granules, and where they were broken down fat-drops in considerable numbers mingled themselves with the granular débris. The cells, when present, were acted on by reagents with much greater facility than usual, but I have not found this peculiarity so marked in the other specimens of the disease which have come under my notice.

Glands in this condition have, as I have before observed, an unusually white appearance by reflected light, and present a remarkable similarity to the microscopic appearances observed in the kidney in the first stage of Bright's disease. This character is always present in some degree in the glands of the stomach, and disappears on the addition of caustic alkalies, but in the condition which I am at present describing it occurs to an unusual extent, and is very characteristic of the affection.
The varying conditions in which the cells are found are probably to be accounted for by the different stages of the disease at which they come under observation, and this may, I think, suffice to explain the difference between Dr. Schäffer's description and my own.

The rapidity of the post-mortem changes may also be concerned in these differences. The cases of typhoid cholera to which I have alluded were probably in a more advanced stage than the others recorded, an opinion supported by the great amount of fat accumulated in the tubes, which would indicate a further degenerative change in the secreting structure.

Slighter degrees of this affection are not very unfrequently met with; the appearances to the naked eye being such, to a greater or less degree, as I have described, and a microscopical examination revealing similar alterations, but in a less marked form. The glands look more dark and granular by transmitted, more white by reflected, light, than is consistent with their normal appearances; but the epithelial cells, though granular to a degree greater than is usually observed, even in the physiological states where this is most marked, exhibit but little increase in size. Many contain double nuclei. This is sometimes, though not very frequently, observed in the human stomach, when it appears otherwise healthy, and appears to have been occasionally met with in the stomachs of animals; but I have certainly seen it most frequently associated with some of the other characters of the catarrhal condition, and it may, I think, be regarded as a sign of increased nutritive activity excited by the inflammatory stimulus.

With regard to the classes of glands affected, I have not been able to detect any marked difference in the changes of those containing a cylindrical and those whose epithelium is of a spheroidal character. Both appear to be equally implicated, though the pyloric portion of the stomach certainly suffers with greater frequency than the other regions. The cylinder epithelium appears to break down with greater facility than the spheroidal, and it is especially
in the pyloric portion that we meet with glands filled with granular débris and fat-drops. I have hitherto met with only one case corresponding to the acute gastritis of authors. It was associated with scirrhous cancer of the muscular coat, which had in some places extended through the mucous membrane. The whole surface of the stomach was intensely injected, and stained with blood. Sections were peculiarly opaque; and the glands, when persistent, were filled almost entirely with a granular débris, a series of appearances which have some similarity to those described by Dr. H. Jones in the stomachs of animals poisoned by arsenic.

With regard to the terminations of recent catarrh, the possibility of a "restitutio ad integrum" must, of course, be admitted; but a series of conditions is not unfrequently met with which I am inclined to classify under the head of "chronic catarrh," as alterations in structure which, in the present state of pathological knowledge, must, I think, be still considered as of inflammatory origin, though many modern pathologists, especially in England, are inclined to remove them from this category, and to regard them simply as the effects of degenerative changes resulting from impaired nutrition. It would be inconsistent with the object of this communication to discuss this question in its full extent, and to enumerate the reasons which still induce me to regard the alterations in question as the results of inflammation. I will, therefore, limit myself to the statement of opinion, that not only in the stomach, but in other organs, most of these changes appear to be consequences or secondary effects of the same exciting causes which in the earlier stages produce appearances to which the term "inflammatory" can be indubitably applied.

The characters observable by the naked eye are, thickening of the membrane, often irregularly, giving rise to a considerable unevenness of surface (but not in all cases identical with the mamillated condition), which appears due at times to prominence of the gastric follicles, at others to increase of the intervening connective tissue. The état mamelonné is described by numerous authors as occurring
in its most marked form in this affection; but I have not seen it constantly present nor occurring in a greater degree than in other stomachs apparently perfectly healthy. Congestion and fine injection of the mucous and submucous tissues may or may not be present. They are not unfrequent, but cases are occasionally met with in which there is no appearance of hyperæmia. A tenacious and, with rare exceptions, an alkaline mucus, is usually found adhering to the surface. There are alterations in colour at various parts, consisting generally (and this is, I believe, the best test of a long-continued subacute, or of previously existing acute inflammatory action) of a slaty-gray discoloration of the surface, extending in patches of variable extent from one to three or four inches in diameter. The surface of the membrane has often, in many parts, a somewhat translucent appearance, and in addition, either in the prominences above alluded to, or in portions not elevated above the level of the rest of the membrane, are seen opaque, dull white spots, varying in size from that of a pin's head to a millet seed, or even attaining a greater diameter. These changes, like those before described, are found with the greatest frequency in the neighbourhood of the pylorus, and assume there the most developed form; but they occur with considerable frequency in the cardiac portion, especially around and to the right of the oesophageal opening, where also I have found the microscopic appearances, which I shall proceed to describe, very well marked. These deviations from the normal structure appear to me to be principally of six kinds:

1. Increase in amount of the connective tissue between the glands and of the submucous tissue, with occasional fatty degeneration of the latter.
2. Thickening of the membrana limitans of the glands.
3. Fatty degeneration, or atrophy and loss of the glandular epithelium.
4. Pigmentary deposit in the tissues.
5. Cystic degenerations in the glands.
6. Occasional fatty degeneration of the vessels.
(1.) Increase in the elements of the connective tissue.—
Dr. Schäffer attributes to this cause the irregularities
which are seen on the surface of the mucous membrane, and
this has appeared to me to be occasionally the case, though
it seems to be more frequently due to a condition which he
has also described, viz., the prominences caused by distension
of the glands themselves with the product of secretion.

With regard to the increase of the connective tissue, I
may be allowed to say a few words on the fallacies to which
the observer is liable in judging of its amount; and they are
of several kinds. In the first place, as pointed out by Dr.
Habershon, the direction of the section influences greatly
the appearance presented by the object under the microscope,
for if not carried in a direction perfectly vertical with the
line of glands, we obtain an object in which an excessive
amount of fibrous tissue appears to be present. There is
also in different regions of the stomach a considerable
variation in the closeness of approximation of the glands.
In the immediate neighbourhood of the cardia, and also
sometimes in the fundus, they are much less closely packed
than in the more central parts; and near the pylorus they
are aggregated into groups, separated by wider interspaces
than occur between the individual glands. Frerichs mentions
that this latter arrangement may occur in other regions of the
organ, and I have myself observed it independently of other
morbid alterations in the cardiac portion. The membrana
limitans of the glands (as pointed out by Dr. Habershon,
whose observations I have repeatedly found confirmed)
breaks down with great facility, and numerous nuclei and
much granular matter are thus set free in the tissue, in
which, by a slight alteration in the focal distance, a deeper
layer of connective tissue can be brought into sight, and the
observer be led to the fallacious opinion that this is generally
increased in amount. Dr. Handfield Jones describes a
condition to which he has applied the term “nuclear
degeneration,” as occurring with great frequency. I have
met with nothing corresponding to his descriptions and
figures (with the exception of one case), but such appearances
as I have above described, and which I am disposed to consider as the effect of cadaveric change. In many cases he speaks of large portions of the glandular tissue being replaced by free nuclei, imbedded in a fibrous stroma; but in the drawings which he has given of these appearances, the nuclei there represented have much more the characters of those of the glandular epithelium than of the connective tissue. I am disposed to doubt whether an independent development of free nuclei can occur under any circumstances, so as to constitute a special form of degeneration; for careful observation has shown that, in most, if not in all, the cases where such a condition has been described—as in tubercle of the lungs or other organs, the enlargement of the patches of Peyer and of the lymphatic system in typhoid fever, and in some forms of sarcoma and cancer—this appearance has been due simply to the early breaking down of the cells which contained the nuclei, by which means they have been set free, and appear, on examination, to form the sole constituent elements of the tissue. Such formations, with the exception of the cells of the glands of Peyer and of the lymphatic system, belong mostly to the class of heterologous products; but in the tissue intervening between the glands of the stomach we have no cause to suppose, in most of these cases, that any new tissue has been developed, and, if we have only to do with a simple increase of a tissue of the same type as that previously existing, that newly formed must, according to the general laws of hypertrophy, present the same characters as the old. At the same time, I must confess that I have seen one case in which the appearances bore some resemblance to the descriptions given by Dr. H. Jones. There was a great number of nuclei free in the tissue, and in one or two places collected in considerable numbers below the line of glands. These were perfect in some parts, but in others in the immediate neighbourhood they had entirely disappeared. The nuclei had a look different from those of the epithelium; they were apparently smaller and clearer, and refracted light more strongly. But the stomach was
much softened, and the breaking down of the glands, in some parts at least, left it open to the suspicion that their appearance in the fibrous stroma was due to this source. If they had any other origin, I am at a loss to explain their presence. The patient had died of extensive tuberculous disease of the lungs and intestines, and the appearance in one or two spots in the submucous tissue of the stomach resembled, under the microscope, to some degree, that afforded by miliary tubercles of the pleura in its earlier stages; but nothing of the kind could be detected by the naked eye. There was in this case a real increase of the inter-glandular fibrous tissue, and other signs of chronic catarrh. I have endeavoured to give a representation of this object in the accompanying drawing (vide Plate II, fig. 7). That a positive increase of the fibrous tissue between the glands does take place there can, I think, be no doubt. It then appears thickened, the glands lie further apart, and, on the addition of acetic acid, a crowd of nuclei are seen, usually appearing elongated and in much greater numbers than are ordinarily seen in these situations, where in the healthy condition little is brought into view by this reagent except the nuclei of the capillaries and a few contractile elements mingled with some elastic fibres. They correspond very closely with the elements of the submucous cellular tissue, though from the direction of the fibres stellate cells cannot be discovered. (Plate I, fig. 4.) The glands are more widely separated, but are not necessarily diminished in diameter, as there is no check to the enlargement by thickening of the membrane, and compression is not, therefore, a necessary consequence. In fact, in some cases, where this increase in the fibrous structures is met with, there seems to be a simultaneous enlargement of the glands, and I have only once been able to observe a positive diminution in the diameter of a gland in chronic catarrh, except in the instances to which I shall hereafter allude under the head of "cystic degeneration," and in the case in which it occurred there were other enlarged ones in the immediate neighbourhood. Whether this condition, if
carried to a higher point, would result in any extensive atrophy of the gland-tissue, I have not been able positively to determine, though, judging from the analogy of other organs in which similar changes occur, such a possibility can hardly be denied; but, except in the instance alluded to, I have neither been able to trace the loss in question, nor any intermediate stages showing a progressive diminution in the size of the glands.

(2.) **Thickening of the membrana limitans** of the glands forms a second of the series of changes induced by chronic catarrh. I do not find this condition described by any of the pathologists who have devoted their attention to these structures, though my observations leave no doubt on my own mind that such a change really exists with considerable frequency, and often accompanied with other signs of morbid change in the membrane, and more especially with pigmentary deposit and fatty degeneration of the epithelium.

There is a source of fallacy to be avoided in observations on this point, in the fact that the membrana limitans of the glands of the stomach does, like that of those in the mouth and pharynx, as pointed out by Kölliker, swell up on the addition both of acetic acid and the solutions of caustic alkalies. This reaction is not constant; but I am unable to offer any explanation of the reason why it takes place in some cases and not in others. The thickening in question may often be seen without the addition of any reagents, though it is considerably increased by them. The membrana limitans is seen under ordinary circumstances as a fine clear line around the gland, perfectly structureless, and not possessing any appreciable thickness; but, in some cases of chronic catarrh, it presents a clear double-defined outline, of a breadth easily recognisable, around the circumference of the follicle, the thickening being generally most marked at the lower end, but often traceable into the upper two thirds of the tube. The glands in which this condition occurs have often an irregular outline, presenting narrowings and contractions in their course. This thickened membrane
appears as a rule perfectly homogeneous, without a trace of structure, though I have once or twice observed a faint appearance of striation in it. The change occurs with greatest frequency in the pyloric region, and affects equally the glands lined with a cylindrical epithelium, and those containing spheroidal cells. It is met with also in the neighbourhood of the cardia, and in the glands of the small curvature. I have not found it in the fundus, nor in the left half of the great curvature. (Vide Plate I, figs. 4, 5, and 8.)

(3.) The state in which the epithelium of the glands is found is a variable one. It may be apparently normal, though this is extremely rare; or it may present but little alteration in appearance, except that the cells are smaller and in diminished numbers, or, may be fattily degenerated to a greater or less degree; or, finally, it may be almost entirely absent, its place being occupied by molecular debris and fat-drops. I have not seen it (except when recent catarrh has coexisted) increased in size, nor presenting the granular look, disappearing with Liq. Soda, which is observed in the acute affection. In the more advanced stages, complete fatty degeneration seems to be the natural course of the disease, and the state of the glands is thus illustrated by the accompanying drawing (Plate I, fig. 4), exhibiting the appearances presented by a vertical section made in the pyloric region of the stomach of a man dying of phthisis, and who for some time before his death laboured under an almost total anorexia. I am decidedly of opinion, from the progressive series of changes which I have been able to trace, that this accumulation of fat is due to a degeneration of the epithelium. In some cases, where this is most marked, the lower extremities of the glands filled out with fat-drops can alone be seen under the microscope; but I should hesitate to ascribe the disappearance of the upper portion to pathological changes. It is very seldom that the glands can be traced to the surface in a vertical section; they generally appear to terminate in a mass of cells, nuclei,
and molecular débris, at a distance of two or three lines from this,—a disintegration which I believe to result from mechanical injury, in the preparation of the object, to the already softened upper layers of the membrane; and it is very possible, when the stomach is already affected by disease, that a more rapid post-mortem disorganization may facilitate this artificial breaking down of the structure. I have not seen any considerable accumulation of fat in the interior and swelling of the epithelium, so as to give rise to any of the forms of granule-cells. They appear either to be set free or to break down before this stage is reached, and the nucleus shares early in the disintegration; at least, it is rare for free nuclei to be found in the granular and fatty débris which occupy the interior of the glands in the more advanced stages of the condition in question.

(4.) Pigmentary deposit is another of the appearances observed in this affection of which mention must be made, though I have but little to add to Dr. H. Jones's observations on this subject. The mucous membrane exhibits in spots of variable size, from one or two to four or five inches in diameter, a dark slaty-gray appearance. It is most marked in general at, or near, the pylorus, but occurs also in other parts, as in the small curvature, or in the central parts of the great curvature. That, in common with other forms of pigmentation, its production is due to changes in the hæmatin of the blood, little doubt can, I think, exist; and in the cases where it occurs the cause is to be found in long continued hyperemia and congestion, attended by escape of blood from the finer capillaries, of which we have such frequent examples in the stomachs of patients suffering from obstructions to the portal or general circulation. It is not uncommonly met with coincidently with similar alterations in the intestinal tract. Its seat seems to be either superficial, or extending through the whole thickness of the mucous membrane. I have seen it, as Dr. H. Jones has described, within the epithelium of the glands, as well as in the inter-cellular tissues, and I have figured one case, where
it could be seen in the cells of the connective tissue, near
the surface of the imperfectly formed villi in the neighbourhood of the pylorus. (Vide Pl. I, fig. 6.)

(5.) **Cystic degeneration of the glands** appears to be a secondary result of one or more of the series of changes which I have endeavoured here to describe. This alteration has been but little noticed, but the number of stomachs in which I have met with it induces me to believe that the appearances produced by this change have been mistaken for solitary glands, similar to those of the intestine; the frequency of which, as occurring in the stomach, has, I think, been greatly exaggerated. I am somewhat at a loss to understand the description which Dr. H. Jones has given of some bodies which he has figured, and whose origin he seems inclined to ascribe to changes in the solitary glands—a class of structures with which these cysts have nothing in common; and this is the more incomprehensible to me, since Dr. H. Jones also recognises degeneration of the glands as a frequent cause of these cystic formations; though I must demur from accepting his further statement, that they originated as "vesicles," which subsequently enlarge, especially since he gives no further information as to the locality or mode of origin of the vesicles in question. The solitary glands when they do occur in the stomach, have their seat below the layer of the tubular secreting structures, and are sometimes even deeply imbedded in the submucous tissue. I have only met with two indubitable instances of their presence in the stomach; but one is figured by Frerichs in Wagner's 'Handwörterbuch der Physiologie,' art. "Verdaunung," and another by Ecker in the 'Icones Physiologicae.' I must also object to the term "nuclear masses," which Dr. H. Jones has applied to these bodies, as inadequately describing their structure, and further tending to induce a confusion with the appearances which he describes as nuclear degeneration, though I have endeavoured to show reasons for questioning the propriety of applying even the latter term to morbid conditions of the stomach. The
glands in question, as met with in the intestine, when from their number they can be easily examined, and with which, I believe, those occasionally found in the stomach entirely correspond, are closed follicles, having a distinct capsule; and, I am inclined to believe, a membrana limitans; but their essential feature, distinguishing them from all the proper secreting structures of the intestinal tract, is the nature of their contents. These are cells, not nuclei (at least when fresh); but the cells are of the same character as those met with in the closed follicles of other organs, and their counterparts are to be found in the cells of Peyer's glands, in the lymphatic system, and in some of the cells of the Malpighian bodies of the spleen; in all which situations, also, the nuclei mostly exceed the cells in number, unless the part be examined perfectly fresh, as the cell-walls rupture and set free their contents with great facility.

The contents of the structures which I am about to describe are of a totally different character, being both columnar and spheroidal epithelium, corresponding with that lining the glands; and, having been able to follow sometimes in the same stomach a progressive series of changes, from the simple gland to the fully developed cyst, I feel no doubt remaining in my own mind as to their real nature. They may, after they have obtained any size, be distinguished by the naked eye as small round bodies, having a peculiar pellucid appearance, situated apparently on or near the surface of the mucous membrane, and differing also in this respect from the solitary glands, which, as before remarked, are more deeply seated. They are best seen in a bright light, falling at an angle of 40°, but are easily overlooked when the light is not good, or the search for them is not very carefully made. Their number varies greatly; sometimes only one or two can be found in the whole mucous membrane, at other times hundreds may be counted. I have hitherto met with them with a relatively much greater frequency in the neighbourhood of the pylorus, and at a distance of from one to three inches from it; but they also occur scattered, and, occasionally, in considerable numbers
in the middle third of the great curvature, extending both to the anterior and posterior surfaces. They are seen, though more rarely, in the neighbourhood of the cardia.

When examined under the microscope, they are seen, when fully formed, to be round or ovoid bodies of variable size, sometimes measuring from 0.42 millimetre to 0.5 millimetre in breadth, and when ovoid having a long diameter averaging 0.52 millimetre. They are generally enclosed in a capsule of fibrous tissue, which may obtain a thickness of 0.006 millimetre. I have only once been able to observe in a fully formed cyst any trace of a thickened membrana limitans; and it is only in some cases that any remains of this structure can be seen. The contents are, as before stated, of an epithelial character, either columnar or spheroidal, according to the part of the stomach in which the cyst is formed, and sometimes both kinds are met with in the same cyst, an appearance due to the constriction leading to the formation of the cyst taking place at the upper part of the follicle, which is always lined towards its orifice with cylindrical epithelium. When once thus encapsuled, the epithelial structures seem to be tolerably persistent. I have repeatedly found them perfectly preserved when the epithelium in the surrounding glands was almost entirely fatty; occasionally a few cells in a state of fatty degeneration may be seen within the cyst; but after some time the cells undergo a species of atrophy; they become very clear and transparent; the cell-wall grows thinner, until it disappears; the nucleus grows indistinct, and finally the cyst becomes filled with a clear mucus-like substance, having some resemblance to "colloid" matter, in which granular débris and a few entire cells still float. A slight pressure generally suffices to burst these bodies, and to set free their contents in the field of the microscope, when their nature can be further ascertained. Such is the cyst when perfectly formed; and in many cases it is almost impossible to trace its direct connexion with any remains of glandular structure; occasionally, however, the gland-tubes can be traced in continuity with them, either at the upper or lower
end; sometimes presenting an appearance nearly normal, at
others wasted to a fine thread; occasionally a second con-
striction may be observed in the cyst, or in the gland at a
short distance above or below it, giving rise to a second
formation of the same character. (Fig. 7, Pl. I, represents
these appearances.) I have, however, met with even more
direct evidence of their mode of origin in an object of which
I have given an illustration in fig. 8, Pl. I, being a gland
undergoing a double constriction in its course. It occurred
in a stomach where numerous other cysts of varying sizes
were present. The gland was lined throughout by a cylin-
drical epithelium, though only faint traces of this were
observable above the upper narrowed part, and in the lower-
end some fat was seen. The membrana limitans was thick-
ened as far as the upper constriction, and presented a
homogeneous, transparent, highly refracting, double outline.
There was considerable thickening of the fibrous tissues
around this gland, and also in other parts of the stomach;
and this had taken place to a most marked extent around
the constricted parts. At both of these, but especially at
the upper, where the lumen of the tube was almost obliter-
ated, there was a marked striation, looking as if the mem-
brana limitans were thrown into folds, or as if the growth
of fibrous tissue were encroaching on this structure; an
idea supported by a somewhat indistinct appearance of
elongated nuclei at this part.

In all the cases which have yet come under my notice
this change was always associated with one or more of the
alterations previously described, though they are not equally
present at all times. There may be considerable slaty dis-
coloration of the membrane, with but little thickening of
the intervening fibrous tissue. The most constant atten-
dant change is that of fatty degeneration of the glandular
epithelium; this may occur to a slight extent, both in the
neighbourhood of the pylorus and cardia, without any other
apparent morbid changes in the structure, and can scarcely
be regarded, in such cases, as of special pathological im-
portance; but when met with to any marked extent, either
in these situations or in other parts, so as to replace the
normal epithelial cells, it must be considered as morbid.

Professor Simpson, in his collected works, has described,
under the title of "pellicular inflammation," a condition of
the gastro-intestinal mucous membrane, attended with dis-
turbances of the digestive functions, and consisting of a
vesicular eruption, which appears to bear some resemblance
to the conditions of the stomach of which I have given an
account. My friend, Dr. A. R. Simpson, who first pointed
out to me this paper in his uncle's writings, also drew my
attention, during a post-mortem examination which he was
performing, to the simultaneous occurrence of cystic forma-
tions in the stomach, rectum, and posterior part of the
fauces, and more especially in the glands of the uvula. I
have since met with two similar cases, and have no doubt
of the true cystic character of the formations in the rectum,
which I have represented in fig. 1, Pl. II. It may, per-
haps, still require further observation to determine whether
the changes in the upper part of the pharynx are true cysts,
or simply glands distended with the products of secretion.
I am inclined to the belief that the former will be found to
be the case, and have, in fig. 2, Pl. II, represented one
burst at the upper end; but I have not been able to trace
any intermediate stages in the process of their formation.
I have not been able to find anything similar in other parts
of the intestinal tract, though it seems extremely probable
that the glands and other structures of the mucous mem-
brane of the intestine may be liable to the same processes
of morbid change as that of the stomach. Dr. H. Jones
has found the glands of the duodenum enlarged, and I have
also observed the same appearance coincident with other
alterations of the stomach. Professor Simpson remarks,
that the patients suffering under the condition which he de-
scribes often pass masses of a white substance by stool; and
in the intestine of the first-mentioned case—a patient who
died of caries of the bones of the pelvis, complicated with a
hydrocephalus of the left side, caused by the impaction of a
calculus in the ureter—there were seen numerous white
masses mingled with the feces, which appeared to consist chiefly of epithelium, but no other morbid alteration could be detected here.

(6.) Fatty degeneration of the connective tissue is not very unfrequently found associated with these alterations. Dr. Jones has described and figured these appearances, and I have met with it three or four times in the course of my observations, though not in the position in which he has seen it, viz., between the glands, but in the elastic tissues below them. The cells in this part become filled with fine granules of fat, and are at the same time somewhat enlarged, and have a very granular look, which does not disappear either with acids or alkalies. In one or two cases I have also seen associated with it a fatty degeneration of the coats of the vessels, both in the finer capillaries which pass between the glands, and also in those of the submucous tissue. As far as I have yet seen, this has been associated with disease of the heart, or similar changes in the larger arteries.

Often coincident with these changes, but at other times occurring apparently independently of them, are other forms of fatty degeneration, of which some have been already noticed by writers on diseases of the stomach, though others have hitherto excited but little attention.

The most frequent of these are found as small, white, highly refracting spots, frequently of about the size of a millet-seed, scattered over the surface of the mucous membrane; they occur most frequently in the pyloric portion, but are often met with in the neighbourhood of the cardia; and, on examination by the microscope, are found to depend on a fatty degeneration of the epithelium of groups of glands, which are entirely filled with fat-drops.

Another form, noticed by Dr. H. Jones, in the fifth volume of the 'Transactions of the Pathological Society,' consists in dull, opaque, white spots, generally of about the size of a split pea, apparently quite superficial, but not
elevated above the line of the surrounding membrane; and which, on microscopic examination, are found to depend on deposits of fat in the upper layers of the membrane, which are rendered perfectly opaque by their presence, so that their real nature can only be ascertained at the edges, where fat-drops may be seen in all parts of the tissue. In one case I was able to trace the apparent primary seat of this affection. The patient was a man who had been for some time under Professor Bichow's observation. He had first suffered from pneumonitis, had subsequently an attack of parotitis and suppuration of the cervical glands, and finally died with symptoms of albuminuria. On examination post mortem, tubercle was found in the lungs; the liver was fatty; the kidneys presented thickening of inter-tubular substance and fatty degeneration of the epithelium; the other viscera were healthy. The mucous membrane of the stomach was injected throughout, but more especially so in the fundus and small curvature; the injection extended also through the left two thirds of the great curvature. The surface was covered with a tough, gelatinous-looking mucus, which was found to consist, in large proportion, of enlarged spheroidal and columnar epithelium and free nuclei. The membrane had a thickened and swollen appearance. In the middle third of the small curvature, and extending over its anterior surface into the great curvature, was a number of small hæmorrhagic spots, varying in size from a pin's point to a pin's head, and of a deep reddish-brown colour. The left half of the stomach presented all the appearances which I have before described under the head of recent or acute catarrh, viz., enlargement of the epithelium, and an abnormally granular character of their contents, irregular enlargements of the glands in their course, and fatty accumulations in scattered points. Some of the hemorrhagic spots had the appearance of being surrounded by a whitish zone, which again passed into a more pellucid tissue. On a section being made through such a point, it was found, that while the glands at the spot where the extravasation had taken place were almost broken down, the tissue was filled with
granular débris, blood-discs, hematin-crystals, and pigment, and was stained throughout of a red colour; the glands in the whole circumference of such a spot, extending through a series of five or six outwards, were almost filled with epithelium undergoing a fatty degeneration, while external to these the tissue presented the characters before described.

In the pyloric portion the process seemed to be of older date, as shown by an extensive fatty degeneration of the epithelium of the glands, accompanied by thickening of the membrana limitans, and some, though but slight, increase of the connective tissue between them. Near the pyloric ring there was a white spot, similar to those before described, and which could be seen by the naked eye to be in the superficial strata of a thin section made with a Valentin's knife. The central parts of this spot were almost perfectly opaque, but a thin line, representing the membrana limitans, could be traced along the upper border. It was only at the sides and lower edge that the true fatty nature of the change could be recognised; and here fat-granules, some of them of a peculiar brownish colour (the origin and nature of which are questionable), were found to penetrate all parts of the tissue, and the normal elements were with difficulty, and in some cases not at all, discernible. At the lower edge a very interesting series of changes could be perceived in the cells of the connective tissue. They were seen to be enlarged and filled with fat, giving to this part the peculiar appearance of strings and stellate groups of fat-granules, crossing and interlacing with one another, while around some of them the outline of the cell-wall was still discernible. At some points there were appearances as if the coats of the arteries were also undergoing this change. Very little of the glandular structures could be seen at this spot, but their lower ends could be still detected here and there resting on the thickened corium, the cells of which also appear enlarged, and in some places fatty. The glands around were in a highly fatty condition. (Vide fig. 5, Pl. II.) These changes appear to me to represent a fatty
degeneration of the elements of the tissue, but commencing in what may appear an unusual manner, and specially affecting the cells of the connective tissue. It appears to lead to occasional loss of substance, which I have been able, in one or two instances, distinctly to trace to this cause, and one of which corresponded so closely with a description given of a superficial erosion by Dr. H. Jones, that I will quote his words: "The mucous surface of the stomach was thickly studded with depressions of a circular or irregular form, and varying in size from the diameter of a silver penny to one fourth of that magnitude. They were rather paler than the surrounding membrane, but many of them were dotted with black points. They existed all over the stomach, except for an inch or two near the pylorus. Their margins were not sharp cut, but evenly rounded ones. The depressions were much more translucent than the surrounding tissue, and it appeared clear that a loss of substance had taken place. In vertical sections, under the microscope, the mucous membrane in the vicinity was found to be quite healthy, the tubes perfectly natural; but in the depressed part the tubes were reduced to a mere granular débris. The basement-line of the surface was lost. There was a great deal of oily matter in the disintegrating tissue, and just beneath it. There was no particular change in the submucous tissue. The surrounding tissue passed rather abruptly into the disintegrating, and there was no deposit or morbid formation of any kind in the parts affected. No injected vessels were seen by the microscope, nor any pigmen
tary deposit. The change in this case seems to have been more akin to sloughing than ulceration."

In the case which I observed, the patient was a woman dying of puerperal fever. The stomach was pale; no injection could be seen anywhere. The mucous membrane was throughout rather soft; that in the fundus was already considerably disintegrated. In the central part of the posterior surface of the small curvature, extending into the great curvature, were six spots, nearly circular, varying in diameter from one eighth to three quarters of an inch, where there
was evident loss of substance on the surface of the membrane. The surface in the depressed part looked smooth to the naked eye, and the edges were evenly rounded off. There was no redness of the membrane, no injection of the blood-vessels at or around these parts. A vertical section carried through several of these showed the same appearances in all. In the central depressed part the upper part of the tubes could be traced, though faintly, through the midst of a granular débris; what remained of them was granular and fatty to a high degree. They retained the same character towards the edges, and scarcely a trace of epithelium could be seen in their whole course. This fatty degeneration passed gradually into the usual healthy structure, within a few lines of the edge of the depression. The other parts of the stomach were healthy.

In another case, there were somewhat similar changes, but presenting a rather different appearance to the naked eye. The patient was a man, æt. 26, who died of the rupture of a strangulated inguinal hernia. The other organs were healthy. The stomach was stained throughout with bile; the mucous membrane soft, but nowhere injected; its appearance to the naked eye was, on the whole, healthy, with the exception of two white spots in the central part of the great curvature, which were rather elevated at the edges above the surrounding membrane, and one of which had a cup-like depression in the centre. A third similar one was found in the small curvature near the pylorus. The appearances of these are represented in fig. 3, Pl. II. In fig. 4 the same superficial deposit of fat is seen as I have before described. The change occurred in the pyloric region, and the basement-line of the rudimentary villi is still perfect. In fig. 3, where depression existed in the centre, we find a more advanced stage of the same. The whole thickness of the mucous membrane had apparently undergone a fatty degeneration, being filled with fat-drops, which had broken down the membrana limitans. Among the débris thus produced, faint traces of the glands could be still perceived. The dark granular appearance thus produced ceased rather
abruptly on each side, but the glands for a short distance showed a fatty degeneration of their epithelium. These cases are not without interest in relation to some forms of erosion or ulceration of the stomach, which still remain involved in considerable obscurity. The principal seat of the change being in the cells of the connective tissue, suggests an analogy with some forms of atheroma—to which attention was drawn by Mr. Gulliver, in the 'Medico-Chirurgical Transactions' of 1843—in the arteries, and especially that described by Professor Virchow, under the name of "fettige usor" (and of which an account has recently been given in the 'British and Foreign Medico-Chirurgical Review'), where the accumulation of fat in the cells of the middle coat leads to their disintegration, and finally to a loss of substance at the parts. The comparative frequency with which the condition in question is met with in cases of chronic catarrh would point to an inflammatory origin, of which this process represents the last stages, though the limited form in which it appears to occur may, perhaps, cause some doubt still to exist on this point, and render it desirable that its mode of production should be made a subject of further inquiry.

With regard to the forms of catarrhal inflammation, which I have here attempted to describe, I would venture to call attention to an interesting analogy which appears to me to be traceable between them and the acute and chronic inflammatory affections of the kidneys. The difference in structure of the two organs must, of course, give rise to some difference in the appearances presented; for the most complicated vascular system of the kidney, its more compact structure, and the long course and convoluted character of the secreting tubes, which do not so easily allow of the escape of the altered epithelium, nor of swelling taking place without producing pressure on the blood-vessels, and the various phenomena which are the secondary effects of this disturbed circulation, give rise to numerous pathological

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1 'Gesammelte Abhandlungen von Rudolph Virchow,' pp. 504 et seq.
appearances in this organ which are not met with in the stomach, where the structures are simple in their arrangement, and the laxity of the tissue allows either swelling or contraction to occur, without necessarily producing such important secondary changes. In the essential characters of the affection of the glandular system, there is, however, a striking similarity. In the first stages we find increased vascularity and swelling of the tissue, which, in the kidney, produces the swollen and cloudy look by which this change is recognisable by the naked eye, and in which, under the microscope, the glands are seen to present an unusually white appearance by reflected, and a darkly granular look by transmitted, light, disappearing on the addition of alkalies, and which are found to depend on an increase of the protein contents of the secreting cells. Precisely the same change is seen in the epithelial cells of the stomach, both in the glands and in those found in the thick tenacious mucus which covers the surface. Slight amounts of fatty change are also seen in the epithelium of the glands of the stomach as in that of the kidney, even in the acute stage; but in neither does it attain a very marked character at this period. In the later stages we have a different series of alterations presenting very similar characters in both organs, viz., those affecting the epithelium and those affecting the connective tissue. The epithelium does not appear to suffer so extensively in the chronic affections of the stomach as in those of the kidney. This may be due in part to the greater facility with which it is set free, and replaced by new cells; but in both we find an alteration of the same nature, viz., fatty degeneration. That this reaches a higher point in the kidney may also, perhaps, be referable to the fact, that its nutrition is more impaired by the increased production of interstitial tissue, affecting more especially the glomeruli, and by the contraction to which this condition gives rise causing shrinking and an anemic condition of the tissue, to which it is evident that the stomach is much less liable; but the essential character of the process in causing the development of an increased amount of the connective
tissues, is in both cases the same; it occurs in both in the membrana limitans, which thickens; in occurs in the interstitial tissue, the effect of which, in the stomach, is chiefly marked by widening of the spaces between the glands; and it is found in the tissues upon which they rest. In some cases the increased nutritive activity extends also to the muscular coat, but of this I have not met with any very striking examples in the course of my investigations, where the thickening of the coats of the organ has generally appeared to be rather due to a change affecting the corium than to a hypertrophy of the deeper structures. The cystic degenerations to which I have drawn attention complete this parallel, which, if borne out by subsequent inquiry, tend, I think, to supply another link in the history of the laws of the affection of glandular structures by the inflammatory process.

In the course of these investigations I have met with a striking instance of a peculiar affection of the mucous membrane, associated with similar changes in other organs, and produced by a form of degeneration of the tissues, which has of late attracted considerable attention, under the names of "lardaceous," "bacony," or "albuminoid," change; and in which the interest of the profession has been still further excited by the discovery of Professor Virchow that the parts so affected give a peculiar blue tinge with iodine, a reaction which has induced him to employ the term "amyloid degeneration" in describing this state.

The patient in whom the appearance which I am about to describe occurred was a woman, set. 34, who died in an excessive state of marasmus. The liver was enormously enlarged, and showed the characteristic appearances of this disease, giving also the above-mentioned reaction with iodine; this was further found to exist in the kidneys, in the whole of the intestinal tract, in the tissue of the lungs (while the mucous membrane of the bronchi appeared free), and in the rete Malpighii of the skin. The muscles were unaffected. The stomach was everywhere pale, the mucous membrane was thin, and had a peculiar glistening appearance.
Sections made in all parts refracted light to a remarkable degree; but the change was most marked in the superficial strata, though it could not be specially traced in the coats of the vessels, where it often appears to have its chief seat, and where it was found in the intestines in the case in question. The whole glandular structure was, however, affected. There was hardly a trace of normal epithelium to be seen, but the interior of the glands was filled with a homogeneous mass, strongly refracting light and giving the characteristic reaction with iodine. Large masses, retaining the form of the glands, and having similar properties, could be separated on tearing the tissue with needles. The reaction with iodine was most marked towards the upper surface, but it extended also into the deeper parts. The vessels in the submucous tissue did not appear to be much affected. On the addition of Liq. Sodae, numerous fat-drops were to be seen in the lower part of the tubes. This was most particularly the case in the cardiac and pyloric portions, but their connexion with the epithelium could not be distinctly traced. The squamous epithelium in the lower part of the oesophagus was similarly affected, but the change did not extend into the submucous tissue, and the muscular coat, both here and in the stomach, was entirely unaffected.

Had no other cause existed, the amount of marasmus in this case could be accounted for by the impairment which the digestive functions alone must have undergone, for scarcely a trace of the essential elements of glandular action, viz., the epithelium, could be found in the stomach; and from the condition of the villi in the intestine, it was reasonable to infer that the process of absorption must have suffered in an almost equal degree.

This may, perhaps, be the fittest place to mention another species of morbid alteration, of which, however, I have only observed one instance, and as to the nature and mode of production of which I must confess myself to be in some doubt; though I hope that if the attention of other observers is attracted to it, other cases may be met with tending better to its elucidation. The stomach was taken
from the body of a male patient, of about the middle age, whose history could be only imperfectly ascertained, though it appeared that he had had a large quantity of blood abstracted without apparent cause, and had taken a considerable amount of mercury for supposed syphilis.

The blood, during life, contained a great excess of white corpuscles, and the same condition was found post mortem, but no other disease could be detected. The lungs and heart were healthy; the liver was of normal size, and presented no unusual appearances; and the other viscera were healthy.

The stomach was pale; a few cysts, of the character before described, could be found in the mucous membrane. This had throughout a peculiar shining, and at the same time a translucent, appearance, which was exaggerated in limited spots, of about two lines in diameter, giving it a mottled look; while here and there were small patches of the dead opaque white colour which attends extensive fatty degeneration of groups of glands.

The translucent parts were found to exhibit, under the microscope, the appearances which I have attempted to delineate in fig. 6, Pl. II; though this was presented, to a greater or less degree, by sections made in all parts. There were seen scattered through the tissue, large, round, or oblong bodies, varying in size from the dimensions of twice or three times that of the spheroidal gland-cells to smaller bodies of about half that size. They were specially aggregated in the upper layers of the mucous membrane, and here also they were found larger than in other parts, and encroached upon and greatly obscured the upper parts of the glands. In the deeper layers they could be especially traced, but greatly diminished in size, in the tissue intervening between the glands, which were much further apart than normal. Near the pylorus, where the change was most marked, they entirely filled and replaced the structure of the rudimentary villi of this part. They refracted light very strongly, and I at first thought them to be large fat-drops, or some inorganic salts, but they disappeared en-
tirely, both on the addition of liq. sodae and acetic acid. They did not give any reaction with iodine alone, nor with iodine and sulphuric acid. In some parts, they appeared to be especially accumulated around the capillaries passing up between the glands; but the connexion between them and the vascular system could not be very distinctly traced. Nothing of this kind could be found in the submucous elastic layer, nor in any other of the coats. Their reaction would lead to the belief, that their constitution was that of a protein substance, and the situation in which they are found would justify the supposition that their origin must be sought in some degeneration or other change of the connective tissue; for though, when attaining a large size, and accumulated in great numbers, they encroached on the glandular structures, yet even between and below such points the epithelium was preserved more or less perfectly, though in many places it had undergone a fatty degeneration. Professor Virchow, to whom I showed some preparations of these appearances, informed me that he has seen similar ones in the ovula Nabothii, and in cysts of the ovaries, but was in doubt as to their nature and origin.

I have collected in the following table the cases in which disease was found in my examinations of a series of 100 stomachs, taken indifferently. They give a sum total of fifty-seven cases, in which either recent or chronic catarrh existed, viz., recent catarrh alone in twenty-one cases; chronic catarrh alone in nineteen cases; recent and chronic catarrh combined in seventeen cases. There is a considerable difference between the proportions thus expressed and those given by Dr. Handfield Jones, who for 100 cases, returns only twenty-three cases of catarrh; but this is in part explicable by the difference of meaning attached to the term. I apply it to all cases where I find a catarrhal condition of the glandular epithelium, while of the twenty-three cases recorded by Dr. H. Jones, the glands in twenty are spoken of as being but little affected.

In seven of the twenty-one cases of uncomplicated recent
catarrh, I found but little injection, though often considera-
ble thickening of the membrane; and I then founded my
opinion of the state present entirely on the condition of the
epithelium, which was precisely similar to that of numerous
other cases presenting the usual character of catarrh, as
recognizable by the naked eye.

<table>
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<tr>
<th>Acute Catarrh of Stomach</th>
<th>Chronic Catarrh</th>
<th>Acute and Chronic Catarrh combined</th>
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<tr>
<td>Pneumonia, Acute</td>
<td>Tubercle of Lungs (uncomplicated)</td>
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<td>&quot; Chronic</td>
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<td>Morbus Cordis</td>
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<td>Tubercle of Lung and Intestine, Bronchiectasia, Morbus Cordis, Cysta in Rectum and Soft Palate</td>
<td>Tubercle of Lungs, Tubercular Pericarditis, Old Valvular Disease of Heart, Liver fatty, Kidneys granular</td>
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<td>Tubercle of Lung, recent Bright’s disease</td>
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<td>Morbus Cordis, Cirrhosis of Liver, recent Pneumonia</td>
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<tr>
<td>Endo-Pericarditis, first</td>
<td>Tubercle of Lungs,</td>
<td>Pneumonia, (uncomplicated)</td>
</tr>
<tr>
<td>stage of Bright’s</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>disease</td>
<td>Morbus Cordis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Suppurative Peritonitis</td>
<td>Capillary Bronchitis,</td>
<td>Morbus Cordis, Gangrene of Lung</td>
</tr>
<tr>
<td>Cholera; kidneys in</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>first stage of Bright’s</td>
<td>Chronic Bright’s disease, Fatty Liver</td>
<td>Morbus Cordis, Gangrene of Lung</td>
</tr>
<tr>
<td>disease</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Typhoid Fever</td>
<td>Gangrena Pulmonum,</td>
<td>Pneumonia, recent Bright’s disease</td>
</tr>
<tr>
<td>Morbus Cordis, Capillary</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Bronchitis</td>
<td>Morbus Cordis</td>
<td></td>
</tr>
<tr>
<td>Morbus Cordis</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Tubercle of Lungs</td>
<td>Melanosis of Lung,</td>
<td>Delirium Tremens</td>
</tr>
<tr>
<td>Tubercle of Lungs and</td>
<td>Pleurisy</td>
<td>1</td>
</tr>
<tr>
<td>Tubercular Peritonitis</td>
<td>Cystic Disease of Ovaries, Peritonitis</td>
<td>Suppurative Parotitis, Kidneys fatty</td>
</tr>
<tr>
<td>Diabete</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Carcinoma Ventriculi</td>
<td>Hernia</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>21</td>
<td>15</td>
</tr>
<tr>
<td>Total</td>
<td>19</td>
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</table>

I have given in this table a short summary of the principal attendant pathological conditions, which, though less complete than I could have wished to make them, still, I think, present some points of interest with regard to the etiology of the disease.
One remarkable fact which appears from this table, especially in the contrast of recent and chronic catarrh, is the greater proportionate frequency with which the former is associated with acute, and the latter with chronic, affections of other organs. Recent catarrh of the stomach was met with in two cases of variola; in two of typhoid fever (four cases of this were examined); in two of cholera; in four cases of simple acute pneumonia; and in two others, in one of which the pneumonia was accompanied by heart-disease, and in the other with phlebitis; and in one of chronic pneumonia; in four out of nine cases examined of various puerperal inflammations; in three cases of phlebitis from various causes; and two of suppulsive parotitis. The frequency with which it appears to occur as a complication of acute diseases, which are associated with the swollen and cloudy appearance which marks the first stage of Bright's disease of the kidneys, struck me as very remarkable. I have before alluded to the analogy which I believe to exist between the affections of the glandular structures involved in the inflammatory process, and it would seem as if the same exciting cause sometimes sufficed to produce similar changes in the cellular structure of both organs; but in one case I examined, in which death took place from acute idiopathic Bright's disease, I found the stomach perfectly healthy. Septic and acute febrile disease seem especially to possess this tendency, and it was especially in these cases that the coincidence was observed. That it is not an invariable law, is shown by the fact that these associated changes were only met with in four out of nine cases of puerperal fever.

The tendency of diseases obstructing the venous circulation to induce a catarrhal condition of the stomach, which has been pointed out by Dr. H. Jones and other observers, meets with additional confirmation from this table, where it is seen that catarrh may arise as a complication in cases where either the heart, the lungs, or the liver, offer obstructions to the free passage of the blood; but the disease which appears to lead to its evolution with peculiar
frequency, and to present it in the most advanced and persistent forms, is tuberculosis. Of the total number of cases of catarrh here recorded, whether acute or chronic, tubercle is seen to be present in sixteen out of fifty-seven, or in 28 per cent. (The total number of tubercular patients examined amounted to thirty-one.) This is perhaps not more than might have been expected, when we recollect the frequency of dyspepsia as a symptom of phthisis, the occurrence of which it has been often difficult to explain; especially since tubercular formations in the gastric mucous membrane are of extreme rarity, not one case being recorded in the work of Louis on this disease. I have looked for them in vain in the stomachs which I have examined, though I have alluded to one case in which there was a suspicion of a formation of this kind in the cellular tissue below the glands.

Of cases in which the patients had been acknowledged drinkers of spirits, I have met with alterations of structure in three. They partook of the characters of both recent and chronic catarrh, but in one only could I trace any history of previous gastric disturbance.
DESCRIPTION OF ENGRAVINGS.

PLATE I.

Fig.
1. Recent catarrh of stomach, showing the irregular outline and granular appearance of the glands, 90 diam. Without reagents.
2. The same magnified 340 diam., showing the glands filled with enlarged and swollen epithelial cells. Without reagents.
3. Cells extruded from the glands in a state of acute catarrh, 400 diam. 
   a. b. c. Enlarged and granular spheroidal epithelium.
   d. The same treated with strong acetic acid.
   e. Swollen columnar epithelium.
   f. Free nuclei.
   g. Enlarged cells, containing double nuclei.
4. Chronic catarrh, 290 diam. The upper part of the glands not seen, the breaking down being probably due to post-mortem change.
   a. A mass of pigment in upper layers of membrane.
   b, b, b. Free fat-drops.
   c. Thickened membrana limitans.
   d, d, d. Entire fatty degeneration of the epithelium.
5. Chronic catarrh. The preparation, treated with acetic acid, shows the increase of connective tissue (the nuclei of which are thus brought into view) between the glands; thickening of the membrana limitans of the glands. The cloudy appearance of the epithelium is here due to the action of the acid. 340 diam.
6. Pigmentary deposits in the villi of the pyloric region, in a case of chronic catarrh. The pigment is seen at (a), within the cells of the connective tissue; at (c) it is free among the elements of the tissue. 340 diam. Treated with acetic acid.
7. Cystic degeneration of a gland in the pyloric portion. The gland-tube is seen continuous with the lower part of the cyst; glands around, with epithelium in state of partial fatty degeneration; 100 diam. Without reagents.
GLANDULAR STRUCTURES OF STOMACH.

Fig.

8. A gland-tube which has undergone a double constriction in its course; probably an earlier stage of the foregoing; 340 diam. Without reagents.
   a. Thickening of membrana limitans.
   b, c. Points of constriction where the connective tissue is greatly thickened.
   d. Fatty degeneration of epithelium.

9. A cyst from the pyloric portion of the stomach, magnified 340 diam.; without reagents; showing the contents to be cylindrical epithelium; at (a) is seen the capsule of fibrous tissue surrounding it; (b, b) glands in a state of chronic catarrh.

PLATE II.

1. Cystic degeneration of the glands of the rectum; 340 diam.
2. Cystic change (?) in the glands of the uvula.
3. Fatty changes in upper layers of mucous membrane, leading to erosion of the surface; 100 diam.
4. A similar change in the pyloric portion; membrana limitans of the membrane preserved; 100 diam.
5. A change somewhat similar to the above, in which the cells of the connective tissue (a, a, a) are seen in a state of fatty degeneration of their contents. (b) Fatty degeneration of the cells of the corium.
6. Appearances described at page 390 of accompanying paper, showing large round and oval bodies accumulated above and between the glands. They disappeared entirely on addition both of Liq. Soda and acetic acid.
7. Appearances resembling (?) the "nuclear degeneration" described by Dr. Handfield Jones, showing great numbers of nuclei scattered through the fibrous tissue, and especially accumulated at the base of the glands. (Qy. Cadaveric change ?)
ON THE INFLUENCE
OF THE
CERVICAL PORTIONS OF THE SYMPATHETIC NERVE AND SPINAL CORD
UPON THE
EYE AND ITS APPENDAGES,
ILLUSTRATED BY CLINICAL CASES, WITH OBSERVATIONS.

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Of late years direct and rigorous experiment has done much in placing us in possession of more correct ideas respecting many parts of the human frame, and especially of the nervous system. Objections, however, have been entertained against the conclusiveness of experiments upon healthy animals as illustrative of morbid actions in man; and these objections may be reduced to three in number. First, the supposed essential difference between results obtained experimentally and those consequent upon disease in man; secondly, the great and almost insuperable liability to implicate numerous and important parts in the attempt to arrive by operation at any given structure, thus complicating the very subject which we are striving to render clear; and thirdly, the modification of results obtained, owing to unavoidable haemorrhage and the exhaustion of sensibility. It is as regards the comparative freedom from the last two objections that experiments upon the sympathetic nerve in the region of the neck, the properties and anatomical con-
stitution of which have ever divided the opinions of physiologists, may be most favorably considered, and their results accepted as trustworthy. The large size of the vessels and nerves of this region, the ease with which they may for the occasion be dislocated without injury or danger to the animal, rendering the sympathetic, as it were, quite superficial and accessible, the simple arrangement and the ready dissection of the parts, all these particulars have caused our knowledge of the influence exerted by the cervical sympathetic upon the heart, lungs, and eye, to be much added to of late.

The main points which I propose to bring before the notice of the Society in the following paper, may be summed up as follows:

In the first place, I shall commence with certain prefatory observations and a short historical review of the most important results obtained as regards the eye and its appendages, by experiments which have from time to time been performed upon the cervical parts of the sympathetic, and the spinal cord with which it is connected. I shall then proceed to relate the histories of clinical cases, old and new, in which a contracted state of the pupil had become a subject of interest, induced apparently by the pressure of thoracic aneurisms, equivalent in degree to the experimental action of the sympathetic in animals.

In the second place, I shall bring forward clinical cases in which a similar result had been observed in connexion with aneurism of other arterial branches than those within the thorax.

In the third place, I shall adduce instances in which pressure upon the sympathetic (but from other causes than aneurism) had produced a like effect, as in the case of enlarged glands, carcinomatous and other deposits, &c.

In the fourth place, bearing in view the intimate connexion between the sympathetic main branches in the neck and the cervical part of the spinal cord, I shall draw attention to certain cases in which a contracted pupil had been observed in injuries of the spinal cord itself.
ON THE EYE AND ITS APPENDAGES.

In the *fifth place*, I shall bring forward clinical cases in which a *dilated* state of the pupil was apparently produced by pressure, from various causes, upon the sympathetic, the pressure being so slight or transient as merely to act as a stimulus to the dilator fibres of the pupil, enabling them to overbalance the resisting contractors.

In the *sixth place*, and lastly, calling to mind one or two points in the anatomy of the sympathetic in the neck and within the cranium, I shall relate cases in which symptoms arose from pressure upon the sympathetic or certain nervous structures with which it is connected, such as might, in addition to these effects upon the iris already alluded to, have been anticipated from anatomical considerations. These symptoms are strictly analogous to the various phenomena produced in the lower animals by section of, or extreme pressure upon, the sympathetic.

The influence of the "sympathetic" portion of the nervous system on the modifications of sensibility and vascularity, and of the temperature of external parts supplied by it, has been within the last few years abundantly proved experimentally by Brown-Séquard,¹ Claude Bernard,² Schiff,³ A. Kussmaul, and A. Tenner,⁴ &c., although these authors differ in some important respects as to their interpretation of several phenomena witnessed by them. Moreover, Bernard⁵ ascertained that, in addition to alterations of the temperature of external parts, division of the cervical part of the sympathetic induced a much increased temperature within the cerebral hemisphere on the side upon which section of the nerve had been made;⁶ and also that the blood in the jugular vein was rendered warmer on the side operated upon

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¹ ‘Mémoire lu à l’Académie des Sciences,’ Janvier 16, 1854.
² ‘Comptes rendus de l’Académie des Sciences,’ Mars 29, 1852.
⁵ ‘Mémoires lu à la Société de Biologie,’ 1853, p. 94.
⁶ See also Henle’s and Pfeuffer’s ‘Zeitschrift,’ vii, p. 206, 1855.
than on the opposite one. But, in addition to modifications of vascularity and temperature, experiment shows that an altered state of muscular parts may be effected by interference with those branches of the sympathetic system which are distributed to them. For instance, Valentin and Henle, and, still later, Budge,¹ have demonstrated that contractions of the heart and larger blood-vessels are producible by stimulation of the sympathetic branches derived to them. E. H. Weber,² of Leipzig, Arnspengen,³ and others, made many observations on the decided effects of the pneumogastric (a nerve considered, I believe, by Brown-Séquard and others as being to a great extent a sympathetic one) upon the movements of the heart; and E. P. Pfüger⁴ shows that arrest of the movements of the small intestine follows galvanic irritation of the splanchnic nerves.

Now, although it is only in quite recent times that the physiological relation between the sympathetic and the various vessels and muscular parts to which it is extensively distributed has become at all adequately appreciated, yet for a number of years many observers have at times instituted experiments tending to show the effects of the sympathetic upon the eye. I will here pass in very brief review some of the most interesting results which have been arrived at by various observers in this direction, so far only as they have relation to the present communication, a passing consideration of which will best lead to a full understanding of the cases which I now present to the notice of the Society.

The first experimenter who appears to have recognised, amongst other effects of injury to the sympathetic nerve in the cervical region, those upon the pupils of the eyes, was Pourfour du Petit. Experimenting as he did between the years 1712 and 1727, upon animals in which the sympathetic nerve in the neck and the pneumogastric are so intimately connected as to be incapable of separation, as in the dog, for example (unlike the rabbit or the cat, in which these

¹ Frerich's 'Tages-bericht,' 1852, No. 441.
³ Virchow's 'Archiv.,' vol. ix, 1856.
⁴ Quoted in Schmidt's 'Jahrbücher,' vol. lxxxix, No. 1, p. 16, 1856.
two nerves lie together in one sheath, but are disjoined), he found that when the united cord of the vagus and sympathetic was divided, the following phenomena were observable. In many animals he noticed on the side operated upon a most marked contraction of the pupil, partial closing of the eyelids, with protrusion of the cartilaginous third eyelid at the inner angle of the eye, and also a redness and apparent swelling of the eye, with, consequently, the secretion of pus from its surface. It is at once obvious that these effects are very similar to those of section of the fifth pair of cranial nerves, when divided near the Gasserian ganglion, as they were first described by Fodern, Herbert Mayo, Majendie, and Sir C. Bell. They however differ materially in the following particulars, viz., in their accession at a much earlier period after injury of the nerve, and in their greater limitation, inasmuch as the cornea very seldom indeed becomes ulcerated, and the inflammation appears to be confined to the conjunctiva, whilst in section of the fifth pair within the cranium, as described by Valentin, the iris, after profuse suppuration of the surface of the eyeball, becomes distended with blood, the chambers of the eye and the pupil become filled with exudations, and the cornea ulcerated. Petit, aware of the anatomical connexion between the sympathetic in the neck and the pneumogastric, sagaciously perceived that it was in regard of the section of the sympathetic alone that these phenomena were

1 'Memoirs on the influence of the so-called Intercostal Nerve on the Eye,' 1727 ('Histoire de l'Académie Royale,' avec les 'Mémoires,' &c.)
2 Emmert ('Archiv. f. d. Physiolog. Von Reil und Autenreith,' Bd. xi, p. 117), who experimented much upon the necks of animals, found that in the dog, wolf, and fowmart, this connexion between the vagus and the sympathetic in one sheath was most intimate, whilst, on the other hand, in the cat, goat, sheep, bull, horse, ass, and pig, he found that it was only very slight, although it did exist. In the hare, rabbit, and guinea-pig, the two nerves are quite separate, as in man.
3 'Journ. de Physiologie expérimentale,' tome iii, p. 227, 1823.
4 'Anatomical and Physiological Commentaries,' No. 2.
5 'Journal de Physiologie experimentale,' tom. iv, p. 178, 1824.
6 'De functionibus Nervorum Cerebraliun, &c., pp. 109-114, Bernae et Sangalli, Helvetiorum, 1802.'
called forth. After Petit, a number of experimenters more or less confirmed his observations. The chief of these were Peter Mollinelli, 1 who has recorded five observations describing the results of ligation and section of the sympathetic and par vagum, as united in the neck of the dog, Justus Arnemann, 2 Cruikshank, 3 Dupuy, 4 Cammerer, 5 Mayer of Bonn, 6 Brachet, 7 Reid, 8 and Valentín. 9 But on reading their productions, several discrepancies are manifest, and it is evident that they experimented with very different powers and with very varied intentions of observation.

Dupuy, Professor of Veterinary Surgery at the College of Alfort, made a number of experiments along with Breschet, and under the eye of Dupuytren, upon the cervical sympathetic in the horse.

Like Petit, he also noticed that division of the nerve was followed by contraction of the pupil, descent of the upper eyelid, and redness of the conjunctiva, &c. In addition, he observed a peculiar dryness and adherence of the skin, swellings of the eyelids, and general emaciation; moreover, he noticed an increased temperature of the ears, with augmented perspiration and eruptions upon the skin, thus anticipating in a remarkable manner much that has more recently been brought forward as regards the influence of the sympathetic upon the capillaries of the skin and other parts. Brachet also found section of the sympathetic cervical nerves to be followed by cerebral congestion, agitation, somnolence, and stupidity.

1 'Commentarii Academiae Bononiensis,' tom. v, pp. 280-297, 1755.
2 'Versuche über d. Regeneration der Nerven,' Göttingen, 1787.
3 'Philosophical Transactions,' part i, 1735, but written in 1776.
7 'Recherches expérimentales sur les fonctions du Système Nerveux Ganglionnaire,' 1830 and 1837.
I need not in this place proceed with any minuteness to
detail the various phenomena connected with the eye, or
other parts, as called forth by the experiments of the above-
mentioned observers on the sympathetic. It suffices to
state that the following were the most prominent aggregate
results so obtained, which I will notice as having special
relation to the object of this paper;—a contracted condition
of the pupil, an inward rolling of the eyeball (convergent
*strabismus*), and a lowering of the upper eyelid to a greater
or less degree (partial *ptosis*). The various experimenters
differed in their observations, but although most interesting,
it would here be out of place to tarry and show in what
particulars they differed or agreed, or upon what circum-
stances their discrepancies depended.

These various effects (as well as others which I will not
now particularise) followed division of the cervical part of
the sympathetic in the middle of the neck of the dog, in
which animal, as well as in the horse, it is found that the
motor fibres of the iris contained in the cervical cords of
the sympathetic, and (following Valentin) the *vagus*, are
not confined to the upper ganglia, but pass through a larger
extent of the trunks of the sympathetic cords. Reid, how-
ever, found that in the case of rabbits operated upon at
the same part of the neck, neither did decided redness of
the conjunctiva, nor yet any alteration in the state of the
pupil, take place at all; and this fact has since been con-
firmed by others. But if, in the case of the rabbit, instead
of dividing the sympathetic at the middle part of the
neck, we remove the superior cervical ganglion, or its com-
munications with the cervical nerves, or its branches asc-
cending to form the plexus around the carotid artery; or if,
again, we remove the ganglion of the pneumogastric or the
communications between the cervical nerves and this gan-
glion, then these results do follow, the pupil becoming small,
oblong, and angular. These phenomena are to be explained,
according to Reid, by the difference in the distribution and
connexion of the nerves in the region of the neck in dif-
ferent animals. For instance, whilst the nervous filaments
which in the rabbit pass from the spinal cord into the sym-
pathetic, and thence into the iris, come from the superior cervical nerves only, they are found, on dissection, in the case of the dog and horse, to come from the lower cervical nerves in addition. Other differences also in the distribution and connexions of the sympathetic fibres in the neck are found to exist in mammals. It will be remembered that in man the communicating branches between the cervical nerves and the sympathetic pass from all the cervical nerves, and join the sympathetic at their ganglia as well as in the course of their prolongations, and branches also pass from the cervical nerves to the ganglion of the pneumogastric.

Valentin, considering the effects of these and other experiments not here mentioned, as well as the acknowledged influence of the third pair of cranial nerves upon the iris, in the lenticular ganglion, came to the following conclusion. He determined that the movements of the membranous iris were under the control of a double nerve-force, emanating from two distinct portions of the nervous system. He demonstrated that that branch of the inferior division of the third pair which is distributed to the inferior, and probably involuntary, oblique, or rotating muscle of the eye, and which contains sensory as well as motor fibres, presides, by means of its connexion with the lenticular ganglion through its short root, over the actions of the circular muscular fibres, viz., those drawing the veil towards a central point; whilst the pupil-dilating fibres, viz., those radiating from the margin of the pupil towards the attached or circumferential edge of the iris, were under the dominion of the sympathetic branch or branches passing from the cavernous plexus which also contain motor as well as sensory fibres. These latter branches are derived secondarily from one or more of the cervical sympathetic, and (according to Valentin) from the vagus-nerve ganglia, but primarily and essentially from the cervical

1 This proposition of Valentin's as to the part played by a double nerve-force in the movements of the iris, so far as the subject of this paper is concerned, is not affected by the views of Schiff, who thinks that the dilator-fibres of the pupil, enjoy both a cerebral and a spinal source of nerve influence.
nerves and spinal cord by branches of junction between them. Thus the iris enjoys a double innervation; the one cerebral, the other spinal; each antagonising and balancing the other. This counterpoise of the opposing fibres of the iris is well illustrated by the dilatation of the pupil which, as a general rule, occurs along with ptosis and abducent squint, when the third cranial nerve is divided experimentally, or much pressed upon by an inter-cranial tumour, &c., the iris-dilating or extensor fibres being then unopposed by the sphincter or flexor fibres. It is also well exemplified by the following circumstances. When a longitudinal opening is made into the iris of a rabbit, the artificial pupil becomes shorter and rounder, so long as the eye is healthy and the opposing flexors and extensors of the iris are not prevented from duly balancing each other; but if in the same animal the "nervi molles" be divided, or, as Valentin terms it, the "fons spinalis" be destroyed, then the artificial orifice, formerly round in form, becomes no longer so, but greatly elongated and arched, assuming a direction more or less parallel with the margin of the natural pupil.

Such being the state of our knowledge in regard to this matter, Budge and Waller, confirmed by experiments,1 in 1841, the fact that those branches of the sympathetic which control certain motions of the iris have not their real origin from the main trunk or the ganglia of the cord in the neck, but that they arise from the spinal cord, passing through the spinal nerves to the cervical sympathetic; and in those conclusions Wagner and Ruiter2 agreed.

By following the nerves step by step until they found a place where irritation produced no effect, and by gradually removing portions of the cervical cord and watching the results on the pupils, they went on to prove, as it appeared to them, that in certain animals those branches which control these movements of the iris were connected solely with that portion of the spinal marrow itself as a centre which

1 Vierordt’s ‘Archiv für Physiol. Heilkunde,’ 1852; Ergänz. Heft.
2 ‘De actione Belladonae in Iridem,’ Dissert. Traj. ad Renum, 1849.
reaches from the sixth cervical to the fourth dorsal or thoracic vertebra.

Within the above limit any stimulus, chemical, galvanic, or mechanical, applied to the cord, if not too great, produces dilatation of the pupil, just indeed as if applied to the cervical branches of the sympathetic or to the branches passing to the sympathetic from this portion of the spinal cord. To this part, therefore, of the spinal cord, they gave the name of "regio cilio-spinalis." It should be stated, that Majendie had previously mentioned disorganization of the eyeball as following division of the cervical part of the spinal cord, although he did not bring forward any explanation of it.

Budge determined that the anterior and middle columns of the spinal cord are to be considered as the truest seat of influence to the iris, the influence of the motor roots of the nerves on the iris being infinitely greater than that of the sensory. He experimented largely upon mammalia, fishes, and frogs, as to the movements of the iris, and found that the latter animals were most useful as regards many experiments, partly because they bore a larger amount of chloroform than the others, and partly because in their case the influence of the sympathetic upon the eye was most clearly shown. In addition to the above-named centre of nervous influence to the iris from the lower part of the cervical cord, which Budge terms the "untere central-stelle," that observer\(^1\) has lately described a second one, called by him the "obere central-stelle," situated high up in the neighbourhood of the so-called hypoglossal, or ninth pair of cranial nerves, the branch or branches anastomosing between this nerve and the upper cervical ganglion of the sympathetic being the medium of the influence or communication.\(^2\)

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1 'Ueber die Bewegung der Iris für Physiologen und Ärzte,' Braunshweig, 1855, p. 109.

2 Dr. Brown-Séquard has lately informed me that, in his belief, injury of almost any portion of the spinal cord will to a certain extent affect the pupil corresponding to the side injured. It seems, however, that if this be so, the influence of such an injury upon the pupil diminishes in pro-
Bidder and Volkmann found that in frogs\textsuperscript{1} that part of the cord from which the iris-influencing nerves proceed corresponds to the second and third cervical nerves.

Budge, and, to a certain extent, Ruiter also, conclude that the first division of the fifth cranial nerve has specific branches of motion to the iris, but that the vagus nerve and the communicating branches of the upper cervical with the sympathetic have no possible influence upon the iris. The motor influence of the fifth upon the iris is thought by Budge to be proved by the contraction of the pupil, which is seen to follow division of the fifth, even although the third pair be divided so as to remove the possibility of any reflex action through that channel.

Valentin, moreover, thinks he proves that in the rabbit an antagonism between various parts of the spinal nervous origin itself exists, and that the innervation supplied through the vagus ganglion (which this observer looks upon as having an influence upon the movements of the iris) controls the muscular fibres nearest to the lower end of the pupil, whilst that from the upper cervical ganglion presides over the muscular fibres nearest the upper end. It appears to him to be so from the fact that, on dividing the ganglion of the vagus and its roots, the margin of the pupil, which is rendered oblong, becomes sharp and angular above, and the inferior margin rounded; but if the upper cervical ganglion of the sympathetic be divided, the sharp margin of the pupil is below and the rounded one above.

In addition, Budge and Waller found that any injury to the whole or half of the central parts of the spinal cord, alluded to above, or to the entire nerves issuing thence, or to the anterior branches only of these nerves, removed all power of action from the extensor or dilator muscles of the pupil, leaving the pupil-contracting fibres to the active and unfettered influence of such fibres of the third cerebral portion to its distance from the cervical region; and for practical purposes, the "cilio-spinal regions" of the neck must be considered to exist.

\textsuperscript{1} 'Die Selbständigkeit des Sympath. Nerven-Systems,' Leipzig, 1842.
nerve as preside over them, and (as they think) to the motor influence of the ophthalmic division of the fifth.

Brown-Séquard and Bernard came to the same conclusion as to the general details of these experiments mentioned by Budge. In addition to other phenomena, they found\(^1\) that in cases where contraction of the pupil and vascularity of the conjunctiva, \&c., had resulted from division of the sympathetic in the neck, galvanism of the upper end of the divided nerves produced a *dilatation* of the pupil and opening of the eyelids, as also cessation of the conjunctival vascularity, and diminution of the increased temperature, \&c. These effects are in some particulars not so strongly marked when they follow galvanism of the sympathetic nerve, which has not been previously divided. Schiff\(^3\) agrees with Bernard and R. Wagner in stating that this galvanizing induces positive protrusion of the eyeballs.

H. Müller\(^4\) has also described dilatation of the pupil as following galvanism of the cervical sympathetic after decapitation at the sixth cervical vertebra (less, however, than that produced by belladonna), and its continuance so long as the stimulus was applied, the pupil of the side opposite that on which galvanism was used being at the same time not at all affected.

Corroborations of the above-made statement as to the effects of section and galvanism of the sympathetic, \&c., are to be found in the writings of Schiff (loc. cit.) of Callenfels, and others.\(^5\) It is worthy of remark that Ruiter\(^6\) found the size of the pupil on the side operated upon, to be, in comparison with that of the opposite eye, exactly as two to three in the rabbit, and in the dog as one to three. In accordance with this statement, another of Valentin's is interesting, viz., that in the dog the cerebral influence upon the iris, exercised through the third cranial nerve, as op-

\(^1\) See Brown-Séquard's *Memoir,* 1854; also Gaz. Médicale, 1854.
\(^3\) Verhandlung. d. Würzburg Gesellschaft.
\(^4\) Henle's and Pfeffer's *Zeitschrift,* vii, 1855, p. 157.
\(^5\) *De actione Belladonae in Iridem,* Op. cit.
posed to the spinal which acts through the channels of the sympathetic, is greater than in the rabbit.

It ought to be added, that Professor Reid found that pressure also upon the cervical part of the sympathetic produced contraction of the pupil, which, on the removal of the pressure, completely regained its natural size.

The above particulars comprehend, as I believe, the sum of our present information with regard to the peculiar dependency of the movements of the iris on certain branches of the sympathetic and that portion of the spinal cord from which they emanate, as shown by experiment.

The question is naturally suggested as regards the contracted state of the pupil in connexion with the ptosis and strabismus which in so many cases followed division of the cervical sympathetic,—What is the signification or physiological purpose, of these changes in the eye and its appendages? I believe these phenomena may be looked upon as having some final reference to the condition of the retina, just as we find that in early life the pupil is generally contracted, and thus adapted as a screen to the delicacy of this tender nervous expansion; or again, as in old age, when, as Bichat observes, the pupil is generally dilated in proportion to the insensibility of the retina or to the opacity of the originally transparent refracting media, by which the entrance of light is hindered. And why in these experiments the retina should require such careful protection, such a tendency to contraction of the pupil and closure of the eyelids in order to exclude a superabundance of light, may easily be conjectured when we call to remembrance the anatomical constitution of that structure, a most delicate nervous sheet, the integrity of whose capillary vessels as to tone, &c., is of the very utmost necessity to that of its function; and when we also bear in mind the numerous examples which we possess of disturbance to the capillary circulation produced by injury to the sympathetic, whose ultimate branches are distributed along with the vessels.

A better instance of this capillary disturbance, by way of illustration, we cannot have than in the distension of the
pericardial blood-vessels and the exudations poured from them out around the heart after injury to the thoracic ganglia of the sympathetic, as shown by Bernard and Schiff.

We may not then injudiciously or unreasonably suppose that in the matter of the retina the very numerous and closely packed capillary vessels of this membrane become affected, whenever, by experiment or disease, the sympathetic nerves from which they are supplied become seriously injured, and that a distinct relation exists between the unnatural condition so produced and the experimental phenomena above mentioned, which are, as regards the retina, protective in their aim. Pathological instances of this kind of confederate or consensual action for conservative purposes might, if needful, be quoted in abundance. A case in point, which I have lately fallen in with, is so notable that I will briefly describe it.

A man, aged 45, experienced the effects, during the night, of a very vivid flash of lightning. He was at once struck down and unable to rise for some seconds, but was not at all deprived of consciousness. At the time he felt great pain in the eyes, but this soon went off, leaving him blind and unable to open them. He continued blind for seven months, at the end of which time the form of the eyes was found to be good and the cornea clear, but the pupils were exceedingly contracted. The moment the eyelids were forcibly raised from before the pupil the patient cried out in ecstasy that he saw the light, but added that it gave him such acute pain that it could be no longer borne, and begged that the eyelids might be allowed to close. In the course of time the patient regained his sight, the retina, as it is observed, still retaining excessive sensibility. It appears to me that in the above case the firm spasmodic closure of the eyelids and the contracted pupil possessed a definite relation to each other, and had special reference to some particular condition of the blood-vessels of the exquisitely sensitive retina, induced by the impress of the over-strong light.

1 'Memoirs of the Medical Society of London,' vol. ii, p. 508.
The above-mentioned phenomena may be concomitant only with the unwonted condition of the retinal capillaries, but it is also possible that they may stand in relation to that condition as reflex movements, according to the suggestion of my friend Dr. Baly.

As a summary of the various particulars contained in the previous part of this communication, I would say that there seem to exist anatomical and experimental grounds for the subjoined statements.—That certain movements of the iris (the contraction and dilatation of the pupil) are under the control of certain fibres of the sympathetic nerve emanating from the carotid and cavernous plexus, as are also frequently to some degree, at least in lower animals, the movements of the levator of the upper eyelid and the external rectus muscle of the eyeball. That these sympathetic nervous twigs are derived secondarily from the great sympathetic trunks in the neck, but primarily from certain parts of the spinal cord, by communication between these grand trunks and the spinal nerves. That consequently, the same effects produced upon the iris, the levator palpebræ, and external rectus, by interference with the sympathetic in the neck, will follow if the communications passing between it and the cervical part of the spinal cord, or the cord itself, be similarly affected. That, as a rule, paralysis of the dilator fibres of the iris (permitting contraction of the pupil), and, in many animals, partial paralysis of the levator palpebræ and external rectus muscle, follow section of, or extreme pressure upon these

1 In connexion with this supposition, I would allude to the experiments, before quoted, of Brachet, who found cerebral congestion and somnolence to follow division of the sympathetic in the neck—phenomena doubtless resulting from some unnatural capillary condition; and to those of Bernard and Callenfells, who determined by actual experiment that the temperature of the brain, and of the blood returning from it, and the size of the meningeal vessels, was increased by the experiment.

2 It may here be stated, that it is not in all the experiments that contraction of the pupil, or even elevation of superficial temperature, follows division of the sympathetic; and much more variable appears to be the influence of the sympathetic upon the upper eyelid and external rectus muscle. Upon what this difference depends is not known.
parts of the nervous system, whilst mere irritation by electricity, and stimulating, chemical, and mechanical agents, induce a dilatation of the pupil.

Bearing in mind the results of the above-mentioned experiments upon healthy animals, it is to be expected that under disease any pressure or lesion of the cervical sympathetic or its ganglia, or its connexions with the spinal cord, would tend to produce effects on the iris similar in kind at least to those arrived at by experiment.

With the beautiful physiology of this subject in view, it was that Dr. Gairdner, of Edinburgh,\(^1\) shrewdly interpreted the rationale of contraction of the pupil, which he found to occur in certain cases of thoracic aneurism in which the enlarged vessel pressed upon some parts of the ganglia or branches of the sympathetic. He has brought before the Medical and Chirurgical Society of Edinburgh, cases in which the contracted state of the pupil existed, apparently owing to interference with the sympathetic in the upper part of the thorax. These cases of his I will briefly allude to along with those placed on record by others, numbering them so as to include in a series those which I shall myself bring forward in which the pupil of the eye only had become affected, or in which, in addition to this symptom, other symptoms also appeared, apparently arising from interference with the sympathetic nervous supply. In Dr. Gairdner's cases the only symptom dwelt upon by him as being produced by such interference is the *contraction* of the pupil.

**Case 1** was that of a quarryman, &t. 40,\(^2\) affected by aneurism of the arch of the aorta, involving the sympathetic

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1 Since writing the above, Dr. Gairdner has sent me a pamphlet written by Dr. Robert MacDonnell, of Montreal, and reprinted from the 'Montreal Medical Chronicle,' June, 1858, entitled, "On Contraction of the Pupil, a symptom of Intra-thoracic Tumours," containing the history of a case observed by him as early as 1850, in which pressure on the sympathetic at the root of the neck had produced contraction of the pupil, and also *ptosis*. To this case I shall refer further on, at p. 432.

nerve, as shown by post-mortem examination, and in all probability the lower cervical ganglion, as also the various spinal nerves and the vertebral artery. During six weeks of observation the left pupil was seen to be much smaller than the right one, but both of them acted under the influence of light. It is stated that "for a good many weeks before death the difference in the size of the pupils had become scarcely recognisable." This case terminated fatally by hæmorrhage into the oesophagus.

**Case 2** was one recorded by Dr. Walshe¹ of aneurism of the aorta, greatly compressing the innominate artery, and causing the right radial pulse to be almost imperceptible. In Dr. Walshe's account I find it stated that "the left pupil during life was observed to be one eighth of an inch in diameter; the right one was not more than half the size, and both round and moderately brisk. After death both were larger than during life; the right one, that which during life was so notably the smaller, was very distinctly larger than the left one."

**Case 3** was one described by Dr. Gairdner, of a journeyman shoemaker, who, along with signs either of thoracic aneurism or disease of the aortic valves, with hypertrophied heart, had contraction of the left pupil. He acquainted me with the particulars of this case in December, 1856, and I have lately heard from him that in this patient, who is still in improved health (May, 1858), there exists a "remarkable irregularity of temperature,—cold sweats followed by flushing, accurately limited to that half of the face on which the pupil is affected." Dr. Gairdner also tells me that recently he has had the opportunity of seeing "another case of unilateral sweating in connexion with contraction of the pupil on the same side and signs of heart disease or aneurism."²

¹ 'Diseases of the Lungs, Heart, and Aorta,' 2d edit., p. 759.
² Since writing the above, Dr. Gairdner has informed me that the first of these two patients affected with unilateral sweating, has lately died,
Case 4 was under Dr. Gairdner's own treatment, and was one in which a contraction of the pupil was associated with thoracic aneurism.

Case 5 was also under Gairdner's observation, and was a "well-marked case of innominate aneurism." The right pupil was contracted as compared with the left. The patient, a woman, is probably still alive.

Case 6 is published at large by Dr. Williamson, of Leith. In this the contracted pupil (a symptom which continued to exist even after death) along with certain neuralgic symptoms was considered during life to be the means of diagnosis between aneurism and aortic valve disease. After death, intra-thoracic aneurism was found on the same side as the contracted pupil. This case has lately been alluded to in connexion with the subject of contracted pupil as consequent upon aneurismal pressure, at p. 482 of the 'Archives générales,' April, 1858.

Case 7, published by Dr. Banks, of Dublin, King's Professor of Physic, was that of a woman, alt. 24, who had a contracted pupil in connexion with a supposed aneurism in the upper and left part of the chest. The sight of the affected eye was good, and the contraction of the pupil, though constant as compared with the pupil of the other eye, was susceptible of change, like its fellow, according to the amount of light permitted to fall upon the eye. The patient left the hospital in the same condition, and from a letter kindly sent to me, April 29th, 1857, I learnt from Dr. Banks that he had seen nothing of the patient since her dismissal from the hospital.

but that a post-mortem examination could not be obtained. He also states that in this case the peculiar symptoms were at the last in abeyance, the pupils being equally small.

2 'Dublin Hospital Gazette,' January 15th, 1856. This case has been quoted in the 'Dublin Quarterly Journal of Medical Science,' vol. xxv, p. 498.
Case 8 is one related by Dr. Willshire,¹ of a supposed enormous dilatation of the ascending sorta and arch, with the vessels arising therefrom, occurring in a woman, æt. 60. The right pupil was always more contracted than the left one, but it quickly dilated on the application of atropine to the eye.

Case 9 is that of a man, æt. 58, related by Dr. Hope,² who was affected by aneurism of the ascending sorta, of the size of a cocoa-nut, with general dilatation of the sorta, and who died of pneumonia. He had had headaches for about six weeks, and, in addition to other symptoms, complete blindness of the right eye and incomplete blindness of the left one. Both of the pupils were contracted. No special brain symptoms existed, and after death the brain was found healthy, but there was thought to be slight dwindling of the left optic nerve.

The above-named cases are the only ones, excepting one quoted by Dr. Reid (here numbered as Case 25), which are, so far as I yet know, placed upon record, or privately noticed by others, wherein an alteration had been observed as a presumable result of interference with the sympathetic branches supplied to the iris, by pressure from intra-thoracic aneurisms.³ It will be remembered that the only symptom

¹ 'Lancet,' 1856, vol. i, p. 678.
² 'Diseases of the Heart and Great Vessels,' 3d edit., 1889, p. 608.
³ In a letter quite recently received, and since the writing of the above, Dr. Gairdner informs me that he has met with four other cases in which a contracted state of the pupil was connected with intra-thoracic aneurism. In the first the symptoms were “clear.” This case was seen with Dr. Simpson. In the second a small aneurism pressed towards, but did not directly involve the sympathetic on the right side. On the same side the pupil was contracted. In the third case Dr. Gairdner was called to the patient the night before he died suddenly. Both the pupils were remarkably contracted, and on this phenomenon, combined with other symptoms, a diagnosis of aneurism was founded. On post-mortem examination a large aneurism was discovered enroaching on both sides of the vertebral column all the way down the thorax. In the fourth case,
of interest in connexion with these aneurisms, which are all of the thoracic sort, was a contracted state of the pupil. It occurred to me that aneurisms of other arteries than those in the thorax might, by exercising similar pressure, produce similar results as regards the pupils of the eyes. I thought it might be so with the subclavian or the carotid in the neck; and I was fortunate enough to find one or two instances recorded in which alteration of the pupil was produced by aneurism of the carotid.

The following forms a very striking instance:

Case 10.—A man, aged 41, had an aneurism of the left carotid artery measuring five and a half inches by four. Headache, cough, dyspnœa, and dysphagia were induced. The pupil of the left eye was particularly noticed by his attendants as being contracted, and vision of the left eye was impaired. Mr. Coates, of Salisbury,1 tied the artery, and on the eighth day after the operation the pupil, which had been contracted, was observed, according to the published notes, as "having almost recovered its natural state of dilatation and sensibility."

The above case, although meagre as to particulars, seems to me to afford a remarkable illustration of the connexion between injury of the sympathetic in the neck by aneurismal pressure, and altered conditions of the iris; the latter recovering its natural state in proportion as the aneurism lessened after the operation, thus relieving the nerve of the unwonted pressure.

It also suggested itself that probably pressure from other

one seen with a pupil of St. Andrew's, at the Dispensary, the signs were "all pretty clear." The patient died, but no post-mortem examination could be obtained. He also mentions two or three other cases in which a contracted pupil was found and a thoracic aneurism guessed at, but not proved to exist; and also several other cases in which, along with this modification of the pupil, there was "no reasonable ground to suspect pressure on the sympathetic."

1 Johnson's 'Medical Journal,' vol. ii, 870, March, 1822.
ON THE EYE AND ITS APPENDAGES.

sources than aneurisms might in a similar way affect the pupil. This might be so in certain cases where the lymphatic glands were greatly enlarged and indurated by inflammatory processes or occupied by specific products, as the scrofulous or carcinomatous; or where the general tissues about the main course of the sympathetic in the cervical or upper thoracic region were similarly affected; or again, it might be the case where abnormal bony growths existed in certain parts of the thorax or neck, or even of the cranium, as of the basi-cranial bones, involving the neighbourhood of the cavernous sinuses.

Here I am bound to mention, that in my hopes of finding such cases I was in a degree anticipated by Dr. Reid, who mentions a case (one which I shall insert as my 25th) in which a contraction of the pupil was produced by pressure from malignant deposit in the neck.

Whilst on the watch for cases such as I have just alluded to, one presented itself to my notice in St. George's Hospital in the course of the year 1856. It was as follows:

Case 11.—A man, aged 22, was admitted with a great enlargement of the cervical lymphatic glands on the left side, forming a painful tumour of the size of a small orange. There was cough, with occasional stridulous breathing, and, subsequently, great dyspnœa and bloody expectoration. Extensive disease also of the axillary glands came on. The pupils of both eyes were noticed as being dilated and equal in size, but on the twenty-first day after his admission the cervical glands had enormously increased, and the pupils were seen to be very unequal, that of the left eye being much the smallest. Both pupils contracted when the eyes were directed either inwards or outwards, and only after a short space of time did the right pupil regain its comparatively large size. On the twenty-third day the tumour was of the size of a swan's egg. There was no difference as detected by the hand (the thermometer unfortunately was not used) in the temperature of the ear or side of the head, &c. As before, the left pupil was much smaller than the
right one, but both pupils contracted when the eyes were turned outwards or inwards, and this was the case independently of the varied access of light. Oftentimes they were seen to become equal when the eyes were kept very widely open, and when suddenly turned to the bright light of a window.

On the patient's looking about or withdrawing the eyes from the light, the right pupil would again become dilated, and thus the two pupils again became unequal. After death I found the left lung, and the mediastinal, cervical, and axillary glands, all occupied by firm carcinomatous deposit, effecting considerable pressure upon the sympathetic and its connecting branches, the trachea and oesophagus being greatly pushed to the right side.

Further on I shall adduce, as Case 19th, an instance of scarlet fever in which the pupil was affected by pressure from enlarged glands in the neck.

I may here mention that I have notes of a case in which there was extensive inflammation of the pleural membranes and mediastina passing up the neck, and involving all the deeply placed tissues about the vessels and nerves, with great effusion of firm inflammatory products as high as the base of the cranium, in which the pupils became remarkably contracted. This condition appeared to be owing to interference with the sympathetic; but as the patient was taking calomel and opium, which might have affected the pupils, I am constrained to have some doubts about it, and therefore will not include it amongst this set of cases.

Judging from our knowledge of the connexions between the sympathetic and the spinal cord, and from the various experiments upon the consequences of injury to the spinal cord itself in animals, to which at an earlier part of this communication I alluded, I thought it probable that similar symptoms to those which we have seen to follow mechanical interference with the sympathetic in animals, might arise when the upper part of the spinal cord was suffering from disease or injury in man. I have not been disappointed in my search for cases related, in which exactly such symptoms
as I anticipated were exhibited. One or two such cases I will quote, as follows, merely selecting such details as bear on the point in question:

Case 12 was that of a man who was the subject of extravasation of blood into the substance of the spinal cord corresponding to the fifth and sixth cervical vertebrae. Both the pupils were noticed as becoming greatly contracted. The patient died in forty-eight hours after the attack, remaining quite sensible to the last, and showing no cerebral symptoms whatever. This case is related by Sir B. C. Brodie.1

Case 13 was one of fracture and dislocation of some of the lower cervical and dorsal vertebrae, owing to a fall from a haystack, and is also related as under the care of Sir B. C. Brodie.2 The patient, æt. 71, had oppressed breathing and noticeable contraction of both pupils. His senses were entire and collected, and he remained quite conscious to the last, gradually sinking. After death the centre of the medulla spinalis opposite to the injury was remarkable as being of a brownish-red colour, and highly congested.

It is not expressly said that the spinal cord was softened, but such a change in colour would most probably not have taken place without some amount of disorganization.

Case 14 was that of a man, æt. 45, the subject of fracture of the fourth and fifth cervical vertebrae, related by Mr. Vincent, of St. Bartholomew's Hospital.3 Diminished sensibility and power of motion existed in all the limbs, and there was priapism. The respiration was mainly diaphragmatic. Both of the pupils were noticed as being contracted. On post-mortem examination the spinal cord at the affected part was found much swollen and softened, and blood was extravasated outside the theca vertebralis.

Case 15 was that of a man, æt. 39, who by a fall had

1 Transactions of the Royal Medical and Chirurg. Society, vol. xx, p. 149.
3 Johnson's 'Medical Journal,' vol. viii, p. 452.
fractured one or two cervical vertebrae, and was brought into St. George's Hospital in 1849. On the day after admission he was found to have lost power in the right leg, and partially also in the left leg; and on this day it was noticed that both pupils were contracted, but especially the right one. No other mention is made of the eyeballs or pupils. The spinal cord, after death, was found to be considerably encroached upon and softened, both as to its anterior and posterior columns, the central part being very vascular. The brain was quite healthy.

Case 16 was that of a man, aged 48, who was admitted into St. George's Hospital in June, 1849, having fallen from a considerable height against a piece of timber. There was loss of sensibility and power of motion in the lower limbs, with numbness of the arms and diaphragmatic breathing.

In the evening of the day of admission it was noticed that the patient moved his arms less easily, and that the pupils of both eyes had become contracted. Two days after admission his pupils were observed as being very highly contracted, and great pain and numbness were complained of by the patient. No brain symptoms came on, and on the day of his death, which was the fourth after the accident, the patient spoke quite rationally to the nurse. After death, fracture of the lower cervical and upper dorsal vertebrae with displacement, was found, and much blood was seen extravasated within and around the cervical cord of the part; the entire circumference of the cord, from a point corresponding to the fifth cervical as far as the second dorsal vertebrae—almost exactly coinciding with Budge's "regio cilio-spinalis" (see reference at former part of the paper, p. 406)—being greatly softened. The cranium and its contents were quite natural.

In all the above cases quoted from other authorities, or newly related, the special symptom brought prominently forward, as resulting from a certain degree of interference with the sympathetic and its branches in the neck, or that
part of the spinal cord with which it is connected, was a contracted state of the pupil. (See Note A in Appendix.)

This pathological phenomenon in the human body quite accords with the contraction of the pupil produced by division, ligation, or extreme pressure of these parts of the nervous system, in experiments upon the lower animals.

But in the description of many of the experiments to which I have alluded (p. 408) it was stated that a certain amount of stimulus, mechanical, chemical, or galvanic, applied to the cervical sympathetic, would produce not a contraction, but a dilatation of the pupil, and moreover, that when contraction of the pupil and other symptoms had been already produced by section or extreme pressure of the nervous structures before described, they could be reversed, as it were, on the application of galvanism to that part of the nerve or spinal cord still holding unbroken connexion with the iris; and in the place of contraction, a dilated state of the pupil could be at once established. Galvanism, also, of the upper part of the spinal cord, which was otherwise whole and untouched, would produce the same dilated state of the pupil.

The knowledge of these facts helped me to account for certain pathological instances in which, under apparently similar circumstances to many of the cases which I have already enumerated, not a contracted state of the pupil was produced, as in them, but, on the contrary, a dilated state. This dilatation of the pupil in the human subject is analogous in its causation, I believe, to the dilatation produced in experiment by the application of certain stimuli to the sympathetic nerve. We know that if a stimulus applied to a motor nerve be carried only to a certain pitch, the nerve is irritated, and we have, as a result, contraction of the muscle to which the nerve is distributed; but if the stimulation be carried to an extreme, then, on the contrary, it produces paralysis of that muscle. (See Appendix, Note B.) In like manner, in the cases which I am about to describe, I would seek to explain the dilatation of the pupil, on the supposition that the pressure upon the sympathetic trunks and fila-
ments and the interference with the spinal cord were so slight, comparatively, that it merely acted as an irritant, just as gentle galvanism might have done, the sympathetic fibres to the dilator pupillae being stimulated. Should the pressure in such cases remain in so slight a degree, the dilatation of the pupil would also continue, but should it increase so as to paralyse the sympathetic or spinal fibres, then the action of the sphincter of the pupil would preponderate and contraction of the pupil ensue. The quickness with which contraction will follow dilatation of the pupil in such cases will depend most likely, in part, upon the general character as to susceptibility of the nervous system in any given person; but chiefly upon the exact position at which the pressure may be exercised, whether it be of an aneurism or other tumour; and also upon the rapidity of increase in size of the object effecting the pressure and its consistency. For example, it appears likely that the same growth situated low down in the thorax (as, for example, an aneurism in the lower part of the arch of the aorta) would not be nearly so harmful to the sympathetic as if it were close to the upper outlet of the chest, where the grand sympathetic trunks on either side are large, more prominent, and confined within a narrow bony circle, and where also they are more elaborate in their connexions.

Again, modifications as to interference with the sympathetic by aneurism or tumour, &c., might well be expected, according to the number and anatomical arrangement of the sympathetic fibres around the part affected, which may vary considerably in man, as we know they do in animals.

I will now proceed to give one or two cases which seem to illustrate the occurrence of dilatation of the pupil, owing to a slight amount only of pressure upon the sympa-

1 In connexion with the susceptibility of external impressions made upon portions of the nervous system, the fact ascertained experimentally by R. Miquel ('Archiv d. Vereins f. Gemeins. Arbeiten,' &c., 1853, Hgt. iii. p. 386) is interesting, that the results of pressure upon the sciatic and median nerve are more decided and persistent in proportion to the general exhaustion of the nervous system.
thetic, or to some slight change in the spinal cord, serving to stimulate and not to paralyse such fibres as pass eventually through the spinal nerves and sympathetic to the iris.

Case 17 was that of a woman, æt. 40, whom I watched closely in the wards of St. George's Hospital, in the year 1856. She was admitted with a pulsating tumour, of the size of a hen's egg, situated at the third intercostal space on the right side. There was a sharp diastolic click audible over both scapulae behind, and specially at the middle of the right one; and subsequently a systolic murmur was heard over the tumour, and dyspnœa, with cough, came on. Afterwards, the tumour was found to have appeared on the left of the sternum, and stridulous breathing occasionally occurred. The pupils were noticed as having become slightly dilated. Later on in the history of the case the "right pupil became still more increased in size, so as to contrast notably with the left one." Death took place shortly afterwards. On post-mortem examination, an aneurysm of the size of a cocoa-nut was found destroying a part of the right side of the sternum, and the contiguous portion of the third rib, and arising from the upper part of the ascending aorta, this vessel itself being also greatly dilated.

Case 18 was that of a man, æt. 57, who was brought into St. George's Hospital with extensive occupation of the left side of the neck by "scirrhous infiltration," the skin being tuberculated, indurated, and discoloured. He lay in a state of indifference, and it was noticed that the pupil of the left eye was dilated, that of the right one being natural. He sank, owing to dysphagia. In addition to the skin, most of the structures of the entire depth of the left side of the neck were found after death to be affected by the deposit, and there can be no doubt that the sympathetic filaments would fall in for their share of pressure, although but slight in degree.

Case 19 was one which lately I observed at St.
George's Hospital, of a young man affected by scarlet fever. He had ulcerated sore throat, and extensive enlargement of both sides of the neck, but particularly of the left side. The pupils of both eyes were fixed, but very dilated, the left one being much the more so. After death, the cervical lymphatic glands were found to be very greatly enlarged, and in one or two places suppurating; and most of the deep tissues, with vessels and nerves at the sides of the neck, were indurated, and matted together by inflammatory products. These changes were especially observable on the left side of the neck.

Case 20 was one which, by Dr. Sibson's courtesy, I saw under his care at St. Mary's Hospital. An abstract of the notes which he gave me is as follows:—Rosetta James, aged 42, was admitted Oct. 18th, 1857, with swelled and congested face and neck, distended jugular vein on the right side, and pulsation at the second intercostal space on the right side of the chest, with fulness and dulness of this region, extending from the edge of the sternum about one inch outwards. Other symptoms existed, leading to the diagnosis of an intra-thoracic aneurism. No mention exists of any peculiarity in the appearance of the pupil until December 11th, when it is stated that the left pupil was more dilated than the right. On the 12th February, it is stated that the impulse of the aneurism was stronger, and felt over an area of three inches from the second to the fourth costal cartilages; and that both pupils were dilated, but the left one much the most so. On exposing the eyes to the light of a candle, "both pupils contracted, but the left one still remained the largest." Attacks of giddiness were also complained of, and the patient felt at times "as if she were going out of her mind." On March 8th, it is recorded that "whilst asleep, the pupils were contracted, but the left one was the largest." "Both of them speedily dilated when she awoke."

Case 21 was one under the care of Dr. Cockle, who
kindly gave me the following abstract of it\(^1\):—The patient suffered from a very large aneurism of the ascending and transverse part of the aorta, as was found after death; and on dissection it was ascertained that, besides other structures, the sympathetic nerve on the left side was involved to a considerable extent. During life, it was observed that, in addition to pain, cough, dysphagia and dyspnœa, one of the pupils (unfortunately it is not particularised) was much larger than the other, the vision being at the same time dimmed. Moreover, before death a peculiar state of mortification was set up in the side of the nose, corresponding to the side chiefly affected by the aneurism. In the early part of the history, the pupils are described as being found contracted, and no difference between them was observed. It may therefore be concluded that one of them became altered during the course of the disease, and whilst the patient was under notice. The aneurism, still showing distinctly the implication of the main sympathetic branch, is now in the museum of St. Mary’s Hospital, where I had the opportunity of inspecting it.

Case 22 was that of J. M., aged 57, who was brought into St. George’s Hospital May 30th, 1849, with infiltration of most of the textures of the left side of the neck, and the cervical glands with carcinoma. On admission he was in a state of half stupor, caused, as it was thought at the time, by pressure upon the cervical blood-vessels. It was noticed, that the pupil of the left eye was considerably dilated, whilst that of the other one was natural. Pressure by the hand on the right side of the neck induced syncope.

On \textit{post-mortem} examination the external jugular vein was found to be obliterated by coagulum, and the carotid artery, internal jugular vein, and pneumogastric nerve, on the left side, were found surrounded by the cancerous growth, so that it can hardly be conceived that the sympa-

\(^1\) This case will be seen related in vol. ix of the \textit{Pathological Society’s Transactions}.\^
thetic trunk behind could altogether escape some pressure which might yet not suffice to paralyse it.

Case 23 was that of P. O. H—, æt. 61, who was brought into St. George's Hospital September 21st, 1857, having fallen down backwards in the act of lifting a heavy load of hay. He was at the time collapsed, and suffering pain at the upper part of the dorsal region, where the spinous processes appeared more than usually prominent. He had lost all power of movement in both lower and upper limbs; and it was noticed, when he was admitted, that both pupils were unusually dilated, but that the pupil of the right eye was more dilated than the left. On a further observation, the same state of the pupil was noticed. The patient died in eight days, and, on post-mortem examination, "minute points of ecchymosis were seen in the substance of the spinal cord, at the lower part of the cervical region, which was also softer than in other parts," and it was noticed that the "white matter had, in parts, a pinkish hue."

The last five cases show simply a dilated state of the pupil, owing, I believe, to a comparatively slight interference with the cervical sympathetic, or that part of the spinal cord with which it is connected, no further change in the pupil being observed. It is presumable, that this dilation, should the pressure have been increased, would have been exchanged for contraction, on the supposition which I have previously advanced.

The next case which I will adduce is a very remarkable one, inasmuch as a dilatation and contraction of the pupil were noticed as succeeding each other in a rapid and somewhat inexplicable manner. The phenomena were, I think, clearly traceable to the extraordinary condition of the sub-fascial tissues of the neck.

Case 24 was that of a German lady in Dublin, æt. 25, who, in August, 1856, suffered from phlegmonous in-
flammation in the neck. In consequence, a tumour formed on the right side of the neck, extending from the ear to the lower border of the thyroïd cartilage, and seated beneath the cervical fascia. Great pain existed, and suppuration came on, marked by repeated rigors. It was now observed that the pupil of the right eye was greatly dilated, so much so, indeed, that but a very little of the iris was to be seen. Soon after this dilatation of the pupil was noticed, the patient got relief from the pain and slept, and, on awaking, the pupil hitherto so much dilated, was found to be of the natural size. In the evening a rigor again came on, and the pupil was now contracted; a paroxysm of pain followed the rigor, and the pupil became dilated as before. These alterations in the size of the pupil were observed several times; and, moreover, during this time, the iris, which naturally was of a gray colour, acquired a dark brown tinge.

The abscess in the neck was now opened, the rigors ceased, and the pupil remained dilated, but would contract under the stimulus of light, the power of vision being apparently entire. As the state of the neck improved, the affected pupil returned to its natural size. In the summer of 1857, whilst in London, this lady again suffered from an abscess in the same region, and is reported to have had a similar return of the affection of the pupil. In 1858 another abscess formed on the same side of the neck, but at a lower part. The pupil became again dilated, and so continued until the abscess was nearly healed. The dilatation was not, however, nearly so extreme as in the earlier attack.

For the history of the above case I have to thank Dr. Kidd, of Great Brunswick Street, Dublin, who, in a most generous way, has placed it, hitherto unrecorded, at my service.

For the general effect of dilatation of the pupil, as resulting from the stimulus of a moderate pressure, analogous to that produced by galvanism, we are in a measure prepared

1 This change of colour was no doubt the result of an alteration in the amount of blood in the capillary vessels of the iris, and was due to interference with the vaso-motor nerves of these capillaries.
The influence of the sympathetic

by a knowledge of the frequency of similar dilatation in cases of mere irritation, as by worms, of the intestinal tract whose innervation is from the sympathetic;¹ as also by the frequency of such dilatation in cases of burns, scalds, lacerations, and other mechanical injuries, physiologically comparable to the influence of the sympathetic on the heart causing palpitation, &c., as in certain diseases of the abdominal viscera; as also in cases of general debility, when we know the cerebral influences to be impaired, and, by comparison, the sympathetic and spinal ones to be intensified.

Hitherto, all the cases and observations brought forward have had reference simply to some altered state, a dilatation or contraction of the pupil, phenomena parallel to similar ones, induced in the eyes of the lower animals by direct experiment. This altered condition of the pupil in disease resembles that established in animals by section of the sympathetic, &c., inasmuch as the dilatation or contraction is not absolute and complete, but may still be modified by the action of light, atropine, &c. Thus, in Case 20 the pupils are mentioned as both of them contracting, although the left one remained still the largest, and, in the same case they were contracted whilst the patient was asleep, but dilated on her awaking. Again, the same response to the impression of light was exhibited in Cases 7 and 24; and to that of atropine in Case 8; and the same effect will be seen hereafter, as in Case 25, to have followed the application of belladonna.

So also in the lower animals. Dr. Reid observed that in a dog, whose sympathetic and vagus nerves he had completely divided (second experiment), the contracted pupil still continued to be affected by light; and when the dog was dying, poisoned by prussic acid, the pupils of both eyes became equal, and very much dilated. Mollinelli² also, in his third observation, speaks of the pupil being made to contract still further, even when already contracted by division of the sympathetic.

¹ Dr. Waterfield has lately informed me of a case in which almost total blindness was apparently produced by the presence of a tape-worm, and at once remedied by its expulsion.

In one or two of the cases which I have watched, I have found that the contraction or dilatation of the pupil varied in a remarkable and unaccountable way at various times; and it is interesting, in connexion with this fact, to remember, that in certain cases of disease of the brain, rapid and well-marked variations in the size of the pupils are wont to occur at times most unexpectedly. It may here be called to mind, that in dilatation of the pupil from paralysis of the oculo-motor nerve, as by a tumour pressing upon it, and consequent want of contractile power of the sphincter of the iris, there is, of course, no change in the pupil on the increased admission of light upon the eye; and this is the case also, I believe, in complete dilatation of the pupil from the use of belladonna, as resorted to for the purposes of couching.

It will be remembered, that I pointed out other symptoms besides the contracted pupil, as following experimental interference with the cervical sympathetic or spinal cord in animals, and these were chiefly ptosis and convergent strabismus. These phenomena I believe may be accounted for rationally, and on anatomical grounds. (See Appendix, Note C.) I thought it, therefore, probable, and was very wishful, that I should find instances in which either of these symptoms had been associated and noticed in man, along with a contraction of the pupil, owing to interference with the sympathetic. Such a combination of circumstances I find in the case which I before mentioned, page 415, as being alluded to by Professor Reid, and more fully described by Mr. Hare, house-surgeon at the general infirmary at Stafford.¹ This was not a case in which the sympathetic nerve was affected by an aneurism, but by a malignant tumour; and, as Reid only spoke of it as respects the contracted pupil, I will here give a general summary of the particulars.

Case 25, was that of a man, æt. 40, who was attacked by

¹ 'Medical Gazette,' September 29, 1838, p. 16.
pain down the left arm and shoulders, across the chest, and up to the left eye and side of the face. A tumour was observed in the inferior triangular space of the left side of the neck, resembling an enlarged and serofulous gland. The left pupil was contracted, and the left eye "partially closed," but no inflammation of the conjunctiva seems to have been observed.

After a time weakness, and eventually paralysis, of sensation and motion in the lower limbs came on, followed by trismus and dysphagia, and death by suffocation, the growth in the neck having become much larger, and very hard. The contracted pupil could be dilated on the application of belladonna around the orbit. After death the tumour was found to be of a scirrhouus nature. It had implicated the large vessels and the spinal nerves with the great sympathetic at the lower part of the neck, the lower cervical ganglion of the sympathetic, the thoracic duct, and the recurrent laryngeal nerve. It was also found lying on the brachial plexus; and having penetrated the foramina, by which the last cervical and the first dorsal nerves escape from the spinal canal, it had become attached to the external surface of the spinal dura mater. The ptosis, the trismus, and the contracted pupil, caused the observers of this case to anticipate some disease of the central nervous structures; but no disease whatever, either of the brain or spinal cord was found, and the above-mentioned symptoms were spoken of as having been essentially sympathetic in character. Whatever may be the interpretation of the trismus, dysphagia, &c., there is, in my mind, no doubt of the connexion between the ptosis, as well as the contracted pupil on the one hand, and the interference, by pressure, with the cervical nervous structures on the other hand.

One or two more cases I will quote, illustrative of a more or less complete paralysis of the upper eyelid, along with some interference with the sympathetic nervous structures.

Case 26. Was that of a man, set. 50, under Sir Astley Cooper's care, affected by aneurism of the internal carotid
 ON THE EYE AND ITS APPENDAGES. 435

artery, which produced distressing throbbing pain in the left side of the head, and giddiness, with almost total loss of consciousness and sight, on stooping; also cough, dyspnœa, and hoarseness.¹

It was noticed that ptosis of the eyelid on the affected side "gradually" came on until the eye was fully half closed, the sight of the eye remaining good, and no other special symptoms arising.

The common carotid was tied, with absolute success, and the peculiar pain in the head was quickly relieved, but unfortunately no history of the state of the eyelids after the operation has been recorded, although I think we may very fairly and reasonably conclude that the eyelid resumed its natural position, as the patient is reported to have become in all respects perfectly well.

Of course, the importance of the ptosis was not at the time recognised, or it would have received special attention. There is no mention of the condition of the eye-ball or pupil during the whole course of this case, but it was, however, noticed that the ear of the patient on the side implicated, was frequently affected by a feeling of coldness, succeeded by heat.

I consider this case to be of special interest, as showing the gradual accession of paralysis of the levator of the eyelid, as the aneurism of the carotid increased.

CASE 27 related by Mr. Butcher, of Dublin,² was that of a man, æt. 56, affected by extensive carcinoma of the glands of the axilla, and of the neck, on the left side, displacing the trachea, and compressing the œsophagus. Dyspnœa and dysphagia came on, and by degrees, indeed almost imperceptibly, a drooping of the upper eyelid on the affected side, which Mr. Butcher states that he watched for many days. On being much roused or spoken to, the eyelid was tardily raised and kept open. In a letter, dated December 7th, 1856, Mr. Butcher informed me that he had not

¹ 'Transactions of the Royal Medical and Chirurg. Society,' vol. i, p. 22.
² 'Dublin Medical Journal,' November, 1856, p. 239.
perceived that the action of the iris or the axis of the eye on the side affected had in any way differed in this case from those on the other side.

With regard to this case, it might be asked how the ptosis can be explained, whilst, at the same time, no affection of the pupil was recorded? In reply, it might be stated that possibly the pupil was affected, although it might be so only to a slight extent. An amount of difference in the pupils, even considerable, often passes unnoticed, unless specially looked for, whilst ptosis is very obvious.

It is to be remarked that, in none of these cases have we any strabismus noted, such as existed in several of the experiments on animals, illustrating the effects of interference with the sympathetic, &c.: but the possibility of the occurrence of strabismus, as a clinical result of such interference, is well illustrated by the cases mentioned by Sir C. Bell, in which\(^1\) squinting in children was produced by over-eating. This squint was at first temporary, and only observed on occasions of distended stomach; but at times became confirmed.

Such are some of the cases, in which, along with injury to the cervical sympathetic, or cervical part of the spinal cord in man, a more or less complete condition of ptosis has been noticed, suggesting a comparison with those experiments on animals, in which the same symptom was produced by section of or extreme pressure on, the sympathetic.\(^2\)

\(^1\) 'The Nervous System of the Body, with an Appendix of Cases and Consultations,' 1844, 3d edit., p. 389.

\(^2\) In the case of carcinoma within the thorax and neck, before alluded to (p. 412), as being described by Dr. Robert MacDonnell, of Montreal, there was in addition to a contracted state of the pupil, ptosis of the upper eyelid and frequent epistaxis from the nostril on the side affected by the malignant growth. The contracted pupil (as in Case 20, and others before mentioned) nevertheless answered to the influence of light. In his remarks on this case, the author alludes to the connexion between the sympathetic and the third nerve as the probable cause of ptosis; and this conjecture, made so completely apart from, and independent of my own solution of the subject, I look upon as being of essential service in support of my view in question.
ON THE EYE AND ITS APPENDAGES.

I have described, as briefly as I could, all the pathological facts which, at this opportunity, I have thought fitting or necessary to bring forward, as indicating the results upon the eye, and its appendages, of interference with the sympathetic thereto distributed, as occurring in the neck, or of injury to that part of the spinal cord with which the sympathetic branches or ganglia are connected.

Of course I have been careful to exclude cases in which the pupils might become affected by other causes, and which we ought to estimate before deciding upon the clinical importance of a contracted or dilated pupil in any given case of supposed pressure upon the sympathetic or its tributaries.

Setting aside those cases in which disease or injury of the brain, intra-cranial irritation or effusion, uremia and various poisonings are accompanied by an irregular condition of the pupil, we must not overlook the effects of such remedies as are well known to influence the pupil, and by which one might easily be deceived. In some people, also, a contracted or dilated state of the pupil is habitual, but it is very rare, although such cases do exist, to find that the pupil of one eye is contracted or dilated without the other being likewise so, unless some previous disease or injury have caused this difference.

We may also be misled by the fact of the pupil having been injured by mechanical or inflammatory lesions of the eye, or by alterations in its size, in adaptation to cataract, opacity of the lens, &c. Of course, all doubt of such complications becomes out of the question, almost to a certainty, when the progress of alterations in the pupil is actually watched at the bed-side, as it was in several cases which I have just adduced.

It is out of the scope of this paper to dwell at length upon the subject, but I cannot close it without alluding to the possibility of alterations in the size of the pupils as a result of injury to other and more distant parts of the sympathetic system than those immediately in the cervical or thoracic regions. This appears to be not improbable, from many facts. For instance, the temperature of the entire body
was found by Bernard to be affected by injury to the sympathetic in the neck; and Arnemann mentions persistent diarrhoea as a result of wounding of this nerve. Moreover, Budge found that galvanism of the posterior part of the lower extremity of the spinal cord produced a very striking increase in the number of the heart's contractions, a circumstance coinciding with those cases of disease in which pressure upon various parts of the back produces syncope and palpitation of the heart, &c. Romberg speaks also of tabes dorsalis, an affection strictly of the spinal cord, and generally of its lumbar portion, as being accompanied at times by a contracted state of one or both pupils; and recently, also, Brown-Séquard has stated that injury and removal of the supra-renal capsules pretty often produce, amongst other effects, decided contraction of the pupil of the eye on the side corresponding to that on which the operation is performed; a fact no doubt due to implication of the sympathetic connected so freely with these small viscera.

May it not be that the contraction of the pupil, accompanying lesions of certain parts of the brain also, may exist by virtue of injury to such nervous strands as pass between those portions of the brain and such parts of the spinal cord as are (according to our knowledge obtained experimentally,) central points, as it were, to branches joining the cervical sympathetic. Some encouragement to this view is afforded by the fact, that diseases of cranial centres appear to induce disease of spinal centres, by depriving them, as it were, of a wonted impulse, thus throwing them more or less out of activity. And Türck has shown how secondary changes, as seen by the microscope, may be produced in the spinal cord, merely by intra-cranial causes, as in certain cases of hemiplegia.

It may be, and this idea may here be suggested with propriety, that the pain in the head and eyes, the giddiness, unusual sensations, deafness, double sight, dimness of sight,

1 'Mémoires de la Société de Biologie,' 1855.
2 'Archives générales,' vol. ii, 1856, p. 587.
3 'Weiner Zeitsch,' vol. vi, 1860, p. 6, and vol. viii, 1862, Bd. ii., p. 511.
or even blindness, which are so very often coincident with thoracic aneurisms, most frequently existing on the same side, and which are independent of any visible cerebral change after death, are owing to some interference with sympathetic nerve-influence to the intra-cranial capillaries. In accordance with the above suggestions is the fact that, in case 26, that of Sir A. Cooper, the peculiar pain in the head, which had existed along with the carotid aneurism, was quickly relieved by the ligation of the vessel, and consequent diminution of the aneurism. The relief which followed the removal of the pressure upon the sympathetic, as it appears to me, yielded sufficient evidence that the cerebral distress was not dependent upon any inherent disease of the blood-vessels, apart from their connexion with the sympathetic, accredited to them, and to which they are subordinate.

Before closing this communication, I would draw attention to a point which, connected as the subject is with several of the experiments before quoted, naturally presents itself on consideration of the cases which I have just detailed. The question might well be put, whether, during observation of any of those clinical cases, any undue heat or sensibility of the skin, or any visible altered vascular action in the eye or elsewhere, ensued from pressure exercised upon the sympathetic? The only clear indication of this appears in case 3, in which it will be remembered that flushings following cold sweats were noticed, exactly limited to that half of the face on which the altered state of the pupil existed. We have, however, something of the kind also, I think, in the 21st case, in which an unaccountable "mortification" of the integument of the nose took place on the side corresponding to that of the aneurism, an effect no doubt due to the interception of such nervous influence as presides over the capillaries of this portion of the face.¹

Just as in the experiments on the lower animals, the vessels

¹ In Case 26, also, that in which ptosis was noticed in connexion with a carotid aneurism, it is stated that, on the side affected, the patient's ear was frequently affected by a feeling of coldness succeeded by heat, phenomena most likely due to sympathetic disturbance.
of the ear become affected, do I infer that the vessels of the retina become influenced; its tolerance of light being subverted in those cases of sympathetic interference wherein a contraction of the pupil is caused in man. But of this I have no distinct proof, as the parts are so removed from view.

In all observations laying claim to accuracy, upon such cases as have been alluded to in the foregoing pages, it will in future, be necessary to notice, as carefully as possible, by instrumental means, the temperature of the various parts of the body, especially the face and head; but, although we may anticipate deviations from the natural state of temperature in many cases, especially if very fine and delicate instruments be used, yet it must not be expected that such alterations will be in all cases manifested, especially if any considerable time have elapsed since the commencement of sympathetic interference. Of course we may well foresee, that in this respect, the effect of suddenly cutting off the influence of the sympathetic by section, in the lower animals, would differ strikingly from the results obtained by such gradual pressure as would generally be exercised by an aneurism or growing tumour in man. Moreover, in a much higher degree would anatomical differences between man and the lower animals be likely to account for variety in results; but this variety in results might exist without at all affecting the validity of parallelism between the class of pathological phenomena above examined, and the experiments quoted. Even in dogs experimented upon, Valentin found that the effects upon the iris of dividing the sympathetic, varied considerably in extent according to the colour of this muscular curtain. Schiff also states, that in the rabbit, a manifest difference regarding the modifications of temperature in the ears, as produced by sections of the sympathetic exists, according as the rabbit be of the long or short-eared variety.

Such are the applications theoretical and practical, which I have ventured to make of the experiments entered upon by several observers, with the view of determining the
effects of injury to the sympathetic upon the pupil and certain appendages of the eye. I have not sought to establish the dictum, that either contraction or dilatation of the pupil is to be considered as pathognomonic, or as a test of intra-thoracic aneurism, cancer, or other tumour, or of any specified disease whatever. On the contrary, I have carefully examined several cases both of aneurism and other causes of pressure in the thorax and in the neck, and I confess that I have found several in which no unnatural condition whatever of the pupil was to be noticed. All I have desired to do, is to establish the fact, that alterations in the movements of the iris and other phenomena connected with the eye may be produced by, and become symptoms of pressure, exercised in various ways on certain parts of the sympathetic, or by pressure upon, or structural alterations of those portions of the spinal cord with which the sympathetic is connected. That these changes do not always accompany the various phenomena above alluded to, is only to say, that they are not invariable criteria, just as vomiting, for instance, or pain in the head, are not the necessary attendants of cerebral disease; but yet they are so frequent as to merit the character and denomination of symptoms thereof, and to have become a means of diagnosis. I will not now occupy time by inquiring why these pupil symptoms are variable, appearing in some cases, and not in others. Most likely the solution is to be met with in the anatomical connexions of the nerves about the cause of pressure, the character of the growth, and the rate of rapidity of its formation, and in the stage of the disease,¹ as well as upon other circumstances yet to be made out.

In these observations, it has been my endeavour to render experimental physiology subservient to practical medicine, to give what assistance was in my power, towards the edification of pathological science upon such a foundation as alone is legitimate and truly scientific. If it shall be

¹ It will be remembered that in the history of one or two of the cases before detailed, the amount of alteration in the pupil varied at different periods of observation.
thought that I have in any degree succeeded in enriching or enlarging the boundaries of a field which the labours of others had in their degree cleared, I shall have reason to congratulate myself, in that my efforts will not have been fruitless.

APPENDIX.

Note A. (See page 421.)—I have no doubt, that if I had had the requisite time to scrutinise all the cases recorded in the various periodicals, or, if it had been desirable to multiply examples, I should have found several other cases in which some affection of the pupil had been noted, though, at the time, not understood, in connexion with aneurisms, tumours, injuries, &c., about the neck, so situated as to interfere with the sympathetic and the cervical region of the spinal cord. I do not, however, think that a very great number of cases would be so met with, having this implication of the pupil, seeing, indeed, how little attention is ordinarily paid at the bed-side to the exact state of the eye, except in cases of disease of the brain. Unless specially looked for, the condition of the pupil has been, hitherto, in a marvellous manner disregarded in disease, even in disease of the brain itself, and, in support of this observation, I cannot resist stating, that it was not until the year 1818 that any positive notice was taken by the profession of the poisoning by opium being attended by any alteration of the pupil.¹ The first distinct report of this fact was by Dr. Kinnis, who spoke of the pupils “being fixed” “and contracted to the size of a pin’s head,” when relating a case of recovery from the effects of opium.² Now, considering how many observers, long before that period, must have watched the results of poisonous doses of opium, is it not a little curious that none had described either contraction, or what sometimes occurs, dilatation of the pupil in connexion therewith? I have looked in vain

¹ See the statements by Dr. Sibson in the 'Medical Gazette,' 1848, p. 268.
² 'Edinburgh Medical Journal,' 1818, p. 603.
through the writings of several authors who wrote before the period above mentioned, for evidence of any knowledge of a relationship between the phenomenon in question and opium-poisoning; and even for some years subsequent to the case related by Dr. Kinnis, I find no mention of the fact in the writings of various medical men who wrote on practical subjects.

**Note B.** (See page 421.)—The fact of spasms of a muscle preceding a condition of paralysis, or *vice versa*, is often illustrated in cases of disease where pressure is exerted on the nerves of the extremities. We also have experience of it in the pupils of the eyes, as when a contracted pupil precedes dilatation, consequent upon pressure, as by a tumour upon the third cranial nerve. And a notable instance exists in the spasm of the facial muscles, which not infrequently precedes facial paralysis of the same side—a state which may also follow the paralysis, when the muscles and nerves are returning to their natural condition.

**Note C.** (See page 429.)—It will be remembered, that in man the cervical sympathetic trunks on both sides, whose connexion with the spinal nerves has been previously alluded to, send up processes (varying in number, according to the statement of Weber, even in the same animals) which, surrounding the internal carotid artery whilst in the carotid canal of the temporal bone and before its division within the cranium, form a large network, the carotid and cavernous plexus. From this plexus, besides sundry branches to other parts, which are not to my present purpose, we find communications joining the third and the sixth pair of cranial nerves, as well as the ophthalmic or lenticular ganglion. The branch to the ganglion is of course the medium of innervation to the dilator of the pupil. The communications between the plexus and the sixth pair of nerves in man has been found by Valentin, to be effected by several important branches. Cuvier, who made observations on the calf, sheep, wolf, and porcupine, speaks of this communication as consisting of "a number of filaments;" but, in some animals, accord-
ing to Weber, the branches to the sixth pair are entirely absent, and this may be the case at times in man also.

In man, the communication between the sympathetic plexus and the third cranial pair, is ordinarily effected by two or three branches which join this nerve just before its division into its two major branches, and consequently before the subdivision is given off, destined to the elevator of the upper eyelid and superior rectus.

All these branches, i. e., the one to the lenticular ganglion, that to the external rectus or abducent muscle, and that to the third pair, will be no doubt affected in a similar manner, though possibly in a different degree, by section of, or extreme pressure upon the sympathetic main branches or trunks in the neck, &c. Just as we get paralysis of the dilator of the pupil, and consequently contracted pupil, produced by virtue of the branch of the carotid plexus, given off to the ophthalmic ganglion, so have we more or less paralysis of the abducent muscle, by virtue of the twigs furnished to the sixth pair, which supplies that muscle, allowing thus of convergent squint; and more or less paralysis of the elevator of the upper eyelid by virtue of the twigs to the third pair, producing ptosis, the orbicularis palpebrarum still acting and overcoming that muscle. Possibly, in some cases, more or less of the strabismus may also be caused by virtue of an implication of sympathetic twigs distributed to other branches of the third and the fourth pairs, from which nerves the oblique muscles are supplied.

We have other instances in the human body, besides the external rectus and levator palpebræ, of a single muscle being supplied by different nerves, of which good illustrations are found in the buccinator, the muscles at the angles of the mouth, and the sterno-cleido-mastoid muscles; and, since in such a case both sets of nervous branches must operate upon the muscular fibre, it cannot be doubted, that in proportion as either nervous source becomes weakened, the muscle loses power, and so becomes overbalanced in action by any antagonising muscles. It is most probable also that the sympathetic twigs distributed to the muscle, or joining the nerves of the eyeball, are only concerned in involuntary and consensual movements.
AN ACCOUNT OF A CASE OF CALCULUS IN THE BLADDER REMOVED BY LITHOTRTITY, IN WHICH A COMMUNICATION EXISTED BETWEEN THE BLADDER AND INTESTINE.

BY CHARLES HAWKINS, Fellow of the Royal College of Surgeons; Vice-President of the Society; Consulting Surgeon to Queen Charlotte's Hospital; Inspector of Anatomy, etc.

Received May 11th, 1858.—Read June 9th, 1858.

I have ventured to bring the following case before the Royal Medical and Chirurgical Society because I believe it to be in some respects peculiar, and the operation having been followed by a successful result, it may induce those who may meet with similar cases to have recourse to the same means of giving relief. As far as I know, lithotrity has not been performed under like circumstances.

On July 14th, 1857, Sir Benjamin Brodie and myself were consulted by a gentleman, aged 55, suffering from all the symptoms of stone in the bladder in a most aggravated form. He was much worn by a constant desire to pass water, accompanied with very great pain; the urine was alkaline, depositing much ropy mucus; his pulse was quick, and his appe-
tite bad. His bladder was examined, and a stone readily detected.

The following history of his case is in his own words:

"It was in February 1853, I first discovered a substance about two inches in length, which I had passed with my water; it had a most offensive smell. I said nothing about it, although I was passing it every day, because it gave me no pain or inconvenience, until June in the same year, when I showed some of the matter to my ordinary medical attendant, who would scarcely believe that it came with my water. He ordered me some medicine, which in no way diminished the quantity I passed. I consulted the same gentleman again in the following November, when he felt satisfied that what I passed with my water was fecal matter, and he told me that there must be an opening from the bladder into the bowel, he gave me little hope of being able to give me any relief. During the year 1856, I ceased to pass any of the substance, but early in this year the symptoms of disease of the bladder set in, and continued with great severity during the whole year. In the beginning of the year 1857, I saw two other surgeons in consultation, when an instrument was passed into the urethra, and one into the rectum at the same time. I suffered much pain from this examination, and afterwards passed blood from the rectum. These gentlemen came to the same conclusion as my own medical man, that an opening existed between the bladder and bowel; they both gave a most unfavorable opinion of my case, and thought that nothing could be done for my relief. It was not until I saw Sir B. Brodie and you that stone in the bladder was detected."

It was decided that, notwithstanding the history of the case, an attempt should be made to remove the stone, as the patient was sinking from the mischief it was causing in the bladder. But his general health was so much impaired and the absence of continued sleep for now upwards of a year had rendered him so very nervous, that I thought it unwise to commence any operation until I had made an attempt to
improve his state, and allay the irritability of his bladder. I advised him to return into the country, and to take some quinine and acid, and introduce into the rectum every night an opium suppository. He derived some benefit from this treatment, and the bladder-symptoms were a little abated. He returned to London on July 20th; on the 25th, I performed the operation of lithotrity. The patient was anxious that he should be put under the influence of chloroform, and Dr. Snow administered it. I may state, in passing, that it is not my usual practice to have recourse to chloroform in lithotrity; I do so occasionally, on account of some special circumstances, but it is the exception, not the rule, in my practice. It is unnecessary to enter into my reasons for this on the present occasion, as I hope I may at some future period be permitted to bring before the Society my experience in this operation. The bladder held comfortably five or six ounces of water, a stone was readily seized, which was so large that the lithotrite could barely be opened sufficiently wide to grasp it; it was very soft, and gave way under the pressure of the finger. The patient bore the operation remarkably well, and soon began to pass fragments of triple phosphate; his bladder-symptoms were much relieved, and he continued to pass stone without any inconvenience till the 29th.

On the 31st, I again operated (Dr. Snow giving chloroform); the fragments continued to pass easily, and he now slept for upwards of an hour without being disturbed.

On August 5th, I repeated the operation, and at the patient's request, without chloroform. He had found on the previous occasions that it was a long time before he recovered from its effects, remaining in a confused and an uncomfortable state of feeling for upwards of twelve hours. The operation was borne quite as well as it had been when chloroform had been used; the bladder held more water. I was able to do as much, and the patient complained of scarcely any pain, the fragments passed as easily as after the former operations, and all his symptoms continued to abate.

On the 13th, I operated again, with like success. On visiting him the next morning, I found him much depressed,
with a quick and feeble pulse, cold skin, and some drowsiness. He had not been able to pass any water in the night and unfortunately had not sent for me; but he said that early in the morning his bowels had acted, since which he had been easy. Upon examining what had been passed by the bowels, I found a large quantity of urine mixed with a considerable quantity of blood: it was evident that some obstruction to the passage of the urine by the natural way had occurred that could not be overcome, and that the bladder had given way, I concluded, where the old opening had existed. I prescribed astringents, and ordered lumps of ice to be introduced into the rectum, and confined his diet to cold drinks. Mr. Cæsar Hawkins, who met me in consultation on this day, concurred in this treatment; it was continued for two or three days. Neither at this time, nor at any other when I examined the rectum with the finger, could I detect any opening into the bladder. For about twenty-four hours all the urine came by the rectum, it was then passed by the urethra, untinged with blood: and in forty-eight hours no blood was passed by the rectum. In four or five days he was quite as well as he was before the last operation, but he continued to pass some urine through the rectum.

Not considering it advisable to continue any operative proceedings at this time, he left London on the 20th, without any further examination of the bladder being made; his general health had much improved and the bladder-symptoms abated. I prescribed decoction of pareira brava and hyoscyamus.

The symptoms of stone however continuing, he returned to London on October 5th. On the 7th, I examined the bladder, which held six ounces of water well; stone was readily detected, which I crushed; he bore the operation remarkably well. On the 21st, all the symptoms of stone were gone. I examined the bladder, and could not detect any; he was anxious to return home on business, and left London the next day. Towards the end of the year the symptoms of stone returned. On January 25th, 1858, he came to London again in excellent general health, but with unmistakable symptoms of stone in the bladder.
On the 27th, I examined the bladder, at once seized a stone, and crushed it. From the fragments he passed I believed it to be a new formation.

On the 30th, I repeated the operation.

On February 2d, I examined the bladder, and could not detect any stone. The urine was quite healthy. He neither suffered pain nor inconvenience, slept nearly all night, and the next day he left London with directions to pass a gum catheter, and to well wash out the bladder with warm water daily, so as to prevent, if possible, fecal matter lodging in the bladder, and again forming a nucleus for stone, as he had been passing fecal matter with his water, from time to time, for the last three months, and on the last two occasions of my operating, fecal matter passed through the catheter when I injected the bladder. I had given him the same directions when he left London before, but circumstances had prevented his following them, hence the new formation of stone.

Since his return home the patient has remained perfectly well. A few days ago I received the following note from him:

"I am very happy in being able to inform you that I continue quite well; free from all pain. I am better than I have been for years. I pass my water very freely. I use the instrument every night. I have discovered once a considerable quantity of feces; once a small quantity; but at other times my water has been quite clear; never any blood." He ends his note with this laudable expression of gratitude for what surgery has done for him: "when I am dead, if my bladder or any other portion of my body will be of any use for the benefit of my fellow-creatures, it is at your service."

It is difficult to give an idea by weight of the size of the stone removed, as it consisted entirely of triple phosphate; but if all the detritus had been collected it would have filled a three or four ounce bottle.—
Dr. Ogle, of St. George's Hospital, who kindly examined it for me, gives the following account of it: "it was formed of the triple phosphate, having as a nucleus a number of little foliaceous-looking masses, of about one eighth to one fourth of an inch in size; after the addition of a little acid they were quite obviously seen to be vegetable in character, presenting numbers of vegetable cells in a good state of preservation."

From the foregoing history it is evident that at some period ulceration must have taken place in the bladder or bowel, most likely, in the first instance, in the latter, resulting in the communication, which existed previous to the symptoms of stone showing themselves; and as no fecal matter passed with the urine for upwards of a year previous to the operation, it may be concluded that the opening had closed, and continued so until the occasion of the retention of urine, in the night of August 12th, when it was again opened.

It is not a little singular that so much mischief should have taken place, resulting in a communication between the bladder and intestine, occasioning so little inconvenience to the patient, for until he perceived the fecal matter in his urine he experienced no inconvenience of any kind with reference to those parts.

Notwithstanding all that had taken place, and the very great irritability of the bladder that existed in this case, and I scarcely ever saw greater suffering, I never had a patient that was so little distressed by the operation, or passed through all its stages more satisfactorily, until the unfortunate occurrence of the 13th of August; and it is surprising that after the communication with the bowel again existed, the bladder was still able to retain between six and eight ounces of water, and bear without any ill effect the presence of the instrument, and the manipulations necessary for seizing and crushing the stone.
A CASE
OF
DISLOCATION OF THE HUMERUS,
UPWARDS AND INWARDS,
WITH FRACTURE OF THE CORACOID PROCESS OF THE
SCAPULA;
ACCOMPANIED BY A DISSECTION OF THE PARTS INVOLVED IN THE INJURY.

BY

T. HOLMES, M.A. CANTAB., F.R.C.S.,
CURATOR OF ST. GEORGE'S HOSPITAL MUSEUM.

'Received June 8th, 1855.—Read June 23d, 1858.

Dislocations of the shoulder are described in all English works on surgery with which I am acquainted as occurring in three directions only, viz., downwards into the axilla, inwards beneath the clavicle, and backwards on to the dorsum scapulae. Besides these complete dislocations, another partial dislocation upwards is admitted, on the authority of two cases communicated to this Society by Mr. Soden, in the year 1841, in which, the long tendon of the biceps being thrown out of its groove, the head of the bone rests on the edge of the glenoid cavity, touching the lower border of the clavicle and acromion process.
DISLOCATION OF THE HUMERUS.

The dislocation which I am now about to describe differs in some respects from any of these forms of injury; and as it has, I believe, never been dissected, and the numerous preparations in our London Museums do not show anything like it, it may be interesting to the members of the Society to examine the preparation and compare it with the account which I shall give of it.

The case was as follows:

John B—, aged 50, was admitted into St. George's Hospital, on April 7th, 1858, under the care of Mr. Tatum. He had fallen from a great height (said to be above thirty feet) upon a heap of stones, striking the head, the left side of the chest, and the left elbow. He was brought in, suffering from symptoms of concussion of the brain. On examining the left arm, the elbow joint was seen to be extensively laid open, the end of the ulna being broken into numerous pieces, several of which were afterwards removed. There was a large prominence of bone in front of the outer part of the clavicle, which had a rounded form, and involved the tuberosities of the humerus, and seemed to consist of the whole head of the bone. There was, however, crepitus on moving the arm, which made some persons suspect a fracture about the upper part of the humerus. The arm appeared to be shortened, and the elbow was directed away from the side. Endeavours were made to replace the humerus, but it was evident that chloroform would be necessary for that purpose. The arm was, therefore, put upon a bent splint for the present.

Considering the form of the displaced bone, the obstacle which existed to reduction, and the crepitus (which seemed external to the humerus), Mr. Tatum came to the conclusion that the head of the bone was dislocated, and that the crepitus arose from fracture of the coracoid process, a diagnosis which was confirmed by the dissection. The effort at reduction was necessarily postponed until he should be in a fit condition for the administration of chloroform. He remained, however, in a state of insensibility for the
DISLOCATION OF THE HUMERUS.

next three days, and no operation could have been safely attempted until some time after this. On the 15th, he seemed so far recovered that the next day was fixed upon for attempting the reduction; but on that day he was seized with shiverings and dyspnœa; and the symptoms of secondary deposit soon showed themselves. He fell into a typhoid state, and died on April 21st, a fortnight after the injury.

Post-mortem examination showed recent pleurisy on both sides, and a single secondary abscess in the right lung. There were no morbid appearances in the brain. The eighth rib on the left side was fractured, but the periosteum had not been torn through. The wound leading into the cavity of the elbow had sloughed, and there were several loose pieces of bone (fragments of the olecranon process) lying in the wound. The head of the radius had been thrown outwards, and was resting on the external condyle; it was partly deprived of cartilage, and felt rough and carious.

The head of the humerus was found immediately under the skin, having passed through the fibres of the deltoid muscle, and having the cephalic vein on its inner side. It had fractured the coracoid process in its passage upwards, and was resting behind on the stump of this process, and on the clavicle, with a small portion of the coraco-acromial ligament, which remained un torn. Internal to it (besides the fibres of the deltoid and the cephalic vein) was found the fractured extremity of the coracoid process, with the muscles attached to it, viz., the pectoralis minor, short head of the biceps and coraco-brachialis. External and somewhat posterior to it was the acromion process, separated from it by some of the fibres of the deltoid. Below and a little external to it was the glenoid cavity, the tip of which lay on a horizontal plane quite below the level of the dislocated head of the bone. The long tendon of the biceps remained still attached to the scapula, and was therefore situated below, and external to, the head of the humerus. The bone, in passing out of the glenoid cavity, had injured this tendon slightly, so that some of its internal fibres had
been broken away from the muscle, and remained floating freely with a tuft of muscular fibres attached to them. The coracoid process had been fractured near its base. The coraco-acromial ligament remained attached to both fragments, so as to prevent their being separated to any great extent. The tip was drawn a little downwards and inwards by the muscles attached to it. The head of the humerus had rested directly upon the stump of the coracoid process, which had caused slight abrasion of the articular cartilage corresponding to it. The humerus had been slightly turned upon its axis, so that the great tuberosity was relatively more forward than natural. The sub-scapularis muscle was intact. The muscles attached to the greater tuberosity of the humerus were torn through, except that a portion of the teres minor remained. The capsular ligament had been lacerated at its upper and inner part, forming a large hole for the passage of the head of the humerus.¹

The possibility of an injury such as I have above described has not escaped M. Malgaigne, the most recent and most approved author on this subject; but, though he met with a case in some respects resembling the above, he had not the opportunity of confirming his diagnosis by post-mortem examination. His case is as follows:²

*Supra-coracoid dislocation.*—Of this dislocation, unknown to all the authors whom I have consulted, I have seen one example, the result of accident, and another which appeared to be from disease. The former alone is related here.

A man, 68 years of age, was thrown from a vehicle with great force, on to the point of the shoulder, the arm being at the time pressed against the trunk. Attempts were

¹ This is not seen in the preparation, as the greater part of the capsular ligament has been removed, in order to show clearly the tendon of the biceps and the situation of the glenoid cavity.
² Malgaigne on Fractures and Dislocations, vol. ii, p. 530.
made at reduction immediately afterwards, by a bone-setter. Two months and a half had elapsed since the accident.

The head of the humerus was found to be dislocated forwards and upwards, above the coraco-acromial ligament, corresponding externally to the inner border of the acromion, internally covering the coracoid process, reaching upwards to the lower surface of the clavicle, and raising the deltoid to such a degree that a pin pushed in upon the most prominent part gave only eight millimetres (one third of an inch) for the thickness of the soft parts, while more forward it had separated the deltoid and pectoralis major so far as to have got within six millimetres (one quarter of an inch) of the surface of the body. The arm presented a shortening of not more than half a centimetre (less than one quarter of an inch).

M. Malaigne attempted to reduce this dislocation by means of pulleys applied to the arm, which was raised to a right angle with the body, but without success, though the movements were thought to have been rendered more free and extensive. He had some idea of dividing subcutaneously the coraco-acromial ligament, which seemed to oppose reduction; but, on reflection, thought it more prudent to abstain, and the dislocation remained unreduced.

For another case, in many respects similar to that which I have above recorded, I am indebted to Mr. Prescott Hewett. Some five years back, a stout, middle-aged woman was admitted into St. George's Hospital with an injury of the left shoulder-joint, which had occurred two days previously.

On examining the joint, Mr. Prescott Hewett, who that week happened to have charge of the accidents, came to the conclusion that the humerus was dislocated. The head of the bone was quite out of the socket, and appeared to be lying to the upper and inner side of the glenoid cavity, and immediately below the bend of the clavicle. Rotation of the arm gave a distinct and well-marked crepitus, which led to a difference of opinion among the surgeons who examined the case. By some it was thought that there was a fracture
of the neck of the humerus, and by others that there was a fracture involving the glenoid cavity.

Mr. Prescott Hewett reduced the dislocation with the heel; the shoulder immediately resumed its rounded form, and all crepitus disappeared. The case presented no further difficulty, and the woman was made an out-patient in a few days.

Now, if we compare the case which I have related with those of which the symptoms or post-mortem appearances are described in our authors, we shall see that it differs from them in this essential particular; that the head of the bone had passed entirely above the level of the glenoid cavity. Whether in M. Malgaigne's and Mr. Prescott Hewett's cases the dislocation was complete it is not possible, in the absence of anatomical details, to assert with confidence; but in the present case, no one who looks at the preparation will, I think, feel any doubt that the head of the bone is thrown entirely out of the glenoid cavity, that all its parts are above the level of the upper border of that cavity, and that it has passed all the obstacles which could oppose its further progress; so that it lies immediately below the skin, and it is only by the accidental cessation of the force that it has been prevented from passing still higher. The position of the humerus is such that, in the natural situation of parts, the vertical line through the centre of the glenoid cavity, produced upwards, would cut the head of the humerus a little external to its centre. The dislocation may, therefore, be regarded as in the upward direction.

One necessary condition of this injury is the displacement or rupture of the long tendon of the biceps; and in this respect the case before us resembles those reported by Mr. Soden; another is the fracture of either the coracoid or acromion process, since, if these two processes remain entire, the head of the bone has not room enough to pass entirely out of the glenoid cavity, but remains in the position indicated in the representation (a very faithful one) of Mr.
Soden's preparation, which will be found in the volume of the 'Transactions' before referred to.

With respect to diagnosis, the difficulties will arise mainly from two causes, viz., the amount of swelling which must always accompany an injury so violent as is necessary to give rise to this displacement, and the crepitus which will probably be caused by the fracture. The former is, of course, common to all severe injuries, and the influence which it exerts in confounding diagnosis may be shown by innumerable histories; the latter cause of error has this special circumstance attending it, that the head of the bone, resting as it does on the stump of the coracoid process, gets eroded by its contact with the sharp edge of this piece of bone, so that, in a few days, two rough surfaces of bone are found lying against each other, and the least movement of the humerus communicates a sensation of crepitus to the hand which is exactly like (in fact, is essentially the same as) the crepitus of a fracture. The portion of the head of the humerus, which is deprived of its cartilage and roughened by contact with the stump of the coracoid process, is very clearly seen in the preparation.

1 The crepitus in Mr. Prescott Hewett's case may have been caused by fracture of the coracoid process, but there are other instances on record of crepitus being felt in what were thought to be cases of simple dislocation.
ON
A CASE
OF
PREMATURE PUBERTY.

BY
ROBERT BATH SMART, M.R.C.S.

(Communicated by Richard Quain, F.R.S.)

Received June 21st.—Read June 22d, 1868.

I desire to place on record a case of premature puberty which has recently come under my notice. That the relation of this case to others may be more readily apprehended, I have prefixed a brief reference to the examples of this condition already published in this country.

The earliest instance of premature puberty which I have been able to find in an English volume is in the 'Philosophical Transactions' for 1745, where there are two letters, entitled "Some Account of the Gigantic Boy at Willingham, near Cambridge." This child, named Thomas Hale, was born Oct. 31, 1741; he was a lusty boy at birth, and the parts of generation were remarkably large. The minister of the parish writes:—"He grew to admiration three quarters of a year, having only the breast sustenance, when his mother died suddenly, and, as is supposed, he was accessory thereunto, by drawing away her vital nourishment." The change of diet, consequent on his mother's death, had no retarding influence on his growth, as he continued to flourish amazingly, and, when he was two years and ten months old, Mr. Dawkes, surgeon, at Huntingdon,
communicated the particulars of his case to Dr. Mead. At this time, he was three feet eight inches and a-half high, and weighed, in his clothes, four stone two pounds. He possessed extraordinary strength, and could easily lift and throw from him a blacksmith's hammer, which weighed seventeen pounds. When provoked by other children, he did not fight with his fists and legs, but laid his opponent prostrate by sheer strength, treating thus even boys of eight or ten years of age. His voice was "like a man's very groom." The genital organs were of large size, and the pubes covered with long thick and crisp hair. "The lanugo first appeared when he was near a year old, which gave great uneasiness to his parents."

This case was considered at the time so incredible, that the surgeon, before submitting his statement to the Royal Society, thought it advisable to confirm its veracity with affidavits from the midwife and the churchwardens of the parish.

Twelve other examples of premature puberty in both sexes have since been published in this country, and references are to be found, in our medical literature, to other instances, recorded by foreign authors. The following table exhibits the cases alluded to:—

<table>
<thead>
<tr>
<th>Title of Work</th>
<th>Author's Name</th>
<th>Sex of Child</th>
<th>Age at which Menstrae first appeared</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phil. Trans., Vol. 43, 1745</td>
<td>Dr. Mead</td>
<td>Males 1</td>
<td>1 never menstruated.</td>
</tr>
<tr>
<td>&quot; &quot; &quot; &quot; Med. Chir. Trans., Vol. 1, p. 277</td>
<td>Mr. Anthony White</td>
<td>Females 1</td>
<td>1 9 months.</td>
</tr>
<tr>
<td>&quot; &quot; &quot; &quot; 2, &quot; 17</td>
<td>Mr. Cooke</td>
<td></td>
<td>1 3 years.</td>
</tr>
<tr>
<td>&quot; &quot; &quot; &quot; 2, &quot; 116</td>
<td>Dr. Martin Wall</td>
<td></td>
<td></td>
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<tr>
<td>&quot; &quot; &quot; &quot; 4, &quot; 204</td>
<td>Sir Ashley Cooper</td>
<td></td>
<td></td>
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<tr>
<td>&quot; &quot; &quot; &quot; 11, &quot; 446</td>
<td>Dr. Gilbert Brechet</td>
<td></td>
<td></td>
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<tr>
<td>&quot; &quot; &quot; &quot; 12, &quot; 76</td>
<td>Mr. South</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Med. and Phys. Journ., Vol. 25, p. 117</td>
<td>Dr. Cookson</td>
<td>Males 1</td>
<td>1 3½ years.</td>
</tr>
<tr>
<td>Midland Med. Reporter, Vol. 1, p. 137</td>
<td>Dr. Birswe</td>
<td>Females 1</td>
<td></td>
</tr>
<tr>
<td>Med. Gazette, 1832, p. 9</td>
<td>Dr. Ledseaun</td>
<td></td>
<td></td>
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<tr>
<td>&quot; &quot; &quot; 1840, p. 548</td>
<td>Dr. Peacock</td>
<td></td>
<td></td>
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<tr>
<td>North. Journ. Med., 1845, p.70</td>
<td>Mr. Whitmore</td>
<td></td>
<td></td>
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<tr>
<td>Taylor's Med. Jurispr.</td>
<td>Mr. Embling</td>
<td></td>
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<tr>
<td>Lancet, 1848, p. 137</td>
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The child who is the subject of this paper first came under my observation in August last, when she presented the same remarkable features as those which now distinguish her. No detailed examination, however, was made until the present month, May, 1858.

Mary Deane, aged five years and four months, was born on January 7th, 1853, at Lombard Street, Deansgate, and has lived in Manchester all her life. The exact date of her birth has been ascertained from the Register.

She is of a robust habit; her complexion is clear, rosy, and slightly inclined to be fair; she has grey eyes, and rather dark brown hair; her height is three feet seven inches, and she weighs, in her clothes, fifty-two pounds and a-half.

It will be seen, from the accompanying photographs, that her general appearance is wonderfully like that of an adult female, of short stature, and the resemblance is maintained in her functional economy, the menses recurring every month, as regularly as in their normal manifestation. The configuration and relative proportions of the torso differ widely from the type of childhood. The subcutaneous cellular tissue is abundantly supplied with fat, by which the unseemly angularities of unripe years are cushioned over and lost in the rounded contour of maturity.

There is nothing unusual in the volume or shape of her head, neither inspection nor mensuration discloses any indication of cerebellar enlargement. The hair of the poll is not more abundant than in other children of her age. Her countenance has a childish expression; the teeth all belong to the first or "milk" set.

A trifling peculiarity is observed about the left eye, which cannot be opened so wide as its fellow, causing an appearance of partial ptosis. This is owing to a curtailment of the palpebral fissure at the outer canthus, which is probably a congenital malformation, as the mother says it was always so: there is no decided mark of a cicatrix, nor do the cilia
extend beyond the present commissure, as might be expected in anchylo-blepharon from opthalmia tarsi.

The throat is full, but the thyroid cartilage is not particularly prominent. The voice is strong, without any other peculiarity.

The bust is full and womanly, as is well seen in the photographs. The breasts are larger and more protuberant than those of most girls who have recently attained puberty, and the characteristic lobular structure of the gland is easily perceived on manipulation. The nipples are well developed, and the integument of the breasts is traversed by the branching blue lines of the enlarged mammary veins, which it is said become especially apparent at the catamenial periods. The axillae are destitute of hairs.

The child is wider across the hips than others of her age. The prominence of the nates and the forward curve of the lumbar spine are well marked, and there is a corresponding excess in the bulging of the abdomen. The pubes is sprinkled over with fine light-brown hair, about an inch in length; the mons veneris is prominent. The labia externa and nymphae are large, the latter included within the fissure of the vulva. The vagina is capacious, and has a fringe-shaped hymen.

The limbs are stout and muscular, the thighs, especially, being very fat and large; the bones are straight and strong. The contrast to the more slender limbs of her elder sister is extremely striking.

The intellectual and moral faculties of this child do not exhibit any signs of precocity at all commensurate with the forward development of the body. Her disposition is lively, but her mother says she has "her little tempers," in which she shows an obstinate resistance to authority, and she is less manageable than her sisters. She is certainly less intelligent than her elder sister; her education, however, has been neglected entirely, her mother being unwilling to send the child to school, lest tricks might be played with her. There are no positive indications of sexual propensities; she is coarse in her manners; but, perhaps, not more so than
other children of her class and age; there is an absence of all that grace and gentleness which accompany natural puberty in the female sex. She is not at all shame-faced; she does not shrink from being examined, nor does she blush when stripped; she has manifested no preference for the society of the opposite sex, indeed, she is reported to avoid the company of boys. Her mother describes her as being "old-fashioned in her ways," as being fond of babies and household employments, and as liking dress and display, loving to strut in some tawdry drapery hung fantastically from her shoulders.

Previous History.—According to the statement of the mother, she was a rather large child at birth, but presented no indication of the remarkable development so soon to follow. The head had very little hair upon it, and the fontanelles were open, remaining unclosed till she was nearly eighteen months old. Her first tooth appeared at the end of nine months, and she had entered upon her third year before the set was completed. She did not go alone until she was sixteen months old.

She escaped all the more serious disorders incident to dentition. She never had convulsions, nor had she ever chorea or any other symptom of spinal irritation or cerebral disease. She had measles, and afterwards scarlatina; the latter, when she was about two years of age. She has never had worms.

An inquiry into her habits does not reveal an exposure to any of the assigned exciting causes of early puberty. She has occupied the same bedroom as her parents, and has never slept with any of the other sex; her sisters have been her usual associates. In diet she has fared like the rest of the family.¹

¹ The effect of diet on sexual development is shown in certain of the lower animals. The "neuter" larva of the bee is changed into a perfect female or "queen" by superior aliment alone.
The earliest phenomenon of sexual development observed by the mother was a slight down on the pubes, which was discovered a few weeks before the menstrual discharge. The catamenia made their first appearance when the child was three years and six months of age; for six months before their occurrence she seemed to be the subject of some inward ailing, and the mother feared “she was going in a low way.” The child was weak and inert; she did not care to play with other children, but moped indoors. She was heavy and drowsy, but did not complain of headache. She used sometimes to suffer from pains in the back and groins, not over the hypogastrum. For some months she had a leucorrheal discharge.

The first menstrual flow lasted about a week, and was very thick and black. It has returned regularly every month since, once only it was delayed for six weeks. The catamenia, which are now natural in quality, but not much in amount, generally last four days, sometimes a day more or less. At these periods she used to wear a napkin, but now refuses to do so, and her linen has been observed by me to be stained in consequence. There is often an appearance of “whites” a few days before the eruption of the catamenia, and, at such times, she complains of aching in her back and belly.

After the establishment of the catamenia she rapidly gained in flesh, and has since been hearty and well. She has never been given to fainting fits, or any other form of hysteria.

History of Family.—The mother of this child will be aged twenty-six next July. She is a small, spare brunette, and was formerly a factory-hand. She began to menstruate at the age of sixteen, and was married in two years after. She has had four children, all of whom are girls, and are still living. The first-born, Jane, aged seven on the 24th March last. The second, Mary, is the subject of this narrative. The third is aged three years and a half, and the youngest sixteen months. The mother is conscious of being
subjected to no extraordinary influence during her second pregnancy. The labour was tedious, but, in other respects, natural. There is nothing remarkable in the appearance of Mary’s sisters; Jane, the eldest, is thin and darker, resembling her mother; although Mary’s senior, she is below her in stature, and weighs nearly a stone less.

The father, 26, is of light complexion, and of medium height; he is a “maker-up” in a warehouse. It has not been ascertained at what age he acquired virility.

No example of sexual precocity has been known to occur in the ancestry, or in any collateral branch of the family.

### Table of Measurements

<table>
<thead>
<tr>
<th></th>
<th>Mary Deane</th>
<th>Jane Deane</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>5 yrs. &amp; 4 mo.</td>
<td>7 years.</td>
</tr>
<tr>
<td>Weight</td>
<td>52½ lbs. avoir.</td>
<td>40½ lbs. avoir.</td>
</tr>
<tr>
<td>Height</td>
<td>43 inches</td>
<td>42 inches</td>
</tr>
<tr>
<td>From root of nose over vertex to occipital protuberance</td>
<td>12½ &quot;</td>
<td>12½ &quot;</td>
</tr>
<tr>
<td>Between meatus auditorii ext. across vertex</td>
<td>13½ &quot;</td>
<td>12½ &quot;</td>
</tr>
<tr>
<td>From occipital protuberance to mid-point on circumference of a vertical plane passing through meatus auditorii</td>
<td>6½ &quot;</td>
<td>6½ &quot;</td>
</tr>
<tr>
<td>From root of nose to same point as above</td>
<td>6½ &quot;</td>
<td>5½ &quot;</td>
</tr>
<tr>
<td>Circumference of cranium above orbits</td>
<td>20 &quot;</td>
<td>20 &quot;</td>
</tr>
<tr>
<td>Half circumference in front of meatus auditorii</td>
<td>9½ &quot;</td>
<td>9½ &quot;</td>
</tr>
<tr>
<td>Ditto behind meatus auditorii</td>
<td>10½ &quot;</td>
<td>10½ &quot;</td>
</tr>
<tr>
<td>From symphysis to angle of lower jaw</td>
<td>3½ &quot;</td>
<td>3½ &quot;</td>
</tr>
<tr>
<td>Circumference of throat opposite thyroid cartilage</td>
<td>10½ &quot;</td>
<td>9½ &quot;</td>
</tr>
<tr>
<td>Circumference of shoulders</td>
<td>26 &quot;</td>
<td>24½ &quot;</td>
</tr>
<tr>
<td>From most projecting point of shoulder to the other, before</td>
<td>12 &quot;</td>
<td>11 &quot;</td>
</tr>
<tr>
<td>Ditto</td>
<td>14 &quot;</td>
<td>13½ &quot;</td>
</tr>
<tr>
<td>Circumference of thorax opposite nipples</td>
<td>25½ &quot;</td>
<td>21 &quot;</td>
</tr>
<tr>
<td>Ditto at scrobiulus cordis</td>
<td>22½ &quot;</td>
<td>20 &quot;</td>
</tr>
<tr>
<td>Ditto of abdomen opposite umbilicus</td>
<td>25½ &quot;</td>
<td>20 &quot;</td>
</tr>
<tr>
<td>Ditto of hips over buttoks</td>
<td>22½ &quot;</td>
<td>21½ &quot;</td>
</tr>
<tr>
<td>From ant. sup. spin. process of ilium to opposite ditto, measured over the protuberant hypogastrium</td>
<td>7 &quot;</td>
<td>6 &quot;</td>
</tr>
<tr>
<td>Ditto estimated callipers-fashion</td>
<td>6 &quot;</td>
<td>6 &quot;</td>
</tr>
<tr>
<td>From ant. sup. spin. process of ilium to opposite ditto, passing horizontally backwards</td>
<td>15½ &quot;</td>
<td>13 &quot;</td>
</tr>
<tr>
<td>From ant. sup. spin of ilium to symphysis pubis</td>
<td>4½ &quot;</td>
<td>4 &quot;</td>
</tr>
<tr>
<td>From symphysis pubis under perineum as far as point of spine on level with topmost crests of ilia</td>
<td>12 &quot;</td>
<td>10 &quot;</td>
</tr>
<tr>
<td>Between great trochanters, (callipers fashion)</td>
<td>8½ &quot;</td>
<td>8 &quot;</td>
</tr>
<tr>
<td>Circumference of thigh at widest part</td>
<td>15 &quot;</td>
<td>12½ &quot;</td>
</tr>
<tr>
<td>From great trochanter to sole of foot (standing)</td>
<td>21 &quot;</td>
<td>20 &quot;</td>
</tr>
<tr>
<td>Length of foot</td>
<td>6½ &quot;</td>
<td>6½ &quot;</td>
</tr>
</tbody>
</table>
In the preceding measurements the elder sister has served as a convenient standard of comparison. The differences observable between the two children are most emphatic, where the data relate to girth, and are chiefly due to the greater volume of the soft parts in the younger girl, and less to the expansion of the osseous framework. The most practical importance is attached to the determination of the pelvic diameters, which do not, however, seem to be notably increased.
INDEX.

Abdominal parietes, case of communication with the stomach through, produced by ulceration from external pressure . 11
Age, influence of, upon the mortality of fever . 297
Alkalies, caustic, influence of, upon the therapeutic properties of henbane, belladonna, and stramonium . 53—61
Ammonia salts, preservative power of . 69
Aneurism, arterio-venous, of the temporal vessels, case of, treated by ligature of artery and vein . 1
Arteries, table of, in a case of congenital deformity of both upper extremities . 101
Atropa belladonna, influence of liquor potassae on the therapeutic properties of . 53—61

Belladonna, influence of liquor potassae on the therapeutic properties of . 53—61
Birkett, Mr. John, case of fibrous polypus of the urinary bladder, with observations and a table of the recorded cases . 311
Bladder, urinary, fibrous polypus of the calculus in, removed by lithotripsy, in which a communication existed between the bladder and intestine . 441

Calculus in the bladder, case of, removed by lithotripsy, in which a communication existed between the bladder and intestine . 441
Colchicum, on the action of, upon the urine in gout . 348
Communication between the bladder and intestine, in case of calculus, removed by lithotripsy . 441
Coracoïd process of the scapula, fracture of, in case of dislocation of the humerus . 447
Coulson, Mr. William, case of hydatids of the tibia . 307
## INDEX.

<table>
<thead>
<tr>
<th>Topic</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Datura stramonium, influence of liquor potassæ on the therapeutic</td>
<td>53–61</td>
</tr>
<tr>
<td>properties of</td>
<td></td>
</tr>
<tr>
<td>Decidua, membranes, which surrounds the ovum in cases of tubal</td>
<td>137–153</td>
</tr>
<tr>
<td>gestation</td>
<td></td>
</tr>
<tr>
<td>Deformity of both upper extremities, anatomical description of a</td>
<td>75–103</td>
</tr>
<tr>
<td>case of</td>
<td></td>
</tr>
<tr>
<td>tables of the muscles, arteries, nerves, &amp;c., in ditto</td>
<td>100–3</td>
</tr>
<tr>
<td>Dislocation of the humerus, upwards and inwards</td>
<td>447</td>
</tr>
<tr>
<td>Etiology of continued fever, or causes of the prevalence and</td>
<td>219</td>
</tr>
<tr>
<td>mortality of its different forms</td>
<td></td>
</tr>
<tr>
<td>Excision of the knee, cases of</td>
<td>193</td>
</tr>
<tr>
<td>Excrements, human, analysis and immediate principles of, in the</td>
<td></td>
</tr>
<tr>
<td>diseased state</td>
<td>119</td>
</tr>
<tr>
<td>synoptic table of method to be adopted for</td>
<td></td>
</tr>
<tr>
<td>their analysis</td>
<td>136</td>
</tr>
<tr>
<td>Eye, influence of the cervical portions of the sympathetic nerve and</td>
<td>397</td>
</tr>
<tr>
<td>spinal cord upon</td>
<td></td>
</tr>
<tr>
<td>Fever, continued, etiology of, or causes of the prevalence and</td>
<td>219</td>
</tr>
<tr>
<td>mortality of its various forms</td>
<td></td>
</tr>
<tr>
<td>prevalence of, and influence of months and seasons,</td>
<td></td>
</tr>
<tr>
<td>sex, age, occupation and station in life,</td>
<td></td>
</tr>
<tr>
<td>recent residence and birth-place, on ditto</td>
<td>223–286</td>
</tr>
<tr>
<td>table showing the prevalence of, at different places, 1817–57</td>
<td>224</td>
</tr>
<tr>
<td>annual prevalence of the different forms of, in London, as compared</td>
<td>226</td>
</tr>
<tr>
<td>with other towns</td>
<td></td>
</tr>
<tr>
<td>localities of London in which each form is most prevalent</td>
<td>247</td>
</tr>
<tr>
<td>mortality from, and influences affecting ditto 286–306</td>
<td></td>
</tr>
<tr>
<td>rate of mortality from, in the London Fever Hospital, as compared</td>
<td>266</td>
</tr>
<tr>
<td>with that of other hospitals</td>
<td></td>
</tr>
<tr>
<td>rate of mortality in the different forms of</td>
<td>291</td>
</tr>
<tr>
<td>overcrowding, with deficient ventilation and destitution, as cause</td>
<td>258</td>
</tr>
<tr>
<td>of typhus</td>
<td></td>
</tr>
<tr>
<td>putrid emanations from decomposing organic matter in drains, cesspools, churchyards, &amp;c., and organic impurities in drinking-water, as causes of typhoid</td>
<td>263</td>
</tr>
</tbody>
</table>
INDEX.

Fever, continued, question of the contagion of . . . 278
Fibrous polypus of the urinary bladder . . . 311
Fistula, gastro-cutaneous, table of recorded cases of . . . 20
Fox, Dr. Wilson, contributions to the pathology of the glandular structures of the stomach . . . 361
Fracture of the coracoid process of the scapula, in case of dislocation of the humerus . . . 447

Galvanism, action of, upon the contractile structure of the gravid uterus . . . 157
case of profuse hemorrhage in early months of pregnancy successfully treated by . . . 167
placental presentation treated by . . . 173
Garrod, Dr. Alfred B., therapeutic communications (No. 1): on the influence of liquor potassae, and other caustic alkaline solutions, upon the therapeutic properties of henbane, belladonna, and stramonium . . . 53
ditto (No. 2) ditto . . . 61
Researches on gout (Part 1): the urine in the different forms of gout . . . 325
(Part 2): the influence of colchicum upon the urine . . . 348

Gestation, tubal, on membra decidae surrounding the ovum in . . . 137, 153
Glandular structures of the stomach, contributions to the pathology of . . . 361
Glaucoma, on some points in the pathology and morbid anatomy of . . . 111
Gout, researches on . . . 325
on the action of colchicum upon the urine in . . . 348
the urine in the different forms of . . . 325
urine of subjects suffering from acute . . . 329
the urine in cases of chronic . . . 336
urine of individuals who had suffered from, at the time of freedom from the disease . . . 345
Gräf's discoveries relative to glaucoma, notices of . . . 112

Hæmorrhage, case of profuse, in early months of pregnancy, successfully treated by galvanism . . . 167
Hawkins, Mr. Charles, account of a case of calculus in the bladder removed by lithotripsy, in which a communication existed between the bladder and intestine . . . 441
Henbane, influence of liquor potassae on the therapeutic properties of . . . 53, 61
Holmes, Mr. T., case of dislocation of the humerus, upwards and inwards, with fracture of the coracoid process of the scapula. 447
Hulke, Mr. J. W., on some points in the pathology and morbid anatomy of glaucoma. 111
Humerus, dislocation of the, upwards and inwards, with fracture of the coracoid process of the scapula. 447
Humphrey, Mr. George M., on excision of the knee. 193
Hydatids of the tibia, case of. 307
Hyoscyamus, influence of liquor potassae on the therapeutic properties of. 53—61

Intestine, communication of, with the bladder, in case of calculus, removed by lithotripsy. 441
Inversion of the uterus, of twelve years' duration, successfully treated. 183

Knee, excision of the, cases of. 193

Lee, Dr. Robert, on the membrana decidua which surrounds the ovum in cases of tubal gestation. 137
Supplement to ditto. 138
Ligature of the artery and vein in a case of arterio-venous aneurism. 1
Liquor potassae, influence of, on the therapeutic properties of henbane, belladonna and stramonium. 53—61
Lithotripsy, case of calculus in the bladder, with a communication between the bladder and intestine, removed by. 441
London, localities of, in which each form of continued fever is most prevalent. 247

Mackenzie, Dr. F. W., on the action of galvanism upon the contractile structure of the gravid uterus, and its remedial powers in obstetric practice. 157
Marchet, Dr. W., on the analysis and immediate principles of human excrements in the diseased state. 119
Membrana decidua which surrounds the ovum in cases of tubal gestation. 137—153
Monstrosity, anatomical description of a case of, in both upper extremities. 75
Moore, Mr. Charles H., account of a case of arterio-venous aneurism of the temporal vessels, treated by ligature of both the artery and the vein. 1
Mortality from continued fever. 286
INDEX.

Murchison, Dr. Charles, case of communication with the stomach through the abdominal parietes, produced by ulceration from external pressure, &c. 11

Contributions to the etiology of continued fever, or an investigation of various causes which influence the prevalence and mortality of its different forms 219

Muscles, table of, in a case of congenital deformity of both upper extremities 100

Nerves, table of, in a case of congenital deformity of both upper extremities 102

Nerve, sympathetic, influence of the cervical portions of the, upon the eye 397

Ogle, Dr. John W., on the influence of the cervical portions of the sympathetic nerve and spinal cord upon the eye and its appendages 397

Illustrative cases 412

Ovum, on the membrana decidua surrounding it in tubal gestation 137–153

Placental presentation treated by galvanism 173

Polypus, fibrous, of the urinary bladder 311

Table of recorded cases of 322

Potasse, liquor, influence of, on the therapeutic properties of hembane, belladonna and stramonium 53–61

Puberty, case of premature 455

Ross, Catherine, case of (gastro-cutaneous fistula) 12

Scapula, fracture of the coracoid process of the, in case of dialocation of the humerus 447

Sex, influence of, upon the prevalence of continued fever 235

On the mortality of ditto 296

Silvester, Dr. Henry R., contribution to the science of teratology 73

Smart, Mr. Robert Bath, on a case of premature puberty 455

Smith, Dr. Tyler, case of complete inversion of the uterus, of twelve years' duration, successfully treated 183

Spinal cord, influence of the cervical portions of the, upon the eye 397
INDEX.

Stomach, contributions to the pathology of the glandular structures of the case of communication with, through the abdominal parietes, produced by ulceration from external pressure fistula of, table of recorded cases of

Stramonium, influence of liquor potassae on the therapeutic properties of

Temporal vessels, case of arterio-venous aneurism of the, treated by ligature of both the artery and the vein

Teratology, contribution to the science of

Tibia, case of hydatids of the

Tubal gestation, on membrana decidua surrounding the ovum in lists of preparations of Fallopian-tube conception in the museums of St. Bartholomew's and Guy's Hospitals

Typhoid fever, see FEVER, (Continued).

Typhus fever, see FEVER, (Continued.)

Ulceration from external pressure, production of communication with the stomach through the abdominal parietes by

Urinary bladder, fibrous polypus of the

Urine in the different forms of gout of subjects suffering from acute gout in cases of chronic gout of individuals who had suffered from gout, at the time of freedom from the disease on the action of colchicum upon, in gout

Uterus, inversion of the, of twelve years' duration, successfully treated gravid, action of galvanism upon the contractile structure of

Vessels, temporal, arterio-venous aneurism of