

COLUMBIA LIBRARIES OFFSITE
HEALTH SCIENCES RESTRICTED



HR00821500

RECAP



SERIAL

v 32

L


1896

Columbia University
in the City of New York

College of Physicians and Surgeons



Library



Digitized by the Internet Archive
in 2010 with funding from
Open Knowledge Commons

SAINT
BARTHOLOMEW'S HOSPITAL
REPORTS.

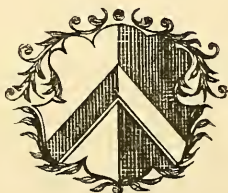
SAINT
BARTHOLOMEW'S HOSPITAL
REPORTS.

EDITED BY

SAMUEL WEST, M.D.

AND

W. J. WALSHAM, F.R.C.S.



VOL. XXXII.

LONDON:

SMITH, ELDER, & CO., 15 WATERLOO PLACE.

1897

med
33-36838

Printed by BALLANTYNE, HANSON & Co.
At the Ballantyne Press

IN EXCHANGE.

- The Practitioner. Editor, Dr. Malcolm Morris, 8 Harley Street, Cavendish Square, W.
- Royal Medical and Chirurgical Society's Transactions, 20 Hanover Square, W.
- Guy's Hospital Reports.
- St. Thomas's Hospital Reports.
- Westminster Hospital Reports.
- Pharmaceutical Society's Journal and Transactions.
- American Journal of Medical Science.
- Madras Medical Journal.
- Société des Sciences médicales de Lyons.
- Surgeon-General's Office, War Department, U.S.A., per Mr. Wesley, Essex Street, Strand.
- Revue des Sciences médicales, M. le Docteur G. Hayem, Rédacteur du Journal, aux soins de M. Masson, 17 Place de l'École de Médecine, Paris.
- Le Progrès Médical.
- Annales de Dermatologie et de Syphilographie, Dr. A. Doyon, Ueiage, near Grenoble, France.
- The Chicago Medical Journal and Examiner, Dr. Byford (Messrs. Keen, Cook, & Co., Chicago, Illinois).
- The Transactions of the American Medical Association, Washington, D.C., per John B. Hamilton, M.D., Chicago, Illinois. Smithsonian Institution.
- Centralblatt für Chirurgie, herausgegeben von F. König, E. Richter, R. Volkmann (Messrs. Breitkopf & Härtel, Leipzig).
- Transactions of the American Gynecological Society, Dr. James R. Chadwick, Clarendon Road, Boston, Mass., U.S.A.
- Mémoires de la Société de Médecine et de Chirurgie de Bordeaux, Dr. A. Demons, 45 Cours de Tourny, Bordeaux.
- The Journal of Nervous and Mental Disease, edited by Charles Henry Brown, M.D., 25 West 45th Street, New York.
- The Liverpool Medico-Chirurgical Journal, Liverpool Medical Institution, 72 Rodney Street, Liverpool.
- Transactions of the New York Academy of Medicine.
- The John Hopkins Hospital Reports, Baltimore, Maryland, U.S.A.
- The Bristol Medico-Chirurgical Journal, Assist.-Editor L. M. Griffiths, 9 Gordon Road, Clifton, Bristol.
- Transactions of the College of Physicians, Philadelphia, per Smithsonian Institution.
- Sheffield Medical Journal, Arthur J. Hall, M.B., 263 Glossop Road, Sheffield.
- King's College Hospital Reports.
- British Medical Association, 429 Strand, W.C.
- Transactions of Medical Society of London, 11 Chandos Street, Cavendish Square, W.

CONTENTS.

	PAGE
LIST OF SUBSCRIBERS	xi
 <i>ART.</i>	
I. In Memoriam. Sir George Murray Humphry. By C. B. Lockwood	xxxii
II. In Memoriam. William Marrant Baker. By Alfred Willettxxxix
III. Clinical Cases from the Wards. By W. S. Church, M.D.	I
IV. Cases from Mr. Smith's Wards. With Remarks by George V. Worthington, M.B.	19
V. Clinical Aphorisms from Dr. Gee's Wards. 1895-1896. Collected and Edited by Thomas J. Horder	29
VI. Cases of Enteritis and Colic simulating Intestinal Ob- struction. By A. M. Mitchell, M.B. With Remarks by Mr. Smith.	61
VII. A Case of Hepatic Cirrhosis, with Obstruction in the Superior Vena Cava. By Sir Dyce Duckworth, M.D., and Archibald E. Garrod, M.D.	71
VIII. On Ether Rash. By Edgar Willett	79
IX. Fetid Empyema. By Samuel West, M.D.	83
X. Profuse Uncontrollable Diarrhœa in a Man recently re- turned from the Tropics. By Samuel West, M.D.	87
XI. On Hernia of the Vermiform Appendix. With a Series of Cases. By W. McAdam Eccles	93
XII. On the Occurrence of Rigor and Collapse in Typhoid Fever. By W. P. Herringham, M.D.	107
XIII. The Relation of Gout and Rheumatism to Dupuytren's Contraction of Palmar Fascia. By Charles Edward Hedges, M.B.	119
XIV. On the Occurrence of the Diphtheria Bacillus. By F. W. Andrewes, M.D.	145
XV. Skin-Grafting by Thiersch's Method. By H. J. Paterson	161
XVI. A Case of Disseminated Sclerosis with Palatal and Laryngeal Anæsthesia. By F. E. Batten, M.D., and W. J. Horne, M.B.	173
XVII. Reports of Cases in the Surgical Wards. By Thomas J. Horder, M.B.	181

ART.	PAGE
XVIII. A Case of Double Empyema, with Recovery. By W. E. N. Dunn	213
XIX. Note on the Value of Ehrlich's Reaction in the Diag- nosis of Enteric Fever. By Herbert J. Paterson, M.B.	217
XX. The Leucocytosis of Scarlet Fever. By R. Sevestre, M.D.	225
XXI. On the Relationship of Cardiac Hypertrophy and Œdema to Chronic Renal Disease. By H. Holmes, M.B., and A. A. Kanthack, M.D.	261
XXII. On Secondary Infective Lesions in Fatal Cases of Lobar Pneumonia. By H. W. Lance, M.B., and A. A. Kanthack, M.D.	309
XXIII. On the Treatment of Anæmia by Oxygen Inhalations. By F. M. Burnett, Esq., M.B.	321
XXIV. The Prognosis and Causes of Death in Cases of Diph- theria Treated by Antitoxin at St. Bartholomew's Hospital. By J. H. May, M.B., and A. A. Kan- thack, M.D.	335
XXV. On the Ætiology of Chorea. By H. Morley Fletcher, M.D.	383
XXVI. Some Cases of Deformity. By Arthur Heath, M.B.	399
XXVII. The Pathology of Alcoholic Paralysis. By Howard H. Tooth, M.D.	407
XXVIII. Acute Atrophy of the Liver. By T. Lauder Brunton, M.D., F.R.S., and F. W. Tunncliffe, M.D.	425
Proceedings of the Abernethian Society for the Session 1895-96	443
List of Specimens added to the Museum	449
Books presented to the Library	534
Summary of Scholarships and Prizes	536
List of Prizemen	537
Hospital Staff	540
INDEX	544

STATISTICAL TABLES WITH REPORTS BY MEDICAL
AND SURGICAL REGISTRARS.

LIST OF ILLUSTRATIONS.

	PAGES
TEMPERATURE CHARTS ILLUSTRATING DR. CHURCH'S CASES	8, 14
TEMPERATURE CHARTS ILLUSTRATING DR. HERRINGHAM'S CASES	108-113
SKIN GRAFTING	171
CHARTS ILLUSTRATING LEUCOCYTOSIS OF SCARLET FEVER	241-259
CHARTS ILLUSTRATING TREATMENT OF ANÆMIA BY OXYGEN	322-332
CHARTS ILLUSTRATING ÆTIOLGY OF CHOREA . . .	388-393
SOME CASES OF DEFORMITY	400-402
MICROPHOTOGRAPHS OF DEGENERATE NERVES IN ALCO- HOLIC PARALYSIS	412

LIST OF SUBSCRIBERS.

- ABERCROMBIE, Dr. J., 23 Upper Wimpole Street, W.
ADAMS, Dr. JAMES, Barnes, Surrey
ADAMS, JOHN, 180 Aldersgate Street, E.C.
ADAMS, Dr. J. O., Brooke House, Upper Clapton, N.E.
ALDOUS, G. F., Charlton House, Compton Gifford, near Plymouth
ALLEN, Dr. HENRY MARCUS, 20 Regency Square, Brighton
ANDERSON, A. R., 5 East Circus Street, Nottingham
ANDERSON, K., Johannesburg, South Africa, c/o General Post Office
ANDREW, Dr. J., Rahere, Branksome Avenue, Bournemouth West
ANDREWES, Dr. F. W., 35 Welbeck Street, W.
ANDREWS, S., Basingstoke
ARATHOON, H. C., H.M.S. "Excellent," Portsmouth
ATKINSON, T. R., 1 West Cromwell Road, W.
ATKINSON, H. N. C., Warneford Hospital, Leamington, Warwick
AUDEN, G. A., Library, St. Bartholomew's Hospital, E.C.
AVERILL, Dr. C., Park Green, Macclesfield

BADCOCK, S. H., Library, St. Bartholomew's Hospital, E.C.
BAILEY, Dr. H. V., Pekin, Illinois, United States, America
BAILEY, R. C., 21 Welbeck Street, W.
BALGARNIE, Dr. W., The Dutch House, Hartley Wintney, Winchfield, Hants
BARBER, SYDNEY F., Jessop Hospital for Women, Sheffield

- BARROW, B., Southlands Park Road, Ryde, Isle of Wight
 BARRS, Dr. ALFRED GEORGE, 22 Park Place, Leeds
 BARTON, J. K., 2 Courtfield Road, Gloucester Road, S.W.
 BATEMAN, F., Whitchurch, near Reading
 BATEMAN, H. E., 48 Micklegate, York
 BATTEN, FREDERICK E., 124 Harley Street, W.
 BATTEN, Dr. R. D., Campden Lodge, Campden Hill Road, W.
 BEAUCHAMP, Dr. SYDNEY, 95 Cromwell Road, South Kensington, S.W.
 BECKETT, F. M., St. Audrey's, Ely, Cambridge
 BELBEN, F., Hoo Meavy, Branksome, Bournemouth
 BELDING, D. T., East Dereham, Norfolk
 BENJAMIN, J. K. K., Darrington, near Shrewsbury
 BERRY, JAMES, 60 Welbeck Street, Cavendish Square, W.
 BEVAN, H. C., Blaina, Monmouth, South Wales
 BINDLOSS, E. F., Melbourne, Royston, Cambridge
 BIRD, ASHLEY, 30 Windsor Terrace, Penarth, Glamorgan
 BIRD, Dr. R., Surgeon-Captain I.M.S., c/o Messrs. Grindley & Co., Agents, Calcutta
 BLAKENEY, H. T. W., Mount House, Dorking
 BLAKER, N. P., 29 Old Steyne, Brighton
 BLAMPEID, J. W., Three Oaks, St. Lawrence, Jersey
 BLOXAM, JOHN A., 75 Grosvenor Street, W.
 BOKENHAM, T. J., 10 Devonshire Street, Portland Place, W.
 BOLTON, J. U., 12 The Crescent, Scarborough
 BONTOR, Dr. S. A., Elm Grove House, Great Berkhamsted, Herts
 BOSTOCK, E. INGRAM, Horsham, Sussex
 BOSWELL, Dr. A., Ashbourne, Derbyshire
 BOTT, H., Brentford, Middlesex
 BOULTER, H. B., Barnard House, Richmond, Surrey
 BOWDEN, Dr. R. T., Rochford House, Ramsgate
 BOWES, C. K., Herne Bay, Kent
 BOWLBY, A. A., 24 Manchester Square, W.
 BRIGGS, G. F., 14 Great Ormond Street, W.C.

- BRINTON, Dr. R. D., 8 Queen's Gate Terrace, S.W.
BROOK, Dr. W. H. B., James Street, Lincoln
BROOK, CHARLES, Minster Yard, Lincoln
BROOKSBANK, H. L., Thornbarrow, Windermere, Westmoreland
BROWNE, Dr. OSWALD, 43 Bedford Square, W.C.
BRUCE CLARKE, W., 46 Harley Street, Cavendish Square, W.
BRUNTON, Dr. T. LAUDER, F.R.S., 10 Stratford Place, W.
BULLAR, J. F., 7 Carlton Crescent, Southampton
BURD, Dr. LYCETT, St. Mary's Street, Shrewsbury
BURN, Dr. W. B., Beechwood, Balham Road, Upper Tooting, S.W.
BURN, T. W. B., 75 Mill Road, Cambridge
BURNETT, F. M., 12 Prince Arthur Road, Hampstead, N.W.
BURNIE, W. GILCHRIST, 1 Drowton Street, Bradford, Yorkshire
BUTLER, C., 370 Brixton Road, S.W.
BUTLER, T. M., The Firs, Guildford
BUTLER-SMYTHE, A. A., 76 Brook Street, W.
BUTLIN, H. T., 82 Harley Street, W.
BYNS, H. S., Library, St. Bartholomew's Hospital, E.C.
- CALVERT, Dr. J., 36 Queen Anne Street, W.
CAMMIDGE, P. J., 49 Grosvenor Road, Canonbury, N.
CARLYON, Dr. T. B., St. Mary's, Exmouth, Devon
CARPENTER, E. G., The Priory, Beaufort Road, Surbiton, Surrey
CARTER, D'ARCY B., Belmont, Shipley, Yorkshire
CARTER, Dr. F. H., Glenholme, 99 Upper Richmond Road,
Putney, S.W.
CAUTLEY, E., 15 Upper Brook Street, W.
CAVE, Dr. E. J., Crewkerne, Somerset
CHAMPNEYS, Dr. FRANCIS H., 42 Upper Brook Street, W.
CHAPMAN, H. F., Hill Rise House, Richmond, Surrey
CHAPPLE, A. D., Clevedon, St. George's Road, Weybridge,
Surrey
CHIPPERFIELD, T. J. B. P., 52 Edge Lane, Liverpool
CHITTENDEN, T. H., 23 Onington Square, Pont Street, S.W.
CHOLMELEY, Dr. H. P., 15 Onslow Crescent, South Kensington

- CHRISTOPHERSON, CECIL, 6 Carlisle Parade, Hastings
CHURCH, Dr. W. S., 130 Harley Street, W.
CLARKE, A. H., Macquarie Street, Hobart, Tasmania
CLARKE, Dr. ERNEST, 3 Chandos Street, W.
CLARKE, J. M., 28 Pembroke Road, Clifton, Bristol
CLOSE, Dr. J. B., 2 Pryne Street, Hull
COALBANK, I., Teddington Lodge, Bushey Park Road, Ted-
dington
COATES, Dr. G., 30 Brechin Place, South Kensington, S.W.
COCKER, W. HENRY, Bloomfield, South Shore, Blackpool
COLEMAN, ALFRED, Midhurst, Hillbury Road, Tooting Common
COLENZO, Dr. R. J., 91 Cromwell Road, Kensington, S.W.
COLES, Dr. C., High-Cross Street, Leicester
COLLINGRIDGE, Dr. W., 65 Tressillian Road, St. John's, S.E.
COLLINS, W. C. G., Ivy Cottage, Aylesford, Kent
COLLYNS, J. B., Dulverton, Somerset
COMBER, T., Pickering, Yorks
CONOLLY, CHARLES HAMILTON, Stuart Villa, Crescent Gardens,
Wood Green
COOKE, ALFRED S., Badbrook House, Stroud, Gloucestershire
COOMBS, Dr., Bedford
COOPER, A., 9 Henrietta Street, Cavendish Square, W.
COOPER-KEY, Dr. A., 30 Wilton Place, Belgrave Square,
S.W.
CORRIE, ALFRED, Fleet-Surgeon, R.N., H.M.S. "Resolution,"
Channel Squadron, Portland.
COSENS, C. H., 49 Oxford Terrace, Hyde Park, W.
COWIE, Dr. A. J., Halifax, Nova Scotia
COWLEY, J. S., Upton-on-Severn, Worcestershire
CRACE-CALVERT, G. A., Esslemont, 10 Macaulay Road, Clapham,
S.W.
CRAVEN, R. M., 14 Albion Street, Hull
CRIPPS, W. H., 2 Stratford Place, W.
CRONK, H. G., Repton, near Burton-on-Trent
CROSSE, R. E., East Dereham, Norfolk

- CROSSLEY, E. W., The Dean, Triangle, Halifax, Yorkshire
CROUCH, A. P., 3 Princes Buildings, Weston-super-Mare
CROWFOOT, Dr. W. M., Beccles, Suffolk
CRUMP, J. A., c/o T. Underhill, Son & Crump, Great Bridge,
Tipton, Staffordshire
CUMBERBATCH, A. E., 80 Portland Place, W.
CUMŞTON, C. G., Square de Champel, 8 bis, Geneva, Switzer-
land
CUTHBERT, C. F., 84 Barton Street, Gloucester

DALBY, J., 4 Rowderton Road, Newton Abbot
DALE, C. B., Hatfield, Herts
DAVEY, Dr. ALEXANDER G., 9 Belvedere Street, Ryde, Isle of
Wight
DAVIDSON, HAROLD, White House, Teddington
DAVIES, Dr. ARTHUR, 23 Finsbury Square, E.C.
DAVIS, Dr. T., Beechcroft, Clevedon, Somerset
DAY, DONALD D., 4 Upper Surrey Street, Norwich
DAYMAN, BARNFIELD, Millbrook, Southampton
DEAN, C. W., Infirmary, Lancaster
DINGLE, Dr. W. A., 46 Finsbury Square, E.C.
DINGLEY, ALLEN, 11 Upper Woborn Place, W.C.
DIXON, F. J., General Hospital, Birmingham
DORAN, ALBAN H. G., 9 Granville Place, Portman Square, W.
DOWLING, Dr. N., Portland, Victoria, Australia
DRAGE, Dr. C., Hatfield, Herts
DUCKWORTH, Sir DYCE, 11 Grafton Street, Piccadilly, W.
DUDFIELD, R., 19 Bloomfield Road, Maida Vale, W.
DUNN, PHILIP, Stevenage, Herts
DUNN, H. P., 54 Wimpole Street, W.
DURHAM, ARTHUR E., 82 Brook Street, W.
ECCLES, GEORGE H., Sherwell House, Plymouth
ECCLES, W. M'ADAM, 124 Harley Street, Cavendish Square, W.
EDELSTEN, Dr. E. A., Quernfield, Latchford, Warrington
EDWARDS, C. R., Charlestown, Nevis, West Indies

- EDWARDS, F. S., 55 Harley Street, W.
 EDWARDS, H. NELSON, Moreton House, Shrewsbury
 ELKINGTON, THOMAS, Fenny-Compton, Leamington
 ELLIOTT, Dr. J., Whitefriars Lodge, Chester
 ELLIS, Dr. J. W., Swavesey, Cambridgeshire
 ELLIS, Dr. W. G., Medical Superintendent, Lunatic Asylum,
 Singapore, Straits Settlement
 EVANS, ERNEST, Hertford
 EVANS, E. L., 11 Gloucester Terrace, W.
 EVANS, Dr. J. TASKER, Jun., Hertford
 EVANS, Dr. NICHOLL, Cheshunt, Herts
 EVANS, Dr. F. W., 21 Charles Street, Cardiff
 EVERETT, E. W., 60 Pitt Street, Norwich
 EVILL, F. C., 43 Gloucester Gardens, Hyde Park, W.
- FABER, J. GREY, Castlegate, Stockton-on-Tees
 FAVELL, R., 348 Glossop Road, Sheffield
 FERGUSON, Dr. G. B., Altidore Villa, Pitville, Cheltenham
 FIELD, F. A., Friern Lodge, 449 Lordship Lane, S.E.
 FIELDING-CLARKE, F., Rosedene, Omsberley Road, Worcester
 FIRTH, Dr. C., 196 Parrock Street, Gravesend
 FLETCHER, A. C., The Charterhouse, E.C.
 FLETCHER, Dr. H. M., 98 Harley Street, W.
 FLINT, Dr. ARTHUR, Westgate Lodge, Westgate-on-Sea
 FOULERTON, ALEX. G. R., 22 Orrington Gardens, South Ken-
 sington
 FOWLER, C. H., Potter's Bar, Middlesex
 FOX, HERBERT, 25 South Parade, Southsea
 FRANCIS, A. G., 31 Albion Street, Hull, E. Yorks.
 FRANCIS, H. A., East Sooke, Victoria, British Columbia, Canada
 FRAZER, F., Library, St. Bartholomew's Hospital, E.C.
 FURNER, WILLOUGHBY, 13 Brunswick Square, Brighton
- GABB, C. B., 3 Wellington Square, Hastings
 GALE, F. W., Kaikoura, New Zealand

- GARDNER, Dr. H. W., 22 Swan Hill, Shrewsbury
GARROD, Dr. A. E., 9 Chandos Street, Cavendish Square, W.
GAY, JOHN, 119 Upper Richmond Road, Putney
GAYFORD, Dr. C., 52 Fleet Street, E.C.
GAYTON, Dr. F. C., Surrey County Asylum, Brookwood,
Woking
GEE, Dr. S., 31 Upper Brook Street, W.
GELL, Dr. H. WILLINGHAM, 36 Hyde Park Square, W.
GIFFARD, D. W., 5 Pavilion Parade, Old Steyne, Brighton
GIFFARD, H. E., Denham House, Egham, Surrey
GILBERTSON, Dr. J. B., 2 Starkie Street, Winckley Square,
Preston
GILBERTSON, J. H., The Limes, Hitchin
GILL, R., M.B., F.R.C.S., 72 Wimpole Street, W.
GILMOUR, R. WITHERS, Newholme, Addlestone, Surrey
GIMSON, Dr. W. G., Witham, Essex
GIPPS, A. G. P., Royal Naval Hospital, Plymouth
GIRVIN, J., c/o Principal Medical Officer, Cork District,
Ireland
GLEDDEN, Dr. A. M., c/o L. Bruck, 13 Castlereagh Street,
Sydney, N.S.W.
GLYNN, Dr. THOMAS R., 62 Rodney Street, Liverpool
GODSON, Dr. CLEMENT, 9 Grosvenor Street, W.
GOODSALL, D. H., 17 Devonshire Place, W.
GORHAM, R. V., Sans-Souci, Yoxford, Suffolk
GOW, Dr. W. J., 27 Weymouth Street, W.
GRANT, Dr. DUNDAS, 8 Upper Wimpole Street, W.
GREEN, F. K., 3 Gay Street, Bath
GREEN, S., Army Medical-Staff, c/o Messrs. Holt & Co., Agents,
17 Whitehall Place, London, S.W.
GRELLET, CHARLES S. BANCROFT, Hitchin, Herts
GRIFFITH, Dr. WALTER, 96 Harley Street, W.
GROSVENOR, W. W., 18 Clarence Street, Gloucester
GÜTERBOCK, Dr. PAUL, Berlin, per Messrs. Lessor & Co., 2 South
Parade, St. Mary's, Manchester

- HABERSHON, Dr. S. H., 70 Brook Street, Grosvenor Square, W.
 HAIG, Dr. ALEXANDER, 7 Brook Street, Grosvenor Square, W.
 HAINES, Dr. J. W., Library, St. Bartholomew's Hospital, E.C.
 HALL, Dr. ARTHUR J., 263 Glossop Road, Sheffield
 HALL, Dr. DE HAVILLAND, 47 Wimpole Street, W.
 HALL, F. A., 4 Albion Street, Lewes
 HAMER, Dr. W. H., Ladywell, Dartmouth Park Hill, Highgate, N.
 HARDING, C. O'B., West House, Chiswick Place, Eastbourne
 HARDY, F. W., Surgeon-Captain, Ceylon
 HARRIS, J. D., 45 Southernhay, Exeter
 HARRIS, SAMUEL, Quorn, Loughborough, Leicestershire
 HARRISON, Dr. A. J., Failand Lodge, Clifton, Bristol
 HARRISON, Dr. CHARLES, 30 Newland, Lincoln
 HARRISON H. LEEDS, 43 Marine Parade, Worthing
 HAYNES, E. L., 10 Bridge Road, Stockton-on-Tees
 HAYNES, Dr. F. H., 23 Lansdowne Place, Leamington
 HEATH, A., Library, St. Bartholomew's Hospital, E.C.
 HEATH, W. L., 90 Cromwell Road, South Kensington, S.W.
 HEMBROUGH, Dr. J. W., The Moat Hall, Newcastle-on-Tyne
 HENSLEY, Dr. PHILIP, 4 Henrietta Street, Cavendish Square, W.
 HERBERT, C. H., Library, St. Bartholomew's Hospital, E.C.
 HERRINGHAM, Dr. W. P., 13 Upper Wimpole Street, W.
 HEWETT, AUGUSTUS, 1 Cambridge Park, Twickenham
 HILL, Dr. ALEXANDER, Downing College Lodge, Cambridge
 HILL, J. WRIGHT, 117 Albert Road, North Woolwich, E.
 HILLABY, A., Richmond House, Pontefract
 HIND, A. E., 60 New Street, St. Heliers, Jersey
 HIND, HENRY, Blythholme, Stockton-on-Tees
 HOGARTH, R. G., Milford House, Salisbury
 HOGG, A. J., Leslie Lodge, Haven Green, Ealing, W.
 HOLDEN, Dr. G. H. R., 168 Castle Hill, Reading
 HOLDEN, LUTHER, Pinetoft, Rushmere, Ipswich
 HOLLIS, Dr., 8 Cambridge Road, Brighton
 HORTON-SMITH, Dr. P., 53 Queen's Gardens, Hyde Park, W.
 HORDER, T. J., Library, St. Bartholomew's Hospital, E.C.

- HORNE, W. J., 8 Glazbury Road, West Kensington, W.
HOUGHTON, P. A., The Châlet, Lingfield, Sussex
HOWARD, Dr. H., Williamstown, Melbourne, Victoria
HOYLAND, S. S., 10 Museum Street, Ipswich
HUGHES, D. WATKIN, Wymondham, Norfolk
HUGHES, J. B., Roe Street House, Macclesfield, Cheshire
HUGHES, S. H., Library, St. Bartholomew's Hospital, E.C.
HUMPHRY, C. H., Lower Camden, Chislehurst, Kent
HUMPHRY, F. A., 25 Marine Parade, Brighton
HUMPHRY, Dr. L., 3 Trinity Street, Cambridge
HUSBAND, W. E., 56 Bury New Road, Manchester
HUSSEY, E. L., 24 Winchester Road, Oxford
HUTCHINSON, J., 15 Cavendish Square, W.
HUTTON, E. R., 18 West Green Road, Tottenham
- LIFFE, W., 41 Osmaston Street, Derby
IREDALE, J., Mablethorpe, R.S.O., Lincolnshire
- JACKMAN, T. S. H., 11 Stoke Newington Road, N.
JACKSON, ARTHUR, 53 Wilkinson Street, Sheffield
JACKSON, H. F. V., Potter's Bar, Middlesex
JACOBSON, G. OSCAR, St. David's Street, Presteign, Radnorshire
JAMES, PHILIP, 1 Bolton Street, Wellington, New Zealand
JAMES, Dr. EDWIN M., Belgrave Mansions, Grosvenor Gardens,
S.W.
JENKINS, Dr. E. J., The Australian Club, Macquart Street,
Sydney, Australia
JESSOP, E., 81 Fitzjohn Avenue, Hampstead, N.W.
JESSOP, W. H., 73 Harley Street, W.
JOHN, D., Nepperhan Avenue, Yonkers, New York, U.S.A.
JOHNSON, J. G., Evandale, Tasmania
JOLLIFFE, W. J., Yofford House, Isle of Wight
JONES, Dr. H. LEWIS, 9 Upper Wimpole Street, W.
JONES, Dr. ROBERT, London County Asylum, Claybury, near
Woodford, Essex

JOWERS, L. E., 51 Marina, St. Leonards-on-Sea

JOWERS, R. F., 29 Norfolk Square, Brighton

KAY, A. R., 35 Chester Terrace, S.W.

KAY, W., Bentley Cottage, Bentley, near Farnham, Hants

KEATS, W. J. C., Laurel Villa, Maryon Road, Charlton, S.E.

KEETLEY, C. R. B., 56 Grosvenor Street, Grosvenor Square, W.

KENDALL, Dr. T. M., Hyde Park, Sydney, Australia

KENNEDY, WILLOUGHBY, Burke House, Beaconsfield, Bucks

KESTEVEN, W. H., Hillwood, Waverley Grove, Hendon, N.W.

KIDD, Dr. P., 60 Brook Street, W.

KING, R. H., 9 Adelaide Road, Leamington Spa

KINGDON, J. A., 2 Bank Buildings, E.C.

KINSEY, R. H., 2 Harpur Place, Bedford

KNIGHT, H. J., Brooklands, Rotherham, Yorkshire

KOCH, Dr. W. V. M., Port of Spain, Trinidad, British West
Indies

LANGDON, THOMAS C., Northgate House, Winchester

LANGTON, JOHN, 62 Harley Street, W.

LATHAM, Dr. P. W., 17 Trumpington Street, Cambridge

LAUHLAN, H. D., Stanley Villa, Muswell Hill, N.

LAWRENCE, H. CRIPPS, 12 Sussex Gardens, Hyde Park, W.

LAWRENCE, L. A., 4 Queen Anne Street, W.

LEE, W. E., 25 St. Peter's Hill, Grantham, Lincolnshire

Leeds School of Medicine, per W. F. Husband, Esq., Sec., Leeds

LEGG, Dr. WICKHAM, 47 Green Street, Park Lane, W.

LEWIS, H. K., Medical Library, 136 Gower Street, W.C.,
four copies

Library of St. Bartholomew's Hospital, E.C.

LITTLE, H. SELBY, 106 London Street, Reading

LOCKWOOD, C. B., 19 Upper Berkeley Street, W.

LOW, Dr. C. W., Stowmarket, Suffolk

LOWE, Dr. WALTER GEORGE, 5 Horninglow Street, Burton-on-
Trent

- LOWE, G. J. R., 5 Cornhill, Lincoln
LOWNE, B. T., 18 St. Quintin's Avenue, North Kensington, W.
LUCAS, ALBERT, 9 Easy Row, Birmingham
LYSTER, A. E., Long Eaton, near Nottingham
- MACDOUGALL, Dr. J. A., 1 Boulevard Carnot, Cannes, France
MACKINDER, Dr., The Cedars, Gainsborough
MACREADY, J., 51 Queen Anne Street, W.
MAHER, C. H., College Street, Sydney, New South Wales
MAIDLOW, Dr. W. H., 14 Park Hill Rise, Croydon
Manchester Royal Infirmary, the Secretary, Manchester
MANTON, J. A., Shrewsbury House, Sheffield
MARK, LEONARD P., 61 Cambridge Street, Hyde Park Square, W.
MARSH, HOWARD, 30 Bruton Street, Berkeley Square, W.
MARSH, Dr. N. P., 7 Abercromby Square, Liverpool
MARTIN, P., Abingdon, Berks
MARTYN, REGINALD, 8 The Beacon, Exmouth, Devon
MASTERMAN, E. W. G., English Hospital, Jerusalem
MATHEWS, F. E., Welsh Row House, Nantwich, Cheshire
MATHEY, Dr. A., Georgetown, Demerara, West Indies
MAUDE, A., Winterton House, Westerham, Kent
MAUND, J. H., Brackley House, Newmarket
MAW, H. T., Holmesdale, Nutfield, Surrey
MAXWELL, J. P., 49 Highbury Park, N.
MAY, Dr. E. HOOPER, Tottenham High Cross, Middlesex
MEADE, R. H., Mount Royd, Bradford, Yorkshire
MENZIES, J. IRVINE, 47 Earl's Court Square, South Kensington,
S.W.
- MILSOME, Dr. J. R., Addlestone, Chertsey
MITCHELL, A. M., Library, St. Bartholomew's Hospital, E.C.
MITCHINSON, Dr., Lindum Holme, Lincoln
MOBERLY, SYDNEY C. H., Stretton, near Warrington
MOORE, Dr. NORMAN, 94 Gloucester Place, Portman Square, W.
MOORE, THOMAS, 6 Lee Terrace, Blackheath, S.E.
MORRICE, G. G., Kincardine House, Salisbury

MORRIS, Dr. C. A., 29 Eccleston Street, Eaton Square, S.W.

MORRIS, EDWARD, 7 Windsor Place, Plymouth

MOSELEY, C. K., 14 Northgate Street, Ipswich

MURIEL, C. J., 42 St. Giles Street, Norwich

MURIEL, J., Hadleigh, Suffolk

MYDDELTON-GAVEY, E. H., 124 Harley Street, W.

NANCE, H. CHESTER, 55 St. Giles' Plain, Norwich

NEWMAN, Dr. A., 70 Macklin Street, Derby

NEWMAN, Dr. W., Barn Hill House, Stamford

NEWSTEAD, J., 9 York Place, Clifton, Bristol

NEWTON, LANCELOT, Alconbury Hill, Hunts

NICHOLSON, G. B., Library, St. Bartholomew's Hospital, E.C.

NUNN, P. W. G., Maplestead, Christchurch Road, Bournemouth

ODLING, T. F., per Hickey & Borman, 14 Waterloo Place,
S.W.

OGLE, Dr. J. G., South Redlands, Reigate, Surrey

O'GRADY, E. S., 33 Merrion Square North, Dublin

O'KINEALY, F., Surgeon-Captain, I.M.S., c/o Messrs. King,
Hamilton & Co., 7 Hare Street, Calcutta, Bengal

OLDFIELD, F., 174A Boyson Road, Camberwell Gate, S.E.

ORELLA, Dr. F. R. COOPER, Medical College, San Francisco,
California, U.S.A.

ORMEROD, Dr. J. A., 25 Upper Wimpole Street, W.

ORTON, G. H., 1A Campden Hill Road, Kensington, W.

PAGET, Sir JAMES, Bart., F.R.S., 5 Park Square West, W.

PALMER, Dr. E. C., Lancaster House, Lincoln

PARDINGTON, Dr. G. L., 47 Mount Pleasant Road, Tunbridge
Wells

PARK, Professor ROSWELL, Medical Department, University of
Buffalo, New York

PARKER, C. A., Colne Valley, Rickmansworth, Herts

PARKER, G. D., 78 Dorset Street, Hulme, Manchester

- PARKER, G. R., 34 King Street, Lancaster
- PATERSON, H. J., Library, St. Bartholomew's Hospital, E.C.
- PAWLETT, T. L., Essex County Asylum, Brentwood
- PEACEY, DR. WILLIAM, Rydal Mount, St. John's Road, Eastbourne
- PEARSE, R. E. FRANKLYN, The Hospital, Jagersfontein, Orange Free State, South Africa
- PETTIFER, E. H., 50 Southgate Road, N.
- PHILLIPS, H., Royal Free Hospital, W.C.
- PIERCE, DR. BEDFORD, The Retreat, York
- Plymouth Medical Society, per Russell Rendle, C.E., 7 Buckland Terrace, Plymouth
- POLLARD, W. H., Tollesbury, Essex
- POLLARD, DR. R., Torquay
- POWELL, C. M., Betworth, Exmouth
- POWER, HENRY, 37A Great Cumberland Place, Hyde Park, W.
- POWER, D'ARCY, 10A Chandos Street, Cavendish Square, W.
- POYNDER, F. C., 8 Guildford Place, W.C.
- PRICHARD, AUGUSTIN, 4 Chesterfield Place, Clifton
- PRICKETT, DR. MARMADUKE, 12 Devonport Street, Gloucester Square, W.
- PRITCHARD, DR. OWEN, 37 Southwick Street, Hyde Park, W.
- QUATREY-PAPAFIO, DR. B. W., Momo's Hall, Aera, Gold Coast, West Coast Africa
- QUENNELL, JOHN C., Brentwood, Essex
- RADFORD, The Library, St. Mary's Hospital, Manchester, per Librarian
- RANKING, DR. J. E., 18 Mount Ephraim Road, Tunbridge Wells
- RAVEN, THOMAS FRANCIS, Barfield House, Broadstairs
- READ, H. G., 1 Portland Place, W.
- READ, DR. MABYN, 42 Foregate Street, Worcester
- REECE, DR. R. J., Local Government Board, Whitehall, S.W.

- REES, J. M., 53 Devonshire Street, Portland Place, W.
 REID, JAMES, 12 Lower Bridge Street, Canterbury
 REID, T. WHITEHEAD, St. George's House, Canterbury
 RENDALL, Dr. P., Devonshire Club, St. James's Street, S.W.
 REYNOLDS, H. W., 28 St. Saviour Gate, York
 RICE, Dr. EDWARD, 90 Woodstock Road, Oxford
 RIGGE, J. A. M., Henley-on-Thames, Oxon
 RISK, E. J., Surgeon-Major, Army Medical Staff, c/o Messrs.
 Holt, Laurie & Co., Bankers, 17 Whitehall Place, London
 RIVERS, W. H. R., Library, St. Bartholomew's Hospital, E.C.
 ROBERTS, CHAS. H., 21 Welbeck Street, W.
 ROBINSON, HAYNES, 35 St. Giles' Plain, Norwich
 ROBINSON, G., Harpur Place, Bedford
 ROGERS, W. J., 107 Lordship Lane, S.E.
 ROUGHTON, Dr. E. W., 33 Westbourne Terrace, Hyde Park, W.
 Royal Medical and Chirurgical Society, 20 Hanover Square, W.,
 per J. Y. W. MacAlister
 ROYDS, W. A. S., St. Mary Bourne, near Andover, Hants
 RUMBOLL, C. F., Lowborne House, Melsham, Wilts
 RUNDLE, H., 13 Clarence Parade, Southsea
 RUSHWORTH, NORMAN, Beechfield, Walton-on-Thames
 RUST, H. R. G., Wethersfield, Braintree
- SALMON, Dr. A. G., Bodmin, Cornwall
 SANTI, P. DE, 91 Harley Street, W.
 SAUNDERS, A. L., The Limes, Grimston, King's Lynn
 SAUNDERS, F. W., Chieveley House, Chieveley, near Newbery,
 Berks
 SAUNDERS, G. R., Balgounie, Wanganui, New Zealand
 SAVORY, Dr. C. T., 6 Douglas Road, Canonbury, N.
 SCOTT, Dr. JOHN, Bromley, Kent
 SELBY, P. G., Bruson House, Greenstreet, near Sittingbourne,
 Kent
 SHADWELL, H. W., "Fernleigh," Arniston Road, East Mosley,
 Surrey

- SHARPIN, A. L., 2 Kimbolton Road, Bedford
SHARPIN, E. C., Bedford
SHAW, JOSEPHUS, 151 Lower Road, Rotherhithe
SHAW, Dr. T. CLAYE, Middlesex County Lunatic Asylum, Banstead Downs, Sutton
SHELLY, Dr. C. E., Hertford
SHORE, Dr. T. W., The Warden's House, St. Bartholomew's Hospital, E.C.
SIDEBOTHAM, E. J., Erlesdene, Bowdon, Cheshire
SIMMONS, H. C., Standerton, Transvaal
SIMPSON, S. H., Romsey, Hants
SKELDING, H., St. Loyes, Bedford
SLATER, Dr. D. J., 4 Courtfield Road, Gloucester Road, S.W.
SMITH, E. CLOETE, The Limes, Ingatestone, Essex
SMITH, H. L., Buckland House, Buckland Newton, Dorset
SMITH, Dr. T. GILBART, 68 Harley Street, W.
SMITH, THOMAS, 5 Stratford Place, Oxford Street, W.
South London Medical Reading Society, per Dr. H. Taylor, 180 Kennington Park Road, S.E.
SOUTHEY, Dr. R., 32 Grosvenor Road, Westminster, S.W.
SOUTTER, J., Souttergate Hedon, Holderness, Hull
SPACKMAN, H. R., Penn Fields, Wolverhampton
SPICER, Dr. W. T. H., 47 Welbeck Street, Cavendish Square, W.
SPREAT, F., The Firs, Oakleigh Park, Whetstone, N.
SQUARE, J. E., 22 Portland Square, Plymouth
Stamford Infirmary, Medical Book Society, Stamford
ST. CYR. DUMAINE, Aux Cayes, Haiti, West Indies
St. Bartholomew's Hospital, The Governors of, thirty copies
STEEDMAN, J. F., 110 High Road, Streatham, S.W.
STEELE, H. F., Stoke Ferry, Brandon, Norfolk
STEER, A. W. T., Trevear, Penzance, Cornwall
STEPHENS, J. W., Tymaw, Cardigan
STEVENS, Dr. A. FELIX, 13 High Street, Stoke Newington, N.
STEVENS, C. R., 6 Middleton Street, Calcutta

- STEVENSON, N., 51 Wimpole Street, W.
 STONEY, P. BUTLER, Holborn Hill, Millom, Cumberland
 STREET, Dr. A. F., Westgate-on-Sea
 STRUGNELL, F. W., Grove End Road, Highgate Road, N.W.
 STRUGNELL, Dr. W. T., Merton Lodge, 213 Brixton Hill, S.W.
 STUBBS, P. B. TRAVERS, Rochester, Salt River, P. O. Cape,
 South Africa
 STYAN, Dr. T. G., Chapel Place, Ramsgate
 Suffolk Medical Book Society, care of Messrs. Pawsey &
 Hayes, Ipswich, per Hayden
 SURRIDGE, E. E. N., High Leigh, Knutsford
 SYLVESTER, K. F., Trowbridge, Wilts
 SYMONDS, Dr. H., Carnarvon Hospital, Kimberley, South
 Africa
 SYMPSON, Dr. E. M., Deloraine Court, Lincoln
- TAIT, E. S., 48 Highbury Park, N.
 TAIT, H. B., Lincluden, Sunnyside Road, Hornsey Lane, N.
 TAYLER, Dr. G. C., Trowbridge, Wilts
 TERRY, HENRY G., 16 Green Park, Bath
 THOMPSON, G. H., Buxton, Derbyshire
 THORNE, Dr. R. THORNE, 45 Inverness Terrace, Kensington
 Gardens, W.
 TOOTH, Dr. H. H., 34 Harley Street, W.
 TRECHMANN, MAC., L., Clyde Villa, Selhurst Road, South Nor-
 wood, S.E.
 TREVAN, F. A., Staff-Surgeon R.N., care of Messrs. Banton,
 Mackrell & Co., 26 Budge Row, Cannon Street, E.C.
 TRINDER, A. P., West Lulworth House, West Lulworth, Dorset-
 shire
 TROLLOPE, Dr., 9 Maze Hill, St. Leonards-on-Sea
 TROUTBECK, H., 4 Dean's Yard, Westminster, S.W.
 TURNBULL, Dr. G. L., 76 Ladbroke Grove, Notting Hill, W.
 TURNER, E., 159 Brigstock Road, Thornton Heath, Surrey
 - TURNER, F. H., The Gothics, East Bergholt, Suffolk

- UPTON, A., 52 Lansdown Place, Brighton
UPTON, H. C., 28 Medina Villas, Hove, Brighton
- VAUGHAN, ALFRED E., Crewe Cottage, Crewe
VERANO, L. L., Gibraltar
VERRALL, T. J., 97 Montpellier Road, Brighton
- WALCH, DR. C. C., 54 Macquarie Street, Hobart, Tasmania
WALKER, E. G. A., Ightham, Sevenoaks, Kent
WALLER, T. H., Museum Terrace, Chelmsford
WALLIS, F. C., 26 Welbeck Street, Cavendish Square, W.
WALLIS, G., 6 Hills Road, Cambridge
WALSHAM, W. J., 77 Harley Street, Cavendish Square, W.
WARD, S. E., Rectory Lodge, Brasted, Sevenoaks
WARDE, DR. A. W., 82 Guildhall Street, Folkestone, Kent
WARING, H. J., 9 Upper Wimpole Street, W.
WATERHOUSE, J. H., M.D., Maltby, Rotherham, Yorks
WATTS, H., County Asylum, Gloucester
WEBB, H. S., Welwyn, Herts
WEST, DR. SAMUEL, 15 Wimpole Street, W.
WHARRY, DR. R., 6 Gordon Square, W.C.
WHITE, C. PERCIVAL, 144 Sloane Street, S.W.
WHITE, C. POWELL, 67 Queen Street, London, E.C.
WHITE, W. H., 39 Maningham Lane, Bradford, Yorkshire
WHITEHEAD, H. E., 475 Caledonian Road, N., two copies
WHITMORE, W. TICKLE, 7 Arlington Street, Piccadilly, S.W.
WHITWELL, A. F., 27 Castle Street, Shrewsbury
WILKINS, H. G. G., The Green, Ealing
WILKS, DR. GEORGE, Ashford, Kent
WILKS, J. H., Rutland Park, Sheffield
WILLARD, S. D., 143 Victoria Street, Westminster, S.W.
WILLETT, A., 36 Wimpole Street, W.
WILLETT, E., 25 Welbeck Street, W.
WILLEY, THOMAS, Library, St. Bartholomew's Hospital, E.C.
WILLIAMS, J. T., Rossall House, Barrow-in-Furness, Lancashire

- WILLSON, A., 1 Church Street, Horncastle, Lincolnshire
WINGATE-SAUL, Dr. W. W., Fenton-Cawthorne House, Lancaster
WINKFIELD, ALFRED, 26 Beaumont Street, Oxford
WOMACK, Dr. F., 9 Dennington Park Road, West Hampstead,
N.W.
WOOD, FREDERICK, 12 Lewes Crescent, Brighton
WREFORD, H., 18 Belsize Grove, Hampstead, N.W.
WRIGHT, Dr. J. C., Park Road, Halifax
WYER, Dr. OTHO, Epperston House, The Avenue Road,
Leamington

YARROW, Dr. G. E., 26 Duncan Terrace, Islington, N.
York Medical Society, 1 Low Ousgate, York, per Hon. Sec-
retary

*The Subscription List in each year will be closed on the
First of October.*

NOTICE TO SUBSCRIBERS.

It is particularly requested that Subscriptions be remitted without delay, as an acknowledgment of the receipt of the volume. If not paid for before the First day of July 1897, the volume will be charged as a Non-Subscriber's copy.

Post-Office Orders to be made payable at the General Post-Office to Mr. P. FRANCIS MADDEN, the Library, Saint Bartholomew's Hospital.

Price to Subscribers, Six Shillings; to Non-Subscribers, Eight Shillings and Sixpence.

An Index to the first twenty volumes, prepared by Dr. Church, is issued in a separate volume, price 3s. 6*d.* to Subscribers, 5s. Non-Subscribers.

January 1897.

In Memoriam.

SIR GEORGE MURRAY HUMPHRY.

BY

C. B. LOCKWOOD.

On September 24, 1896, George Murray Humphry passed away, and St. Bartholomew's lost one of the best and ablest of her sons. He was born on July 18, 1820, at Sudbury, in Suffolk. His father, William Wood Humphry, was a barrister-at-law, and, like others of the family, a person of consideration in the town and county in which he dwelt. When Sir George Humphry was born, English towns had grammar-schools, which played no unimportant part in the lives of succeeding generations of inhabitants. In those days of difficult communication, the grammar-schools afforded the sons of the local gentry a sound and classical education. Therefore, we learn that Sir George Humphry went first to the Grammar School at Sudbury, and afterwards to Dedham. His school-days were soon over. When sixteen years old he set off to Norwich, to become the apprentice of J. G. Crosse. This surgeon was a skilful operator and of scientific bent. In after years his pupil spoke of him with great respect and admiration. There can be no question but that he exercised a powerful influence for good upon the mind of his pupil. Next door to his surgery he had a large room fitted up as a library and museum. At night, when the day's work was done, he was wont to sit there and pore over his specimens, and spend his spare time in mounting new ones. This room was open to the pupils, and it is easy to imagine what an influence such opportunities and such an example must have had upon the keen and receptive mind of Humphry. Doubtless this was the beginning of that love of morbid anatomy which characterised him throughout his life. In other respects the pupilage at Norwich must have borne good fruit.

But a few years passed, and he went to Cambridge, where he had a large and successful country practice, similar in many respects to that of his first master and teacher.

When nineteen years old the Norwich pupil came to St. Bartholomew's. Since then at least six of his family have come and gone, and they have one and all upheld the name. Of his teachers, Sir George Humphry in after years spoke oftenest of Latham, Lawrence, and Paget. To the last he must have been greatly drawn, for Paget was then curator of the Museum, and engaged in the pathological work which was destined to make him famous. Also, during his student days, he showed his talent for anatomy by winning the gold medal in London University. It is true that this was awarded for anatomy and physiology, but in those days (1840) there was not such a gulf betwixt those subjects as we see now. The lectures of Dr. Latham impressed him deeply. He over and over again spoke of the clear and emphatic style of that distinguished physician. Ofttimes his remarks were quoted, and no one could doubt but that, when called upon to lecture himself, he took Dr. Latham's style as his model. It is not to be inferred from this that he was a servile imitator. On the contrary, his mind was too strong and original to betray such evidences of mediocrity.

In 1842 he obtained the diplomas of the Apothecaries' Society and of the Royal College of Surgeons of England. Those examinations were not so rigorous as they now are, but, nevertheless, no longer accorded with Smollett's well-known description. In the same year the post of surgeon to Addenbroke's Hospital at Cambridge fell vacant. His abilities and meritorious conduct won the help of Mr. Paget and of others, and thus he became, as he was wont to say, the youngest hospital surgeon in England. The work of this appointment was carried out with extreme energy. A course of surgical lectures was the earliest fruit. Always an enthusiastic and altogether admirable teacher, he at once began the work of his lifetime. Henceforth generations of youth owed to him the greatest boon that one human being can confer upon another, namely, the gift of independent and accurate thought. The lectures were an innovation, and permission to deliver them was obtained after opposition. Those who opposed could not have foreseen that these lectures marked such an important epoch in the history of the Cambridge Medical School. But soon his remarkable energies were diverted into a new channel. Strange as it may seem to us, the Chair of Anatomy in the University of Cambridge was at that time filled by a Reverend

gentleman, Doctor W. Clark. This gentle and worthy professor also undertook comparative anatomy and zoology. Under the circumstances, it is easy to understand that that branch of anatomy which is most essential to medicine and surgery may not have had the attention to which it was justly entitled. The good Dr. Clark soon became apprised of the advent of one who was highly qualified to afford him the help which the growing demands of human anatomy had rendered necessary. He accordingly invited Humphry to undertake the lectures and demonstrations on this subject. This was one of the various causes which led him to become a Fellow-commoner of Downing College (in 1847), and to proceed in due course to the degrees of Bachelor of Medicine (1852) and Doctor of Medicine (1859). Some time before, when the order of Fellows of the Royal College of Surgeons of England was founded in 1844, he, by virtue of his post at Addenbroke's, was elected a Fellow; a prelude to high and honourable offices in the College. In later years he became a member of the Court of Examiners, Arris and Gale Lecturer, and Hunterian Orator. The high offices of Vice-President and President he could not hold, because his Cambridge work was as much as his strength would allow him to do. He was too faithful in his allegiance to neglect the interests of his rapidly-growing school.

The labour of organising that which was almost a new anatomical department must have been great. A proper supply of subjects for dissection was most difficult to obtain, and many would have been daunted by the prejudices which had to be overcome, and by the unpopularity which had to be incurred, before a proper supply could be secured. During these early years he collected the materials for and wrote his profound and philosophical treatise on the Human Skeleton. No one can read this without a deep sense of the care and sagacity of the author, and of his truthfulness and learning. How many succeeding lecturers in anatomy have not delved into this rich and precious mine for jewels wherewith to adorn their teaching? No small part of the merit of this work was due to the clear and truthful drawings with which it was illustrated. These were drawn from the specimens by his wife, an artist of no mean gifts, and who was steeled by her devotion to her husband to perform a task so uncongenial to a cultured and delicate lady. Other works upon Human and Comparative Anatomy followed. His essay on the Limbs of Vertebrate Animals, and Observations on Myology, are accurate and thorough, and interspersed with shrewd and thoughtful lucubrations. The merit of these essays is apparent when we see that so many turn to them for

information, and quote them as authoritative writings. By these labours Humphry's scientific reputation was established, and in their midst the Royal Society elected him one of its Fellows in 1859. Some of his writings were not of such a severe type as those which have been mentioned, as, for instance, his book on the Human Foot, and that on Old Age, an inquiry which he pursued with characteristic energy and enthusiasm, and was at great pains to see himself many of the old people of whom he wrote. Some, too, will learn for the first time that he was the author of a useful and very interesting guide to Cambridge.

To trace the career of Humphry is to trace the rise of the Cambridge Medical School. In 1866 the Rev. Dr. Clark resigned, and a new Professorship of Human Anatomy was founded. Henceforth Dr. Humphry was known as Professor Humphry; a name by which most of us knew him best, and which always seemed to be so thoroughly fitted to his personality. At first the emoluments of the new Chair were inconsiderable, and hardly sufficed for the expenses; but by the year 1883 they began to reach a substantial sum. Thereupon Professor Humphry voluntarily made way for a successor who could devote all his time and energies to the Professorship of Anatomy, whilst he himself undertook, without remuneration, a Professorship of Surgery, which the University founded.

But little perspicacity is needed to tell any one that at each of these steps a master-mind was at work for the advancement of the Cambridge Medical School. Those who are familiar with such undertakings know the difficulties which have to be overcome, the opposition which has to be conciliated, and the rarity of men who have the ability and tact to carry them to a successful issue; they also know that the victory cannot be won without some having suffered, and without some traces of resentment lingering in the minds of the vanquished. At this time, too, we have to remember that Professor Humphry was engaged in a laborious surgical practice, and occupied with work at the College of Surgeons, the Medical Council, and the learned societies. Surgical practice alone made great demands upon his endurance, long and dismal journeys by road in one of the bleakest parts of England, often to do an operation which alone would have severely taxed the strength of any one. A mere enumeration of the offices he filled and the honours which were conferred upon him would prove the extraordinary activity and industry of his life and the esteem in which he was held. President of the Cambridge Medical Society, President of the Cambridge Graduates' Club, President of the Pathological

Society of London, first President of the Anatomical Society of Great Britain and Ireland, Honorary Fellow of Downing College, Fellow of King's College, LL.D. of the University of Edinburgh, and Honorary Doctor of Science of the University of Dublin, all were his. Further, in 1893 the honour of knighthood was conferred upon him. Surely no one would deny that that guerdon was well won.

As a surgeon Sir George Humphry attained a high position. To a shrewd and penetrating mind he added a profound knowledge of anatomy and pathology, dauntless courage, and great manipulative skill. Like many who have excelled in the art, a great operation was for him a severe trial, thought of with apprehension, and undertaken with a heavy heart. But once engaged, his steadfast courage carried him through difficulties which would have appalled a weaker man. His work on Resection of the Knee-joint did much to place that operation upon a firm basis. His essay on the Coagulation of the Blood in the Venous System during Life is replete with original and striking observations. It is in other respects characteristic of the author. Thrice he had thrombosis of the veins of the leg. He laughed at those who said he would die; and when he recovered, the essay was the memorial of his illnesses. His other contributions to the science of surgery are not few, but better than all of them is the tradition he has left to the Cambridge School of correct and careful observation, followed by clear and rational inference. When busy with the beloved task of teaching students how to see and think, he had ever upon his lips the words, "Eyes first and much, hands next and least, tongue not at all." To see him going round the wards of Addenbroke's with his class was a privilege to any one, and a liberal education in the art of imparting knowledge.

Like all good surgeons, he had a genuine love of morbid anatomy. The Cambridge Museum was his particular pride. Nothing afforded him greater pleasure than to display its treasures, except, perhaps, the acquisition of new ones. No trouble or expense was too great when a choice specimen could be obtained, and the cataloguing of them was a labour of love. Long and often did he sit in the Museum surrounded by his pupils, teaching them pathology, and telling them never to forget to send him whatever they could obtain for the Museum.

He who worked so hard and accomplished so much was never of strong physique. Of rather more than middle height, his body was thin and slender, almost emaciated; his complexion pale, and his aquiline features sharp and delicate, betokened

keen intelligence. His natural pallor was perhaps increased by the darkness of his hair, moustache, and beard, which remained black until the end. Although his features have oftentimes been delineated on canvas or in marble, no artist has quite caught that expression of keen attention and thoughtfulness which told that they characterised no ordinary man. The changing mood of his thoughts was reflected in his face, and was as hard to fix upon canvas as the changeful ripple of the sea. Standing was always a fatigue to his frail body, and was followed by pain in the back. At one time his friends were in dread lest he should be carried off by consumption. Despite these physical disadvantages, he lived a life of incredible activity. In thinking of all he did we are reminded of the herculean labours of that genius John Hunter. After a hard day's work in Cambridge he thought little of going to London to take part in the work of a society. Even in the midst of his last illness he still, with indomitable courage, busied himself with affairs, attended various functions, and entertained his old pupils and friends for the last time, endeavouring to soften for them the sadness of farewell.

When he spoke, his steadfast eyes gave a penetrating glance. His questions were clear and to the point. All that he said was couched in the clearest language. Without possessing that gift of oratory which enthral the senses and captivates the mind, nevertheless he could achieve that which is the end of all true oratory, for he nearly always carried his point and convinced his audience. His keen and subtle intellect often suggested arguments which were original and striking, and were calculated to display the depths of his knowledge.

In his home life Sir George Humphry was singularly happy. In 1849 he married Mary, the daughter of Mr. Daniel Robert M'Nab, surgeon, of Epping, in Essex. This true gentlewoman was a fitting helpmate. We have already seen how she made the drawings for many of his writings. In addition, her artistic sensibilities did much to adorn and beautify his home. The entertainment of numberless under-graduates and visitors seemed to be a never-failing pleasure to the household. None was more welcome than the friendless student, to whom this most kindly hospitality might be a source of pleasure, and even an influence for good. In this way many a new pupil learnt that he who had hitherto been thought of as Don and Professor had another and most charming side to his character. It was always very pleasant to see how fully their most thoughtful and kindly host interested himself in their ways and studies, and how much he sympathised with them in their difficulties.

Indeed, his mind always retained a singular youthfulness, due, no doubt, to this most wise and kindly intercourse.

That his pupils admired and looked up to him goes without saying. No one can talk long to any of them without his name being mentioned, or some wise and pregnant apothegm quoted. No wonder, too, that he won such a great share of their kindly regard and affection. Their destinies and careers were clearly remembered, their visits to their old master gladly welcomed, and advice and help freely bestowed. Until the last his mind was busied with the latest work, and he delighted to discuss whatever was new with his pupils and with his numerous friends amongst the younger and rising men. His mind was fully alive to and sympathetic towards the discoveries of bacteriology and their application in surgical practice. Unlike some of his contemporaries, he appreciated to the utmost the great discoveries which revolutionised surgery during the latter years of his active career.

This brief narrative tells the leading traits of Sir George Humphry's character. But, in addition, were unending deeds of private charity and liberality, which, although unspoken, were not unknown; deeds, too, of unostentatious friendship, filling many with deep-felt gratitude. When at length his last lingering illness reached its inevitable goal, and it was known that he had laid his

“ . . . just hands on that golden key
That opes the palace of eternity,”

deep and sincere were the words of sorrow and regret.

In Memoriam.

WILLIAM MORRANT BAKER.

BY

ALFRED WILLETT.

William Morrants Baker, until some $4\frac{1}{2}$ years ago one of the Surgeons to this Hospital, died on October 3, 1896, at his residence in Sussex—Nutbourne Manor, Pulborough—in his fifty-seventh year. Morrants Baker's professional career well-nigh terminated in the spring of 1892, when, in consequence of the steadily progressing advances of disease—locomotor ataxia—his physical strength had become so reduced that, feeling he could no longer conscientiously attempt to carry out the duties of his office, he placed his resignation as Surgeon in the hands of the Governors. From this date onwards, leading the life of an enforced recluse, and residing either in Woburn Square, London, or at Nutbourne, but with ever-increasing loss of muscular power, Morrants Baker gradually faded away until death released him from sufferings endured heroically. Probably for the last ten years of his life, Morrants Baker had rarely if ever passed a day free from pain, whilst often he suffered most acutely; yet he was never known, at least to his friends, to murmur at his lot, which he faithfully accepted as the all-wise decree of Providence.

William Morrants Baker came of a Hampshire family, born at Andover on the 20th October 1839, the second son of a solicitor in practice in that town. His father died comparatively young, and whilst the subject of this notice was still in his teens. Morrants Baker was educated at the Andover Grammar School, upon leaving which he was apprenticed to a local surgeon, Mr. Payne. Of his boyhood not much is known. Morrants Baker has told of his partiality for all school-boy games; he was a very fair cricketer and a powerful hitter; but,

probably from being myopic, he evinced but little inclination or aptitude for sports in general, although as a young man he was very partial to boxing, and with the gloves could hold his own. In later life he was often to be seen watching matches at "Lord's" or "The Oval;" indeed he was a very good judge of all-round cricket.

Entering at St. Bartholomew's at the age of 19, on October 1, 1858, Morrants Baker at once attracted the attention of, and soon became a marked man among, his fellow-students; for even at that date his personality was remarkable in many ways. His frame was large, well set, inclining to be massive; his features refined and regular, forehead high and broad; then with abundant hair and luxuriant whiskers, he appeared so much older than he really was, and so much senior to the majority of his fellow-students: this aspect was increased, moreover, by a very sedate demeanour and a thoughtful, earnest expression. Indeed, at this time Morrants Baker was a remarkably handsome and attractive man, standing 5 feet 10 inches in height, and not far short of 13 stones in weight, broad-shouldered and robust; he possessed an open, honest, taking manner that charmed all with whom he was associated. From the first he gave himself up to his medical studies, and quickly gathered around him a group of hard-working students; for even thus early Morrants Baker was a leader in all good ways. His influence and example were ever exerted in the right path, and never would he countenance in act or deed anything that was low or debasing to the fair name of "gentleman." His fellow-students liked and respected him, and soon understood, not so much by word as by a more telling expression he would put on, that if any man wished to be of his set, he must be also of his way of thinking, acting, and speaking. Throughout his student's career, Morrants Baker's conduct and work were characterised by the same quiet earnest attention to detail and determination to master every branch of his profession. This was most praiseworthy in one who had accepted, rather than chosen, medicine as his future calling. It scarcely needs telling that Morrants Baker's work and influence were quickly recognised by his teachers. Paget, West, Kirkes, and Callender more especially encouraged and advised him in his course of study. Morrants Baker gained the Senior Scholarship in 1861. He held all the customary student's appointments; presented himself for all his examinations at the earliest dates, passing the final F.R.C.S. in November 1864. He was never House-Surgeon at St. Bartholomew's, or at any other hospital; but on October 1, 1861, he became Resident Midwifery Assistant to Dr. West—the last

officer, in fact, whom Dr. West nominated, and when this gentleman resigned the appointment of Physician-Accoucheur, the Treasurer and House Committee, upon the advice of the Medical Council, who fully recognised Marrant Baker's special capacity for the position, placed him in temporary charge of Martha Ward and of the Maternity Department until the election of Dr. Greenhalgh as Dr. West's successor. For a few years at this period of his career he was Sir James Paget's assistant in private practice.

Marrant Baker's first teaching appointment at St. Bartholomew's was that of Assistant Demonstrator of Anatomy in 1863, then newly instituted. The successive positions in the Hospital and School to which he was elected may now be given briefly in simple chronological order. When Callender was appointed to lecture on anatomy with Holden in 1865, Marrant Baker was promoted to be full Demonstrator of Anatomy and Physiology. In 1867 he was elected Warden of the College, and very shortly after succeeded Callender in the office of Secretary to the Committee of Medical Officers and Lecturers usually called the "Medical Committee," in distinction from the "Medical Council," the former being the governing body of the Medical School, whilst the latter, composed of the Staff, being the advisers of the Governors of the Hospital in all medical matters. In 1869, upon the late Sir William Savory's resignation of the Lectureship of Physiology and General Anatomy, this important appointment was entrusted to Marrant Baker; but after a few sessions Dr. Klein was associated with him at his own request, relieving him of the histological part of the course, whilst later, viz., in 1885, he, after sixteen years of office, resigned altogether the lectureship, Dr. Klein from this time delivering the complete course as sole lecturer. In 1870 Marrant Baker was appointed Casualty Surgeon, being the first and only holder of this office; he took over the supervision of the morning work on the Surgical side of the Casualty Department; he was also elected Assistant-Surgeon to the Hospital in 1871, upon Callender succeeding Sir James Paget in the Surgeoncy as Junior Assistant-Surgeon. Marrant Baker continued in charge of the same duties allotted him as Casualty Surgeon, but when Howard Marsh was elected an Assistant-Surgeon, Marrant Baker passed into the Surgical Out-Patient Room, and began his career as a clinical teacher. In 1875 he was given the charge of the Skin Department. Lastly, Marrant Baker became full Surgeon to the Hospital in 1882, when the Governors proceeded to increase the Surgical Staff from eight to ten, adding a fifth Surgeon and a fifth Assistant-Surgeon. Thus Marrant Baker

held the Surgeoncy ten years almost exactly, resigning in 1892 owing to ill-health, as above mentioned. The Treasurer and Governors of St. Bartholomew's Hospital, in grateful recognition of Mr. Marrant Baker's faithful and distinguished services, when accepting his resignation, elected him a Governor of the Institution.

Outside his Alma Mater, Marrant Baker was for some years Surgeon to the Bartholomew Close Dispensary, and to the Evelina Hospital for Children. At the Royal College of Surgeons Marrant Baker was one of the first appointed separate Examiners on Physiology on the Board, and with intermissions for a year held this office from 1878 to 1887, whilst from 1887 to 1892 he occupied a seat upon the Court of Examiners as an Examiner in Surgery. He also examined in Surgery for the Universities of London and Durham.

Marrant Baker never sought election on the Council of the College, feeling debarred, when his turn came to stand, by the state of his health.

As a student, he took an active share in the work of the Abernethian Society, frequently reading papers and regularly joining in the discussions.

He became a Fellow of the Royal Medical and Chirurgical Society in 1865; he joined the Pathological at about the same time, whilst he was an original member of the Clinical and Dermatological Societies, in each of which he held various offices.

Marrant Baker contributed freely to medical literature; chiefly will he be remembered for the long period of his connection with "Kirke's Handbook of Physiology," viz., from 1867, when he produced the sixth edition of this book, and the first edited by him, to 1892, when the thirteenth edition appeared. In the tenth, which was brought out in 1880, as well as in the three later editions, Marrant Baker had the valuable assistance of Dr. Vincent Harris. Thus he for many years upheld the reputation of this work as the leading popular text-book both in London and other centres of medical education. But in addition to this continuous labour for twenty-five years on "Kirke's Handbook," he contributed extensively, chiefly in the shape of articles upon doubtful points in surgery, to the several societies to which he belonged, to our own Hospital Reports, and to the medical journals. Among them may be mentioned a communication to the *Lancet* on "The Removal of the Tongue by Median Division," in which he desired to show that by splitting the tongue the two halves could be removed more satisfactorily as regards getting well beyond the disease, and also that in successively removing the two halves, the

divided lingual arteries were more readily secured, and troublesome hæmorrhage in the course of the operation more certainly avoided. In the *British Medical Journal*, Marrant Baker advocated the temporary fixing together of femur and tibia after the operation of excision of the knee-joint by long sharp-pointed steel pins passed obliquely across the line of junction of these bones. He also contributed to this journal "A case of Charbon, with external symptoms (malignant pustule), excision, recovery," with a remarkably typical coloured drawing of the skin affection.

To the Royal Medical and Chirurgical Society, Marrant Baker contributed "Statistics of Cancer," "Necrosis without Suppuration," "On the Use of Flexible Rubber Tracheotomy Tubes;" and, with Mr. Bowlby, "Diffuse Lipoma." In this paper the authors propounded the theory that diffuse lipomata were directly attributable to the habitual consumption of an excessive quantity of spirits. The evidence adduced has not been shaken, and the dependence of this disease upon the habitual consumption of large quantities of alcohol has ever since been generally recognised. In "Necrosis without Suppuration," Marrant Baker collected all the cases he could find in support of one of his own at St. Bartholomew's Hospital, when, after consultation with his colleagues, who shared his conviction that a large uniformly swollen but not tender thigh was a case of sarcoma of bone, he had amputated at or just below the hip-joint, finding upon examining the limb afterwards that the case was one of necrosis, which had advanced to complete exfoliation of a long sequestrum, absolutely without suppuration. Few in the present day are aware that the common red rubber tracheotomy tubes were Marrant Baker's invention, but in the paper above mentioned he introduced them to the profession. When a tracheotomy tube has to be worn habitually, the convenience and comfort of a yielding elastic tube over a rigid metallic one is undeniable. To the Pathological and Clinical Societies Marrant Baker also contributed frequently, chiefly cases of interest.

At the meeting of the International Medical Congress in London in 1881, Marrant Baker read two papers which are printed in the *Transactions* of that Congress. The first, on a subject with which he was particularly familiar, was called "The Diseased Conditions of the Kidney which admit of Surgical Treatment, illustrated by three Cases: (1.) Nephrotomy in which Nephrectomy was subsequently performed; (2.) Nephrotomy; (3.) Renal Lithotomy." The other, "On Prurigo or Eczematous Prurigo or Pruriginous Eczema." In "Heath's

Dictionary of Surgery" he contributed the article on "Diseases of the Tongue," and articles on some minor subjects, whilst with Bowlby he wrote the articles on "Carcinoma," on "Sarcoma," and on "Malignant Tumours."

It was, however, for the St. Bartholomew's Hospital Reports that Marrant Baker wrote most freely, being for many years a well-nigh regular contributor. It is only necessary to mention them:—"On tumours containing fluid blood;" "The inheritance of cancer and its relations to questions concerning the local or constitutional origin of the disease;" "On the relation of life to other forces;" "On ranula;" "Case of ligature of the common iliac artery;" "Erythema serpens;" "A case of spontaneous dislocation of both femurs;" "A case of femoral aneurism cured by digital pressure;" "Fatal wound of the ascending pharyngeal artery by a tobacco pipe;" "Formation of synovial cysts in the leg in connection with disease of the knee-joint;" "On aneurisms which do not pulsate;" "Two cases of acute arthritis of infants;" "Cancer of the rectum removed by operation;" "On the treatment of delirium with insomnia;" "Introductory note to orders and ordinances of the Hospital of Bartholomew the Less;" "The formation of abnormal synovial cysts in connection with the joints;" "Perforating wounds of the orbit;" "On whitlow;" "Submaxillary cellulitis: syn. cyanche cellulitis of Gregory, angina externa, angina ludovici, cyanche sublingualis rheumatico-typhoides;" "Brief note on the relief of pain in certain cases of cancer of the tongue."

This last article appeared so recently as 1894, being in vol. xxix. for 1893.

In his student days, and for some few years later, Marrant Baker read papers at nearly every session of the Abernethian Society. In October 1885 he delivered the introductory address before this Society, the subject being "The two Foundations of St. Bartholomew's Hospital." In this address he brought forward all that had been previously known, and much more which he and Dr. Norman Moore had unearthed of the "first" foundation of the Hospital, as distinct from the Priory, by Rahere in 1123, and much about Rahere himself, and then he proceeded to relate the "second" foundation in 1544, or, as most persons would call it, the "*re-foundation*" by Henry VIII. of pious memory, two years later, when his Majesty endowed the Hospital from the sequestrated estates of the monks of the Priory and Hospital at the Reformation with a revenue of 500 marks a year, stipulating that the citizens of London should contribute a like amount. This address was printed by and circulated among the members of the Society, and serves to form

a memorable link between Marrant Baker and the Abernethian Society.

Marrant Baker originated many improvements in surgical appliances, but it should be put on record that probably he may be regarded as the inventor of the "duplex" burner for lamps and gas jets, for certainly early in the "60's" he had a reading-lamp constructed, in which, by introducing a metal bar of about $\frac{1}{4}$ in. width in the long axis of the flame immediately above the burner, he succeeded in dividing the flame and thereby considerably augmenting its illuminating quality by effecting more perfect combustion of the oil.

Having thus shown how active-minded and actively engaged Marrant Baker was all through his hospital life, no memoir of him would be complete or adequate which did not depict him as a lecturer and teacher, as a surgeon and clinician, as a man of culture outside his profession, as an adviser, friend, and colleague, or fail to show the influence he exerted in his day in the school, or bring home some of the lessons his life and work have for those who succeed him.

As a lecturer and teacher of physiology, Marrant Baker won a deservedly high reputation, not by reason of any innate brilliancy of his intellectual faculties, or of persuasive eloquence, or of commanding force of character, but by a thorough knowledge of his subject, gained by laborious study, by an anxious willingness to impart all he knew, and by taking infinite pains to fulfil the charge entrusted to him most thoroughly and conscientiously. And although as Lecturer on Physiology he undoubtedly, as successor to Paget and Savory, started at a discount, yet he proved himself to be very much more than a merely respectable or passable lecturer on his subject, for unquestionably he was superior to many of his compeers in London, and came very near to being a model lecturer in physiology, as the subject had up to his day been taught; for he could teach thoroughly well a subject he knew, in clear language, with happy expression, and with abundant illustrations from or references to disease; his experiments were well chosen and telling; he scrupulously eschewed everything that was common-place, vulgar, or clap-trap. Thus during his long tenure of the lectureship he held from first to last the attention of his class, and did most ample justice to his selection for the position. He lectured to the largest classes ever known; and it should be told to his lasting credit that it was at his own strong desire and initiative entirely that Dr. Klein became associated with him as Lecturer on Histology. Marrant Baker was the first to foresee and to press upon his colleagues

the necessity for advanced teaching in his own department; and equally at Marrant Baker's wish it was carried through at his expense, no addition being made to the emoluments of the co-lecturers.

As an operating surgeon and clinical teacher Marrant Baker was distinguished naturally by many of the qualities he exhibited as a lecturer. His operative measures were always well planned and methodically executed. He was most observant, prudent to the verge of over-caution, for he would take nothing for granted of which he was not morally certain. He was thorough in getting to the bottom of a case, sparing neither time nor pains over its investigation, but having made up his mind on the course to adopt, he was stopped by no difficulties of execution nor flinched before any obstacles that assailed him. Notably were these qualities seen in some of his renal operations, as also when on one occasion he laid open a gluteal aneurism, and ended by placing a ligature upon the common iliac artery. Marrant Baker never took cordially to antiseptic surgery; he had been very much imbued with Callender's views of strict cleanliness, and he believed rather in deodorisers than in antiseptics, and quite failed to grasp asepticism. His favourite topical applications were Sanitas oil, Sanitas lotion, and Condy's fluid. He took extreme care with the dressing of his wounds, and especially in securing the comfort of his patients; and it must be added that on the whole his cases did well. He shone more in the wards and at consultations than in the operating theatre; in clinical teaching his forte was in exposition. He had an acute perception of the affinities of morbid processes, and he had also a very retentive recollection for the external appearances of disease, so that he often made very shrewd guesses in puzzling cases. His diagnostic powers were unusually accurate, and he had a quiet, convincing way of unfolding the points of a case, so that his class, when going round with him, rarely failed to learn something important, or something on which in the future they would set store. At consultations he was perhaps seen at his best; very sagacious, not easily turned aside from the important factors of a case, he would as surely brush away trivial or misleading elements. His opinion was always regarded as worthy of the highest respect. The cases which he himself would show were always exhaustively worked up and stated in a telling, lucid form. His opinion was sought and valued by his colleagues, probably beyond that of any other member of the Surgical Staff since Sir James Paget's time. Marrant Baker advocated the performance of excision of the knee in adults, and was adverse to the operation in chil-

dren, upon grounds which every one will appreciate. He was very successful in these operations, and would often relate with legitimate pride how, after employing his "pins," an adult patient had been got up, and was walking about with a sound wound under a month from operation.

Marrant Baker, when his health began to fail him, was rapidly becoming well established in a large consulting practice; he was trusted alike by the profession and by patients. Without doubt, he would have taken a leading position as a consultant had but health and strength been extended to him. As examiner, Marrant Baker was remarkably fair, able, and discriminating. He was a very popular examiner with candidates, and had the reputation of never plucking if he could help it, whilst his attention and courteous demeanour charmed every one.

Turning to Marrant Baker's position in the school, as an adviser of the young, as the sincere loyal friend of all his colleagues, who retain an abiding sense of the void, still unoccupied, which his resignation effected, it is possible that the time lies in the still distant future to do impartial justice to his memory. Others have been more looked up to, revered, or feared, but no one was ever more universally beloved and trusted. This feeling arose from the conviction that Marrant Baker was in all things guided and moved by principle rather than by expediency; that he possessed an even-balanced judgment; that he was incapable of doing an underhand action, or intriguing for his own advantage. Marrant Baker would take sides with none, and so was the friend and confidant of all. Students and young men just qualified eagerly sought his advice; he had quite marvellous powers of gauging a man's powers, and what he was capable of doing. His long tenure of the Wardenship doubtless ripened his judgment of young men and their ways, and thus well fitted him to become their philosopher, friend, and guide, and he was this to very many. He could both check a vain enthusiasm and stimulate a legitimate ambition, and in both instances appear to give to each the exact advice they desired.

In spite of his numerous professional preoccupations, Marrant Baker had many interests outside his calling. For classical music he had cultivated a really good critical judgment. He did not perform upon any instrument or sing, but the Saturday and Monday Populars were his great delight, and as often as he could he would attend them. He devoted whatever spare time he could snatch to reading high-class works outside his profession; he was always well versed in general contemporary

literature, and could talk well and freely upon it. He was a considerable collector of engravings, of old ones chiefly; he possessed probably the best existing collection of prints of St. Bartholomew's Hospital, and it always gave him real pleasure to decipher and to demonstrate the various changes and metamorphoses the old place had undergone in its well-nigh seven centuries of existence. In perfecting this collection he was greatly helped and advised by that good friend the late Mark Morris, the old steward, who had a sixty years' (or two generations) knowledge of Bartholomew's men.

Then, later in life than most men, Morrant Baker took to whist; like all things he handled, he studied and learned the principles of whist thoroughly, and played the correct game, so conscientiously at times indeed, that he has been known deliberately to sacrifice the odd trick rather than contravene any of the principles of the game. He belonged to one or two social whist clubs, and enjoyed the relaxation a rubber gave.

Morrant Baker was a man of a deeply religious turn of mind, as every one who knew him would feel certain of. He was consistently devout in the exercise of his religious tenets. He greatly enjoyed the services at "All Saints" and at Wells Street Churches, but he was most unobtrusive in these matters, and generously charitable towards the opinions of others.

Morrant Baker was forward in promoting all real charitable works, and was ever ready, as far as prudence permitted, to assist with his purse, but he loathed publicity in what he did. Many of the less prosperous men whom he had known as students turned to him in their adversity, and, if deserving of assistance, never went away empty-handed. With others, he largely helped to supply a great want in the extern midwifery department, by providing nurses who visited at the homes—often devoid of necessaries—of the recently confined patients, and ministered to their creature comforts. And again with the assistance of two charitable ladies, who acted as his almoners, systematic inquiry was made into the circumstances of the families of patients admitted to his wards, and relief given to such as needed it.

In 1868 Morrant Baker married an Andover lady, Miss Annie Mills, the sister of the late Joseph Mills, for so many years the administrator of anæsthetics at St. Bartholomew's Hospital, and so well known to, and highly esteemed by, a numerous succession of Bartholomew men. Mrs. Morrant Baker and a family of two sons and four daughters are left to mourn the loss of an affectionate and devoted husband and father. The eldest son has entered his father's profes-

sion, and is a student at St. Bartholomew's. Two of Marrant Baker's brothers were also in the medical profession; one, Mr. Henry Baker, survives, and is well known as one of the Surgeons to the Royal Orthopædic Hospital.

It may be difficult, but surely not impossible, to appreciate, at some approach to a correct estimate, the influence for good which any one man has had, by imparting a definite personal impress of his own character upon so complex a body as a large medical school, composed of a number of teachers and a very much greater number of the taught, both of whom are being perpetually changed and renewed. For the converse of this proposition is but too true; it is most easy to trace the harm which one man can do by precept and by example against his fellow-men. It is then but meet and right to award a high place in the roll of famous members of the staff of St. Bartholomew's Hospital and Medical School to a man like Marrant Baker, whose whole career was a constant incentive both to his colleagues and to his younger brethren on the threshold of professional life, to work honestly, to do justly, to think no evil, and to love all men.

SAINT BARTHOLOMEW'S HOSPITAL REPORTS.

CLINICAL CASES FROM THE WARDS.

BY

W. S. CHURCH, M.D.

The following cases appear to me to present sufficient features of clinical and pathological interest to render it desirable that they should be recorded in our Hospital Reports.

CASE I.—*Disease of the Aortic Valves, with adherent Vegetations—
Hæmorrhage into the substance of the Spleen.*

Henry S., aged 46, an engineer, applied at the Hospital on January 9, 1896, suffering from cough and shortness of breath, accompanied with slight hæmoptysis. He was admitted to the Surgery Ward, and during the night brought up about ̄ij of blood; the next day, as he still continued to bring up some blood, he was admitted into Mark.

He had had rheumatic fever when young, and an attack of rheumatism five years ago; but up to four months before admission had enjoyed good health, and experienced no difficulty in doing his work.

On admission to Mark, the man looked ill; his voice was hoarse and husky. His chest was well formed, slightly hyper-resonant on percussion, and the expiratory sound somewhat prolonged. The action of the heart was forcible, the apex-beat in the fifth intercostal space, half-an-inch outside the nipple-line. The area of cardiac dulness increased in all directions.

The cardiac sounds were natural at the apex; at the base a diastolic murmur, loudest over the pulmonary area, and conducted down the sternum to the ensiform cartilage, was present. Pulse 100, jerking and water-hammer in character; considerable pulsation was present in the carotids.

The liver was somewhat enlarged; the spleen could be felt also enlarged, but not very greatly so. The bowels were reported to have been very costive. There was a trace of albumen and blood in the urine, but no œdema anywhere.

Abstract from the Daily Notes of the Case.—January 17.—Doing well; no more hæmoptysis; a trace of blood in the urine.

January 21.—Has had some slight hæmoptysis again; some pain in the left side, where there are a few crepitant sounds.

January 24.—Better, no pain nor hæmoptysis; the diastolic murmur is louder than it was, and musical in character. Pulse 72. Still a trace of blood in the urine.

January 31.—Sleeps a great deal both day and night, but feels better; diastolic murmur no longer musical.

February 1.—Complains of pain on swallowing. Tonsils, both arches of the palate, uvula, and pharynx intensely red and congested; much tenacious mucus adhering to them. The pulse 100, and the temperature, which up to this time had been natural, 103° . In the evening there was some redness of the nose and eyelids, and the temperature rose to 103.8° .

February 2.—Well-marked erysipelas of the face. He had a severe though short attack of erysipelas of the head and face, his temperature becoming normal again on February 11.

During the erysipelas, although there was no change in the character of his pulse, the diastolic murmur became fainter and fainter, and towards the end of the month no murmur could be heard.

On February 8, when the erysipelas was beginning to fade away, and great improvement in his general condition had occurred, he complained of severe pain about the left elbow. The elbow and arm rapidly swelled, and in forty-eight hours the arm above the joint measured four inches more than the right one; a cordlike body could be felt along the brachial vessels. Two days later, when his face and throat were nearly well, the arm, although no longer painful, measured eight inches more than the right.

On February 17, he had some pain in the left leg, but no swelling was detected, and the pain rapidly subsided.

On February 22, a small abscess which had formed in the left mamma was opened, and about 5j of thick pus evacuated.

From this time improvement took place uninterruptedly, and the patient went to Swanley in fair health on March 13.

Unfortunately there is no note of the cardiac sounds after February 26, when the murmur, which had been growing fainter and fainter, could not be heard, although it states that he had all the other evidences of aortic regurgitation.

On September 5 he was readmitted, and stated that when he left Swanley he was feeling well and able to get about, but was short of breath on exertion. The shortness of breath gradually increased; a fortnight before readmission he went to Southend for change of air, but rapidly became worse, and four days before he applied to the Hospital his legs became very swollen, and he was hardly able to walk.

On admission, he was very short of breath, and had to be propped up in bed. A few râles could be heard at both bases of the lungs; the heart's impulse could be scarcely felt; the area of cardiac dulness was greatly increased; a very distinct diastolic murmur was present, blowing in character at the aortic base, whistling and almost musical down the sternum. At the apex-beat a distinctly double murmur accompanied the heart-sounds. Pulse 92, regular, and characteristic of aortic regurgitation. Temperature normal. The liver very large; its lower border could be felt just above the umbilicus. The spleen much increased in size, its edge being palpable some distance below the ribs. Slight œdema of the lower part of the legs and feet.

September 11.—A great deal of pain in the hepatic region; twelve leeches were applied; the bites bled profusely and were difficult to stop. The bleeding was followed by much relief to the pain.

September 20.—Breathing easier; much pain in the splenic region and left side of the chest. Râles to be heard in the axilla and at the base on the left side. Pulse 84, regular, of the same character. Murmurs unaltered.

October 4.—General condition the same; still has much pain in the left side and splenic region.

October 10.—The temperature has been slightly raised during the last six days, but has never exceeded 99.6°. A slight pleural rub to be heard on the left side. A soft systolic murmur is now to be heard at the aortic base, the diastolic one remains unchanged. The spleen is larger, reaching nearly to the umbilicus and four inches below the ribs. Pulse 76, regular.

October 15.—An herpetic eruption came out over the nostrils,

probably due to the iodide of potassium that the patient had been taking since the 10th. By the 21st the whole end of the nose was a raw, granulating surface. During the next eight days the nose gradually healed, and the man expressed himself as feeling better and more comfortable, but it was obvious that he was fast losing ground, and was looking more worn and exhausted. The condition of the heart, liver, and spleen appeared unchanged, and his temperature remained normal. In the course of the night of October 28th the patient's temperature suddenly rose to 104.4° ; he shivered, and on the morning of the 29th there was a blush of redness apparent about the nose and eyelids, which were œdematous. The erysipelas rapidly spread over the face; his temperature in the course of the day fell to 100° , but rose in the evening to 102.6° ; his pulse became 112. From this time the erysipelas spread, extending to the tissues of the neck. The patient gradually sank, and died on November 2.

During the whole of the period of the patient's second stay in the Hospital there was a slight cloud of albumen present in the urine, and usually a trace of blood.

Post-mortem.—Some remains of bullous eruption on the face; cranial bones, membranes, and brain natural. Œsophagus and glands of neck natural. Slight ulceration of larynx, but no infiltration of the cords. Right lung emphysematous; some small calcified nodules at the apex. Left lung emphysematous; some interlobular adhesions and its base œdematous. Slight excess of fluid in the pericardium. Milk spots on right ventricle. Weight of the heart, 22 oz. The hypertrophy was chiefly of the right ventricle. Pulmonary and tricuspid valves natural. On the auricular surface of the mitral curtains, near the aortic valves, a group of minute vegetations; attached to the posterior aortic flap were several long vegetations, the largest $\frac{1}{8}$ of an inch in length, pointed, fusiform, not at first sight calcareous. The right flap had on it numerous but smaller vegetations, and a perforation the size of a pin's head in it. The coronary arteries showed marked atheroma, but no calcareous change.

The liver weighed 102 oz.; surface hobnailed; typical cirrhosis on section.

The spleen very large, weighing 41 oz. Its head adherent to the diaphragm. In the head of the spleen was a spherical mass of deeply coloured blood-clot, as large as a small orange, and showing a distinctly laminated structure. At the upper surface is a thin layer of pale firmer tissue, but continued so as to form a capsule to the clot. Below, and to the outer side

of the chief hæmorrhage, are two smaller ones, apparently independent. The hæmorrhages have not the shape or appearance of an infarct. There is no bulging over their surfaces, and they are covered by a thin layer of spleen tissue. Kidneys, large; capsules strip off readily; apparently fatty; no infarcts.

DR. KANTHACK'S REPORT.

Histological Examination.—(1.) *Large calcareous vegetation on aortic valve.*—On its superficial parts there is much fibrin and numerous diplo- and streptococci.

(2.) *Mitral valve.*—Is also covered by fibrin, and also shows diplo- and streptococci.

(3.) *Musculus papillaris of the left ventricle.*—Shows diplococci and streptococci in the vessels just around them, and also a few between the muscular fibres.

(4.) *Wall of the left ventricle.*—Numerous diplococci are found distributed exactly as in the musculus papillaris.

(5.) *Blood-stained patch in the aorta.*—Shows in some of the *vasa vasorum* a large number of streptococci.

(6.) *Lung.*—Shows much inflammatory œdema, some alveoli filled up with fibrin, others with round cells; large numbers of streptococci in the vessels, air vesicles and walls of the alveoli.

(7.) *Kidneys.*—There are a fair number of streptococci in the vessels.

(8.) *Liver.*—Also shows streptococci in some vessels.

(9.)—*Spleen.*

(10.)—*Splenic hæmorrhage.*

} Strange to say, no organisms
} were found in these situations.

This, then, is undoubtedly a case of streptococcus endocarditis and septicæmia. There must have been micro-organisms in the spleen, although they were not found.

CASE II.—*Pneumonia of the Right Lung—Mitral Stenosis—Recent Endocarditis.*

James M., aged 31, a porter at Covent Garden. Admitted May 6, 1896. Five days before admission his illness commenced with pain in the right side of his chest and abdomen, shivering and cough.

He states that eleven years ago he was in the Hospital for pleurisy and bronchitis, and in March of this year in the Bromley Infirmary for rheumatism.

On admission, all the symptoms and physical signs of acute

pneumonia from the base up to the spine of the scapula on the right side were present.

The heart-sounds were clear, and *no murmur* was present. Respirations 40. Pulse 116, regular and of good volume. Temperature on admission 102°, rising quickly to 104°. May 7th to 10th he remained much in the same condition. On the morning of May 11th he was very much worse. His pulse had run up to 140, respirations 40, whilst his temperature had fallen to 102.6°. He died in the course of the day.

On the morning of the 11th it was noticed that there was a harsh presystolic murmur present, and a thrill could be felt over the region of the heart.

Post-mortem.—No abnormal appearances were found excepting in the lungs, heart, and spleen. The right lung was bound down by firm adhesions. The entire lung, with the exception of a small portion at the base in front, was consolidated throughout. The upper lobe was evidently the most recently affected, being redder and firmer; the lower lobe was easily broken down by the finger and exuded a brownish liquid on pressure. The lung weighed 66 oz.

The heart weighed 13 oz.; its cavities contained much decolorised blood-clot entangled in the muscular bands. The aortic, pulmonary, and tricuspid valves were quite healthy; there was well-marked stenosis of the mitral orifice, which only admitted the tip of the gloved index finger. The stenosis was of the jellybag type. The valve-flaps and chordæ tendineæ were thickened, somewhat shrunken, and the edges of the curtains adherent; at one spot on the auricular aspect was an area of roughening about $\frac{1}{3}$ inch in diameter, which was clearly due to recent endocarditic changes. There were no exuberant vegetations. At another spot was a small oval orifice in the valve curtain about $\frac{1}{16}$ inch in diameter, with clean-cut edges, smooth, and clearly not recent. There was some hypertrophy of the wall of the left auricle, but none of the left ventricle. There was slight excess of blood-stained fluid in the pericardium, but no pericarditis.

The spleen weighed 9 oz., very soft and flabby; the spleen pulp diffuent on section.

REPORT BY DR. KANTHACK.

The vegetations on the mitral valve are infective, and show numerous cocci, which are almost certainly pneumococci.

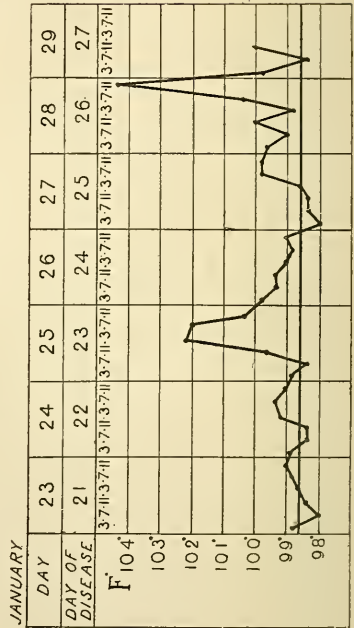
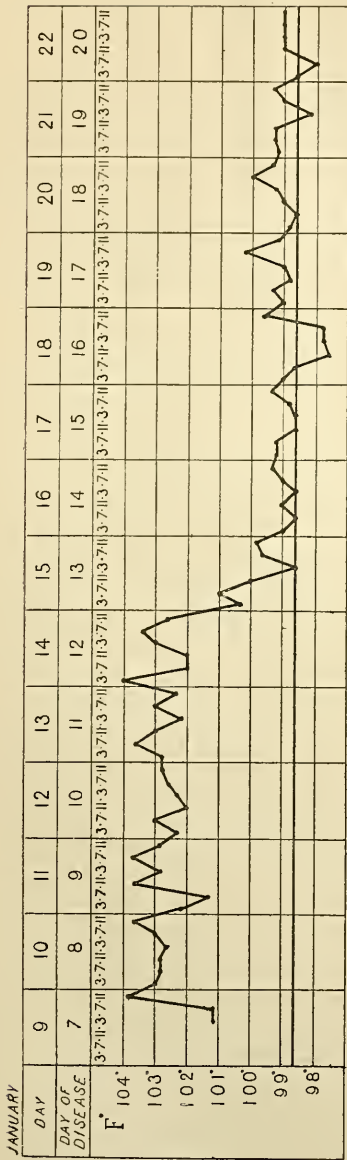
CASE III.—*Pneumonia—Recent Endocarditis with Vegetations—Embolism of the Right Coronary Artery—Sudden Death.*

Joseph P., aged 40, a metal polisher, admitted January 9, 1896. On admission, all the symptoms and physical signs of pneumonia of the right lung were present. He was much exhausted. He looked older than his age, and stated that he had not been feeling well for some seven or eight days, having suffered from constipation and abdominal pain. His severe symptoms came on January 6th, when he had severe pain in the side, and had to give up his work and go home. He had suffered from two attacks of pleurisy, the first four years ago, the second in 1895.

The heart-sounds on admission were noted as clear, his pulse 100, fair volume and regular. Temperature 101.2°. Respirations 30 per minute.

The specific gravity of the urine was 1020, and it contained a trace of albumen.

An examination of the temperature chart appended will obviate the necessity of transcribing the daily notes. It will be seen that the pneumonia ran a severe, but not in any way abnormal course. Defervescence of the fever with a fall in the temperature and pulse, and to a slight degree in the respiration, occurred on the ninth day from the advent of pain in the right side, but owing to his previous indisposition it is uncertain when the pneumonia actually commenced. On the morning of January 14th, the day on which defervescence of the fever began to take place, he complained of very great pain in the right arm near the insertion of the deltoid. The arm, on handling, was extremely tender, but no swelling, redness, or other abnormal condition could be detected. On January 15th, the pulse was found to be irregular and of very poor volume, and four minims of liq. strychninæ were ordered every four hours. On the 16th, the pulse was better, quite regular, and the heart-sounds are noted as "clear and well heard." The arm was still extremely painful and tender, and pain and tenderness were complained of in the calf of the right leg. On the evening of the 19th and 20th there was a slight rise in the temperature, but no increase in the rapidity of the pulse or respiration. On the 20th there was an abundant crop of sudamina on the chest; the respiratory sounds and air entry on the right side had very much improved. The pain in the calf of the right leg had gone, and that in the right arm was much better. From this time until January 25th, the patient went on very well, and presented no symptoms or



physical signs to cause anxiety; but on the evening of the 25th his temperature, without any assignable cause, rose to 102.2° , but fell to normal the next day. On the 27th, a swelling was noticed round the right sterno-clavicular joint. The swelling was tender, ill defined, and rose into the neck and extended downwards to the first rib. On this day and the following, although the patient appeared pretty well, he was much depressed and anxious about himself.

On the evening of the 28th the temperature rose to 104.4° , but there was no rigor, and it fell again as quickly as it rose to normal. Nevertheless, on the morning of the 29th the patient, though apparently doing well, was still more depressed and anxious about himself, and stated that he felt sure he was going to die. A careful examination of him revealed nothing, except that the swelling around the sterno-clavicular joint had increased in size; it felt elastic, but distinct fluctuation was not made out. The respiratory sounds in the lung were clearer and freer than they had been. The pulse 80, regular; the heart-sounds clear and distinctly heard.

At 3.15 P.M. he had sudden præcordial pain and urgent dyspnœa; this subsided after a short time. An hour later he was seized with similar but more severe præcordial pain and dyspnœa, and died in a few minutes.

At the post-mortem investigation all the organs of the body, with the exception of the lungs, heart, and aorta, were found to be perfectly normal. The upper lobe of the right lung was in a state of resolution from pneumonia. It was grey in colour and crepitant, and floated in water; there were four adhesions in the right pleura. The arch of the aorta was dilated, but not greatly so, and the thoracic portion was extremely atheromatous.

The heart weighed 12 oz. Attached to the left cusp of the aortic valves was a single large vegetation, as big as a small nut, which was attached to the valve by a somewhat narrow neck. The vegetation did not interfere with the closure of the orifice. The free end of the vegetation lay very near the orifice of the right coronary artery, which was found to be entirely obstructed by an embolism, whitish in colour, and which was probably detached from the vegetation. There was no other appearance of disease about the heart, and no other vegetations were found.

The abdominal organs were all normal, no embolic infarcts being found in the spleen or kidneys.

On opening the right sterno-clavicular joint, a little pus escaped, and the surfaces of the cartilages were somewhat

roughened. The right shoulder-joint was natural, and nothing abnormal was found in the tissues around the joint, or in the brachial plexus or vessels of the surrounding parts.

Portions of the lung and the vegetation on the aortic valves, and the embolic mass from the coronary artery, were examined in the Pathological Laboratory, and Dr. Kanthack has kindly furnished me with the following report:—

Cultivations were made from the cardiac vegetation, lungs, and spleen.

(i.) In the aortic vegetation are found (1) pneumococcus, (2) staphylococcus pyogenes aureus.

(ii.) In the lungs are found (1) pneumococcus, (2) staphylococcus pyogenes aureus, (3) bacterium coli commune.

(iii.) In the spleen, (1) pneumococcus, (2) staphylococcus pyogenes aureus.

On making histological specimens, which were imbedded in paraffin, are found—

(1.) *Coronary artery.*—A thrombus consisting of fibrin and blood. A few diplococci are found in the thrombus.

(2.) *Vegetation attached to the aortic valve.*—Also contains diplococci in fair numbers.

(3.) *Cardiac wall.*—Here also a few diplococci were found between the muscular fibres.

REMARKS.

These cases all present some features in common. (1.) In all there was recent infective endocarditis, secondary in the first case to erysipelas, in the other two to pneumonia. (2.) Notwithstanding the presence of grave cardiac disease, in one case no abnormal sounds were heard, and in the other two the cardiac murmurs were not constantly present.

Ulcerative or malignant endocarditis had been recognised as distinct from simple endocarditis by many observers for years before its association with micro-organisms was suspected. The attention of physicians in this country, at all events, was directed to the association of micro-organisms with this form of endocarditis by the very interesting Gulstonian Lectures delivered on Malignant Endocarditis by Professor Osler in 1885. Since that time the association of micro-organisms with this form of endocarditis has been abundantly demonstrated.

In Case I. there are several points of great clinical and pathological interest. When the patient was admitted, he was suffering from the effects of aortic incompetence, the result probably of hard work upon valves which had suffered

damage in one or other of his attacks of rheumatism. His case presented no unusual symptoms until his erysipelas occurred. How this was contracted I don't know; there had been no case in the ward for months, and no one else in the ward subsequently took the disease. During the attack of erysipelas the diastolic aortic murmur became fainter and fainter, until at last it entirely disappeared. Unfortunately, the date of its reappearance is not known. What can be the explanation of this? It will be noticed that during the end of the attack of erysipelas, and subsequently, the man had pain and swelling in the left arm and elbow, and there was a cord-like structure to be felt along the course of the brachial vessels; and further, that a small abscess formed in the left breast. It appears, therefore, highly probable that his whole system was infected with micro-organisms at that time, and that the damaged aortic valves were then the seat of infective endocarditis. It will be noticed also that the cardiac vegetation found after death was partially calcified in its interior. Did this form during his first attack of erysipelas? Clearly there was not time for calcification to take place during the second attack of erysipelas. Is it possible that a large, soft vegetation, as it gradually increased in size, might so interfere with the backward stream of blood through the aortic valves as to cause the murmur of regurgitation to become so faint that finally it could not be heard? Improbable as this explanation may appear, I believe it is the true one; nor is the present the only instance in which I have known such a sequence of events. Many years ago, whilst attending a patient who had been under Dr. Andrew's care for disease of the heart, of rheumatic origin, in whom there was a double aortic murmur, the diastolic murmur disappeared. Shortly afterwards the patient died, and post-mortem the aortic valves were found ragged and calcified, and attached to one of them a very large soft vegetation, which was amply large enough to close the stenosed aortic orifice.

Another very interesting question arises in this case. If, as seems probable, his endocardium was affected by micro-organisms during his first attack of erysipelas, how was it that he made so good a recovery? For several months he enjoyed fair health, and when he failed, it was with symptoms of weakened and dilated heart, rather than with those usually accompanying the presence of micro-organisms in the system. Assuming that the first attack of erysipelas was accompanied by a state of general infection, were all the micro-organisms eliminated, or did they remain dormant? Were the specific micrococci of

erysipelas present all the time, the second attack being due to an auto-infection, the herpetic eruption on the nose affording them a favourable nidus for active development? In our present state of knowledge it does not seem possible to answer those questions definitely one way or the other. One other remarkable feature in this case still remains to be noticed—the splenic hæmorrhage. The main hæmorrhage consisted of a mass of laminated clot, about the size of an orange. The clot, though laminated, was not discoloured; the smaller hæmorrhages in the neighbourhood closely resembled the large one, and they all differed entirely from hæmorrhagic infarcts, resembling clot in an aneurysm. Pain in the splenic region was one of the most distressing symptoms that the man suffered from during his second stay in the Hospital; for nearly a month, from September 11 to October 10, the pain was continuous, and towards the close of this period the greatly enlarged spleen was noticed to still more increase in volume.

Case II. calls for no special remarks. I am unable to suggest any reason for the absence of the cardiac murmur during the first few days of the patient's life in Hospital, but I think there can be no doubt of the fact. I tried to find the notes of the case when he was in the Hospital eleven years ago, but was unable to trace him. A man corresponding in age, but with a different Christian name, was found in the Register of John Ward for the year he stated that he was in the Hospital suffering from pleurisy and bronchitis. If, as seems probable, this was really the same man, no cardiac murmur, and apparently no suspicion of heart affection, existed then.

In Case III. until the last four or five days of life there was nothing to lead one to think that the case was other than an ordinary one of acute croupous pneumonia, although the pain complained of in the right shoulder, calf, and side without discoverable cause was puzzling. After death the sequence of events was clear—the attack of pneumonia, the secondary affection of the endocardium and system from micro-organisms, the anginal attack and sudden death consequent on embolism of the coronary artery.

A reference to the temperature chart shows that the primary pneumonia was severe; the crisis took place on the 12th, or, as I think more probable, on the 9th day of the fever. My house-physician reckoned the day of disease from January 3rd, the day on which he first felt unwell, but the pneumonia probably really commenced on the 6th, the day on which he had to leave his work and the pain commenced in the side.

It is worthy of remark that in this case, as in Case I., the

presence of micro-organisms in the heart and system, which may, I think, be inferred from the pain and tenderness in the right shoulder, calf, and leg, did not affect the temperature; it was not until pain and tenderness and swelling occurred in and around the sterno-clavicular articulation and accompanied by suppuration that the temperature rose on the 23rd day of the disease, and then only for a few hours.

The absence of cardiac murmur notwithstanding the existence of a large vegetation attached to the cardiac valves, is not so unusual as might be expected; numerous cases of acute ulcerative endocarditis are now on record in which large vegetations have been found after death, while no abnormal condition of the cardiac sounds had been present during life.

The sudden death must, I think, be attributed to the embolism of the coronary artery; and it is interesting to find that this condition was associated with symptoms closely resembling angina pectoris.

It may fairly be observed that there were two anginal attacks, and that from the first the man rallied for a time; it appears to me possible that in this attack the vegetation temporarily blocked the orifice of the right coronary artery, whilst in the second a portion of it broke off and thus permanently closed it. Cohnheim¹ states that embolism of one of the larger branches of the coronary arteries has been found as the sole cause of death; but the only fully recorded case I am acquainted with is one lately published by Dr. Rolleston.² In his case death followed within ten minutes of the commencement of the attack, and was due to an embolism derived from an ante-mortem clot in the left ventricle, there being no affection of the endocardium in his case. Experimental ligation of the coronary arteries has yielded very contradictory results; the effects not only seem to differ in different sorts of animals, but in the same species very different results have been observed. Mr. Townshend Porter³ has written a very interesting paper on the results of ligation of the coronary arteries, in which he has collected and summarised the literature of the subject, as well as describing his own experiments.

Many cases of death, some of them sudden, are recorded, in which obliteration of one or other of the coronary arteries was found; and Dr. West,⁴ in his remarks on the coronary circulation, mentions a case in which obliteration of both arteries was

¹ Cohnheim, *Lectures on General Pathology* (Syd. Soc. Trans.), vol. i. p. 36.

² *British Medical Journal*, November 21, 1896, p. 1566.

³ *Journal of Physiology*, 1894, vol. xv. p. 121.

⁴ *Path. Soc. Trans.*, vol. xxxv. p. 112.

found in a man aged 56, dying of cancer. Dr. West also demonstrated to the Society that there was a very free anastomosis between the right and left coronary arteries; hence, one can understand that gradual obliteration of one of the arteries may take place without at any time giving rise to much interference with the cardiac circulation.

CASE IV.—*A Case of Typhoid Fever, with Relapses, unusually high Temperatures and frequent Rigors—Optic Neuritis (? of old standing)—Recovery.*

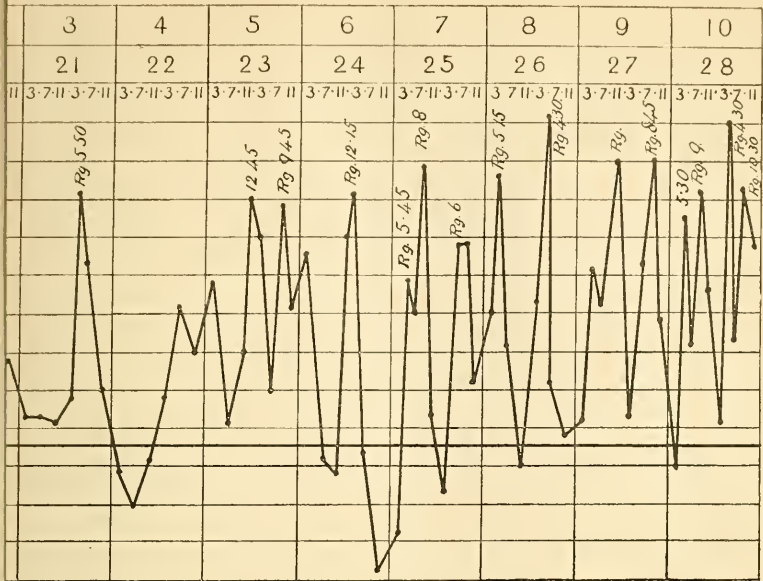
The interest of this case mainly attaches to the remarkable range of the temperature and to the frequency of the rigors. The appended chart shows these features of the case very clearly, and a very brief abstract from the daily notes will fill up what is wanting in the chart, and enable the reader to follow the course of this most interesting case.

Mary Mc—, aged 12, was brought to the Hospital at 6 A.M. on the morning of June 15th with the following history. For about a week she had been languid and had lost her appetite; she went to bed on the evening of June 13th without feeling any worse. On the morning of the 14th she was seized with giddiness, and complained of pain in the head and upper part of the abdomen. She returned to bed again, and had an aperient given to her, and subsequently passed a small motion. She was restless all day, and during the night of the 14th vomited so constantly that her parents brought her to the Hospital as early as possible in the morning.

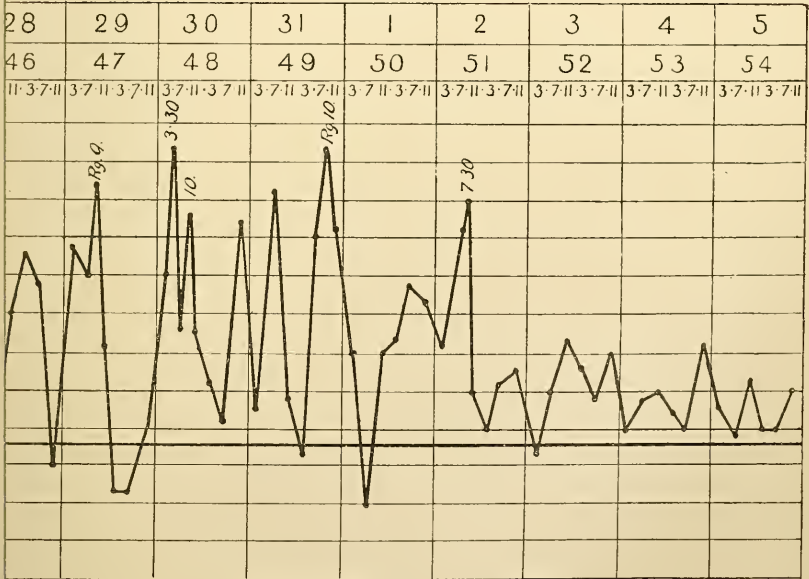
She was admitted to the Surgery Ward to be watched, her temperature then being 104.4°.

The only point in her previous history worth noticing was, that when seven years of age she had been struck by a plank falling on her head, and was stated to have had "concussion." She has ever since suffered from headaches, and they are stated to have been worse lately. She has five brothers and sisters, all healthy, excepting one who is said to have "water on the brain" and convulsions.

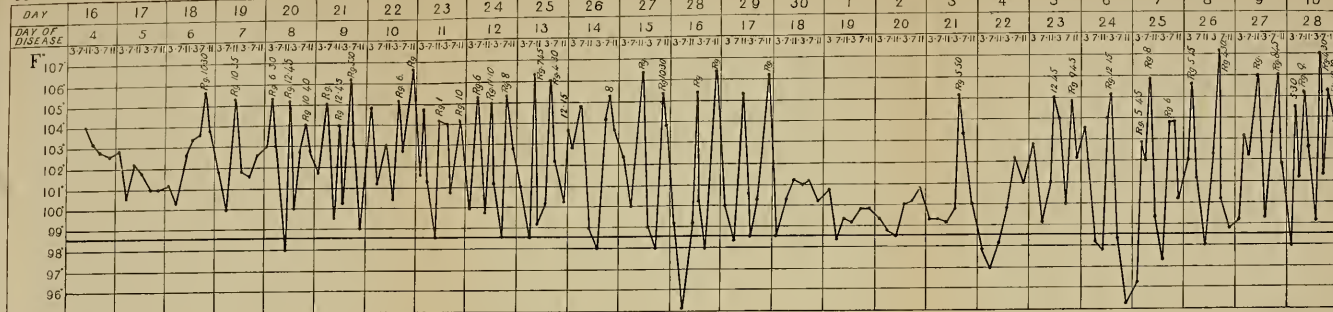
When seen in Faith on June 16th, she was a well-nourished, fair-complexioned child, with a very flushed face; *alve nasi* quiet; the pupils equal, reacting to light; tongue red at tip and edges, papillæ prominent, and slight fur on the dorsum. There were some patches of psoriasis on the elbows and knees. The lungs were normal, and no enlargement of the liver or spleen could be detected; there was no tenderness in the abdomen. The temperature was, at the time of my visit, 103°.



AUGUST

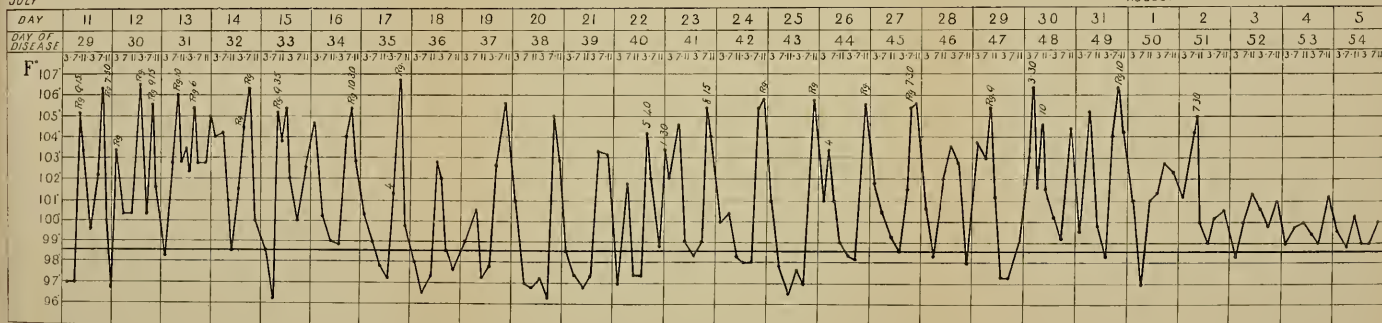


JUNE



JULY

JULY



AUGUST

Respirations 44. Pulse 140. An ice-bag was ordered to be applied constantly to the head.

June 17.—Has passed a fair night, looked brighter, headache less; bowels acted twice, motions loose and brownish. Respirations have fallen to 28, and the temperature to 101°. Pulse varied from 120 to 108.

June 18.—Decided tenderness in the cæcal region.

June 19.—A suspicious-looking spot in the left hypochondrium. (This was the only spot found during the whole course of her illness.) During the night of the 18th she had had a severe rigor, and a second one this morning. During the rigors her pulse became very feeble, and her face and extremities dusky.

June 20.—She was put on three grains of quinine every four hours, and an ounce and a half of brandy in the twenty-four hours. The brandy was increased to two ounces on the 23rd.

June 24.—She had some diarrhœa, which continued until the 30th, the bowels acting five, six, and seven times in the day, the motions loose and light yellow in colour. Examination of the eyes showed well-marked optic neuritis of the right eye, less of the left.

June 27.—The quinine having had no effect on the temperature and rigors, was stopped, and tinct. catechu ℥ xv., sp. ætheris ℥ x. H^s: A. A. ad ʒss. ordered every six hours.

June 30.—The eighteenth day from the advent of the giddiness and pain in the head, there was a decided remission in all her symptoms. The rigors ceased, the temperature fell, reaching the normal on the nineteenth and twentieth days, the pulse also fell to below 100, and this may be considered the end of the primary fever.

The first relapse began with a rigor and rise of temperature to 105° on the afternoon of July 3.

July 10.—The spleen could be felt for the first time to-day. The brandy increased to three ounces.

July 15.—The eyes again examined by Mr. Vernon, who reports that their condition is the same as on June 24.

July 17.—The spleen has enlarged during the last week. The rigor to-day not nearly as severe as usual, lasting only a few minutes. From this date until the 24th her temperature remained lower, and she had no rigors; they recurred on the 24th, but from that time until they ceased on the 31st, were never severe, and could not be compared with the ones occurring from the 5th to 14th of July.

Towards the close of July her general condition became very bad; her pulse varied from 140 to 120. She was very anæmic

and apathetic; could with difficulty be got to take nourishment.

August 1, 2, 3.—Considerable diarrhœa and some vomiting. On August 2, the fifty-first of her illness, her temperature reached 105° , but that was the last time that it rose to any considerable height. The chart is not carried on beyond August 5, but the temperature did not become really normal until August 13. When she once began to improve she made rapid and steady improvement, and went to Swanley on September 23. On August 19 the eyes were again examined, and were found in the same condition.

I have seen the girl several times since her return from Swanley, and she appears in perfect health.

REMARKS.

Although the evidence is not absolutely conclusive, I think there can be no doubt that this was a case of typhoid fever. A study of the chart shows that her illness falls into three periods—(1.) From admission to the 30th of June, which I consider was the primary attack, followed by a relapse commencing July 3rd and lasting until the 17th; then another period of remission, but not so complete as the former one, followed by a second relapse commencing on the 22nd or 23rd, and lasting until the 2nd of August, when defervescence of the fever commenced for good.

During the fourteen days which she passed in the Hospital, of the primary attack her temperature reached or exceeded 106° seven times, and she had twenty-two distinct rigors, of which some were very severe. During this period, notwithstanding the great height of the temperature and the rigors, the child, excepting for a short time during and immediately after the rigors, appeared pretty well, took her nourishment well, and amused herself reading and looking at pictures.

During the first relapse the temperature rose at times even to a higher point than in the primary attack, twice reaching or exceeding 107° , eight times reaching or exceeding 106° , and having twenty-five rigors in the fifteen days the relapse lasted. During this relapse she lost ground much more decidedly, ceased to be able to read or to interest herself in anything, but took her nourishment willingly.

During the second relapse the temperature never reached to quite the same height as in the primary fever and first relapse, reaching or exceeding 106° only twice and 105° seven times; the rigors were much less severe, and only six occurred in the

eleven days during which she had high temperature. Her condition during this second relapse was very alarming; she became greatly emaciated, was with difficulty got to take nourishment, and at times her pulse, though regular, was extremely feeble, even at times when the temperature was down.

During the forty-eight days of high fever which she had in the Hospital, her temperature reached or exceeded 105° fifty-four times, and during the same period she had thirty-three rigors. It is also to be noticed that during the same time on only five days, viz., the fourth, fifth, sixth, tenth, and thirty-first days of her fever, did the temperature fail to fall to or below 100° , and on thirty-six it reached the normal or lower. None of these falls in temperature were due to antipyretic medicine, as none was taken, unless the three grains of quinine given every four hours from June 20-27 be so considered. The ice-cap, which was applied to her head when first admitted, was not continued after the first two or three days, and although cold sponging was occasionally made use of, she disliked it so much, and it appeared to have so little effect, that it was not persevered with. The fact that the temperature, though so abnormally high, rose and fell with such rapidity, led me to consider that there was less need for attempting to keep it down than in cases in which there is a continuous high temperature; the same reason also determined me against the use of cold bathing in this case. I was more alarmed at the frequency and severity of the rigors. In a very considerable experience of typhoid fever, I had never met with any case like the present, although I have known a case of typhoid fever in which frequent rigors occurred in connection with suppuration of the gall-bladder, terminating fatally.

What is the explanation of the rigors in this case? There can, I think, be no doubt that they depended on the elevation of the temperature of the body, and may be regarded as taking the place of delirium, owing to what, for want of better knowledge, we must call an idiosyncrasy in this girl's nervous system. It is to be remembered that she was young, and just as we occasionally see in young children a convulsive attack takes the place of a rigor, so here rigors appeared to take the place of delirium, which was absent throughout the whole of her illness.

The case, when viewed as a whole, appears clear enough, but when first admitted presented very great difficulties for diagnosis. For the first two days I was inclined to regard it as one of insolation, for the weather at the time was exceedingly hot, and the headache, vomiting, and continuous fall of the temperature under treatment with the ice-cap seemed to confirm

that opinion, whilst the great range of the temperature and the rigors were unlike the ordinary condition of typhoid patients during the first fortnight of the fever. The abdominal tenderness, which only existed on the 18th, and the suspicious spot seen on the 19th of June, were in themselves hardly satisfactory evidence of typhoid fever, and the existence of optic neuritis added yet further difficulty to the diagnosis.

In this, as in many other instances of typhoid fever, the examination of the urine with Erhlich's solution was unsatisfactory, as the colour reaction was not marked.

The temperatures were taken in the axilla, and occasionally in the mouth. The thermometer used was compared with others, and no discrepancy found.

In conclusion, I must thank my house-physicians, Messrs. Fox and Woodward, for the care with which these patients were watched, and for the notes taken of their cases.

By the kindness of Dr. A. Garrod I have had references given to me of two other fully recorded cases. One to which reference is made in Virchow and Hirsch's Archives for 1887 (B. ii. p. 219), I have not been able to consult, as it was published by Dr. Korzynski in a Polish magazine, and is entitled, "A Case of Embolism of the Coronary Artery Diagnosed during Life." The other case is to be found in the Medical News (Philadelphia) for August 20, 1892. A full account is given of the post-mortem appearances of a man who died suddenly in the street of whom no history was obtained. In this case there was no valvular disease of the heart, but a roughened patch with vegetations attached was found a little above the commencement of the aorta. Numerous infarcts were found in one kidney, and an embolic mass at the bifurcation of the left coronary artery.

CASES FROM MR. SMITH'S WARDS.

WITH REMARKS.

BY

GEORGE V. WORTHINGTON, M.B.

CASE I.—*Multiple Polypi of Rectum.*

Arthur E., æt. 16. Admitted into Harley Ward, September 21, under care of Mr. Smith, suffering from multiple polypi of rectum.

History.—Admitted into Rahere Ward, October 1890, with the history that four years previously he experienced pain on defæcation, and passed blood with his motions; that nine months previously a lump as big as a cherry came down.

On examination.—On straining, a lump as large as a man's fist came down, composed of rectal mucous membrane studded with many polypi, varying in size from a pea to a gooseberry.

Many were removed, being cut off after a ligature had been applied to their pedicles.

Discharged relieved of his symptoms.

In October 1893 was again admitted (Henry Ward), with the history that one year after his discharge from Rahere was again troubled with pain and bleeding on defæcation, and something coming down (prolapse of mucous membrane and polypi).

On examination, large numbers of soft friable pedunculated polypi were discovered springing from the rectal mucous membrane, which within reach of the finger is in no part free from the disease.

No bleeding on gentle digital examination.

Under chloroform, with use of speculum, mucous membrane seen to be studded with polypi of a dull red colour, varying in size from that of a pea to a marble; did not bleed much.

Two or three of the larger and more movable ones were removed.

Discharged relieved of pain and bleeding on defæcation.

Was seen in Out-patient Department one year after his discharge, with return of old symptoms. Declined to again enter the Hospital.

Since that time has been getting worse, the pain at times being severe and amount of bleeding more; now returns to Hospital for further treatment.

Family history.—Mother suffered from same disease, polypi, being of same variety—*i.e.*, adenoid. Was operated upon at age of 19 in the London Hospital, and again at age of 21 in same Hospital. At 22 was married. At 28 had return of old disease. At 29 died of phthisis.

On September 22 was examined under chloroform. With the finger could be felt a large number of polypi, varying in size from that of a pea to that of a medium-sized strawberry. The disease extended beyond the reach of the finger.

On using the speculum, a large number of polypi came into view, arising by their stalks from the mucous membrane, of a dull red colour, and all covered to a greater or less degree with mucus. They were in appearance and shape similar to ripe strawberries, picked in the rain a day or two before inspection. Bled slightly on examination.

October 2, 1896.—Chloroform administered in the theatre; put up in the lithotomy position and speculum introduced.

Some straining brought down some of the mucous membrane with a large mass of polypi.

About fifteen of the larger were ligatured with silk and cut off with scissors, varying in size from a walnut to a bean.

One nipped off without ligature; pedicle at once commenced to bleed pretty freely; bleeding point taken up and ligatured; with this exception practically no hæmorrhage. Prolapsed mucous membrane returned within the anus; no plugging. Pad of iodoform gauze fixed over anus with T bandage.

Left untouched for forty-eight hours; for the week following rectum washed out daily with boracic lotion by means of a double-way catheter.

Bowels opened on fourth day; no pain or hæmorrhage.

Ligatures came away on sixth and seventh days.

Discharged October, quite relieved of pain and hæmorrhage, and much improved in general health and appearance.

CASE II.—*Growth and Multiple Polypi in Rectum.*

Arthur C., æt. 41, coalminer, admitted to Henry, November 7, 1896, under care of Mr. Walsham, suffering from growth and multiple polypi of rectum and secondary enlarged glands in groin.

History.—Was admitted into Harley Ward, February 28, 1896; gave a history of gnawing pain in rectum and constipation for two weeks previous; bowels quite regular till onset of pain; pain much increased on passing a motion.

On examination, was found to have a growth about size of bantam's egg in the rectum, situated on the anterior and left wall, also a number of multiple polypi of various sizes up to that of a pea, scattered all over the mucous membrane as far as the finger could reach.

The growth was excised; made a complete recovery, having had slight difficulty in retaining fæces for about three weeks.

April 10.—Sent to Swanley; could retain fæces without difficulty. No contraction.

Since has been gaining flesh and doing well till about second week in August, when he noticed pains in rectum. Was examined by a doctor and nothing found.

At end of August patient noticed a lump in right groin; was recommended by doctor to return to Hospital.

Previous illnesses.—Has been subject to piles.

Family history.—One brother died of carcinoma of rectum. One sister was in Royal Free Hospital, under Mr. James Berry, suffering from multiple polypi and carcinoma of rectum, for which she has had inguinal colotomy performed.

Condition on admission.—A tall, gaunt individual, with a sallow complexion. In the groin (R.) are situated some hard enlarged glands, which are not markedly tender, are not adherent to skin nor fixed to the deeper tissues.

In the rectum there is found a hard nodular lump, situated on the posterior wall of the rectum, rather to the left, and just inside the sphincter; its size is about that of a walnut; it is somewhat fixed, and gives some pain on examination; does not bleed on gentle manipulation.

It is quite free from, and apparently has no connection whatever with, the scar of the former operation, which is quite soft and flexible.

The rest of the rectal mucous membrane is studded with numbers of polypi, varying in size from that of a hemp-seed to that of a small bean, and extending as far as the finger

can reach; they are not painful, and do not bleed on digital examination.

November 11.—Growth was excised by Mr. Walsham, and one polypus removed for examination.

The glands in the groin were removed, and found to be hard and carcinomatous.

Wound in groin healed by first intention; stitches removed at end of a week.

November 30.—Wound left by excision of growth not yet healed.

On microscopical examination, the growth proved to be adenoid carcinoma.

The polypus was of the adenoid variety.

REMARKS.

These two cases show well the general characters of this disease, which is a rare one; there are at present not more than five or six specimens in the museums of the London hospitals.

As regards the pathology of the disease, these polypi are, as a rule, of the adenoid variety, as in both these cases. Mr. Cripps states that they are sometimes fibrous in nature, and there is a specimen in St. Thomas' Hospital Museum in which they are composed of lymphatic tissue. This case was reported in *Path. Soc. Trans.*, vol. xli. p. 131, by Mr. Shattock.

They generally occur in large numbers, of various sizes, and extend throughout the whole length of the large intestine, occasionally into the small.

They are usually attached to the mucous membrane by a thin stalk, occasionally by a sessile base.

As long as they remain in the form of a polypus they are innocent; but occasionally, as in Case II. and his sister, and also in a case reported in a series by Mr. Smith in *Hospital Reports*, 1887, the growth extends in the other direction, involving the mucous, muscular, and peritoneal coats of the rectum, and also the tissue beyond; in fact, becomes an adenoid carcinoma, and may be compared to an innocent papilloma or wart becoming an epithelioma by the ingrowth of processes of epithelium.

Perhaps in Case I., if the boy lives long enough, carcinoma of the rectum will be the end.

This disease is prone to occur in members of the same family, of the same or of a different generation, as is shown by both these cases and by Mr. Smith's series.

In Case I. the mother suffered from same disease.

In Case II. the sister suffered from same disease, possibly also the brother, who is said to have died of carcinoma of rectum (perhaps there is some connection between polypi in one and carcinoma of rectum in another member of same family).

The symptoms usually complained of are loss of blood from the bowel, uneasiness and pain, especially on having the bowels opened, and of "something coming down." Hæmorrhage is usually a prominent symptom, and may be very severe, as in one of Mr. Smith's series, leaving patient very anæmic, or may even cause the death of the patient; on the other hand, it may be slight, or not noticed at all, as in Case II.

The pain may be slight or severe; in Case I. it was at times very severe.

Often "something comes down," as in Case I. This may be either one or two polypi, or, as is usually the case, the rectal mucous membrane, with the polypi attached.

Prognosis.—The disease is incurable; the growth may remain as polypi, and so innocent, for many years.

Hæmorrhage may give trouble, and may even cause the death of the patient.

It may become malignant, taking the form of adenoid carcinoma, and eventually causing the death of the patient.

Treatment.—Operation to relieve patient of the prominent symptoms. The largest polypi, where available, may be ligatured and cut off, and this has, as a rule, to be repeated many times, as in Case I.

CASE III.—*Streptomycolosis or Septicæmia Treated with Anti-streptococcic Serum.*

George R., æt. 28, stoker, admitted into Harley, October 23, 1896, under care of Mr. Smith, suffering from necrosis of femur, abscess in thigh, and septicæmia.

Family history.—Nothing of importance, and no previous illness.

History.—Ten years ago knee became red, swollen, and painful, for which he could not account; saw a medical man, who painted it with iodine and ordered poultices; after one month swelling burst, and a large quantity of blood and matter was discharged.

Three months after leg first became bad came up to Hospital as an out-patient, and was advised to come in. Was admitted to Henry, developed erysipelas of leg, and went to Coborn,

where he remained five weeks; was then sent to Rahere, where Mr. Smith removed a piece of dead bone; he again developed erysipelas, and went to Coborn for another six weeks; was then sent to Swanley for three weeks. During the whole of this time had some discharge from wound in thigh; attended Out-patient Department once a fortnight for three months, and has since had no medical attention.

October 14, 1896.—Came up to Surgery to see what could be done for it, and was found to have two discharging sinuses on outer side of lower part of thigh; dead bone found at bottom. Temperature normal.

Saw Mr. Smith on October 23, saying that two days before he had had a shivering fit, and the day before another, and that he felt very ill. Was admitted.

Condition on admission.—Looked ill, and evidently was. Face pale and drawn, tongue furred, pulse full and rapid, respiration accelerated. Temperature 100° .

On lower and outer part of thigh were two sinuses about $\frac{1}{4}$ in. across and $1\frac{1}{2}$ in. apart; skin around shining red, and in parts bluish and œdematous.

These sinuses were discharging thick foul-smelling blood-stained pus; no collection discovered in thigh.

Temperature at 9 P.M. 103° . Ice-bag applied to head and quinine (gr. x.) given.

October 24.—Bowels opened. Temperature still 103° . Quinine (gr. v.) three times a day. Cradled. Temperature falling. Less discharge from sinuses.

October 25.—7.30 A.M., temperature 101.6° ; shortly after this patient had a rigor which lasted twenty minutes. Temperature rose to 104° ; respiration 40; pulse 120.

3 P.M. put under an anæsthetic.

Mr. Smith made an incision joining the two sinuses together and letting out a quantity of foul-smelling, blood-stained pus; bone not loose and could not be removed. Femur was found to be extensively bared of periosteum.

Irrigated with hydrarg. perchlor.; tubes put into the wound, plugged around with iodoform gauze. Dry dressed. Temperature falling, 101° at 7 P.M.

Taking food well (milk, essence, beef-tea, brandy). Delirium at intervals during a restless night.

October 26.—3 A.M. temperature 100.8° ; at 7 A.M. 103.4° ; pulse 120; respiration 35.

2.30 P.M. 15 cc. of antistreptococcic serum injected into cellular tissues of abdominal wall. In half-an-hour pulse had decidedly improved, being slower and of better volume. Tempera-

ture began to fall; at 3 P.M. had been 104.4° , at 7 P.M. 103° , at 11 P.M. 102° .

October 27.—2.15 P.M. 20 cc. serum injected had no effect on temperature, which was 102.4° , but pulse improved.

Has passed a restless night, with delirium at intervals. Is taking well.

11.40 A.M. rigor lasting 20 mins.; temperature rose to 106° .

12.30 P.M. 20 cc. serum injected; cradled. Ice-bag to head. Quinine and brandy.

1.25 P.M. another rigor, lasting 25 mins.; temperature rose to 106° .

3 P.M. temperature fallen to 101.1° .

10.50 P.M. rigor lasting 25 mins.; temperature rose to 105° .

October 28.—3 A.M. temperature 104° ; pulse 125; respiration 35. 20 cc. serum injected. Temperature fell rapidly to 102° at 7 A.M. and 100.4° at 11 A.M.; pulse 100; volume improved. Slept very little during night, constantly muttering.

12.15 P.M. 20 cc. serum injected.

2.10 P.M. rigor lasting 15 mins.; temperature rose to 106.5° ; pulse 120, rapid and full; respiration 40. Ice-bag to head; cradled. Quinine sulph. gr. x.

Temperature at 3 P.M. 103.5° , at 7 P.M. 103.8° .

8 P.M. rigor; temperature rose to 106° . Ice-bag; cradle. Quinine sulph. gr. x.

10.30 P.M. 20 cc. serum injected. Temperature 103.4° .

11 P.M. temperature 101.4° .

October 29.—12.30 A.M. temperature 101.2° , at 1.30 A.M. 101.4° , 2.30 A.M. 101.2° , 3.30 A.M. 101.6° .

7 A.M. temperature 102° ; pulse 112; respiration 30. Shortly after rigor lasting 35 mins., temperature rose to 105° ; respiration 60; pulse 140. Ice-bag; cradle. Quinine. Had rather restless night, slept a little after morphia; delirious; passing urine and motions under him; diarrhoea.

Temperature 11 P.M. 103.5° ; 12.45 P.M. 20 cc. of serum injected.

Temperature 3 P.M. 102.8° , 7 P.M. 103° , 11 P.M. 102.4° .

October 30.—2.30 A.M. temperature 102.4° , 20 cc. serum injected; 3 A.M. temperature 102° , at 5 A.M. 101.4° , 6 P.M. 103° , 7 P.M. 103° , 11 P.M. 104.2° .

2.15 P.M. temperature 104.2° , 20 cc. serum injected, 3 P.M. 103.8° , 4 P.M. 105.4° , 5 P.M. 105.6° , 6 P.M. 104.6° , 7 P.M. 103.8° , 11 P.M. 104.8° .

October 31.—1.30 A.M. temperature 104.8° , 20 cc. serum injected; 2.30 A.M. temperature 104° , 3 A.M. 104° , 3.30 A.M. and 4.20 A.M. 104° , 7 A.M. 102° , 11 A.M. died.

Leg dressed and irrigated with HgCl_2 1 in 1000 twice daily. Four pieces of dead bone came away with irrigation.

Food, which consisted of milk, beef-tea, mutton essence, eggs (beaten up), brandy and champagne, taken well up till 30th.

Patient was always delirious at night.

Dr. Kanthack kindly made cultivations from both the discharge and blood of the patient, and pronounced the case to be a genuine one of streptomycosis or septicæmia, the result of infection by the streptococcus pyogenes, and at his suggestion the serum was pushed.

The preparation used was that of the British Institute of Preventive Medicine.

Post-mortem.—Well nourished.

Outer side of thigh, just above knee, operation wound leading down to femur, which was necrosed from lower epiphysis as high as great trochanter; periosteum sloughy and completely stripped off from and separated from the bone by pus.

Extensive infiltration of muscles of thigh with similar pus.

Abscess, which reached up to gluteal region, was indirectly drained through wound.

Necrosed portion of femur thickened from old periostitis, and near lower end showed marks of recent chiselling at this place; detached sequestrum about 1 in. long lying in a cavity in the bone.

Knee-joint showed old adhesions and inequalities of cartilages, seat of old synovitis; no recent disease of any joint.

Left femoral and iliac veins contained dirty purulent blood-clots.

Kidneys, liver, spleen (especially latter) all swollen and congested; other abdominal viscera appeared normal.

Heart and pericardium normal.

Commencing septic pleurisy on both sides.

Lungs showed well-marked early septic pneumonia; no secondary abscesses anywhere.

Head not examined.

In this case, after what was found post-mortem, it may be considered doubtful whether, and hardly to be expected that, any treatment could have saved the patient's life. The complete necrosis of femur and sloughy condition of its whole periosteum, the extensive infiltration of muscles and other tissues of thigh, added to the general infection, had already made the result almost a certainty.

But after injection it was noticed that, as a rule, the temperature was lowered in varying degree, sometimes with considerable rapidity, at other times slowly.

The time of onset of the fall after the injection was given was not constant, and could not be foretold. On two occasions there was no fall, but a rise; the time during which the effect of injection lasted was also variable.

After each injection the pulse was improved in volume and lessened in rapidity, even when there was no fall of temperature.

The patient on two occasions thought he felt better after injection, but said on the whole the thing did him no good.

It appeared to have no effect on his delirium or general discomfort.

The following is an abstract of a collection of cases of puerperal septicæmia published by Williams, *British Medical Journal*, 1896, p. 1285, partly collected from literature, partly from my own observation:—

Fourteen cases of puerperal septicæmia submitted to serum treatment, of which six were under own observation, eight from literature.

Two fatal cases, twelve recoveries.

Ten definite records of state before and after administration.

Onset from a few hours to eight days after labour; usual local and constitutional treatment to start with in each case.

Administration begun fifth to nineteenth day after labour.

State of pelvic organs noted in nine cases; normal except in two cases (uterine tenderness).

In all cases severe febrile symptoms, some diarrhœa and vomiting.

In only two cases was there made a bacteriological examination.

Serum was injected with a sterilised syringe into areolar tissue of abdominal wall, skin having been prepared antiseptically.

Doses up to 45 cc. Bullock (*Lancet*, i. 96, p. 1216). Large doses with safety; 10 cc. into rabbits, no ill effect.

(The strength of various preparations is uncertain; some standard is wanted.)

Suitable cases.—Only those of strepto-infection or streptomycosis.

Discharges and blood should be bacteriologically examined in each case; if streptococcus pyogenes not present, it is useless to go on.

Effect of serum.—Hot dry skin became moist and active, parched lips and dry tongue became moist, lochia and lactation reappeared. Delirium, insomnia, and restlessness passed off and patient slept. Headache and mental torpor dispelled; exceptionally headache remained for some hours.

In each case except three, degree of temperature and frequency of pulse relieved after each injection.

The temperature, as a rule, fell rapidly; three cases normal in twenty-four hours; effect noticed in from six to twenty-four hours.

Two fatal cases.—(1.) Daily injection of 20 cc.; no observed benefit. (2.) 10 cc. on fourth, fifth, sixth, and seventh day after labour; temperature normal on ninth day; on same evening bilious vomiting and meteorism; vomiting uncontrollable; became semicomatose, and died on eleventh day.

Erythematous rash in two cases.

CLINICAL APHORISMS FROM DR. GEE'S WARDS.

1895-1896.

COLLECTED AND EDITED BY

THOMAS J. HORDER.

A few words of explanation are perhaps appropriate as an introduction to this contribution to the Hospital Reports. Anything of the nature of an *Apologia* I feel is not needed, since I am convinced that what follows, though not previously attempted in this work, falls well within—if indeed it does not exactly cover—the scope of these Reports. For, in their preface to the first volume, issued in 1865, the Editors expressed the “hope . . . that these Reports may represent . . . not the practice of the Hospital only, but the opinions, the teaching, and the experience of the school.” As to the form which this record of clinical experience takes, should any reader at first think it new, a second reflection will doubtless remind him that medicine knows none older.

Most of us are not long in learning that text-books, however excellent, can take us but a short way along the road of medical education. To be brought face to face with the patient in the presence of the master, however, can take us considerably farther. Hence it is that I have thought these Aphorisms, which are for the most part the result of what Trousseau calls “*les causeries familières des salles d’hôpital*,” worthy of a more permanent and a more public place than they have hitherto occupied in my own note-book. To many readers not a few of them may be already familiar,—to some, indeed, an Aphorism may recall particulars of the very “case” the discussion of which gave rise to it,—to all I cannot but think they will prove of value.

In several instances the words used are entirely the original

ones; in the rest, the original language is preserved so far as my memory has made this possible. That any of the language is mine is to be regretted; but that the substance of each Aphorism has been maintained may be inferred from the fact that Dr. Gee very kindly read over the sentences, making those corrections which seemed necessary in view of my availing myself of his permission to print them.

To facilitate reading, I have arranged the Aphorisms under several headings. As it was not desirable to multiply these indefinitely, and the range of subjects touched upon is very wide, some of the headings are necessarily rendered rather comprehensive. For the "Notes" on pp. 57 seq., and the references contained therein, I am alone responsible.

"Il faut voir, toujours voir, des malades. Ces matériaux confus, que l'on amasse sans ordre et sans méthode, sont pourtant d'excellents matériaux; inutiles aujourd'hui, vous les retrouverez plus tard enfouis dans les trésors de votre mémoire."—TROUSSEAU.

I.—Of Some Pulmonary Physical Signs.

1. No other terms convey any information about the breathing sounds than those in the following table:—

Breathing may be	{	i. Vesicular.	{	i. Ordinary.
				ii. Weak.
				iii. Loud (= Puerile).
		ii. Bronchial.	{	i. Ordinary.
				ii. Cavernous.
				iii. Amphoric (rare).

Other terms, *e.g.*, "harsh," &c., are objectionable, because they correspond to no known pathological conditions, and thus add nothing to our knowledge.

2. Intense bronchial breathing indicates the presence of either consolidation or cavity. Hence the condition may be present in (i.) tuberculous disease, (ii.) simple dilatation of the air-tubes in emphysema, or (iii.) cirrhosis of the lung, with dilated air-tubes. The co-existence of dulness would exclude (ii.), much sputa would suggest (i.), and tubercle bacilli in them would confirm it.

3. In little children the so-called "bronchial breathing" is nearly always a bronchophony. When present, it is nearly always heard most distinctly over the junction of the healthy and the consolidated lung.

4. In pleural effusion there is sometimes hardly any breath-

ing sound to be heard, and sometimes there is loud bronchial breathing. If we accept the view that the breath-sounds are all produced at the glottis, then, bearing in mind that both solid lung and fluid are good conductors of sound, we shall rather wonder that the sign of bronchial breathing is not *always* met with in these cases. But if, in those in which it is not met with, we suppose the air-tubes to be compressed, we have an explanation why sounds cannot be properly conducted along them; but this assumption would be very difficult to prove correct.

5. "Consonating râle," termed "cavernous râle" by Laennec—a bad name, because it implied that the sound was always produced in a cavity—indicates that the râle is produced either near consolidated lung-tissue or in a cavity,

6. It is not uncommon for children to be thought the subjects of pulmonary phthisis when post-mortem examination shows no tubercles, but only cirrhused lung and dilated air-tubes. These two conditions, therefore, need careful distinction. Operation undertaken for the purpose of draining a supposed phthisical cavity may reveal the same state of things. But often the operation is attended with good results in spite of this.

7. There is no disease of the chest in which the signs are plainer than in pneumothorax, and yet there is no disease the signs of which are less often understood, and which is, therefore, more often overlooked. The auscultatory signs are amphoric breathing, bell-sound, metallic tinkle, and—if fluid be present—succession splash. The conduction of the bell-sound is good, whether the medium be liquid or air: the excellence of its conduction depends upon a homogeneous medium.

II.—Of Phthisis.

8. Phthisis spreads by means of dried sputa, and any other mode of spreading must be considered trifling in comparison.

9. Phthisis at the bases of the lungs is not uncommon, but its existence is often overlooked because the apices alone are examined in suspected cases.

10. One may be quite sure that in any case of phthisis the disease is much more extensive than the physical signs would seem to indicate.

11. Most cases of phthisis progress more rapidly, and terminate sooner, than is usually thought. In any case, if physical signs of phthisis are once present, the probabilities are very

much against recovery; but if the disease is detected before this and treated, there is hope that it may be cured.

12. Before the appearance of physical signs it is the presence of some or all of the following facts which leads one to a diagnosis,—hæmoptysis, cough, loss of flesh and colour, slight rise of temperature, and hereditary liability to the disease.

13. The great value of therapeutics in this disease, therefore, lies in their application before physical signs have developed at all; for if you wait for physical signs, you wait too long.

14. Always say three things to a patient whom you suspect to be phthisical—

(i.) Get yourself weighed—by the same machine each time—to see if you are losing weight.

(ii.) Use a thermometer two or three times each evening, to see if there is any fever.

(iii.) Save your sputa to be tested (for bacilli).

If, besides auscultation and percussion, these three points give negative results, you may safely hope there is no phthisis.

15. Above all, never give a definite opinion as to how long a patient suffering from phthisis will live; for the only certain thing about it is, that if you do, you will invariably be wrong.

16. Almost every chronic affection of the apex of the lung is tuberculous in nature.

17. Aphonia in phthisis is not always due to local tuberculous mischief; it is sometimes the result of mere debility, the vocal cords being quite natural in appearance.

18. Phthisis may begin as bronchitis, as pneumonia, as pleurisy with or without effusion, as pneumothorax; and in either case the beginning may be quite sudden.

III.—*Of Hæmoptysis.*

19. The commonest cause of hæmoptysis is phthisis; the next commonest cause is disease of the heart, which leads to congestion of the lungs or to embolic infarction of those organs.

20. Excluding cancer, which, if present and causing hæmoptysis, can usually be detected, the larynx is *not* a source of bleeding. Nevertheless, patients with hæmoptysis very often wish the physician to believe that the blood comes “from the throat.”

21. In a case where blood is brought up, if the sputa are themselves bloody, this proves the blood to have come from the lungs.

22. Hæmoptysis in phthisis never proves fatal except in the last stages of the disease, and where a large cavity exists.

Hence there is no cause for great alarm when the condition occurs.

23. Hæmoptysis sometimes occurs, on and off, for many years—all through life, it might almost be said. In these cases, it probably is due to the presence of a small cavity, often not larger than a cherry, and giving no physical signs on examination of the chest.

24. Hæmoptysis during an attack of asthma with bronchitis may be very great.

25. Hæmoptysis is not uncommon at the beginning of an attack of pleurisy with effusion; it is then probably due to the collapse and congestion of the lung.

26. In a young man an attack of hæmoptysis is quite sufficient indication for treating him for phthisis. It is not so in the case of a young woman. Young women seem to bleed much more easily than young men, so that here considerable importance should be attached to the question of sex. Again, in the case of a young man, it is not necessary that the sputa should be actually coloured by blood; a few streaks of blood, like threads of scarlet silk, running through them, are sufficiently characteristic.

27. The treatment of hæmoptysis by drugs is not satisfactory. Perhaps the best drug is ipecacuanha in emetic doses, when its action is probably due to the depression of the heart which precedes the vomiting. Now that emetics are no longer in fashion, other drugs have replaced ipecacuanha. Of these, gallic acid, though often given in sufficiently large doses to produce a green coloration of the sputa, thus proving it to have reached the affected region, does not stay the hæmoptysis. Ergotin seems sometimes to be of use, but at other times conspicuously fails. Opium is of value here as it is in all cases of hæmorrhage. But, in the end, the patient who was wont to treat his own attacks of hæmoptysis by "going to bed, sucking ice, and feeding on milk," summed up in brief the essential points in our most successful treatment of this condition.

IV.—*Of Bronchitis and Emphysema.*

28. An ordinary case of bronchitis is not febrile for more than a week or so. Later than this the temperature becomes a valuable sign in diagnosing the disease from phthisis or scattered tubercle.

29. It is hardly easy to over-estimate the frequency of collapse of quite a large part of the lung in children from very

slight causes—very little pulmonary catarrh producing a small amount of mucus in the air-tubes, the presence of enlarged glands along the trachea, &c. Hence signs of consolidation do not of themselves necessarily justify a diagnosis of pneumonia or tubercle.

30. I have satisfied myself that bronchitic signs may be wholly restricted to one lung, and the condition still be mere bronchitis. But when this is the case, there is always the suspicion that something worse is at the bottom of the condition—*i.e.*, tubercle.

31. There is no more potent cause of chronic bronchitis than alcohol.

32. Diarrhœa setting in spontaneously in a case of bronchitis relieves the pulmonary complaint; but do not attempt to induce diarrhœa in treating a case of bronchitis, for purgation does no good.

33. Acute emphysema of children may cause very marked protrusion of the front of the chest, accompanied by dyspnœa. If examined post-mortem, the lung in this disease is found to be white and bloodless, and scrutiny of its surface reveals the dilated air-cells.

34. Emphysema consists of (i.) distension of the lung alveoli, and (ii.) destruction of them; the former occurs more largely in children, the latter in adults. Hence the prognosis is good in emphysema of children, bad in emphysema of adults.

35. Chronic bronchitis and emphysema may simulate asthma in the character of the dyspnœa, the attitude of the patient, &c. When this is so, it may be of use to try anti-asthmatic remedies.

36. Fœtor is by no means a *sine qua non* in the pus from a bronchiectasis.

37. Contrary to what might perhaps be expected, operations upon gangrenous cavities of the lung are sometimes attended by very good results.

V.—Of Stinking Expectoration.

38. The causes of stinking expectoration are five—(i.) empyema; (ii.) gangrene of the lung; (iii.) phthisis; (iv.) diseased bronchial tubes; (v.) abscesses of the bronchial glands.

(i.) The physical signs in a case of expectoration of an empyema are often very slight, because (α) the cavity is small and deep-seated, or (β) the cavity contains air.

(ii.) Usually only a small piece of lung is affected. It is not

necessarily fatal. One may often get very good specimens of lung tissue from the sputa in these cases.

(iii.) In those cases where the cavity contains a small slough, or where the wall of the cavity itself sloughs, both the cavity and the slough may be very small.

(iv.) Here we have two varieties:—(a) Where the tubes are dilated, and the secretions putrefy in them. This condition may be present in cirrhosis of the lung. (β) Where the secretions of a bronchitis putrefy in undilated tubes.

(v.) A rare condition, and difficult of diagnosis.

VI.—Of Pleurisy and Empyema.

39. It is safe to say that pleurisy is always due to an infection. Nevertheless, it would be absurd to deny the influence of injury, exposure, or local cold as determining factors in the onset of an attack. Either of these may produce a suitable soil for the infecting microbe.

40. Tubercle is one of the commonest causes of a clear serous effusion. Cases may occur where, on drawing off the fluid, advanced phthisis is discovered, with the existence of cavities in the lung, the detection of which was quite impossible by auscultation previous to the tapping. Moreover, many cases of serous pleural effusion end fatally within ten years from tuberculosis. Again, tuberculous peritonitis is so often recovered from, that probably tuberculous pleuritis is also recovered from, cases never proved to be tuberculous having really been so.

41. Vomiting is an almost invariable symptom at the onset of pleurisy in children. Hæmoptysis, again, is not uncommon in both children and adults.

42. Temperature is no guide to the nature of a pleural effusion, whether serous or purulent. Neither is the duration of the effusion. But rigor and a discharge from the ear, if associated, probably point to an effusion being purulent.

43. A large heart may so press upon the root of the left lung as to produce collapse of its lower lobe. Such a condition can only be distinguished from pleural effusion by the needle. The symptoms which attend an effusion occurring in such circumstances are more often than not left unrelieved by tapping.

44. Solid tumours of the chest and pleurisy with effusion give the same physical signs; but the former do not displace viscera (*e.g.*, the heart), whereas the latter do.

45. In the presence of a pleural effusion the co-existence of a pericardial effusion cannot be made out by physical examina-

tion of the chest. Post-mortem, many unsuspected pericardial effusions are continually being discovered.

46. Blood-stained pleural effusion, though at one time supposed to be diagnostic of malignant disease of the lung, is not really so; for it occurs in some cases which rapidly recover, and, on the other hand, it is absent in some well-marked cases of cancer, the effusion being quite clear.

47. Serous pleural effusion so often clears up, that it is not good practice to drain one off as soon as discovered. Hippocrates lays down fourteen days as a good time after which interference is to be recommended, and the rule is not a bad one.

48. Serous effusions do not tend to become purulent. In the great majority of instances purulent effusions are purulent from the beginning, and serous effusions remain serous throughout. Rarely, an effusion which is purulent at first may be found to be serous at a later paracentesis, but such a sequence has occurred.

49. Those cases of pleurisy where one suspects tubercle as the cause clear up quite as quickly as others.

50. You never know what may be at the bottom of a pleural effusion, especially a chronic one. Often the treatment adopted is based upon the assumption that the condition is one of simple pleurisy, whereas one of several complications may be present (*e.g.*, an aneurysm of the aorta), which may render evacuation of the fluid very dangerous.

51. In passive pleural effusions, such as the hydrothorax of Bright's disease or heart disease, evacuation of the fluid does not give much relief. If the distress is great, however, it should be done for what little relief does follow. (See Aph. 43.)

52. The chronicity of a pleural effusion affects the prognosis in a similar way, for the tendency is very great for the chest to refill almost as soon as it is emptied in cases of long-standing effusion.

53. Paracentesis of the chest has been known to cause fatal hæmoptysis on the spot, as in a case where it was due to the rupture of a minute aneurysm situated in a small phthisical cavity of the lung.

54. There are three signs for desisting from the evacuation of a pleural effusion—cough, pain in the chest, and staining of the fluid by blood. The coughing which is induced during a paracentesis is probably due to œdema of the lung, set up by the returning blood-stream; this produces an exudation, and, when the coughing is considerable, serous sputa containing much albumin are the result.

55. I was wont to think that empyema confined to an apex

of the lung did not occur, and that I could therefore exclude empyema as a cause of dulness in this region; but I have since seen a case.

56. The microbe in the empyemata of children is usually the pneumococcus; in that of adults it is usually streptococcus and staphylococcus. Hence, perhaps, the better prognosis in the former class of patients, and the greater liability for the condition to clear up after a single tapping.

57. Empyema rarely ruptures spontaneously now-a-days; but if this does occur, perhaps the commonest situation on the left side is at the point where the normal apex-beat of the heart is felt.

58. A small amount of most offensive-smelling pus may be drawn off from the pleura, and yet no further operation be required for the condition to clear up. If the amount present, however, is considerable, the chance of this happening is small. The fœtor may possibly be accounted for by the proximity of the œsophagus.

VII.—*Of Pneumonia.*

59. Pneumonia is not a local, but a general disease; and the brunt of it may fall upon any part—lungs, membranes of the brain, intestines, kidneys.

60. Acute pneumonia in children may run a very short course, the crisis appearing sometimes as early as the fourth or fifth day.

61. Pneumonia may be a cause of otorrhœa; in many cases the pneumococcus is abundantly found in the pus from such a case.

62. The way in which pneumonia clears up depends much upon the state of health the patient was in when attacked. In men who drink hard, the period of resolution may be very long. If evidence of consolidation of the lung be found after six weeks, the character of the consolidation is the same as it would be at the end of one week—*i.e.*, hepatisation. At the end of several months, however, it would probably be of the nature of fibrous degeneration.

63. The mortality from pneumonia in patients who drink hard is very high, and perhaps the reason is to be found in the fact that alcoholism predisposes so much to delirium.

VIII.—*Of Diseases of the Heart and Pericardium.*

64. If more than one valvular lesion be present in a heart, it is rarely that more than one is diagnosed. One of the commonest complications is the existence of mitral stenosis

with aortic insufficiency, and then the development of the typical aortic diastolic murmur may be considerably interfered with.

65. The heart-murmur called after Austin Flint, if it really occurs at all, is very rare. Only three or four well-marked cases have been recorded.

66. Dilatation of the left ventricle may occur in a heart having mitral obstruction. This seems contrary to theory, but the subject of cardiac dilatation cannot be wholly explained by a consideration of pressure-effects. The amount of dilatation in the case above-mentioned may be often astonishing.

67. There is no kind of valvular disease of the heart which may not produce a thrill.

68. Mitral disease causes lividity by interfering with the circulation of the blood, bronchitis by interfering with its aëration, and emphysema in both ways.

69. "Corrigan's pulse," capillary pulsation, visible pulsation and twisting of the arteries, though all typical features of aortic regurgitation, are none of them pathognomonic of the disease. Each may occur without the presence of any valvular lesion, and in old people they are often due to arterial degeneration. "Corrigan's pulse" is common in atheroma of the aortic arch.

70. Venous pulse in the superficial veins of the arms or hands, however, must be regarded as a more conclusive sign of aortic regurgitation, for it only occurs in the course of that disease, and perhaps in a few cases of extreme debility.

71. A rarer sign, but one which is quite pathognomonic of aortic regurgitation, is the conduction of the diastolic murmur into the large arteries, such as the femorals, over which vessels it is heard on auscultation, without using pressure with the stethoscope.

72. It is important to remember that ulcerative endocarditis may produce quotidian fever some weeks before the appearance of physical signs on auscultation of the heart.

73. Very great dilatation of the heart is commoner in children than in adults, and may be produced very rapidly—in a few weeks even.

74. Fatty disease of the heart is one of the most serious conditions of this organ, and yet is devoid of any corresponding physical signs. And this is only part of a general statement that might be made: Affections of the muscular tissue of the heart are usually more serious than those of its fibrous tissue (*e.g.*, the valves), but are usually represented by much less definite physical signs.

75. There is no conclusive way of diagnosing "adherent pericardium;" for when systole of the heart is accompanied by retraction of the wall of the thorax, this is due to the pericardium being adherent, not only (and not necessarily) to the heart, but also to the front and back of the thoracic cavity. Traction is thus made upon the walls of the thorax when the heart shortens during systole. But the diagnosis of adherent pericardium would not be of very great value.

76. Pericarditis is not nearly so common now as formerly, and this refers especially to the large pericardial effusions once seen. The fact can only be attributed to the use of sodium salicylate in the treatment of rheumatic fever. Endocarditis, however, seems as common as ever.

77. In cases of erythema you should always examine the heart, for pericarditis is not uncommonly associated with it, even without any joint or other rheumatic symptoms.

78. Pericarditis is very common in Bright's disease, but is not often detected ante-mortem.

79. In pericarditis it should be remembered that there may be a good deal of effusion of liquid, and yet the friction sound may remain.

80. Dropsy, lividity, scanty urine, and irregular pulse—these are the four symptoms which indicate the use of digitalis, quite irrespective of the nature of the heart-lesion producing them. On the other hand, the use of digitalis is *not* indicated by finding on examination that the heart is dilated, the above symptoms being absent.

IX.—*Of Diseases of the Blood and Blood-Vessels.*

81. From both clinical and post-mortem observations it does not seem proven that the disease called "pernicious anæmia" is a definite entity. Cases so diagnosed during life often turn out on post-mortem examination to be cancerous, where the growth is in situations unsuspected during life—as the kidney—and the cachexia was well marked.

82. True leucæmia is very rare in young children. I have only seen two cases, and hardly expect to see another. The term "leucæmia" is a badly defined one; its use should be restricted to those cases in which the number of white blood cells almost equals the number of red ones, and used in this way it may be said that cases of leucæmia never recover, though they may rally.

83. In important contradistinction to cases of true leucæmia are those (occurring in children) where the spleen is enlarged

—sometimes reaching the right iliac fossa—where there is anæmia, bleeding from the gums, and a liability to bruising. Here, however, the prognosis is not necessarily bad, for the cases recover often. Hence the distinction is valuable.

84. You should never see a case of anæmia in a young woman without thinking of phthisis as a possible cause of it.

85. It is often a question, when called to treat a person suffering from dyspepsia and anæmia, whether the dyspepsia is due to the anæmia, or *vice versa*. In chlorosis, cardialgia and other dyspeptic symptoms are often very marked; and here it is unnecessary to treat them, for they are secondary.

86. There are cases of permanent anæmia in which the blood-forming faculty, wherever that resides, is feeble, and we cannot stimulate it. Iron is no longer a specific. A single hæmorrhage may result in an anæmia which lasts throughout life, as in the case of Paulina, the wife of Seneca, recorded by Tacitus.

87. Iron is not of much use in the anæmia of nephritis, or in any other form of anæmia excepting chlorosis, for which it is a specific.

88. Arsenic is sometimes of great use in the treatment of malarial fever, and especially in those anomalous cases of aperiodic fever in which quinine fails to do good.

89. It is quite common for a patient to live after "rupture" externally of a thoracic aneurysm. Fainting after the first gush of blood tends to stay the flow, and the deposit of laminated fibrin beneath the skin protects from severe loss of blood afterwards. The presence of this fibrinous layer and its thickness or thinness are very important points in determining the length of life in these cases. Much more common than actual rupture, however, is a process of gradual ulceration and leakage or oozing from the vessel.

90. Phlebitis is common in phthisis, as indeed it is in many infective diseases. This would seem to favour the view that the condition is a primary one in the veins, and not the result of thrombosis, though this is doubtless also true in some cases. In this connection it should be remembered that suppurative organisms form an important factor in most cases of phthisis.

X.—*Of Nephritis, Albuminuria, and Dropsy.*

91. You will rarely be wrong if you consider all cases of nephritis not associated with some obvious morbid poison, such as scarlet fever, diphtheria, pneumonia, &c., to be chronic.

92. Quite apart from either diphtheria or scarlet fever, patients

suffering from attacks of sore throat are liable to nephritis, which is of temporary duration, lasting as long as the quinsy. The urine is usually bloody in such cases.

93. It is impossible to distinguish with certainty between the varieties of nephritis during life; much albumin and little urine may be found in a case of chronic interstitial nephritis.

94. Nothing cuts off the flow of urine so much as persistent vomiting. This result may be so marked, that unless the cause be remembered, you may suspect "suppression of urine" as the condition producing the diminution in the quantity of urine passed.

95. Having excluded calculus, hæmaturia in people who are past middle life is most commonly due to granular kidneys.

96. Hæmaturia repeated after long intervals may be the only evidence of the existence of granular kidneys.

97. "Granular kidney" may exist to an extreme degree without any albuminuria. Moreover, in some cases no signs of cardiac hypertrophy may be made out. These cases usually terminate either by the development of ordinary Bright's disease with dropsy, or by the occurrence of cerebral hæmorrhage.

98. Black hellebore and cantharides are the two most efficient drugs for arresting hæmaturia due to granular kidney, &c. The latter drug, however, is a dangerous remedy, sometimes producing foci of suppuration in the kidney, if injudiciously used.

99. It is quite a mistake to suppose that morning urine is best for testing for albumin; often it contains none when that passed later in the day contains a good deal. The same statement is true of saccharine urine.

100. With valvular disease of the heart you can never tell the significance of albuminuria; it may be transient and due to congestion of the kidneys. But if casts are found in the urine as well as the albumin, this is an observation of more value, for it indicates the existence of serious organic disease of the kidneys.

101. An epileptic attack, like an apoplectic fit, may cause a temporary albuminuria.

102. Travelling, and especially railway-travelling, is known to produce a temporary albuminuria in some people. From the consideration of one or two cases, it would seem not unlikely that sugar has appeared in the urine for the first time after a long journey, though, of course, it may have been present in slight amount previously.

103. Dropsy may be seen to be greater upon one side of the body than the other, and the phenomenon is probably purely

mechanical in its cause, depending upon the mode of lying in bed, &c.

104. Do not reduce the allowance of fluid food in dropsy ; it is bad practice, for anything is bad practice which reduces the amount of urine in this condition.

105. Purgatives in dropsy are scarcely of any use ; the practice is a survival. If we cannot influence the condition of the kidneys, we should do nothing to add to the patient's discomfort.

XI.—*Of Gastric Ulcer and Gastralgia.*

106. In ulceration of the stomach, when the patient is young, it is generally a succession of ulcers you have to deal with ; when the patient is old, it is generally one ulcer, which does not heal.

107. A gastric ulcer which has been diagnosed seldom perforates. When perforation takes place, this is generally the first symptom. Under treatment the risk of perforation is very small.

108. Gastric and duodenal ulcers are not to be distinguished clinically. Duodenal ulcers only occur in the "gastric" part of the duodenum, *i.e.*, above the opening of the bile and pancreatic ducts, where the contents of the gut are still acid. The vomit in these cases is sometimes excessively sour, so that the patient's teeth are set on edge by it.

109. In gastric ulcer the pain is commensurate with the acidity of the stomach contents. It is owing to this fact that relief follows the act of vomiting. It is owing to the same fact that the pain usually comes on an hour or so after food is taken.

110. The pain of chronic gastric ulcer is very often relieved by firm pressure upon the epigastrium.

111. The "Carlsbad treatment" in cases of chronic gastric ulcer is sometimes useful, though it has become of less importance since the introduction of the stomach-tube. It consists in the free use of alkalies and aperients. Carbonate of magnesium is a particularly useful drug, for it is a powerful antacid, and the soluble salts of magnesium are good aperients, ensuring thorough emptying of the stomach.

112. Washing out the stomach is of great service in cases of chronic gastric ulcer, where there is no hæmatemesis. No fermentable foods should be given in these cases ; but milk is an exception to this rule, for though it is fermentable, experience shows it to be devoid of ill effects.

113. Simple gastric ulcer is not a common cause of sub-diaphragmatic abscess and pleurisy. The ulcers of the stomach causing pleurisy are nearly always cancerous, and the fœtor of the pus in these cases is usually very marked.

114. Sub-diaphragmatic abscess occurring as a metastatic abscess (*i.e.*, the result of pyæmia) must be very rare. Hence pyæmia (with a sub-diaphragmatic abscess) is usually to be regarded as secondary to the abscess itself.

115. Pain in the stomach is often a neuralgia, and is very common in well-to-do patients. The pain may bear no relation to food, and be quite unrelieved by vomiting. In treating such cases, dieting is generally of no use: patients can digest toasted cheese. *Liquor arsenicalis* is by far the most valuable drug.

116. Neuralgia and debility ("atonic dyspepsia") which is part of a general weakness, are the only two common affections of the stomach in a young man who is sober.

XII.—Of *Diarrhœa* and *Dysentery*.

117. In infantile cholera the temperature of the surface of the body may be as low as 97° , when that of the rectum is as high as 102° .

118. The beneficial effects following the use of mercury in cases of infantile cholera are probably due to its antiseptic properties.

119. Dysentery, like diarrhœa, owns several causes: it is not a disease *sui generis*. But it should not be supposed that the border-line between dysentery and diarrhœa is ill-defined. It is in reality well marked, for tenesmus, hæmorrhage, and the passage of mucus and sloughs, are signs characteristic of dysentery. Moreover, a patient may suffer from dysentery without any diarrhœa,—the motions being solid, but the above signs present.

XIII.—Of *Abdominal Tumours*.

120. In physical examination by palpation, organs and tumours feel more superficial than they really are.

121. It is in persons suffering from chronic dyspepsia in its worst forms that a palpitating aorta is especially felt.

122. A renal tumour on the left side very often pushes the colon in front of it; but this condition is never found on the right side.

123. On careful palpation of the abdomen, the normal urachus can often be felt as a cord, stretching up from the bladder to

the liver, beneath the anterior abdominal wall. In cases of malignant disease of the abdomen, the urachus often becomes infiltrated by the growth, and this band may then be felt thickened, and measuring as much as half an inch across.

124. Carcinoma of the stomach is not seldom very latent, so that the whole organ may be infiltrated without any definite symptom having appeared during life.

125. In cases of malignant disease occurring in the abdomen, it is not uncommon to find post-mortem a chain of enlarged lymphatic glands, stretching up the thorax on one or both sides, and appearing behind the clavicles. One may even be led to a diagnosis of malignant disease of the abdominal viscera by finding these glands on careful examination. Thus, deep jaundice, obscure abdominal pain, but nothing abnormal felt in the abdomen itself, with hard glands behind the clavicle, would point to malignant disease of the head of the pancreas. Be this as it may, the discovery of these hard and enlarged glands is always most important.

XIV.—*Of Diseases of the Liver.*

126. An enlarged spleen may be the only evidence of cirrhosis of the liver.

127. Hæmatemesis in cirrhosis of the liver may be severe enough to cause very marked and prolonged anæmia.

128. Pain is a very common symptom in ascites, but is seldom or never described in accounts of that condition.

129. There is a condition known as "tympanitic ascites," where the abdomen contains fluid but is resonant to percussion. Intestines may sometimes give resonance through a layer of fluid an inch thick.

130. Malignant disease of the liver may occur and the patient gain weight during treatment. Indeed, a patient's gaining weight whilst under treatment does not exclude malignant disease of any organ affected.

XV.—*Of Jaundice.*

131. In jaundice the urine may contain bile before the skin or mucous membranes become tinged by it. Moreover, urinary jaundice may be of very brief duration, and jaundice of the tissues never appear at all. Iodine is a better test for the presence of bile in the urine than is nitric acid.

132. A jaundiced skin may never wholly recover its natural

colour in chronic cases, even when the cause of the jaundice is removed.

133. It is sometimes a good thing to let patients suffering from chronic jaundice due to a gall-stone take exercise. In this way the stone may be dislodged, an attack of colic ensue, and a spontaneous cure be effected.

134. In women who are past middle age, gall-stones are so common, that one is not wrong to be always suspecting them.

XVI.—*Of Cerebral Hæmorrhage and Hemiplegia.*

135. Contrary to current opinions of apoplexy, cases in which the patient is, as it were, struck down by a pole-axe—"stunned," as the word really means—are not common; and when they occur, they are usually due to hæmorrhage into the medulla or pons, though the hæmorrhage may be quite small in amount. The coma of fatal cerebral hæmorrhage is usually "ingravescent."

136. In cases of hemiplegia with coma, rigidity generally indicates hæmorrhage into the ventricles, convulsions generally indicate hæmorrhage into the pia mater.

137. Right hemiplegia, accompanied by aphasia, may be safely said always to be due to softening from arterial obstruction, and not to hæmorrhage. For the extent of a hæmorrhage which would produce this condition would be such as to prove also rapidly fatal.

138. Conversely, embolism from a diseased aortic valve is not likely to cause right hemiplegia if there is no associated aphasia. This may be safely premised even when the conditions for producing an embolus are quite manifest.

139. Moreover, in any case of hemiplegia, loss of consciousness, especially if complete, is in favour of hæmorrhage as its cause, rather than softening. In a typical case of embolism of the cerebral arteries, there is no loss of consciousness.

140. A hemiplegia, therefore, which comes on suddenly and is unaccompanied by any loss of consciousness, is probably due to softening from arterial obstruction. Hysterical hemiplegia also sometimes comes on suddenly, but is much less common. It is generally believed that the face is never paralysed in the latter case, though this is doubtful.

141. Hysterical hemiplegia does *not* come on during an emotion. Unless there be some positive evidence for a hemiplegia being hysterical, it should not be confidently diagnosed as such. Hemiplegia appearing during an emotion is much more likely to be due to a small hæmorrhage.

142. Cases of uræmic hemiplegia are well known, but are rare. Still, the condition should always be borne in mind when there are evidences of granular kidney in a hemiplegic patient.

143. When hemiplegia has come on suddenly, its continuance for more than three days excludes both epilepsy and uræmia as possible causes of it. One has then to discuss actual lesions: (i.) hæmorrhage, and (ii.) softening from arterial obstruction, as likely causes. In favour of (i.) is any evidence of granular kidneys and a hypertrophied left ventricle—conditions so often associated with cerebral hæmorrhage. In favour of (ii.) is the co-existence of aphasia with the hemiplegia.

144. A hæmorrhage into the internal capsule always produces rigidity sooner or later. It may appear as early as the fourth day or as late as the sixth week. Sometimes both legs become rigid—a fact explained by the incomplete decussation of the pyramidal fibres.

145. In cases of hemiplegia it is difficult to say if there is, or is not, any paralysis of the face when the patient is lying upon the side. The examination should therefore be made with the patient lying upon his back or sitting up.

146. Congenital hemiplegia or “birth-palsy” excepted, the hemiplegia of children differs in no respect from the hemiplegia of adults.

147. The pain suffered from after hemiplegia is often of the nature of an arthritis, the shoulder-joint being most commonly affected. At other times tender points appear along the course of the nerve-trunks.

148. The after-condition of old hemiplegics is often very sad. The patients become little different, to all outward appearances, from idiots, suffering as they often do from aphasia and verbal deafness at the same time.

149. The only form of spinal hemiplegia you are likely to meet with is that due to anterior poliomyelitis.

XVII.—*Of Meningitis.*

150. It is rare to get a case of meningitis without the history of a blow upon the head some time before; but the blow seldom or never has anything to do with the disease.

151. No disease is more frequently followed by tubercular meningitis than is hooping-cough.

152. A direct diagnosis of tubercular meningitis is rarely possible, for the only direct evidence is the discovery of tuber-

cles in the choroid—too often only possible when the patient is moribund. But generally a diagnosis may be based upon the fact that the disease is—as shown by the symptoms—cerebral, is in a child, and has a sudden onset.

153. Aphasia is occasionally the first symptom in tubercular meningitis, and may be the only one present for some days.

154. By far the commonest manifestation of general acute tuberculosis in children is tubercular meningitis.

XVIII.—*Of Epilepsy.*

155. Epilepsy does not admit of definition, because we do not know what the essence of epilepsy is, nor has it any criterion; certainly loss of consciousness is no criterion.

156. Fracture of the base of the skull may be produced by the violence of a fall during an epileptic attack, and the special signs of fracture are sometimes quite absent. The coma of the epileptic seizure becomes prolonged into the coma due to hæmorrhage caused by the fracture.

157. Epileptic attacks are common in the course of any form of chronic disease of the brain or its membranes—sclerosis, general paralysis, chronic meningitis, old hemiplegia, &c.

XIX.—*Of Hysteria.*

158. Hysteria is a definite malady. It is a mistake to employ the term to cover all the cases of obscure nervous derangement in women which ignorance prevents our otherwise classifying.

159. Hysteria is generally diagnosed by the method of exclusion, and mistakes are sometimes made in consequence. Yet very often no other method of diagnosis is possible. The most characteristic symptoms are defects in sensation.

160. Optic atrophy may occur in hysteria, but is very rare. When present, it is unilateral, and there is accompanying hemi-anæsthesia.

161. Pain, muscular atrophy, loss of faradic excitability by the muscles, and loss of knee-jerk—either of these tells against hysteria being the cause of a paralysis in which it occurs.

162. Anæsthesia, and especially hemi-anæsthesia, is so commonly functional, that when it occurs as part of the nervous symptoms of a case, hysteria should be discussed as a possible cause of the condition.

XX.—*Of Myelitis.*

163. In medicine you can seldom say that something is *always* the case; but one of the instances most nearly justifying it is the production of bladder symptoms in myelitis.

164. You will not be far wrong if you say that only one disease of the spinal cord *begins* by affecting the bladder, and that is myelitis.

165. The two most useful drugs in the treatment of myelitis are belladonna and strychnine; and there seems a good deal of reason for adhering to the rule in vogue since Brown-Séguard's time—to give the former in acute, and the latter in chronic cases.

XXI.—*Of Neuritis.*

166. Sciatica is most probably always a neuritis, and often traumatic, *e.g.*, due to much driving on a hard seat. In this connection note the frequent occurrence of some degree of wasting of the muscles, loss of sensation, &c., after the attacks.

167. Old writers confused hip-disease and sciatica; Cotunnus of Naples, the discoverer of neuralgia in general, first pointed out the distinction.

168. Loss of knee-jerks may be the sole sign of post-diphtheritic neuritis. In one case it occurred where the jerk was known to have been previously very well marked; there had been abundant discharge of membrane from the trachea, the fauces and larynx being unaffected.

169. Pain must, as a rule, be regarded as a contra-indication in the use of massage, inasmuch as it usually betokens downright irritation of the nerves.

170. In cases of muscular wasting, where the muscles are nicely antagonised, as they are at the ankle-joint, galvanism must be used with great caution, if at all. For by it you may do more harm than good, improving the nutrition of the antagonistic (unaffected) muscles out of proportion to any improvement resulting to those that are wasted. In this way a deformity may be produced, or one which existed before may be increased. So far as I know, the employment of massage in such a case does not entail a similar risk.

XXII.—*Of Some other Nervous Diseases.*

171. Double internal strabismus is often the earliest sign of disease of the pons, because the sixth cranial nerves become so easily affected. Disease of the pons is not uncommon in

children, and is either tuberculous in nature or consists of a diffuse glioma.

172. It is erroneous to regard pin-point pupils and a rise of temperature as diagnostic of a lesion of the pons; any cerebral lesion may cause the latter, and the former is present in the case of hæmorrhage into the ventricles.

173. "Cervical opisthotonus of children" is not a tuberculous affection; it is due to a chronic meningitis of the medulla. It may, however, occur as a symptom in tuberculous affections of this region.

174. There seems no reason for thinking that any other difference than one of degree exists between "paralysis agitans" and so-called "senile tremors."

175. It is extremely improbable that hæmorrhage into the spinal cord ever takes place apart from softening.

XXIII.—*Of Delirium.*

176. Apart from insanity, which may be regarded as a chronic or oft-repeated delirium, this latter condition is mostly due to alterations in the blood, *i.e.*, to a blood-poisoning. This is quite an old notion; thus Shakespeare, speaking of a delirious man, has—"All his blood is touched corruptedly."

177. No conditions are more difficult of diagnosis than those two in which the intelligence is impaired—coma and delirium.

178. If called to a case of delirium with fever, it is useful to consider three things of which it may be a symptom:—

- (i.) Brain disease—such as purulent meningitis, &c.; these cases the ancients would term "idiopathic phrenitis."
- (ii.) Blood-poisoning—including poisoning from drugs (alcohol, belladonna, &c.); from specific diseases (enteric fever, pneumonia, hydrophobia, &c.); and from local diseases (pericarditis, &c.).
- (iii.) Insanity—remembering that insanity is really a chronic delirium; that you may be dealing with the first attack; and that fever is not an uncommon accompaniment of an acute attack of insanity.

XXIV.—*Of Headache.*

179. Some people seem totally incapable of headache, whatever disease they may suffer from (as enteric fever), which is usually accompanied by this symptom in others.

180. Indian hemp is sometimes a drug of great value in the treatment of chronic dull headache. Especially is it of value

in cases of headache in overworked men and in Bright's disease. Its use should be cautiously commenced, especially in girls and women. Persons accustomed to take much alcohol, however, are usually very tolerant of the drug.

181. Iodide of potassium is perhaps of still greater value in cases of headache of a different character,—those acute and violent headaches, often accompanied by a raised temperature, the cause of which is very obscure. The cases are not very uncommon,—headache and fever are the two cardinal symptoms. Full doses of the drug should be given, and are often followed by complete cure, though at first the pain may be aggravated by the iodide. In such cases as these, though a tempting hypothesis, syphilis can often be wholly excluded.

XXV.—*Of Aphasia.*

182. Aphasia is not a defect in the unuttered word, nor in the uttered word; it is a form of paralysis,—of the special movements of speech.

183. In many cases of aphasia attempts to protrude the tongue only succeed in getting it as far as the teeth; this illustrates the definition of this condition given above.

184. The cases of aphasia suddenly appearing during an emotion are probably not hysterical, as Trousseau thought them. They occur often in adult men, and are better described under the term "emotional aphasia." The condition may last for several days.

185. There is no aphasia in disease of the pons.

XXVI.—*Of Some Infectious Diseases.*

186. Hoop is not an absolute criterion of hooping-cough. A child may hoop for weeks with a cough which is not hooping-cough, and yet cannot be distinguished from it, except by the fact that the said child does not convey the disease to other children who are susceptible.

187. Patients suffering from measles are quite as infectious previous to as after the development of the rash. Hence the great difficulty in preventing spread of the disease. Although the fourth day is the commonest for the appearance of the rash, this may be the first sign of the disease, or, again, may be delayed until the seventh or eighth day, the patient meanwhile feeling very ill. Indeed, there is no disease in which the duration of the invasion period is so variable.

188. Mumps may produce many curious nervous symptoms

at times; intense giddiness is one of the most marked of them.

189. There is nothing in the symptoms or signs of influenza which enables you to say, "That is influenza." It is often best, if the patient has had a previous attack, to consult his own feelings in the matter.

XXVII.—*Of Enteric Fever.*

190. It is a safe maxim never to diagnose enteric fever without the presence of rose-spots. Many cases occur without rose-spots, but these can only be safely diagnosed where there is an epidemic.

191. "A rose-coloured raised spot, disappearing on pressure," is not a sufficient definition of the rash of enteric fever. It is necessary to add that "each spot is of short duration, and successive crops of the spots appear." Thus defined, the eruption is quite characteristic of the disease.

192. Enteric fever very rarely begins with a rigor; pneumonia very commonly. The reason probably lies in the fact that in the former disease the temperature rises slowly, in the latter it rises rapidly.

193. Scarlatiniform symptoms,—a rash, sore throat, &c.,—coming on early in enteric fever, are noteworthy, and not uncommon. In such cases diagnosis at first is often impossible.

194. Vomiting coming on early in enteric fever, especially if it be frequent, indicates a severe case; it is then often accompanied by bad headache.

195. Muscular rigidity in enteric fever always marks a severe case, and often, but not always, a fatal one.

196. Deafness is common in enteric fever. Trousseau thought it indicated a good prognosis, and I agree with him; but a statement so grossly empirical ought to be very well established before much importance is attached to it.

197. Hæmorrhage from the bowels in enteric fever, if it occurs not later than the second week, may be disregarded. After that time it is a serious symptom. Trousseau's statement that hæmorrhage in enteric fever is not of much importance, is probably explained by assuming, with Collingridge, that he was referring to the former class of hæmorrhages.

198. Nothing can be done to relieve the tympanites of enteric fever. Passing a rectal tube, puncturing the intestines with a trocar, administering small doses of turpentine or charcoal,—all these have objections or produce no benefit.

XXVIII.—*Of Some Tuberculous Diseases.*

199. A period of improvement in general condition, even an increase in weight, does not exclude the possibility of a child's disease being tuberculous in nature.

200. The existence of tubercles beneath the skin—a “scrofulide,” but not lupus—is a very serious condition, for the patients invariably die. The tubercles are apt to soften, disappear, and signs of general tuberculosis follow.

201. Tuberculous peritonitis may come on quite suddenly; but when it does so, the acute symptoms last for a short time only, and they then lapse into the usual chronic condition. During the acute stage, however, the diagnosis from some form of intestinal obstruction may be very difficult.

202. In chronic tuberculous peritonitis indurations within the abdomen always become manifest sooner or later.

203. Tubercle of the brain substance only occurs as circumscribed tumours. There is, however, a rare condition of miliary tubercular encephalitis, where the tubercles follow the ramifications of the blood-vessels, but this is only found in conjunction with tubercular meningitis.

XXIX.—*Of Chorea and Rheumatism.*

204. Chorea resembles the specific fevers in many points. It runs a definite course; and what is good for it, like what is good for rheumatic fever, is “six weeks.”

205. Choreic patients are nearly always weak-minded, so long, at least, as the chorea lasts. Insanity is not an uncommon accompaniment of the chorea of young girls.

206. I have never seen a fatal case of chorea in which endocarditis was not found on post-mortem examination.

207. Salicylate of soda, guaiacum, and aconite—the three drugs having a reputation for curing sore throat—have all of them more or less specific action in cases of rheumatism. Hence, if with a sore throat there is no other indication, you are justified in treating it as rheumatic.

208. The use of sodium salicylate was formerly often attended by attacks of vomiting, salivation, and delirium, which are now rarely seen. These effects were probably due to impurities in the drug, such as carbolic acid.

209. Salicin is a feeble remedy to give in cases of acute rheumatism; you may give a patient twenty-grain doses every two or three hours without any effect, and then obtain almost

immediate good from the use of sodium salicylate. As a fillip, used like quinine, and in mild cases, salicin is useful.

210. Rheumatoid arthritis, having an acute onset, cannot at first be diagnosed from rheumatic fever.

XXX.—*Of Syphilis.*

211. I have never been able to satisfy myself that any such thing as syphilitic disease of the lung exists. So-called "syphilitic disease of the mediastinum" undoubtedly occurs, but is very uncommon; it is certainly much rarer than aneurysm in this situation, which it may closely resemble in its symptoms.

212. There is too great a tendency to call all children that are weakly, wasted, and have sores on the body, syphilitic. These sores are often due to neglect, dirt, want of food.

213. Syphilis is almost invariably the cause of any sudden blepharoptosis; it is also the great cause of paralysis of the iris—indeed of all forms of ophthalmoplegia.

214. Hæmorrhage into the substance of the brain may occur as a sequel to a gumma of this organ, especially when the patient is a child.

XXXI.—*Of Some Skin Diseases.*

215. Rheumatoid symptoms are very apt to occur in conjunction with three skin diseases, in this order of frequency: Erythema (especially *E. papulatum*), purpura, and urticaria; and these conditions of the skin are apt to interchange.

216. A person suffering from active psoriasis is likely to get it upon any irritated piece of skin. It is quite possible, therefore, that a purpuric or other spot may become the seat of psoriasis.

217. Ordinary facial psoriasis may simulate a syphilide if washed much with soap and water, the spots being no longer scaly, but becoming shiny, and of the colour that is common in syphilides.

218. Blood is a powerful anti-scorbutic; therefore raw meat often protects from scurvy.

XXXII.—*Of Diabetes and Glycosuria.*

219. Diabetes is a disease very often overlooked, partly because the urine escapes being tested for sugar on account of its having a fairly low specific gravity—which really is com-

patible with the presence of a considerable amount of sugar—partly because the patient is a child, and sometimes because there is no thirst or emaciation.

220. Diabetes sometimes becomes evident in two or three members of a family at the same time, so as to give an appearance of contagiousness. This idea is by no means a new one, but there is no actual proof of the disease being contagious.

221. Temporary glycosuria, if not due to the ingestion of large quantities of sugar, probably indicates a tendency to diabetes.

222. Drugs excluded, there is nothing in the urine which reduces Fehling's solution except sugar, not even uric acid or kreatinin.

223. There is probably no such thing as acute diabetes; in cases so termed, sugar in the urine probably existed undiscovered for some time. Symptoms, even thirst, may have been quite latent.

224. The enormous quantities of food which diabetics sometimes consume, to satisfy the excessive appetite which occasionally occurs as a symptom of the disease, never seem to cause dyspepsia. The food is easily digested; indeed, there seems to be quite an exceptional development of the digestive function in these cases.

225. Diarrhœa is a not uncommon symptom in diabetes; it may even be of a dysenteric nature.

226. Diabetes resembles tabes dorsalis in three points—loss of knee-jerk, perforating ulcer of the foot, and darting pains in the limbs.

227. It is dangerous to put diabetics upon a very strict diet suddenly; coma has come on when this has been done.

228. The beneficial effects of opium in the treatment of diabetes are probably due to the power the drug has of allaying the feeling of weariness and discomfort suffered from, and of acting as a "euphoric."

XXXIII.—*Of Some Signs and Symptoms.*

229. Irregular pulse in children after a serious illness—or indeed after an illness which is not serious—is so common as to be of little importance.

230. Difference in size of the pupils is of itself of no value in the diagnosis of disease; it is quite compatible with perfect health.

231. *Tâche cérébrale* is a sign of little or no value.

232. The most infallible early sign of death is loss of faradic excitability by the muscles.

233. During almost any very acute illness the mind may become so clouded that afterwards the patient remembers scarcely anything that has happened in its course.

234. In all nervous diseases sensory symptoms are apt to be very fugitive : cf. Myelitis.

235. In children, finding the apex-beat of the heart in the fourth costal interspace is no sign of disease ; it is not at all uncommon.

236. Convulsions are often the sole symptom of brain-disease of various kinds in children.

237. So little is known about the causation of anæsthesia, that, unless it be a hemi-anæsthesia, perhaps the less said about it for purposes of diagnosis the better.

238. It is sometimes difficult to make out a true hemi-anæsthesia, because in order to do so the sight, hearing, and taste must be tested.

239. All sorts of anæsthesia are met with in disease of the pons, difficult to explain because we know so little of the course of sensory fibres in this situation.

240. Examination of sputum for pus microscopically is of no use, for the naked eye appreciates the only important fact—the *amount* of pus, whether great or small, and not its mere presence, which may always be presumed.

241. Spirit-drinking, much more often than wine or beer-drinking, may produce no facial signs, even after indulgence for many years. One sign, however, which it may produce, is pallor—quite the opposite of the condition produced by the other forms of alcoholic drinks.

242. The “blue line” in cases of plumbism may become apparent quite early in the disease. When acetate of lead was wont to be given freely for cases of hæmoptysis, a “blue line” sometimes developed in three weeks from the commencement of taking the drug.

243. There is, perhaps, no disease in which crippling may not result from the patient lying curled up in bed. In enteric fever it is not uncommon, and bad contractures are occasionally seen in cases of alcoholic neuritis. These deformities should be studiously avoided, or, if this has not been possible, they should be corrected early. There is also a tendency for dislocation of the tibia backwards upon the femur to take place.

244. A muscle may be quite paralysed to faradism, and yet not be paralysed to volition. The opposite condition is, of

course, much commoner, occurring as it does in all cases of upper-segment paralysis.

245. Nummular sputa, though most often found in phthisis, are not diagnostic of that disease.

XXXIV.—*Of Fever.*

246. Almost anything may cause a rise of temperature in a patient. Mental emotion alone may produce a temperature of 104° – 105° for a time.

247. Convulsions in children are often sufficient to cause high temperature,—a rise of 9° – 10° even,—and this hyperpyrexia may prove fatal unless counteracted by the application of cold water. Such a condition may be seen at the onset of almost any specific infective disease, such as varicella.

248. Hæmorrhage may be attended by a rise of temperature quite apart from the disease causing the loss of blood. This is well seen in some cases of hæmoptysis. The rise of temperature in cerebral hæmorrhage is not analogous, however. With Cullen, fever formed part of his definition of hæmorrhage, “*Pyrexia cum profusione sanguinis absque vi externâ.*”

249. Frequently a convalescent patient, whose temperature will not settle down, gets a steady normal temperature on being allowed to get up. With patients convalescent from rheumatic fever this is especially noticed.

XXXV.—*Of Food in Disease.*

250. Pre-digestion of food is of very little value in any disease that I know of.

251. You must not think that fever in itself contra-indicates the administration of solid food; that is not a rule of practice at all.

252. An apparent good condition of a patient fed upon nutrient enemata is often very deceptive, for symptoms of inanition may, and often do, appear very suddenly and without warning. A very little food retained *per os* is much better than a large amount *per anum*, and on this account the former method of feeding should never be wholly abandoned without the direst necessity.

253. A milk diet is certainly not to be recommended in all cases of nephritis. Some patients fare much better upon a meat regimen, and few fare worse.

254. Typhlitis, unlike enteric fever, is a disease of short duration, and therefore nothing is gained by “feeding up” the

patient. Especially avoid those foods after the digestion of which a large residue is left, such as milk. For this reason whey is very good in this disease.

XXXVI.—*Of Childhood and Old Age.*

255. There is no better guide to the degree of proper development of a child than how it cuts its teeth. It should be remembered in this connection that girls are usually more forward in cutting their teeth than boys.

256. Boys are sometimes very backward in learning to talk; but if a boy cannot talk at four years of age, he is—with a single rare exception—either deaf and dumb or an idiot. The former alternative can easily be excluded. The exception mentioned is the condition of congenital aphasia.

257. There is often a remarkable latency about the diseases of old people. Phthisis particularly is often overlooked because of its supposed improbability.

258. As a rule, people do not die “of old age.” There is usually failure of a particular organ, and not of all together. Still, it is sometimes very difficult to say exactly from what an old person does die.

NOTES TO APHORISMS.

27. Medical Register for 1896, vol. ii., part ii., index, No. 131. Charles Jagot, warded in “Luke” for hæmoptysis, from attacks of which he had suffered for twenty years.

48. Medical Register for 1895, vol. ii., part ii., index, No. 258. William Henderson, æt. 22, warded in “Luke,” suffering from pyopneumothorax on the left side. On June 4, five and a half pints of pus were drawn off. On July 12, patient was sent to Swanley Convalescent Home, but was readmitted to “Luke” five days later. On July 18, five pints of clear serum were aspirated from same side of chest. On September 7, seven pints of fluid were drawn off, once more purulent in character. Ultimately several ribs were resected.

76. Compare Dr. Church’s valuable paper on the frequency of cardiac affections in acute rheumatism, with an analysis of nearly 700 cases, published in vol. xxiii. of these Reports. The same conclusions are arrived at with regard to the past and present frequency of pericarditis and endocarditis. Church’s results are quoted by Fagge. On the other hand, Osler, in his somewhat meagre account of rheumatic fever, its complications and treatment, denies that the use of salicylates has diminished the frequency of cardiac affections in any way.

81. This conclusion, arrived at from clinical observation, would seem to tally with the growing belief of a certain school of pathologists, investigating blood diseases along quite different lines. Thus, Dr. Ralph Stockman has “endeavoured to show that so-called ‘pernicious anæmia’ is simply

a congeries of clinical symptoms, and not a disease *sui generis*." *Vide* papers in British Medical Journal for 1895, vol. i. p. 965, &c.; and more recently, 1896, vol. i. p. 1077, "Remarks on the Analysis of Iron in the Liver and Spleen in various Diseases affecting the Blood."

82. The word would seem to be unfortunate in another sense, since, as Dr. Gee points out, "leuchæmia," the form in which it is very frequently spelt (see Fagge's text-book, &c.), is incorrect. The Greeks did not write "leuchippus," but "leucippus."

86. ". . . cui addidit paucos postea annos, . . . et ore ac membris, in eum pallorem albenibus ut ostentui esset multum vitalis spiritus egestum." —*Annalium*, liber xv. cap. 64.

122. Though most authors draw attention to this point in the diagnosis of renal tumours, none of them, so far as I can discover, makes the distinction between right and left sides. Assuming the truth of the statement, the anatomical relations of the colon and its peritoneal covering to the kidneys might possibly explain the difference on the two sides. For the splenic flexure and the descending colon are bound more closely to the kidney and posterior abdominal wall than are the ascending colon and the hepatic flexure. The last-named portion of bowel lies lower down, is in a more superficial plane, and is suspended by a longer mesocolon than is the splenic flexure. Thus the colon on the right side is better able to slip away from the front of an enlarging kidney than is the colon on the left side. Moreover, if an ascending or descending mesocolon is present, as it is, according to Treves, in 50 per cent. of all cases, its relations again fit in with the above statement. For the line of attachment of the left mesocolon is along the outer border of the kidney, whereas that of the right mesocolon runs along the inner border of this organ. The left kidney, when enlarged, would therefore push the colon in front of it; the right kidney, similarly affected, would come to the front external to the colon.

163. *Cf.* Celsus: "Medicinalis ars recipit vix ulla perpetua præcepta."

174. As regards the pathology of the disease, Dubie's conclusion accords with this opinion: "Parkinson's disease has for an anatomical basis the lesions of cerebro-spinal senility, and which only differ from those of true senility in their early onset and greater intensity."

175. Hayem, from the consideration of thirty cases of hæmato-myelia, which he collected and published in his thesis "*Des Hémorrhagies intrarachidiennes*," Paris, 1872, concluded that none of them could be classed as primary, non-traumatic hæmorrhages, and that such a condition did not occur. This opinion, however, is by no means universally held. Berkley, of Baltimore, collected 11 cases from medical literature between the years 1872 and 1889, and prefaced these to an article on "*Syringomyelia*," published in *Brain*, winter part, 1889. Full references are given, and though some of the cases are excluded by lack of proper microscopic post-mortem examination, others of them appear to make Hayem's opinion unwarranted.

203. The only recorded case of this kind is that by Dr. Gee, in his article on "*Tubercular Meningitis*" in Reynolds' System of Medicine, vol. ii. 2nd edit. p. 408. It is the third of the four cases mentioned under "*Accidental Lesions*."

219. In this connection it is interesting to note that Eustace Smith has no account of diabetes in his excellent manual "*On Diseases in Children*." C. West, in his "*Lectures on the Diseases of Infancy and Childhood*," states that he had only met with two instances of diabetes occurring in children, and quotes Prout's analysis of 700 cases of the disease, of which number only one was a patient under eight years old, and only twelve

were between eight and twenty years old. Later observers have obtained very different figures; thus, Stern has collected 117 cases of diabetes in children. The rapidly fatal course of the disease, which he found common in this class of patients, is probably the cause why it was formerly so often overlooked.

222. This conclusion Dr. Gee arrived at after experiments with pure chemicals. I am unable to confirm his results. I find that a drop of a solution of uric acid in caustic soda, when added to boiling Fehling's solution, soon causes a deposit of red cuprous oxide. If, however, the copper be not in excess, the precipitate obtained is more voluminous, and greyish-white in colour—a urate of cuprous oxide. The difference between the two reactions, according to the relative amount of each substance present, is very striking. In the former case, one is left with a blue solution, containing a bright red sediment; in the latter, one has a bulky grey precipitate, with a quite colourless supernatant liquid. Kreatinin appears to reduce Fehling's solution only after boiling for some considerable time, and there is, even then, no separation of cuprous oxide. If, as is usually the case clinically, "reduces" means "causes a deposition of red cuprous oxide," the aphorism is thus confirmed with regard to kreatinin, and with regard to uric acid when the copper salt is not kept in excess.

223. It may be remembered that Trousseau commences his lecture on diabetes by describing such a case—of a man in whom the symptoms were distinctly traceable to a particular day on which he was engaged in mowing grass beneath a hot sun. But, of course, here again there is no proof that sugar was not present in the urine previous to this; and saccharine urine is at present our sole criterion of the disease.

CASES OF ENTERITIS AND COLIC SIMULATING INTESTINAL OBSTRUCTION.

BY

A. M. MITCHELL, M.B.

WITH REMARKS BY MR. SMITH.

The difficulty of diagnosis between true intestinal obstruction and certain forms of colic, or enteritis with constipation, either absolute or nearly so, is briefly referred to in several books, but little or no description is given of such cases, nor is anything said as to the points on which the differential diagnosis depends; and it therefore seemed interesting to record four such cases which were admitted into the Hospital in July 1895, under the care of Messrs. Smith, Willett, Marsh, and Butlin, who have kindly given their leave for the cases to be reported.

The patients were all admitted direct to surgical wards as cases of intestinal obstruction, except the one under the care of Mr. Marsh, which had been admitted two days previously with an extra-capsular fracture of the neck of the femur.

In the first case, that of Mr. Smith's, recovery took place without operative interference, but in the other three cases exploratory operation was deemed necessary, owing to the urgency of the symptoms.

In Mr. Willett's case, about a foot of hyperæmic intestine was found, but no strangulation nor visible cause of obstruction, and a good recovery was made.

In Mr. Marsh's patient the intestines were much distended and congested, but at the post-mortem no mechanical obstruction nor peritonitis was found.

In the last case, that of Mr. Butlin's, three small and easily reduced intussusceptions were found, but they were not strangulated, nor did they appear to be the cause of obstruction, and recovery ultimately took place.

CASE No. I.—MR. SMITH'S.

William Evans, æt. 54, ironmoulder, admitted into Henry Ward under care of Mr. Smith, on Friday, July 19th, suffering from symptoms of intestinal obstruction.

History of present condition.—On Sunday, July 14th, had dinner as usual in the afternoon, consisting of mutton, green peas, and potatoes. At supper he ate shrimps, cherries, and watercress.

At 5 A.M., Monday, July 15th, after passing a motion, he was suddenly seized with pain across the abdomen. Poultices were applied, but pain continued to 4 P.M. The pain has continued in paroxysms till the present time. He has vomited all his food since Monday, latterly his vomit is said to have been fæcal.

He has passed flatus from time to time, but the bowels have not acted.

The urine has been scanty, but micturition has given him relief.

Yesterday patient took a dose of castor-oil of his own accord.

Condition on admission, July 19th.—Strong-looking man with anxious expression. Abdomen slightly distended, moving on respiration, very tender; not sick since admission, but has felt so; passed some flatus in the morning. Nothing abnormal felt on palpating the abdomen, nor per rectum. Pulse 72, regular, and of good volume. Temperature not raised. Tongue furred and dry. He was seen this afternoon by Dr. Church with Mr. Smith, when it was decided to watch the patient, and to defer operative measures.

Given spoonfuls of warm water by the mouth and nutrient enemata, and ext. belladonnæ gr. $\frac{1}{4}$ 4^{ts} horis, but no morphia.

July 20.—Condition much the same; no vomiting. Pulse 65. Warm water enema oij given this afternoon, after which he passed some flatus. A tablespoonful of milk and water, equal parts, given every hour, and nutrient enemata continued.

July 21.—Better in morning. In the evening abdomen became much more distended, pain increased, and pulse not so good. Rectal tube inserted, and much flatus passed, giving considerable relief.

July 24.—Rectal tube passed whenever much distended. Condition has slowly improved; bowels not opened yet. No abdominal tenderness; much less distended. No vomiting. Now taking a pint of milk in the twenty-four hours.

July 15.—Bowels opened for the first time since admission (after ten days' constipation), after an olive-oil enema; patient comfortable.

July 27.—Bowels opened three times; still improving.

Recovery from this time uninterrupted, and patient left the Hospital for Swanley when convalescent.

CASE No. II.—MR. WILLETT'S PATIENT.

Abstract of Notes.

Anne Brown, æt. 58, washer and mangler, admitted to Stanley Ward, July 20, 1895, and transferred to President Ward as an emergency operation, under the care of Mr. Willett, July 21st.

History of present condition.—At 5 A.M., Friday, July 19th, patient awoke with violent abdominal pain and vomiting, continuing till 3 A.M. July 20th. Abdomen became distended. Bowels acted regularly till Thursday, July 18th, and again slightly after a dose of castor-oil on night of July 19th. Has vomited repeatedly after taking soda and milk. Never a similar attack before. No history of injury.

Condition on admission, July 20.—Abdomen slightly distended, very tender, but soft; patient lies with her knees drawn up. Temperature 100.4°. Urine passed was red.

July 21.—After consultation, patient was taken to theatre and laparotomy performed; median incision; abdomen explored. After exhaustive examination, the only abnormality found was about a foot of hyperæmic intestine. The intestines were not much distended. Wound closed.

July 22.—Much improved; only sick once; passes flatus.

Convalescence uninterrupted; bowels opened on July 26, after castor-oil, after seven days' constipation.

August 28.—Patient discharged to Swanley wearing abdominal belt. Mr. Willett is of opinion that the symptoms were due to a severe enteritis.

CASE No. III.—MR. MARSH'S PATIENT.

Abstract of Notes.

Joseph Crossland, æt. 53, stableman, admitted to Abernethy Ward, July 28, 1895, suffering from an extra-capsular fracture of the neck of the femur.

History.—Fell on pavement.

Admitted, and fracture was put up on a long Liston splint with extension.

July 31.—Abdomen became greatly distended, breathing rapid, pulse small and frequent, occasional vomiting. Distress seems great.

August 1.—Bowels opened three times yesterday; twice after enemata to-day.

2 P.M.—Laparotomy performed; median incision, small intestine much distended and congested. Glass tube inserted into small intestine, and some of the contents pressed out. The matter was fluid, thick, and somewhat pus-like. Death 3.45 P.M.

Post-mortem, August 2.—Whole intestine, from œsophagus to rectum, much distended with gas and fluid. No solid fœcal matter. Intestines opened eight feet from ileo-cœcal valve. No leakage into peritoneal cavity. No peritonitis. No ulceration, growth, or other lesion of intestine found. Kidneys slightly granular. Hypostatic congestion of lungs. Liver, spleen, and other abdominal viscera healthy.

CASE No. IV.—MR. BUTLIN'S PATIENT.

Abstract of Notes.

Daniel Harrington, æt. 12, school-boy, admitted into Colston Ward, under care of Mr. Butlin, July 15, 1895, suffering from symptoms of intestinal obstruction.

History of condition on admission.—On July 11th seized with griping pain in the stomach during the night; vomiting began. Brought to Hospital and given Ol. Ricini.

July 13.—Symptoms continuing again, attended Surgery, and Ol. Ricini repeated.

July 15.—Admitted to Colston Ward. Bowels not opened since July 11th (three days since); abdominal pain and vomiting have continued since onset.

Condition on admission.—Well-developed boy; abdomen flat, not distended, moves on respiration, tender; intermittent griping pains. Soft fluctuating tumour sometimes felt at outer edge of rectus muscle. Face flushed, eyes bright, headache. Temperature sub-normal, pulse 72, respirations 24; not collapsed; vomiting continued.

Mr. Butlin, after consultation with Sir Dyce Duckworth, decided to do an exploratory operation.

Operation.—Laparotomy, median incision; three distinct intussusceptions found, one situated by the ileo-colic valve, where the intestinal walls were somewhat thickened and congested, and two others farther along the small intestine. All three were easily reduced, and were not strangulated. The

veriform appendix was somewhat thicker than usual. No other abnormality found.

July 17.—Symptoms not being relieved, Dr. Moore saw the patient with Mr. Butlin. Patient was restless, the bowels had not been opened since admission. Temperature 101° ; pulse 100. He had been fed by nutrient enemata with opium, but being free from abdominal pain and vomiting, no further operation was considered necessary.

July 18.—Passed flatus for first time since July 11th.

July 20.—Much improved; pulse 64, fuller; bowels not open yet.

July 22.—Bowels opened for first time after eleven days' constipation.

From this time patient made a satisfactory recovery.

It will be noticed that three of the four cases are between fifty and sixty years of age.

The attack began in all whilst in bed.

The pain is commonly paroxysmal, severe, and griping, and is only described as constant in one of the above cases.

Vomiting, although occasionally occurring independently, commonly follows the introduction of food into the stomach, especially after the attack has lasted some few hours.

The abdomen in all four cases is described as tender, which would be explained by the inflamed state of the intestines, but there was respiratory movement in each patient.

No movements of coils of intestine were visible.

In Case No. IV. the symptoms were most severe; constipation was absolute, and remained so for some days after the operation.

In neither Cases No. I. nor II. was there any action of the bowels, but in the former flatus was passed from time to time throughout.

With the exception of case No. III. there was an absence of the typical hard and frequent pulse common in cases of intestinal obstruction, nor were these patients collapsed.

The intussusceptions found in No. IV. were multiple, not strangulated, and were easily reduced, and were thought to be due probably to the irregular peristalsis and partial paresis of parts of the intestine rather than the original cause of obstruction, and in their nature allied to "post-mortem intussusceptions."

The symptoms in these cases appear, therefore, to be due to a colic or enteritis rather than to any mechanical cause of obstruction, the intestines being more or less completely paralysed.

In severe cases the whole thickness of the intestinal wall is

Table of Chief Points in the Four Cases for Comparison.

	MR. SMITH'S CASE. No. I.	MR. WILLET'S CASE. No. II.	MR. MARSH'S CASE. No. III.	MR. BUTLIN'S CASE. No. IV.
Age	54.	58.	53.	12.
Previous diet	Green peas, &c., for dinner; shrimps, cherries, and water-cress for supper.	Not stated.	Not stated.	Not stated.
History of onset	5 A.M. next morning, after going to stool.	5 A.M. patient awoke with violent abdominal pain; vomiting.	Third day after admission with extra-capsular fracture of femur, whilst lying on back wearing a long Liston splint.	Seized during night with sudden griping pain in abdomen and vomiting.
General condition	Regular and good pulse, 72, but varied. No shock. Temperature normal. Anxious expression. Tongue furred and dry.	Temperature 100.4°. No shock.	Pulse small and frequent. Much distress.	Face flushed. Pulse 72. Temperature subnormal on admission; rose to 101° after operation. Headache. Not collapsed.
Condition of abdomen	Very tender, slightly distended. Distension increased till rectal tube passed. Moved on respiration.	Very tender, soft, slightly distended.	Great distension.	Flat, not distended, tender, moved on respiration; soft tumour felt at outer edge of rectus muscle.

Character of pain	Paroxysmal gripping pain.	Constant violent pain, relieved by flexing thighs.	"Great distress."	Gripping pain.
Character of vomiting	Vomited everything taken by mouth. Laterally vomit said to have been fecal. Vomiting ceased on admission, nothing being given by mouth for first twenty-four hours.	Repeated vomiting since onset, especially after food (soda and milk).	Occasional vomiting.	Vomiting continued from onset till operation.
Degree of constipation	Bowels not open till eleven days after onset. Flatus passed from time to time throughout.	Bowels open slightly after onset after <i>Ol. Ricini</i> , and then not for seven days. Flatus passed after operation.	Bowels open three times the day before and twice on day of operation after enemata.	Bowels not open till eleven days after onset. Flatus passed third day after operation.
Treatment	Nutrient enemata, belladonna. Passage of rectal tube.	Exploratory laparotomy.	Exploratory laparotomy and enterotomy.	Exploratory laparotomy. Nutrient enemata, morphia, &c.
Condition of intestine	No operation.	One foot of small intestine rather hyperemic; not much distended.	Congested and very distended, containing thick liquid matter. No peritonitis nor mechanical obstruction.	Three non-strangulated and easily reduced intussusceptions, apparently more of the nature of "post-mortem" intussusceptions rather than the original cause of the condition.
Result	Recovery.	Recovery.	Death.	Recovery.

involved, and in this condition it is likely the muscular coat ceases to act.

In other cases the congestion may be more passive in origin, and allied to the hypostatic congestion of the lungs.

In another class of cases it may possibly be the result of septic absorption from the contents of the intestine.

It is to be regretted that in three of the cases there is no record of the immediate antecedents of the attack, as an aid to diagnosis.

The difficulty of differential diagnosis between enteritis and mechanical obstruction is so great, that it is most important to ascertain under what circumstances the attack came on, and the difficulty is still further increased as in many cases an attack of enteritis brings into activity and operation some latent cause of mechanical obstruction. For instance, many cases have come under treatment where, after partaking of indigestible and irritating food, old irreducible herniæ have become strangulated.

In one case a patient who had an old umbilical hernia eat abundantly of mushrooms and crab or lobster. The hernia became acutely strangulated and was reduced by immediate operation, but the patient died with profuse purging.

Other cases have been admitted when the same cause has developed symptoms of acute strangulation, which on the operating table has been found to be due either to bands of adhesions or to persistent vitelline duct; and in more than one case in our experience, when the primary cause was enteritis the secondary was found to be due to kinking from adhesions between the viscera.

The following case is an illustration of this:—

A girl, aged 13, was admitted with severe abdominal pain and attacks of vomiting for the past nine days; after passing a hard motion the pain and tenderness was relieved, and she seemed quite well. Next day the symptoms returned with increased intensity.

The face was flushed, tongue furred, pains in abdomen, which was full and tender and did not move on respiration. She was vomiting the contents of the stomach. Temperature normal. On the second day the symptoms had increased; pulse 100 and small; bowels not open and no flatus passed.

Laparotomy was performed; some clear fluid found in abdomen; no general peritonitis, but intestines somewhat congested. A band the size of a goose-quill extended from the umbilicus to a coil of intestine in the right iliac fossa. The band appeared to press on, but did not constrict, some coils of intestine. The

band was ligatured and a portion removed. The patient made a satisfactory recovery.

In another case a man aged 30 eat nine mince-pies on Christmas day. At night while in bed he was seized with violent abdominal pain and vomiting. On December 27th, the vomit, which had been solid matter, became of a pea-soupy description. On December 28th he was brought to the Hospital about 8 P.M. and operated on two hours later, when a band was felt, but could not be reached and ligatured without a more extensive operation than his condition warranted, so enterotomy was performed.

The patient died next day, and post-mortem an "old adherent diverticulum was discovered, with its fundus attached to the mesentery of the portion of bowel from which it itself sprang. This formed a band across the gut and partially constricted it; the distended small intestine above this partial stricture had fallen down, producing a kink over the band, and hence a volvulus."

Again, an attack of enteritis may bring into activity a quite unsuspected mischief about the appendix, which must have been of very old standing, but had never given signs of its existence.

A boy of five years old was admitted to the Hospital, having had diarrhoea and vomiting three days previously, the attack commencing with abdominal pain. Diarrhoea ceased after the first day, and the bowels had not been open since, but the vomiting had continued.

On admission he was acutely ill, headache, eyes sunken; tongue moist but thickly furred at the sides; abdomen soft and natural, nothing abnormal felt. Pulse 120, regular and fair volume. Temperature subnormal. Vomiting green slimy matter.

On day of admission, coils of intestine were visible, and the abdomen painful and distended. Bowels well opened after soap and water enema and distension relieved, but distended coils still visible.

The patient became slowly worse during the next three days, and then vomiting increased and became faeculent (though the bowels were very slightly opened naturally) on the morning of the fifth day after admission.

Laparotomy was then performed, and the last three feet of ileum were found to be strangulated by a band which was ligatured and divided, and proved to be the appendix, which had been the seat of old inflammation and perforation.

The patient died, and post-mortem 18 inches of the ileum were gangrenous.

Concretions in the appendix must take long to form, and

may remain innocuous until some intestinal irritant (as we suppose) stimulates the secretions of the appendicular glands, distending the appendix, which distension may cause strangulation and sloughing or ulceration from internal pressure.

Circumstances like these illustrate the extreme difficulty of arriving at an exact and accurate diagnosis before operating, and the advisability of keeping an open mind during the operation.

A CASE OF
HEPATIC CIRRHOSIS, WITH OBSTRUCTION
IN THE SUPERIOR VENA CAVA.

BY

SIR DYCE DUCKWORTH, M.D.,

AND

ARCHIBALD E. GARROD, M.D.

W. J., *æt.* 35, was admitted to Matthew Ward on June 29, 1896, suffering from ascites and an obvious degree of jaundice. He was a labourer. Three years previously he had noticed that the veins over his belly had become enlarged. His strength had failed. Two years ago he vomited a pint of black, clotted blood. Two months previously he became yellow, and found his belly swollen. There had been occasional retching in the morning, and sometimes blood in what he brought up. There had been no previous severe illnesses. His habits had been intemperate.

The family history had no bearing on the case.

The patient was a well-developed man. He was in no pain, and could lie in any position. The temperature was natural. Respiration 20 and pulse 80 in the minute, the latter with fair tension and volume. The lungs were resonant all over, some crepitations being heard at the bases. The heart's apex-beat was in the natural position, the sounds clear, with reduplication of the second sound over the pulmonary valves. The belly was protuberant. Hepatic dulness began at the fourth rib, and was so extensive as to reach below the anterior superior iliac spine, measuring 16 inches in the right nipple-line. The liver was readily felt all over this area, firm, smooth, and with a sharp edge. The spleen was not detected. Over the abdomen there coursed a series of enormously distended

veins, the largest being laterally disposed and tortuous, and there was an area of distended small vessels about the scrobiculus cordis. The navel was obliterated and free from a *caput Medusæ*. The current of blood clearly ran, under high pressure, in both an upward and downward direction, but more forcibly downwards whenever examined. The lower limbs were not œdematous, and their veins were not unduly full. The veins over the arms were not specially noteworthy, but there were a few venous stigmata visible. The bowels were rather loose; the urine a little bilious, but free from albumen and sugar.

The diagnosis made was hepatic cirrhosis, due to alcoholic excess; but the condition of the superficial veins and the direction taken by the blood in them from above downwards, indicated that there was difficulty somewhere in the return of venous blood to the right side of the heart. This venous plethora, therefore, could not all be traced to hepatic obstruction in the portal venous system. There was some ascites, but the greater fulness of the abdomen was due to the enormous size of the liver.

The chest was therefore carefully examined for evidence of any signs of obstruction to the flow of blood through the superior vena cava. A faint systolic murmur was heard in the aortic area, and some slight dulness was noted over the mid-sternal region. The cervical veins and those from the arms were noticed to be full, but not remarkably so. The air-entry was less free on the right than on the left side.

A light diet was ordered, and effervescing citrate of potass draughts were given, also the confection of tartrate of iron.

There was occasional vomiting, and some blood was found in the ejecta from time to time. Increasing distension and discomfort in the belly necessitated the operation of paracentesis on September 15. During the several weeks this man remained in Hospital he was fairly comfortable, and left his bed daily. The temperature was subnormal. The urine was small in amount, generally under a pint *per diem*, and containing from 1.4 per cent. to 2.3 per cent. of urea, with bile pigment, and sometimes with pink uratic deposit. There was a tight urethral stricture, which caused dysuria. Thrombosis occurred in some of the distended superficial abdominal veins, especially on the right side. The belly gradually filled up again with fluid, and had to be tapped on October 6. It measured $43\frac{1}{2}$ inches in the greatest circumference. A Southey's trocar was employed, and in six hours 0. xxiss of fluid were removed, the girth being thus reduced to $39\frac{1}{4}$ inches. The liver was now discovered to have shrunk very materially, and was not much below the level of

the navel. Progressive weakness ensued. The ascites continued to increase, and the lungs became œdematous. On October 18 the patient had a rigor, and complained of abdominal pain. Temperature rose to 104.6°. On October 20 the respiration was hurried. Decubitus on right side; œdema of the right arm and side generally. From the pubes to the right shoulder there was a reddened irregular line of cellulitis. This condition was found on the right back, and the involved area was tender to the touch. The mental condition remained clear. The legs became slightly œdematous. At night delirium set in, and death ensued on the morning of October 21. The last recorded temperature was 101°.

Dr. Garrod made an inspection of the body on the following day. The facts elicited entirely justified the diagnosis. Some of them were so remarkable as to deserve permanent record in connection with the clinical features of the case. They are therefore appended.

In the earlier stage of the illness, on admission, there could only be a little doubt as to the exact condition of the liver. The diagnosis lay between cirrhosis due to alcoholic excess, and a variety of uniformly infiltrating carcinoma. The age, history, and general aspect of the patient helped me to decide in favour of cirrhosis with enlargement, and the existence of venous stigmata, which I consider pathognomonic of alcoholic cirrhosis, much fortified this opinion.

Nothing in the antecedent history threw any light upon the nature of the obstruction to the superior vena cava. There was no evidence of an aortic aneurysm, or of any new growth in the thorax. In the absence of such tumours, obstruction such as was here met with is of very rare occurrence. An instance somewhat similar is recorded by Dr. T. Henry Green in the *Path. Soc. Trans.* vol. xxvii. 1896, p. 118. In this case there was fluid and lymph in the right pleura compressing the lung, and a dense mass of lymph encircling the root of the vena cava. The opening of the latter into the right auricle was completely closed. Another instance is recorded on p. 79 of the same volume by the late Dr. Habershon, where tough fibroid tissue was met with in the anterior mediastinum. Dr. Goodhart, who made the examination, believed that the condition was congenital, and that no true superior vena cava had ever existed. There was a doubtful history of syphilis, and the co-existence of fibroid disease of the heart and of a malformed pulmonary valve lends support to the view that this may have been an outcome of that malady.

The occurrence of occlusion of the superior longitudinal sinus

by a fibrous plug, evidently of long duration, was a remarkable coincidence in this case, and unattended by any readily recognisable symptoms during life. This is not a common condition. Dr. John Reid recorded a similar instance of fibrous occlusion of this sinus in the *Edin. Med. and Surg. Journ.* for 1835. In the same volume, too, may be found a record of a case by this observer, in which the vena cava superior was obliterated by a hard cord. This occurred in a woman *æt.* 40, and appeared to be induced by the irritation of adjacent tuberculous and calcareous bronchial glands. The literature bearing on this subject points to two main classes of cases in which occlusion of the superior cava may occur—(1) in which the obliteration is due to thrombosis-concretion, and (2) in which the obstruction is the result of mechanical compression, as by tumour, cancerous or aneurysmal. An excellent account of cases illustrating both varieties is to be found in the *Mem. de la Société Méd. d'Observation de Paris*, p. 391, by M. Oulmont, 1856. I append some further references which may be consulted with respect to this condition. In 1853, M. Barth described a case in which cancer of the left lung and heart led to occlusion of the superior cava by clot. The lower limbs were free from œdema, but the head and arms were swollen, and the superficial veins characteristically dilated. Duchek recorded several examples in the *Vierteljahrschrift für die pract. Heilkunde*, Prag, 1854. A case resembling the one here recorded in respect of mediastinitis as the cause of obliteration was described by Dr. Bennett of Dublin for Dr. Williams of Liverpool, and reported in the *Dublin Journal of Medical Science* for 1875. Another case, due to similar influence, is recorded by Dr. A. V. Meigs of Philadelphia, in a man *æt.* 72, in the *Transactions of the College of Physicians of that city*, 1884.

The diagnosis of obliteration of the superior vena cava is not difficult. The precise cause of it in any case is not always readily to be determined. If no signs of gross tumour exist, we may bear in mind the possibility of chronic inflammatory growth, not gross, though sufficiently effective to compress the large vessel mechanically, and lead to fibrosis within its lumen, or to induce thrombosis, which may in course of time completely seal it up. Compensatory venous circulation is gradually set up, in which the azygos veins largely take part, and the superficial venous channels become more enormously dilated than is possible by any other conceivable obstruction. The prognosis in all such cases is necessarily of the most unfavourable character.

(For the references to published cases, I may mention my indebtedness to Dr. Garrod.)

DR. GARROD'S NOTES.

An inspection was made on October 22nd, twenty-seven hours after the death of the patient,

The skin showed a slight icteric tint; the abdomen was greatly distended; the enormously enlarged and tortuous superficial veins on the thorax and abdomen were collapsed, but were nevertheless clearly visible.

On opening the abdomen, a very large amount of bile-stained serous liquid escaped. The peritoneum was opaque, but its surface was smooth and shining. The omentum was thickened and contracted. There were no adhesions in the peritoneal cavity.

The liver was enlarged, weighing 79 ounces, and its capsule was thickened, and showed the numerous small circular depressions characteristic of perihepatitis. Beneath the thickened capsule the surface of the liver was distinctly hob-nailed, and on section the organ showed the changes characteristic of fatty cirrhosis, the lobules having a pale yellow tint, and being separated from each other by conspicuous strands of fibrous tissue. The portal vein was free from clot and somewhat enlarged. The gall-bladder showed nothing abnormal, and the bile-ducts were patent throughout.

The spleen was much enlarged, and weighed 20 ounces. Its capsule was thickened and opaque, and showed pit-like depressions similar to those seen upon the surface of the liver. On section the organ showed nothing abnormal to the naked eye. The kidneys and suprarenal capsules were natural.

On opening the thorax, the right lung was found to be covered by a greatly thickened pleura, and was everywhere bound down by adhesions.

The tissues of the mediastinum around the superior vena cava had apparently been the seat of an inflammatory process similar to that in the adjacent right pleura, and this had resulted in the obliteration of the vessel from its origin to its entrance into the right auricle. The pericardium showed nothing abnormal. On opening the right auricle *in situ*, the orifice of the inferior vena cava was found to be large, but was otherwise natural, and the same may be said of this vessel throughout its course. At the point of entry of the superior vena cava there was a small orifice, which admitted a probe for a very short distance only.

The right auricle was otherwise normal, as also were the

other cardiac cavities and the valvular orifices. In the commencement of the aorta there were a few patches of atheroma.

The innominate veins were unduly large, but otherwise natural.

At the upper end of the obliterated vena cava was a complete funnel of calcareous material, in the centre of which was a small orifice, into which the tip of a probe could be inserted only for a short distance. Just above this calcareous funnel a large vein from the mediastinum entered anteriorly, just below the junction of the innominate veins.

The vena azygos major was much dilated, and could be followed up to its junction, at an obtuse angle, with the greatly enlarged superior intercostal vein; but not only was the normal point of entry of the vena azygos into the superior vena cava included in the oblitative process, but the vein itself, after its junction with the superior intercostal, became completely lost, and could not be traced over the root of the lung.

The collateral venous circulation appeared to have been chiefly carried on through the enormously dilated superficial veins of the thorax and abdomen, but the large size of the right superior intercostal vein and vena azygos major showed that these vessels took a considerable share in the work, in spite of the obliteration of the latter at its upper extremity. Indeed, many of the deeper veins of the thorax and abdomen showed conspicuous enlargement to meet the altered conditions of the venous circulation.

The left pleura showed no adhesions. The left lung was somewhat emphysematous. On opening the skull, the superior longitudinal sinus was found to be completely obliterated by firmly organised clot, and felt like a piece of split cane inserted into the dura mater. The dura mater itself was thickened and unduly opaque. The other sinuses were patent and normal. The brain weighed 46 ounces, and was otherwise normal. The appearances suggested that an inflammatory process, originating in the right pleura, had spread to the adjacent mediastinum, and that the obliteration of the vena cava was secondary to an old pleurisy; but a study of the recorded cases in which obliteration of the vena cava has resulted from inflammatory changes in the mediastinum shows that, under such circumstances, the right pleura is frequently involved, being sometimes obliterated by adhesions, and in other cases containing fluid. In some instances in which this association of lesions has been observed, the process apparently originated in the mediastinum, having its origin in inflammation around caseous lymphatic glands. Hence it would seem to be by no means

certain that in the case under consideration, in which, however, there was no evidence of a glandular origin, the pleurisy was not a secondary rather than a primary event.

The obliteration of the superior longitudinal sinus had obviously occurred some long time before the death of the patient, and it might be expected that the effects of obstruction of the venous circulation should be manifested in this channel, in which the flow of blood notoriously takes place under conditions of considerable mechanical disadvantage.

It might further be anticipated that thrombosis in this channel, if it occurred at all, would probably take place early, before a collateral circulation had been fully established.

The absence of swelling of the head and neck is a remarkable feature in this case, in view of the complete obliteration of the vena cava. Such swelling has been usually present in such cases, and formed a conspicuous feature in one which has since come to a *post-mortem* examination at our Hospital, in which, as the result of a sarcomatous growth in the mediastinum, the superior vena cava was conspicuously narrowed in calibre, but was by no means completely occluded.

ON ETHER RASH.

BY

EDGAR WILLETT.

By "ether rash" is meant the rash that occasionally occurs during the administration of ether as an anæsthetic. Although its occurrence is fairly well known, so little has been written about it, that the following short account may be of interest.

Writers on anæsthetics, as a rule, dismiss the subject in a few words. Thus Dr. Hewitt¹ writes: "A roseolous rash sometimes makes its appearance upon the chest, neck, and other parts when the vascular excitement is at its height."

Again, Dr. Dudley Buxton² writes: "The pupils dilate and the skin becomes bedewed with perspiration, while a roseolous rash appears in patches about the neck and chest." This is part of a description of what usually occurs while a patient is becoming unconscious from the administration of ether, and while correct in that the dilatation of the pupils and the breaking out of perspiration and the rash *may* all occur, so far as my experience goes, the dilatation of the pupil is the only phenomenon which *always* occurs. The perspiration commonly occurs also, but, in my experience, the presence of the rash is decidedly rare; even when the rash is present, perspiration to any marked extent need not occur also.

In the tenth edition of Erichsen's Surgery, in the article on Anæsthesia³ there is the following sentence: "At this time a bright red rash not uncommonly appears upon the chest and neck;" these are the only references to the occurrence of the rash that I have been able to discover.

Dr. P. A. Morrow of New York, while stating in his book on Drug Eruptions⁴ that his "object has been to collect from all

¹ Anæsthetics and their Administration, by F. W. Hewitt, M.D. Published by Griffin & Co., 1893, p. 156.

² Anæsthetics, their Uses and Administration, by Dudley Buxton, M.D. Published by H. K. Lewis, 1888, p. 59.

³ Science and Art of Surgery, by Erichsen, tenth edition, vol. i. p. 22.

⁴ Drug Eruptions, by P. A. Morrow, M.D. New York, 1887.

available sources well-authenticated observations relating to every form of cutaneous disorder thus far recorded from the action of drugs," makes no allusion to ether, although in his opening chapter on the Pathogenesis of Eruptions caused by the external or internal administration of drugs, the nervous origin of certain rashes is described as being an established fact.

Nor does Dr. Snow,¹ in his classical description of the various phenomena observed during the administration of ether, make any mention of the occasional presence of a rash. This omission in such an accurate observer is probably due to the fact that it had not occurred in his practice when he wrote his book, at a period when anæsthesia from ether and chloroform alike was yet in its infancy.

In order to determine with a certain amount of accuracy how often it occurs, I have for rather over a year carefully looked out for it, and have kept a note of each case when I have observed it, with the following results.

Characters of the Rash.—The rash always appears quite suddenly, and is roseolous in character; it is blotchy and bright pink, or even red in colour, the sharp contrast between the pink or reddened portions of skin and the pale or unaffected parts being very noticeable. It is, of course, due to a sudden but irregular dilatation of the surface capillaries.

In point of time the rash is generally well marked three or four minutes after the administration has commenced; it will be at its height in another two minutes, after which it slowly begins to fade, and at the end of fourteen or fifteen minutes from the commencement it has usually quite disappeared, so that its duration altogether is about ten minutes.

Parts affected.—The neck and upper part of the chest is the commonest situation; it may spread up to the lower jaw, or even on to the cheeks, and it seldom goes lower than the upper half of the mammary gland. I may here mention that it is much commoner in women than in men; it is markedly symmetrical as regards the two sides of the body, and from the parts I have already mentioned as being chiefly affected, it will be noticed that its distribution, as a rule, is confined to the area supplied by the nerves of the superficial cervical plexus.

Frequency.—As to the frequency of the rash, I have separated hospital patients from private patients, to see if there is any difference, and find that, although it occurs with slightly greater frequency in private practice, there is practically no difference. The actual numbers are 4.2 per cent. in hospital practice, and 6.6 per cent. in private; from which it is noticed that its occur-

¹ The Inhalation of Vapour of Ether, by John Snow, M.D., 1847.

rence at all is by no means frequent. These numbers apply only to those cases where ether has been the anæsthetic, as, with one single exception, to be mentioned later, I have never noticed anything approaching a rash during the administration of chloroform, and never from ether in those few cases where it has been thought advisable to commence with chloroform.

Sex.—As shortly stated above, the rash is very much more frequent in women than in men—in fact, I have only notes of three cases where it has occurred in men (twice in hospital cases and once in private), so that it is practically confined to the female sex. In connection with this fact may be noticed the greater frequency of blushing in women.

As to the question of sex, however, I may mention that the most intense and extensive case of ether rash I have seen occurred in an elderly man, aged 54. In his case the whole of the trunk down to the pubes was covered with a well-marked, dark red roseolous rash, which faded in the usual way.

Age.—I find that the age at which the rash is most frequently observed is about 30. Actual numbers make it 32, although I have observed it in girls of 12 and 15, and in patients over 50 years of age. In one case, a lady over 50, who had her breast removed, and who underwent a second operation six months later for recurrence, the rash was well marked on both occasions.

Operation.—There does not seem to be any connection, or, if any, but slight connection, between the part operated on and the development of the rash. If this were so, some slight unconscious reflex action, due to irritation of the skin in the neighbourhood of the rash, might be put forward as a cause. It is true that, in those cases where I have noticed it, the breast has been the commonest part operated on, but when one remembers how frequently this operation is performed on women of 32 or thereabouts, the occurrence of the rash can hardly be said to be caused by the action of the knife in setting up a reflex dilatation of the vessels of the skin in the neighbourhood. Also, it is worthy of note that in the case of the extensive rash on the abdomen of the man already mentioned, the operation was that of inguinal colotomy, and the inguinal region had been “prepared,” and perhaps slightly irritated, by an antiseptic dressing.

Curiously enough, in connection with this question of site of operation, a very instructive case occurred quite recently, during the writing of this paper. A girl aged 16 was brought into the theatre for a slight operation on her right ankle, where a sinus had existed for some time. As the dressing was removed from her foot, it was noticed that there was a well-marked roseolous rash, similar to the other rash, all over both legs and thighs,

but that there was no rash on her face, neck, or chest, although those parts were slightly flushed. A practical question, then, was asked, whether the rash was due to the ether, or whether it was due to erysipelas or one of the fevers, in which case it might not have been considered advisable to perform the operation. The general opinion was in favour of its being due to the ether, and the fact that it entirely faded more quickly than usual—in three or four minutes—confirmed this view. It was quite an exceptional case, however, in that the rash did not appear at all in the usual situation.

Temperament.—I have not been able to trace any relation as regards the temperament, complexion, or general condition of the patient and the presence or absence of the rash. Many exceedingly nervous patients, who are exceptionally frightened at the thought of the operation, have gone quietly under the influence of the anæsthetic without any development of the rash, and in others, who have given no previous indication of fright or nervousness, the rash has been present in the manner described above. Perhaps it is rather commoner in people with fair complexions.

The presence of a rash during the administration of chloroform is, as stated above, exceedingly rare. The only instance in which I have observed it was in a little boy aged 9, a hospital patient, who underwent the operation of osteotomy of the femur. In his case there was at the usual time a fine punctiform rash universally distributed over the neck and upper part of the chest, gradually fading away at the margins. It closely resembled the rash of scarlet fever, and was not blotchy or roseolous. It faded gradually in about ten minutes. This is not, however, an isolated case, as other anæsthetists have informed me they have *very rarely* observed similar instances of a rash due to chloroform.

In conclusion, the practical points to observe are that the ether rash occurs rarely, and generally in women; that it fades in about ten minutes from its onset; and that it need be no bar to an operation.

FETID EMPYEMA.

BY

SAMUEL WEST, M.D.

In fetid empyema the fluid has an extremely fetid, sickening stink, such as is met with in gangrene of the lung or in faecal abscess. It is dirty green or brown in colour, opalescent, turbid, with a pulverulent sediment, and contains more or less altered blood. Microscopically, there are found many red blood-cells, pigment granules, fatty crystals, or plates of cholesterin.

Micro-organisms are numerous, and of many kinds, *e.g.*, proteus, leptothrix, bacterium tetragenese, bacillus of malignant œdema, spirochæta denticola (the spirillum of saliva), bacterium coli comunis, the long and thin bacillus of Löffler (diphtheria), and others. With these may be associated other organisms which do not cause putrefaction, *e.g.*, the pneumococcus, streptococcus, staphylococcus, actinomyces, &c.

The cases of fetid empyema fall into two groups. In the one it is consecutive to putrefactive or gangrenous inflammation in the neighbourhood of the pleura, and in the other it is of spontaneous or primary origin.

GROUP I.

When there is gangrenous or putrefactive inflammation near the pleura, the consecutive empyema may be fetid, though not necessarily so. Thus it is the rule with gangrene of the lung, but rare with other lesions of the lung, even with fetid bronchiectatic or other cavities; if it occur with a pneumonia, the pneumonia must be of a septic, and not a simple character.

It may also follow caries or necrosis of the bones of the thorax, but usually the resulting empyema is not fetid.

It is especially likely to occur where there is a perforating disease of the digestive track, as in connection with stricture of the œsophagus, ulcer of the stomach, or with any faecal abdo-

minal abscess which has reached the neighbourhood of the diaphragm, or where there are lesions in the lung or liver which are likely to become gangrenous, as, for instance, a suppurating hydatid cyst.

In this group the symptoms, prognosis, and treatment depend chiefly upon the primary disease. The symptoms are asthenic in type. The patient is in a state of profound prostration, with low, muttering delirium, a feeble pulse, and dry tongue, and soon passes into the typhoid state. If the primary disease, as well as the empyema, cannot be relieved, death must be the result.

GROUP II.

In the second group, that is to say, where the fetid empyema is primary or of spontaneous origin, and not connected with any other putrefactive lesion, the conditions are entirely different.

It seems that nearly one-half of all the cases of fetid empyema belong to this group. Of twenty cases which Netter examined, seven were due to gangrene of the lung, five to pyemia, and eight could be attributed to no cause at all—*i.e.*, they belonged to the primary group. Schwartz's results are much the same; of fifteen cases, nine belonged to the primary group and six to the secondary.

In most of these cases the pus is fetid, however early in the attack it may be examined, so that it is probably fetid from the commencement. It is true that sometimes a simple empyema may become fetid after operation, but this is quite a rare event, and as a rule it is the exact contrary which occurs; for as soon as the fetid empyema has been opened the fetor passes off and the pus becomes sweet.

I do not know that there is anything in the symptoms of the case by which the fetid nature of the effusion may be diagnosed beforehand.

I am inclined to think that in this group the empyemata are generally localised; at any rate, this has been so with those that I have seen.

The prognosis seems to be neither better nor worse than that of ordinary empyema.

The treatment is the same, except that as soon as the empyema is known to be fetid, the sooner an operation is performed and the fetid pus removed the better. It appears to be desirable at the same time to thoroughly wash the side out, and, if necessary, to repeat the irrigation as often as the fetor returns. A single washing out is often sufficient, and the fetor may then pass off very rapidly, even in a day or two.

I have seen several of these empyemata burst through the lung, and then the diagnosis from a gangrenous cavity in the lung becomes very difficult, if not impossible.

The expectoration of a fetid empyema is, however, no more likely to cause secondary changes in the lung than a simple empyema, and in most of the cases that I can recall complete recovery has taken place without any complication.

I have seen unusual complications follow fetid empyema once or twice. Thus, I remember the case of a young lady who had an empyema, the fetid nature of which was not known until the side was opened, when the stench was so overpowering as to turn the surgeon sick. The case, however, ran its ordinary course, except that shortly after operation the patient developed a severe diphtheritic inflammation of the conjunctiva, which ultimately destroyed the cornea, and necessitated the excision of the eye. In other respects the case ran the ordinary course, and ended in complete recovery.

PROFUSE UNCONTROLLABLE DIARRHŒA
IN A MAN RECENTLY RETURNED FROM
THE TROPICS.

DEATH FROM EXHAUSTION—NO CAUSE FOUND—
P.M. THE ONLY LESION A SLIGHT DEGREE
OF GRANULAR KIDNEY.

BY

SAMUEL WEST, M.D.

Stephen H., aged 42, an inspector of detectives in Singapore, was admitted into the hospital with very severe diarrhœa, vomiting, and some fever, from which he had been suffering for the last three weeks. He gave the following history:—

He reached England about six weeks ago, having come to England on leave, because he had not been in very good health recently. He became very much better, and about a month ago went for a holiday with a friend. After being away a few days he was seized with vomiting and diarrhœa. He then came back to London, and had been ill ever since. For the first day or two he had very severe pains about the level of the liver, and the urine became very much diminished. He could refer his illness to nothing which he had eaten or drunk, and he could not assign any cause for it; he had never suffered from any attack of the kind before. When he returned to London, the bowels were opened eight or nine times in the twenty-four hours; they were always liquid, loose, and copious. He also had very bad morning-vomiting, but was occasionally sick during the rest of the day. He had generally been a heavy drinker, taking, as a rule, half a bottle of whisky in the day, but that was his usual habit out in Singapore. He had been drinking beer in Warwickshire, with a little spirits now and then, but had not been taking any large quantity at the time. For the week before admission he had been getting hardly any stimulants at all, and yet was in a tremulous, shaky condition.

His previous history was the following :—

He had been eighteen years in Singapore, and had had very good health for the first six years ; then, twelve years ago, he had had an attack of dysentery, brought on by getting cold on a steamer, and had been invalided home. He went into the Royal Free Hospital, and a mass was discovered between the liver and stomach, thought to be an aneurysm, but this disappeared after a time. He got better, but not completely cured, and went back to Singapore. During the next three years after his return he had a good deal of fever, malarial in character, and had a very hard life with bad feeding and much fighting. At the end of that three years, nine years ago, he had another very bad attack of dysentery. He was then treated with ipecacuanha. He had never had any dysentery since, but the bowels have always been loose. He had also had malarial fever occasionally since, but had never been laid up with it. He had had several slight sunstrokes and met with accidents, but had never been wounded while fighting. For the last twelve months he had been getting somewhat thinner, had vomited occasionally in the mornings, and had had a somewhat bad appetite. Ever since the last attack of diarrhoea his bowels had been loose and open three or four times a day. Latterly he had become somewhat tremulous and nervous, which he in part attributed to sunstroke, a bad attack of which he had two years ago, but recovered from quickly at a hill-station. During the last year this tremulousness has come on whenever he has had extra work, and it was on this account, and because he was losing flesh and health, that he came home on a year's leave of absence.

His mother died at the age of 62 of diarrhoea with wasting, but this was probably malignant disease. She had been in the same district some time before she died, but there was no connection between her diarrhoea and her residence in that part of the world. The patient had two brothers in the same district, and one suffered in the same way as the patient, but he is not known to have actually had dysentery.

The patient was a spare man with a somewhat earthy complexion, and looked as if he had been a fairly hard drinker in his time. He lay in a listless, apathetic condition, not actually delirious, but taking little notice of what was going on around him. His pulse was 102, the tension a little above the normal, but the artery was not thickened. The temperature was 100.8° on admission, rising to 101.4° in the evening ; the tongue was a little inclined to be dry and coated with a white fur. There was a good deal of tremulousness about his movements, both of his face, tongue, and hands.

There was nothing special to be noticed on physical examination, except that the urine contained a little albumen and a few granular casts (?); these, however, were not found after the first day. The liver was slightly enlarged, extending about three-quarters of an inch below the costal arch in front.

The chief symptom was the profuse diarrhœa. This was almost constant, and the motions, which were large in quantity each time, numbered sometimes as many as twelve or fourteen in the twenty-four hours.

There was nothing special in their character; they were simply liquid and somewhat powdery, but they contained no blood, no mucus, and nothing that would suggest dysentery. For the first few days they were bright green, but afterwards their general colour was the usual brown. They were not particularly offensive, and microscopic examination did not show the presence of any pus.

The motions and the blood were all carefully examined microscopically as well as bacteriologically, and nothing that would suggest the nature of the illness discovered.

The temperature was at its highest, viz., 101.4° , on the evening of his admission, but it rapidly sank, and after three days remained persistently below normal, falling even as low as 97° on several occasions, and did not begin to rise again until a day or so before death. The patient's weakness rapidly increased, as would be expected with the profuse diarrhœa, although he was able to take food. He was given freely of stimulants, and various drugs were employed, but all alike seemed to have but little effect upon the diarrhœa. On the supposition that it might be a case of dysentery, ipecacuanha and opium were employed in large doses, but without any marked effect. The most successful treatment seemed to be a mixture of catechu, cinchona, opium, and *mistura cretæ*, to which subsequently some resorcin was added, but without materially increasing the effect.

A week after admission a rash appeared in the centre of his trunk, and rapidly spread over the whole body, including the face. It was erythematous in character and itched intensely. After a few days it was followed by a brawny desquamation. The itching and redness of the skin persisted, though becoming less intense than at first. This was the only new symptom. The diarrhœa persisted, although during the last week the number of motions did not exceed five or six in the twenty-four hours.

The patient became weaker and weaker, more and more apathetic, and though usually delirious at night and rather

difficult to keep in bed, he lay during the day in a listless condition, and could hardly be roused to take notice of anything that was going on about him. He subsequently died of sheer exhaustion, without any new symptoms, on the eighteenth day after admission, having been ill about six weeks.

The diagnosis was extremely difficult. At first it was thought that probably the patient had some form of dysentery; but this diagnosis was soon given up, for there were no other symptoms which pointed to it. Then it was thought it might be some curious condition, such as tropical diarrhœa, which the patient had brought home with him, and inquiry elicited that one of his brothers who lived in Singapore was liable to attacks of a somewhat similar kind; and it was stated by his wife, though we could never ascertain whether this was actually the case or not, that another resident in Singapore, who was also at home in this country on leave, was suffering from a similar attack.

The possibility also of the condition being one of toxine poisoning, started perhaps by eating tinned meats or prepared fruits, or something of that kind, was considered; but no evidence could be obtained of this, and the patient died without a diagnosis having been really made beyond that of diarrhœa and exhaustion.

On the post-mortem examination the body was found covered with the desquamating mottled rash described; the lungs were slightly emphysematous; the heart a little hypertrophied, weighing $10\frac{1}{2}$ oz., the vessels free from atheroma; the peritoneum healthy; the liver a little larger than normal, weighing 54 oz.; the spleen 8 oz. and normal; the pancreas normal; the suprarenals with some small specks on their surface; but otherwise normal; the kidneys slightly enlarged, weighing 14 oz. together, red on section, the surface slightly granular, and the capsules a little adherent.

The intestines showed no ulceration of any kind, either in the small or large intestines. There was some black pigmentation in the mucous membrane and the upper part of the rectum.

The post-mortem examination, therefore, threw no fresh light upon the case. The only fact was that there was a slight degree of granular kidney, and it was then suggested that this had possibly been a case of uræmia; but there were no other uræmic symptoms, nor anything during life to suggest that this was the nature of the attack. The urine, moreover, had been in fairly good quantity, and although it contained a slight trace of albumen at first, this varied a good deal, and towards the end of life completely disappeared. With reference to this suggestion I can only say that I have never seen anything in uræmia

at all like the symptoms in this case, and that such a diagnosis did not present itself to me as at all probable during life.

I record the case because I have never seen anything like it before in this country, because I could not understand or explain it during life, and because the post-mortem throws no further light upon the subject. The pathological examination was made by Dr. Kanthack, who made an investigation both of the blood and of the fœces without being able to discover anything which would throw any further light on the case.

ON HERNIA OF THE VERMIFORM APPENDIX.

WITH A SERIES OF CASES.

BY

W. McADAM ECCLES.

The vermiform appendix may be found in the sac of an external hernia, or it may occur as the contents of a retro-peritoneal hernia within the abdomen. Both of these malpositions of the appendix cæci are comparatively rare, though, with the increasing number of operations performed, there are now a considerable number of cases recorded and of specimens preserved—such a number, in fact, as may warrant certain deductions being made from them.

I. The appearance of the appendix in a hernial sac may be the sole contents of such a sac, or it may be accompanied by other parts of the viscera.

A. The appendix as the sole contents of a hernial sac.

The question which first arises is as to how the appendix has come to enter a peritoneal pouch protruded beyond the abdominal wall. The normal appendix, some four to five inches in length, with a complete meso-appendix, is free to move about within the abdomen within a certain range around its attachment to the cæcum; but it cannot be brought down to, and much less dragged through, either the right inguinal or femoral ring.

One of two conditions then is necessary for its descent. In the first place, it may itself be considerably in excess of its usual length. This can be either the outcome of congenital elongation, or of a stretching the result of adhesion. In the second place, the cæcum may be abnormally free from the presence of a long meso-cæcum, with the result that the appendix may thus approach dangerously near to a hernial ring. A further point comes up for investigation in the query whether the appendix alone can be the primary and final contents of a hernial sac? It is undoubtedly possible for it to be extended

into the sac of a congenital right inguinal hernia, for in such a case there is a naturally patent processus vaginalis into which any viscus lying near may be forced.

It is certainly somewhat difficult to understand how such a small part of the alimentary tract, having so little of surface area, would be able of itself to form a sac by pressure ; but, on the other hand, is it actually necessary that there should be this special and active distending force of a particular viscus in order that an acquired pouch of peritoneum may be produced ? It does not require any very resistant organ to share in the formation of a pouch ; in fact, it may be true that no pressure of any viscus is absolutely needful. Some sacs with very narrow necks and of an acquired origin have but an extremely thin process of elongated omentum lying within them, and surely this would seem of itself less able to aid by pressure the protrusion of the serous membrane than even the appendix.

If, however, it is necessary to suppose that other of the abdominal contents besides the appendix have been the cause of the hernial sac, it is difficult to account for the very small pouches, and these, moreover, with narrow necks, which appear not infrequently as the coverings of prolapsed appendices.

Doubtless this argument is open to the rejoinder that it is quite possible for the original multiform contents of the sac to be reduced all but the appendix (which might alone have become adherent), and then for the mouth and neck to contract around the small structure passing through them ; and beyond this it is perhaps feasible that strangulation, or at least some constriction of the appendix, may occur. Or, again, the appendix might slip into a sac which had not previously contained it, but had been occupied by some other viscera.

There is an instructive preparation in the Museum of the Royal College of Surgeons of London¹ which bears out the theory that other viscera may form a sac, but that sooner or later the appendix may remain as the sole contents, and that more particularly if it become adherent. In this specimen a somewhat large sac of a right inguinal hernia has at the upper part a portion of the cæcum, and it is not difficult to believe that the whole of that organ was at one time within the peritoneal pouch. Further, this would doubtless have been completely reduced were it not for the fact that the tip of the appendix cæci is adherent to the bottom of the sac, and thus effectually preventing the total return of the cæcum.

It may further be conjectured that if the appendix were to

¹ No. 2635.

become stretched in such a case, the rest of the cæcum might disappear from the sac and its appendix alone remain, held fast within it. In spite of these latter considerations, however, I am inclined to believe that the appendix itself may be the producer and the entire contents of a right inguinal or a right femoral sac, and in a few very rare cases of a left inguinal.¹ I do not know of any instance in which it has been discovered in a left femoral, umbilical hernia, or ventral hernia.

The appendix when herniated is liable to a number of changes, some of which are peculiarly interesting, and others of the gravest import.

(1.) Elongation is almost invariably present. The normal or average length is given as four inches, but the appendix may vary from one to nine inches. This lengthening may be the outcome of traction upon the tube, either by its being repeatedly nipped in the hernial ring, or by its being adherent within the sac.

Moreover, the appendix may be congenitally longer than usual, and this fact itself may predispose to its protrusion.

With this elongation there is sometimes an accompanying diminution in the calibre of the tube in its whole length, or only in that part which still lies within the abdomen.

(2.) Adhesions may form, and seem to be of comparatively common occurrence. Indeed, the appendix would appear to be more frequently adherent than other viscera found in hernial sacs. This adhesion is probably always the result of local inflammation, and it is a well known fact that an abnormally placed appendix is very liable to attacks of inflammation.

In these cases of adhesion the appendix may be found attached to the margins of the mouth of the sac, with the extremity free in the body of the sac, or the tip alone may be the part adherent, or its whole length may be attached.

This adherence, moreover, will bring about irreducibility, and may further lead, as above stated, to elongation and thinning of the appendix. Possibly also it sometimes has to do with the onset of inflammation by producing kinking of the organ. Lastly, the blocking of the mouth of the sac by the adherent appendix may be complete, and bring about the formation of a hydrocele of the body of the hernial sac.

(3.) A cystic dilatation of the protruded portion has been found in several cases. Within the abdomen such a condition may also be found, and would appear to be due to blockage of the

¹ An instance of this is found in the record by Geissler in which the appendix alone was within a left inguinal sac, the cæcum lying in the middle of the abdomen (Geissler in Virchow's "Jahresbericht," vol. ii. 1867, p. 475). See also Case 26.

lumen of the tube, either by contraction (or constriction) or kinking.

Precisely the same may occur in a hernial sac. The mucous membrane of the tube contains glands which are habitually secreting, and if obstruction to the outflow of this secretion occurs, a cystic distension will result.

(4.) Inflammation of the appendix in a hernial sac is decidedly common, just as it is when the appendix retains its normal position. The causes of these inflammatory attacks are probably exactly similar to those of inflammation occurring in the appendix when within the peritoneal cavity. It has been suggested that a truss worn over an adherent appendix is very likely to produce injury, and thus cause inflammation, and possibly this may account for a small proportion of the cases.

As in the abdomen, so in the sac of a hernia, perforation of the appendix may be a termination of the inflammation, and this may lead to symptoms which are very like, if not identical with those which are ordinarily said to be the outcome of strangulation of the appendix. In many of the cases recorded below it was stated that there was a sudden onset to the symptoms, and that there was local pain, vomiting, and in some constipation. These, of course, are symptoms which might well be put down to strangulation, and therefore intestinal obstruction; not so complete, certainly, as in cases where strangulation of a loop of intestine occurs, but similar in every detail to those which are in evidence when appendicitis is present within the abdomen. In this latter condition there is wanting any cause of strangulation of the appendix, though there is very clear inflammation of the tube.

(5.) Strangulation of a true nature no doubt does occur sometimes when an appendix is protruded, though I venture to assert that it is much less frequent than is usually thought. It may be brought about in at least two different ways. In some cases the contraction of inflammatory tissue outside the sac has seemed to be the real cause, but more frequently it appears the appendix has been forced into a sac with a very narrow mouth, particularly that of a congenital inguinal hernia. Here the strangulation is the result of direct pressure of the tissues around the neck of the sac.

In whatever way produced, the symptoms which follow are, as has been stated, hardly those of true intestinal obstruction, but similar to those resulting from a partial enterocele, also from strangulation of Meckel's diverticulum, and occasionally from a strangulated ovary. The vermiform appendix, though somewhat poorly supplied with blood, seems to be well inner-

vated, and it is probably as the outcome of reflex irritation that the symptoms which do arise are present. Though there may be vomiting, it is not so urgent or persistent, nor does it become stercoraceous, as in acute obstruction, and the constipation present is rarely if ever absolute. Still, these cases are open to a considerable amount of uncertainty, and by far the wiser course is to explore the swelling.

Usually on opening the sac the appendix is readily recognised, but at times, owing to the presence of many adhesions, with, in fact, a matting together of the tissues, it is difficult to be certain as to the structure which is being dealt with. The appendix has been mistaken for the sac itself, for a second sac within an outer one, for the Fallopian tube, and even for the urinary bladder. The elongated form, the presence of its extremity, and the smaller size as compared with other hollow viscera, all serve to differentiate the appendix.

The methods of treating the organ vary according to the condition in which it is found. If an operation for a radical cure of a non-strangulated reducible hernia is being undertaken, and the appendix is found in the sac, it may be returned into the abdomen, or it may be amputated in the usual way, and the stump replaced within the peritoneal cavity. If, however, the tube is found adherent, and specially if not of natural size, it is well to free it from its attachments and remove it. Furthermore, if, as in some cases of inflammation, and possibly also of strangulation, it is found perforated or gangrenous, it may be either removed after having been drawn down until healthy tissue is seen, or it may be left in the wound which should remain unclosed.

Death occurs in a certain proportion of these cases from general peritonitis, as a consequence of an infection of the peritoneum from the hernial sac, and in other instances it is due to extravasation of contents of an appendix which has been reduced into the abdomen. Recovery may be complete after operation, or it may be complicated with the presence of an intestinal fistula, as the outcome of perforation. Such a fistula, moreover, may occur spontaneously, as the result of an abscess caused by inflammation and perforation which has of itself opened through the skin or been incised by a surgeon. These fistulae occasionally close without any operation, but most need to have the diseased appendix exposed and removed.

Foreign bodies are occasionally found, as when the appendix is normally situated, within the lumen of the tube. An interesting specimen illustrating this will be found in Guy's Hospital Museum, No. 1166, in which a pin was present.

See also Cases 6 and 17, in both of which a piece of bone was discovered.

To sum up, it may be said that the appendix alone in a hernial sac is not so very rare—in fact, a good deal more frequent than is generally supposed, and that when so occurring, it is almost invariably in a sac protruded on the right side.

Further, a herniated appendix is very liable to become inflamed, and thus to acquire adhesions, so as to be irreducible. The inflammation, if severe, may lead to gangrene or perforation, with the result that symptoms are produced which are not infrequently attributed to a strangulation of the tube.

Lastly, that a true strangulation may occur, but that the signs and symptoms of acute intestinal obstruction in their characteristic form but rarely present themselves, the case being like that of a Richter's or Littre's hernia, where in the former only a part of the circumference of the bowel is nipped, and in the latter Meckel's diverticulum is strangulated.

B. The appendix appearing within a hernial sac accompanied by other viscera.

This class of cases may conveniently be divided into two distinct sections, one where the appendix merely passes with the cæcum into a rupture—a condition by no means uncommon, and the second in which the appendix is protruded and often adherent, and therefore irreducible, but is associated as the contents of the sac with small intestine or omentum.

It is these latter cases which lend so much interest to and bring so much light upon the question of local appendicitis within a sac; for, as in Case 12, acute inflammation of the appendix may be in progress with the signs and symptoms so often produced under such conditions, and these may be taken as evidence of strangulation; but on herniotomy it will be found that a portion of unconstricted intestine or omentum lies by the side of an inflamed appendix.

There are a few rare cases on record where the appendix accompanied by the cæcum has been found in a femoral sac on the right side. I have recorded one which was under the care of Mr. Langton in a previous volume of these Reports.¹

Moreover, some true congenital umbilical herniæ most commonly contain the cæcum and appendix.

II. Retro-peritoneal hernia of the appendix cæci has been met with in not a few instances.

About the cæcum are several well-marked peritoneal fossæ, and others which are not so well defined; in fact, about these latter there is some dispute as to their actual existence.

¹ St. Bartholomew's Hospital Reports, vol. xxvii. p. 180.

The recess which can be seen lying below the cæcum when that viscus is drawn upwards and forwards is the one which is most constant in appearance and development. It is usually termed the sub-cæcal, and is undoubtedly formed as the cæcum gradually descends in the right iliac region. In addition an ileo-cæcal fossa may often be made out lying parallel and posterior to the lowest portion of the ileum.

It has been averred that the appendix can become strangulated in one of these peritoneal pouches; but here again it is easy to conceive that the symptoms which occur may equally well be due to inflammation rather than strangulation.

In some cases where the opening of the retro-peritoneal sac is considerably constricted—for it may even become entirely occluded—strangulation may possibly occur.

The appendix has been found wholly or only partially enclosed within the pouch, and when so displaced is usually much coiled. Occasionally it is firmly adherent; at other times there are only very slight adhesions, easily broken through.

As to the production of this condition, it may be that during the descent of the cæcum the appendix has been as it were left behind and become caught in the sub-cæcal fossa.

Mr. Lockwood has suggested another mode of formation of such herniæ by supposing that while an ordinary scrotal hernia is being formed, the displacement of the parietal peritoneum causes the appendix to remain behind and become imprisoned in a retro-peritoneal fossa in a manner similar to that just alluded to. There is, moreover, certainly no reason why, during the movements of the abdominal viscera in health and disease, the appendix should not pass or be forced into one of the cæcal pouches, and sometimes become fixed there. Such displacement has led in some instances to the belief that the appendix is absent altogether.

In conclusion, I have to thank all those gentlemen who have kindly allowed me to record cases under their care, particularly Dr. Gee and Mr. Butlin.

SERIES OF CASES TO ILLUSTRATE THE VARIOUS POINTS
ALLUDED TO IN THE PAPER.

FIRST SERIES.—*Appendix alone in the Hernial Sac, and with no symptoms of acute inflammation or strangulation.*

Case 1.—Female, aged 48. Appendix in right femoral sac. Patient had had hernia for eleven years. She had never had any vomiting, but suffered from constipation. On admission, a con-

siderable swelling in the right groin. No impulse on cough, but there was fluctuation. The skin was somewhat inflamed and tender. The swelling was irreducible.

Herniotomy was performed. The sac was much thickened, and one inch of the appendix was found adherent in the mouth, and somewhat dark in colour and swollen. The sac contained much clear fluid. The appendix was ligatured and removed, as also was the sac wall. The patient made a good recovery.

The case was under the care of Mr. Butlin, and the full notes will be found in the St. Bartholomew's Hospital Female Surgical Register, 1896, vol. v. No. 1804.

Case 2.—Male, aged 41. Appendix in right femoral sac. The patient died from carcinoma of the stomach, and *post-mortem* the appendix was found to be five inches long, of which four inches were within the abdomen, and the last inch in the sac, adherent at its mouth, and the free extremity somewhat dilated. There was no previous history of the case. The patient was under the care of Dr. Gee. See St. Barth. Hosp. Med. *Post-Mortem* Reg., 1895, p. 193; also Trans. Path. Soc. Lond., 1896-97.

Case 3.—There is a specimen in St. Bartholomew's Hospital Museum, No. 2111A, of an appendix within a right inguinal funicular sac. The upper two-thirds of the appendix is devoid of mesentery; the lower third lies free in the sac.

Case 4.—Appendix adherent in right inguinal sac; found during operation for radical cure. Recorded by the late Mr. John Wood (Trans. Path. Soc. Lond., vol. x. p. 190).

SECOND SERIES.—*Appendix alone in the Hernial Sac, with the record that it was acutely inflamed.*

Case 5.—Female, aged 50. Appendix in right femoral sac. Recorded by Cruveilhier. Inflammation occurred with a fatal result. See Anat. Path., tom. ii. liv. 37.

Case 6.—Male, aged 56. Appendix in right inguinal sac. The patient had worn a truss, but inflammation was produced by the presence of a piece of bone within the lumen of the appendix, and this led to perforation and the formation of an abscess. From this a fistula resulted, which in the end, however, healed. Two months later the patient died from other causes, and the appendix was found to be the sole contents of the hernial sac. The case is reported by Thurnam (Trans. Path. Soc. Lond., 1846-48, vol. i. p. 269).

Case 7.—Male, aged 16. Appendix in right inguinal sac. The testes were undescended. Inflammation occurred; an

operation was performed; the appendix found inflamed and adherent. A fatal result followed. The case is reported by the late Dr. Habershon (*Diseases of the Abdomen*, 4th edit., p. 445).

Case 8.—Male, aged 17. Appendix in right inguinal scrotal sac. It was said to be completely reducible. Inflammation came on, and was thought to be of a tubercular nature. Perforation occurred, and operation was performed and the appendix removed. Subsequently laparotomy was undertaken for consequent suppurative peritonitis. Ultimately the patient recovered. The case is recorded by R. J. Hall (*New York Med. Jour.*, 1886, vol. i. p. 662).

Case 9.—Female, aged 60. Appendix in right femoral sac. No truss had been worn. Inflammation supervened (it is expressly said there was no strangulation). Herniotomy performed, and $3\frac{1}{2}$ inches of appendix removed. The case is recorded by Annandale (*Lancet*, 1889, vol. i. p. 627).

Case 10.—Male, aged 13 months. Appendix in right inguinal scrotal sac. Acute inflammation was in progress. Suppuration resulted. An incision was made. Healing followed. An operation was subsequently performed, and a very adherent appendix was removed. There was no evidence of any symptoms of intestinal obstruction in this case. Recorded by G. H. Monks (*Boston Med. and Surg. Jour.*, 1890, vol. i. p. 543).

Case 11.—In the Museum of the London Hospital there is a specimen (No. 1300) from a case of Mr. Jonathan Hutchinson's. It is from an old man who had the appendix alone in the sac of a right inguinal hernia, which had apparently been in existence for some time. Inflammation came on and an abscess formed. The appendix is adherent to the sac wall, except the lowest inch, which is free. About two inches from its extremity there is a small perforation.

THIRD SERIES.—*Appendix with other Viscera in Hernial Sac, but acute inflammation of Appendix present.*

Case 12.—Male, aged 24. Appendix with a knuckle of small intestine in right inguinal sac. The patient rather suddenly was seized with vomiting, but beyond this there was no evidence whatever of any intestinal obstruction. A portion of the contents of the sac had been reduced by taxis prior to the operation. Herniotomy performed, and the sac found to contain an inflamed adherent appendix, together with a knuckle of small intestine. The latter presented no sign of having been strangulated. The appendix was removed by ligature. During con-

valescence the patient had an attack of parotitis, but in the end completely recovered. The case was under the care of Mr. Butlin. See St. Barth. Hosp. Male Surg. Reg., 1894, vol. v. No. 203.

Case 13.—Male. Appendix with omentum in right inguinal sac. Inflammation and perforation of appendix. An abscess formed, followed by a fistula, from which came a very profuse discharge. It never healed, and the patient died twelve months after. It was thought to be a case of orchitis, until *post-mortem* examination revealed appendix. Recorded by Shaw (Trans. Path. Soc. Lond., 1846-48, vol. i. p. 270).

FOURTH SERIES.—*Appendix alone in the Hernial Sac, with the record that it was acutely strangulated.*

Case 14.—Female, aged 50. Appendix in (? right) inguinal sac. Strangulation was said to have occurred four days before operation. There was vomiting and complete constipation. The appendix was in a fit condition to be returned, and speedy recovery followed. Recorded by Court (Lancet, 1870, vol. ii. p. 401).

Case 15.—Male, aged 19. Appendix in right femoral sac. As a result of strangulation, nausea, vomiting, and constipation ensued. The appendix, on herniotomy, was left adherent in the sac, and recovery succeeded. Recorded by Wölfer (Archiv für klin. Chir., vol. xxi. p. 432).

Case 16.—Male, aged 66. Appendix in (? right) inguinal sac. It was said to be strangulated, but although there was local pain, there was neither vomiting nor constipation. Recorded by De Morgan (Trans. Path. Soc. Lond., vol. xxv. 1873-74, p. 107).

Case 17.—Male, aged 57. Appendix in right inguinal sac. Said to have been strangulated. There was no impulse of cough in the swelling; the pain was severe, but there was no vomiting, and the bowels were open freely. At the herniotomy, $2\frac{1}{2}$ inches of the appendix were cut away. Within the tube was found a small piece of bone. Recorded by C. R. Thompson. (Brit. Med. Jour., 1882, vol. ii. p. 599).

Case 18.—Female, aged 38. Appendix in right femoral sac. Strangulation was said to be present. Pain was great, and there was some vomiting, but the bowels acted well. Recovery occurred after operation. Recorded by Davies-Colley (Guy's Hospital Reports, 1884, vol. xxvii. p. 430).

Case 19.—Male, aged 5 months. Appendix in right inguinal sac, of congenital origin. It was said to have been strangulated.

The infant was very sick, but passed flatus. The skin over the scrotum was much reddened. On herniotomy, the appendix was found sloughing and perforated. A fistula remained, but ultimately there was complete recovery. Recorded by Durham (Guy's Hospital Reports, 1884, vol. xxvii. p. 435).

Case 20.—Female, aged 47. Appendix in right femoral sac. Said to have been strangulated. Vomiting was in evidence, but there was no constipation. On herniotomy, the appendix was found gangrenous. It was ligatured and removed with perfectly satisfactory results. Recorded by Clement Lucas (Guy's Hospital Reports, 1884, vol. xxvii. p. 436).

Case 21.—Male, aged 54. Appendix in right inguinal sac. In this case pain was marked; there was offensive vomiting, and the bowels did not act. Recorded by Pick (Lancet, 1880, vol. i. p. 801).

Case 22.—Female, aged 53. Appendix in right femoral sac. Said to have been strangulated. The patient vomited thrice. Herniotomy four days later. No definite peritoneal sac found. The appendix was found white, but thickened, and contained much mucus. It was ligatured and removed, and the patient made an excellent recovery. Recorded by Keetley (Med. Press and Circ., 1890, vol. i. p. 85).

Case 23.—Female, aged 67. Appendix in right femoral sac. Said to have been strangulated. There was nausea, but no vomiting. The sac was not opened, but the appendix felt and reduced. Recorded by Swasey (New York Med. Rec., 1881, xix. p. 706).

Case 24.—Female, aged 46. Appendix in right femoral sac. A sudden onset of symptoms was said to have occurred twelve hours before operation. Vomiting, but loose motions. Much flatus by mouth. The appendix was found ulcerated and perforated, and the sac much thickened. The appendix was removed. Recovery followed. Case under the care of Mr. Langton. (See St. Barth. Hosp. Female Surg. Reg., 1891, vol. iv., No. 92; also St. Barth. Hosp. Reports, 1891, p. 179).

Case 25.—Male, aged 6 weeks. Appendix in right inguinal sac, of congenital origin. Strangulation was said to have occurred. The appendix was removed on herniotomy, and the infant recovered well. Recorded by Bilton Pollard (Lancet, 1895, vol. i. p. 1114).

Case 26.—Male, aged 58. Appendix in *left* inguinal sac. Patient had noticed a hernia for eight months, which had always been reducible up to three days before he came under observation. At that time, after a rather violent movement, he was seized with intense local pain, and in addition vomited, and

had no action of the bowels. Herniotomy was performed and the sac opened. Fluid with fæcal odour was evacuated. After section of the constricting neck, the appendix was drawn down, and found to be eight inches in length. It had been tightly nipped at the junction of the middle with the lower third, and was gangrenous below the constriction, and perforated at the extremity. A ligature was applied near the cæcum and the appendix cut away. The patient made a good recovery. Recorded by Thiéry (Bull. de la Soc. Anat. de Paris, July 1892).

Case 27.—Female, aged 45. Appendix in (? right) femoral sac. Had been irreducible five days. Operation revealed appendix, which was said to be strangulated. It was removed, and the patient recovered. Recorded by Guinard (Med. Press and Circ., 1896, vol. ii. p. 604).

Case 28.—Appendix in right femoral sac. Said to have been strangulated; there was local pain and swelling. Recorded by Bayer (Centralb. für Chir., 1876, xxxi. p. 689).

Case 29.—Male, aged 63. Appendix in right femoral sac. Was said to be strangulated. There was vomiting, but the bowels acted. The appendix was returned to the abdomen, and death resulted. Recorded by Diffenbach.

LIST OF AUTHORS AND REFERENCES CONSULTED.

External Hernia of Appendix.

- ANNANDALE, *Lancet*, 1889, vol. i. p. 627.
 BAYER, *Centr. f. Chir.*, 1876, p. 689.
 BRIEGER, *Archiv f. Klin. Chir.*, 1893, Band 45, p. 892.
 BRITISH MEDICAL JOURNAL, 1882, vol. ii. p. 579.
 COURT, *Lancet*, 1870, vol. ii. p. 401.
 CRUVEILHIER, *Anat. Path.*, tom. ii. liv. 37.
 DAVIES-COLLEY, *Guy's Hospital Reports*, 1884, p. 430, &c.
 DE MORGAN, *Transactions of the Pathological Society, London*, vol. xxv. p. 107.
 DIFFENBACH.
 ECCLES, McADAM, *St. Bartholomew's Hospital Reports*, 1891, vol. xxvii. pp. 179, 180.
 GEISSLER, *Virchow's Jahresbericht*, vol. ii. p. 475.
 GUINARD, *Medical Press and Circular*, 1896, vol. ii. p. 604.
 GUY'S HOSPITAL MUSEUM, No. 1166.
 HABERSHON, "Diseases of the Abdomen," 4th edition, p. 445.
 HALL, *New York Medical Journal*, 1886, vol. i. p. 662.
 KEETLEY, *Medical Press and Circular*, 1890, vol. i. p. 85.
 KELYNACK, "The Pathology of the Vermiform Appendix," 1894.
 LANCET, 1880, vol. i. p. 801; 1889, vol. i. p. 627; 1895, vol. i. p. 1114.
 LONDON HOSPITAL MUSEUM, No. 1300.

- MEDICAL PRESS AND CIRCULAR, 1890, vol. i. p. 85 ; 1896, vol. ii. p. 604.
MONKS, Boston Medical and Surgical Journal, 1890, vol. i. p. 543.
PATHOLOGICAL SOCIETY OF LONDON, Transactions, vol. i. pp. 269, 270 ;
vol. x. p. 190 ; vol. xxv. p. 107.
PICK, Lancet, 1880, vol. i. p. 801.
POLLARD, Lancet, 1895, vol. i. p. 1114.
POLLOSON, Lyon Medical, May 21, 1893, p. 75.
ROYAL COLLEGE OF SURGEONS' MUSEUM, No. 2635.
SHAW, Transactions of the Pathological Society, London, vol. i. p. 270.
ST. BARTHOLOMEW'S HOSPITAL—
Medical Post-Mortem Register, 1895, p. 193.
Museum, No. 2111A.
Surgical Registers. Female—1891, vol. iv. No. 92 ; 1896, vol. v.
No. 1804. Male—1894, vol. v. No. 203.
Reports, vol. xxvii. pp. 179, 180.
SWASEY, New York Medical Record, 1881, p. 706.
THIÉRY, Bull. de la Soc. Anat. de Paris, July 1892.
THOMPSON, British Medical Journal, 1882, vol. ii. p. 579.
THURNAM, Transactions of the Pathological Society, London, vol. i. p. 269.
WÖLFER, Archiv f. Klin. Chir., vol. xxi. p. 432.
WOOD, Transactions of the Pathological Society, London, vol. x. p. 190.

Other cases are recorded by—

- BAYER, Prager Med. Woch., 1886.
BENNET, Virginia Medical Monthly, 1883-84, vol. x. p. 91.
BRUNNER, Beit. zur Klin. Chir., 1886 and 1889.
CABARET, Jour. de com. Med. Chir., 1842.
HABS, Deutsche Zeit. f. Chir., xxii. S. 323.
HAIDENTHALLER, Archiv f. Klin. Chir., xl. 1890.
JACKLE, Die Erkankung des Processus vermiformis in Bruchsack, Mar-
burg, 1888.
JAVANELLI, Archiv Gen. de Med., 1836, vol. x. p. 483.
KLEIN, Ueber die äusseren Brücke des Processus vermiformis cæci,
Geissen, 1868.
LOEBKER, Mittheilungen aus der Chir. Klin. zu Greifswald, 1884.
LUSCHKA, Archiv f. Path. Anat., 1853, vol. vi. p. 409.
PAGET, STEPHEN, Lancet, 1891, vol. i. p. 928.
THORIN, Hygiea, 1887, p. 762.
TREVES, "A System of Surgery," 1896, vol. ii. p. 723.
WETTE, Die Herniotomien im städtischen Hospital zu Aachen, 1889.
ZDEKAUR, Prager Woch., 1890, No. 27.
ZUCKERHANDL, Wien. Klin. Woch., 1891, vol. iv. p. 305.

References to Retro-Peritoneal Hernia of Vermiform Appendix.

- DUNN, Transactions of the Pathological Society, London, 1889, vol. xl.
p. 114.
LICHTENSTEIN, Cyclopædia of Medicine, vol. vii. p. 550.
LOCKWOOD, Transactions of the Pathological Society, London, 1889, vol. xl.
p. 115 ; 1890, vol. xli. p. 118.

ON THE OCCURRENCE OF RIGOR AND COLLAPSE IN TYPHOID FEVER.

BY

W. P. HERRINGHAM, M.D.

The following case will serve as a text for a few remarks upon rigors and collapse occurring in the course of typhoid fever.

Typhoid Fever—High Temperature at First—Slow and Irregular Lysis—Rigors during Lysis—Recovery.

Mary Anne H., æt. 33, married, was admitted under my care, while I was on duty for Dr. Hensley, on September 1, 1896 (Medical Register, Female, vol. iv. No. 180). She was admitted on the sixth day of an attack of typhoid fever.

She had had rheumatic fever five times. There was some family history of consumption, and she was suckling a baby three months old. At the apex of the heart the first sound was indistinct, but there was no definite murmur.

She had typical rose spots, a dry brown tongue, headache, and slight diarrhœa. The pulse was of good volume and strength, its rate about 96.

Of the course of the disease I need not say much. The temperature lay about 104° until the ninth day, and thence until the seventeenth day lay between 100.4° and 103.4° . On September 13 (18th day) she passed a clot in one stool, and a large blood-stained slough in another. There was a good deal of abdominal pain at the time, and the intestines could be seen moving considerably. She was given opium both by enema and by the mouth, and no further hæmorrhage occurred. She was at this time a good deal depressed in mind. From September 15 to September 17, the temperature was a little lower, for its highest point on the 15th was 102.8° , on the 16th 102.8° , and on the 17th 102° . There was now no diarrhœa, and the fever appeared to be yielding in the regular

manner. But on September 18 began the series of violent oscillations for which I record the case.

September 19.—Has had two rigors during the night, each lasting three-quarters of an hour. Copious sweats. Abdomen slightly full, but soft, and no pain or tenderness. No sign of thrombosis.

20th.—Another rigor, lasting three-quarters of an hour; began at 5 P.M. Profuse sweating.

22nd.—No more rigors; slight œdema of left leg. Appearance much improved.

25th.—œdema of left leg very slight. A little pain about the knee.

26th.—Another rigor in the night. Yesterday the abdomen was distended and uncomfortable.

30th.—A rigor again this morning. Unaltered improvement notwithstanding.

October 1.—A rigor at 6 P.M. Pain in the back always preceded the rigors by about ten minutes, and immediately she felt this she was given quinine, but without any preventive effect.

4th.—A very severe rigor yesterday evening, and another during the night. Quinine was similarly administered, but without effect.

That day the temperature fell steadily, and was never again above 100.4°. It reached a permanent normal on October 17, and the patient convalesced steadily.

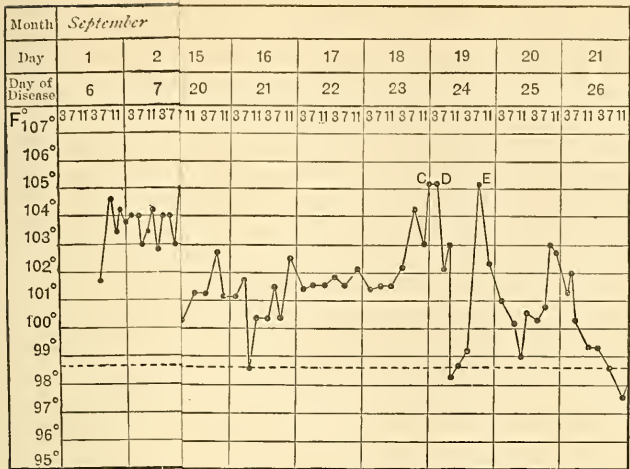
These chills resembled each other. Pain in the back was the first symptom: it lasted usually for five or ten minutes, then the patient became distressed and shivered, the hands and nose became livid, and the pulse very feeble. This condition lasted for from half to three-quarters of an hour, and gradually subsided. It was terrifying to the patient, and a cause of grave anxiety to those who witnessed it. It considerably exhausted her.

The spleen was not palpable on admission, and was not again noted until October 4, when it was enlarged to about two fingers' breadth below the ribs. This enlargement was recent, for the abdomen was frequently and carefully palpated.

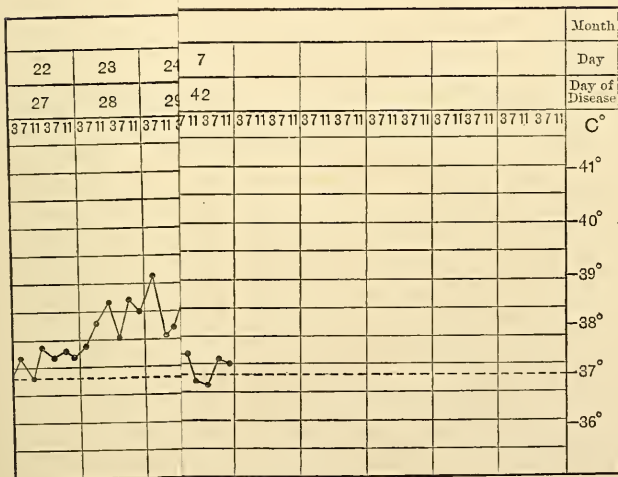
Quinine was given in five-grain doses directly the pain in the back began, but we were wholly unable to prevent the rigors by this means. There is no evidence that it had any effect when given after the rigor had begun.

The cessation of all rigors after October 4 was not due to treatment. Quinine was the only drug used in this case. On October 3 I ordered antipyrin to be used for the next rigor, but no occasion arose for it.

The rigor period ceased suddenly, without any more warning

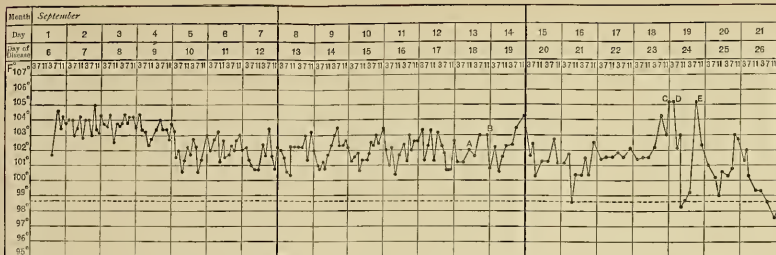


pl.
.

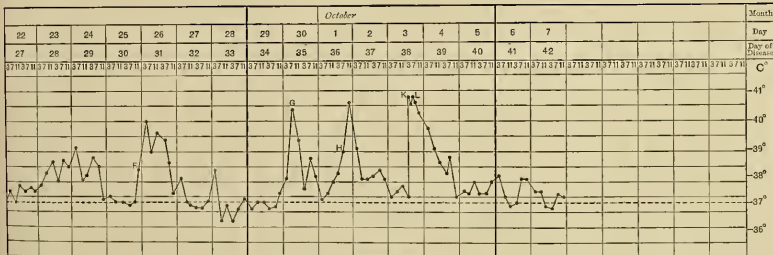


gr v. given at initial symptom.

To face



A Faced blood in stool. Tr. opt. in xx in excreta &c. B Faced large clough in stool.
 C Rigor 3 a.m. D Rigor 6 a.m. E Rigor 5 p.m. Quin. Sulph. gr. v. at 6 p.m. and 5 p.m.



F Rigor at beginning of pyrexia. G Rigor at beginning of pyrexia. H Rigor at beginning of pyrexia. Quin. Sulph. gr. v. given at initial symptom.
 K Rigor. Quin. Sulph. gr. v. given at initial symptom. L Rigor.

To face page 108.

than its onset had given. The rigors had not decreased in severity, nay, the last two were severer than the preceding. There had been no change in the patient's general condition. During this period the bowels had been rather loose, but not constantly nor greatly so. The pulse certainly became more rapid. Up to September 12 (17th day) it only rose to 100 on five occasions, though it was counted regularly six times a day; from September 12 to September 18 the rise in frequency is remarkable. I give the numbers as taken at three-hourly periods.

September 13.—88, 92, 96, 108, 100, 88.
September 14.—116, 112, 108, 90, 108, 96.
September 15.—100, 88, 100, 100, 100, 96.
September 16.—92, 96, 88, 96, 96, 104.
September 17.—84, 104, 100, 110, 112, 104.
September 18.—100, 100, 114, 116, 104, 104.

From September 19 onwards it became much more rapid.

September 19.—116, 136, 102, 96, 120, 120. Three rigors in this period.
September 20.—104, 104, 108, 108, 108, 100.
September 21.—102, 106, 88, 102, 96, 100.
September 22.—80, 112, 100, 112, 104, 100.
September 23.—104, 118, 120, 96, 132, 104.
September 24.—120, 108, 104, 120, 120, 96.
September 25.—84, 92, 108, 96, 96, 120.
September 26.—120, 128, 136, 120, 112, 96. Rigor at 3 A.M.
September 27.—80, 80, 104, 96, 108, 88.
September 28.—96, 80, 96, 100, 108, 96.
September 29.—72, 96, 108, 88, 92, 80.
September 30.—100, 120, 108, 96, 108, 88. Rigor at 7 A.M.
October 1.—84, 104, 104, 108, 144, 120.
October 2.—104, 100, 116, 116, 108, 100.
October 3.—88, 100, 100, 100, 140, 130. Rigors at 3 and 5 P.M.
October 4.—108, 104, 120, 108, 112, 100.

From this time the pulse became steadily slower. After October 11 it was never over 100.

No cause could be found for the rigors, for the slight œdema of the leg, which lasted but a day or two, came on after the rigors had begun.

During the years 1893, 1894, and 1895 there have been ten cases of rigor occurring during typhoid fever from causes other than perforation. There have been four in which a rigor accompanied perforation, but these need not be further discussed.

I will give the cases briefly, arranging them according to the period of the disease at which the first rigor in each case occurred. To avoid repetition, I will premise that in every case, besides typical symptoms, there was the characteristic rash of typhoid fever.

CASE I.—*Severe Typhoid—Rigor during Acme—Rigor during Lysis with abdominal pain—Death—Post-mortem, Suppurating Mesenteric Glands.*

Ada G., 10 (under Dr. Gee, Hope, 1894, No. 103), was admitted February 16, 1894, on the 10th day. She had a rigor on admission, lasting ten minutes, with T. 103.2°, P. 164, R. 60. The temperature lay between 102° and 104° until February 19, when it began to fall. She was very irritable and delirious, but had no severe diarrhœa. The temperature fell steadily till February 25. Mental state remained the same. On February 25, at 6.30 P.M., she complained of pain in the abdomen, and asked two or three times for the bed-pan, but passed nothing. At 6.45 she had a rigor lasting ten minutes, with T. 105.2°. During the night she passed two large solid motions and two looser. From that time until death on March 7, she constantly complained of abdominal pain, and lay with drawn-up legs. But there were no definite signs of peritonitis nor of perforation. She had a very rapid pulse, from 168 to 130, throughout.

Post-mortem, vol. xxi. 58 (Dr. Tooth).—Peritoneum quite natural. Peyer's patches deeply and extensively ulcerated from about a foot above the valve. All the abdominal lymphatics are large and hyperæmic, one contains pus. Other organs natural.

CASE II.—*Severe Typhoid—Rigors at Onset of Lysis, perhaps due to application of ice.*

Charlotte J., 17 (under Sir Dyce Duckworth, Med. Reg. 1895, Female, vol. iii., No. 180), was admitted June 20, 1895, on the (i) 7th day. Till June 25 (12th day) the temperature lay between 102° and 104°. From June 26 to July 1 (18th) it every day surpassed 104°. From this time it fell slowly. She had pneumonia in the left base on June 28, but she had an otherwise favourable attack. Her pulse was slow and of good volume. During the six days of acme she was cradled, ice-cradled,¹ and sponged. July 1, 7.40 P.M., while in an ice-cradle, she shivered. The ice and cradle were removed and she was wrapped in blankets. Temperature rose to 104.6° at 9 P.M. Blankets were removed and the ice-cradle put on again. Temperature fell to 103° at 10 P.M., but rose to 105° at 11 P.M., when she was sponged with ice-cold water. Temperature fell at once to 102.2°. At 1 A.M. on July 2, temperature was 105°. At 1.30 P.M. there was a slight rigor, the temperature being then 104.6°. During the rigor the pulse was scarcely perceptible, and remained irregular for a little time. At 2 A.M. it was of fair volume and regular. From this time temperature fell slowly. She continued very delirious for some time. July 19 she had thrombosis of the right femoral vein. She recovered.

¹ "Ice-cradling" is to hang ice-bags from a cradle inside the bed-clothes.

CASE III.—*Severe Typhoid—Collapse during Acme—Rigor during Lysis while passing stool.*

Annie E. W., 21 (under Dr. Gee, Hope, 1893, No. 197), was admitted September 2, 1893, on the 10th day. She presented the usual symptoms of a high typhoid fever (temperature usually 103° – 104.5° , with 4–6 loose motions daily), but took and slept well until September 7 (15th day). On that day, at 3 A.M., she had a sudden attack of collapse. The respiration became very rapid and shallow, and the pulse almost imperceptible. This lasted for a few minutes, after which both symptoms gradually gave way, the breathing became deeper and slower, and the pulse improved in volume and tension. Temperature at this time was 103.8° , its usual level, but it fell from that point until it reached 100.4° at 5 A.M., and 99.8° at 9 A.M. By 11 A.M. it had returned to 103.8° . No cause was found for the collapse.

Lysis began on September 18 (26th day). On September 22 the morning temperature was 99.8° . At 3.45 P.M., while using the bed-pan, she had a rigor lasting five minutes. After it her hands and feet were cold, the pulse running, rate about 150, the abdomen not distended, some tenderness over the spleen. The motion passed was small, loose, and contained no blood. The temperature at 3 P.M. was 100.6° , at 4 P.M. 102.6° , at 5.30 P.M. 103.6° , at 7 P.M. 103° , at 11 P.M. 101.8° . No cause could be discovered. This was the only rigor. Convalescence was uninterrupted thereafter.

The pulse in this case was usually about 120. But it was considerably lower on September 7. I give the rate taken at four-hourly periods:—

September 6.—120, 118, 120, 122, 128, 120.

September 7.—110,¹ 100, 110, 100, 100, 120.

September 8.—98, 100, 98, 100, 128, 136.

September 9.—132, 120, 114, 120, 120, 112.

After this time it rose to about 132, and so remained for several days after the temperature had fallen to normal on October 9 (47th day). During the evening after the rigor it reached 160.

CASE IV.—*Severe Typhoid—Slight Rigors during Acme, due to Antipyretics—Severe Rigors during Lysis, probably due to Thrombosis—Boils—Insanity.*

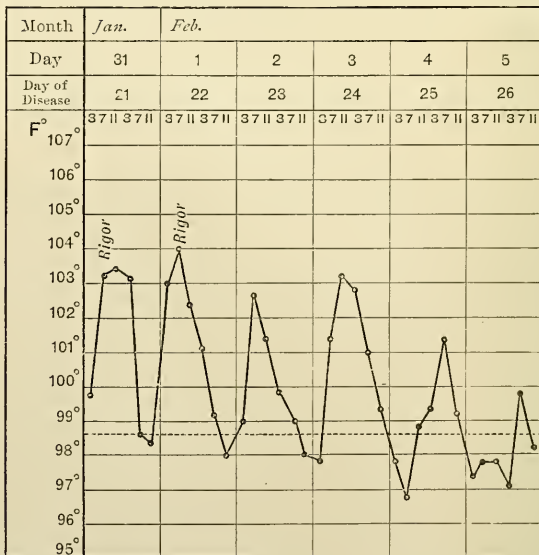
Richard H. B., 37 (under Dr. Church, Mark, 1893, No. 12), admitted November 18, 1892, on the 11th day. He was somewhat delirious, and had some diarrhoea, with a temperature which, rising from 101° – 102° , reached 104.2° on November 25 (18th day), and stayed at that evening level until November 29. On November 30 (23rd day) it began to fall. During the acme he had occasionally been treated, when the temperature was high, with phenacetin, antipyrin, and cold sponging. Once after phenacetin, while the temperature was falling, he had some shivering, and once he shivered after being sponged, though the temperature remained unaltered. On December 2 (the 26th day, the 3rd day of lysis) at 2 A.M. he had a rigor with a temperature of 106.6° ; it fell from this height to 103.6° at 4 A.M., when he had another rigor, without any fresh rise. That morning signs of thrombosis of the veins of the left leg were found. At 7 A.M. the temperature was 100.4° . At 8 A.M. he had another rigor, in which the temperature was 105.8° . At 3 P.M. it was 103° . At 7 P.M. a fourth rigor

¹ Here occurred the collapse.

occurred with a temperature of 107.6° , which fell by 11 P.M. to 101.8° , and to 100° by 3 A.M. on December 3. For the next three days it did not rise above 101° ; but it rose a little on December 6 and 7, on which days some small boils appeared on the back, and on December 6 he had two attacks of severe pain in the left lower scapular region. They were not associated with rise of temperature, were made worse by pressure, and could not be accounted for by anything heard in the chest. On December 11, a similar attack of pain was followed in half-an-hour by the rigor. On December 8, at 6 P.M., he had a fifth rigor with a temperature of 107° , falling to 98.4° by 4 A.M., and remaining moderate for December 9 and 10. On December 11 (34th day), again at 6 P.M., the sixth and last rigor occurred with a temperature of 105° . From this time the temperature fell slowly to the normal, which was reached on December 23 (46th day), and never rose again; and on the same day he began to have distinct delusions (*a*) of wealth; (*b*) that he had a third leg made of wood. On December 12, he was suspicious of the other patients. This insanity began to improve by December 20, and had disappeared by January 9, though he was still very excitable in mind, and liable to sudden quickening of the pulse. He recovered.

CASE V.—*Mild Typhoid—Rigors during Lysis—Cause unknown.*

Robert H. C., 43 (under Dr. Gee, Luke, 1894, No. 72), admitted on January 26, 1894, on the 15th day. For the next four days the temperature was quite low, usually about 100° ; but on January 31 (21st day) it



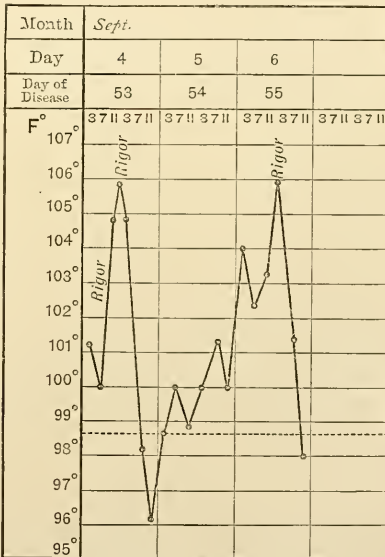
began to oscillate violently, and on January 31 and February 1, at 6.45 A.M., he had rigors. There was no enlargement of the spleen. No cause could be found for the pyrexia. On February 6, it fell to normal, and convalescence was uninterrupted.

CASE VI.—Moderate Typhoid—Rigor during Lysis—Cause unknown.

Albert H., 26 (under Dr. Gee, Luke, 1894, No. 194), admitted June 20, 1894, on the 18th day. For the first four days temperature was over 104°. On June 25 lysis began, and continued steadily until June 30 (28th day). On that day the temperature at 3 A.M. was 100.6°; at 4.30 A.M. a shivering fit occurred, which lasted till 5.10 A.M. During that time the temperature fell to 99°; but it rose again, and was at 7 A.M. 104°; at 11 A.M. 103.2°; at 3 P.M. 103.8°; at 7 P.M. 103.2°; at 11 P.M. 103°; from which point it fell by lysis to normal on July 6 (34th day). No cause could be found for the rigor. It interrupted the regular lysis for two days, but after that time the temperature fell as steadily as it had fallen before the interruption. No other bad symptoms.

CASE VII.—Moderate Typhoid—Relapse, followed by a long period of Rigors—Pelvic Abscess.

Ethel K., 23 (under Dr. Gee, Hope, 1893, No. 227), admitted July 21, 1893, on the 8th day. Primary fever mild. Lysis began on July 28 (15th day), and the temperature reached normal on August 7 (25th day). Two days later a relapse set in, of which the lysis began on August 17 (35th day). The temperature fell steadily for three days (the evening temperatures being August 16, 101.8°; 17th, 101.5°; 18th, 100.8°; 19th, 99.8°), but



on August 20 (38th day) it rose again, and from August 21 to September 10 inclusive, a period of twenty-one days, she had a succession of extremely severe rigors, combined with the utmost irregularity of temperature. Three days near the end of the period are a sample; yet, notwithstanding the extreme irregularity, it is plain that in the steadier periods between the

rigors the temperature oscillated about a gradually sinking mean. On September 10 she had the last rigor with a temperature of 105° , having had nine severe and a few slighter. On September 11 the temperature reached normal, and so remained.

This period of rigors has the appearance of a second relapse, with very irregular temperature, but there were no fresh spots, and no diarrhoea. On August 30 the left foot swelled slightly. This was probably connected with a tumour found shortly after in the iliac fossa, and diagnosed as a pelvic abscess. It burst into the bladder. The rigors might be ascribed to this cause, did they not precede by ten days any symptom that could be ascribed to it.

The rigors were accompanied by great collapse, and sometimes the collapse came on suddenly without the rigor.

She recovered well after this time.

CASE VIII.—*Mild Typhoid—Rigor in Post-febrile Period, probably due to Enema.*

Ethel P., 29 (under Dr. Gee, Med. Reg. 1895, Females, vol. ii. No. 175), admitted October 20, 1895, on the 7th day. She had a mild attack, and her temperature was normal on October 26 (13th day). On November 6 she had an enema in the evening. The temperature, which had been subnormal all day, rose at 11 P.M. to 98.6° ; at 3 A.M. (November 7) 102.8° ; at 7 A.M. 104.2° ; at 11 A.M. 103° ; at 3 P.M. 100.8° ; at 7 P.M. 100° ; at 11 P.M. 98.8° . Yet there was no pain, or any symptom of abdominal disease. On the 8th, at about 3 A.M., she had a rigor with a temperature of 105.4° , which fell to normal the same evening, and so remained. There was a little abdominal discomfort, no vomiting or pain, and no symptoms of thrombosis. She convalesced steadily.

CASE IX.—*Severe Typhoid—In Post-febrile Period one Rigor, and several sudden attacks of Pyrexia.*

James H., 15 (under Dr. Church, Mark, 1894, No. 25), admitted on December 8, 1893, on 15th day. Lysis began on December 18 (25th day), and the normal was reached on December 24. On January 7, at 11.45 A.M., he had a slight rigor, lasting five minutes. It was accompanied by intense headache and a temperature of 105.6° , which next day fell again to normal. On January 12 a sudden rise of temperature to 103.4° began at 11 A.M. and lasted to 3 P.M. The evening temperature was subnormal. A similar rise took place on January 15, the highest point reached being 104.2° at 7 A.M. There was no shivering in the last two attacks. He had a good deal of constipation throughout. He was discharged on January 31.

CASE X.—*Severe Typhoid—Recurrent Collapse during Lysis—Rigors in Post-febrile Period—Cause unknown.*

Emily B., 34 (under Dr. Hensley, Mary, 1894, No. 49), admitted January 8, 1894, on the 22nd day. For the first three days the temperature remained about 103° . On January 13 (27th day), at 4.30 P.M., she became blanched and collapsed, the temperature falling to 95.8° . Her extremities were fairly warm. This lasted about an hour. There was no

hæmorrhage nor abdominal symptoms. On January 15, at 8 P.M., and on January 16, at 9 P.M., were similar and equally unaccountable attacks. On January 18 (32nd day) the temperature reached normal and remained so for four days. On January 22, at 1 A.M., she had a rigor, was pale, livid, and collapsed, with a temperature of 103.8°. This lasted fifteen minutes. Later in the morning she passed a natural solid motion unconsciously, and remained lethargic for the rest of the day. On January 23, at 6.45 P.M., she had a rigor, which lasted fifteen minutes. With this there was no collapse. The temperature was 103° then, and rose to 103.8° by 11 P.M., but then fell again. On January 27 she had thrombosis of the left femoral vein. On February 12 she caught a pleurisy. Temperature fell finally to normal on February 19. She was very anæmic, the red cells being 47 per cent. on February 13; but this improved rapidly under iron. She had a pulse of about 120 at first, which fell about January 17 to 110, and thence by January 20 to 100. It became very rapid (130-140) with, but not before, the rigors.

From these cases it is plain that rigors may occur during the acme (Case I.), at the beginning of lysis (Case II.), during lysis (Cases III., IV., V., VI.), in the lysis of a relapse (Case VII.), or in the post-febrile period (Cases VIII., IX., X.).

They occur at all ages and in both sexes. Cases IV. and VI. probably, and Case V. certainly, were alcoholic men. None of the women lay under this suspicion. Ague is not mentioned in any one of them, and is so uncommon in England, and especially in London, that it may be disregarded.

There are some cases in which the exciting cause may be suggested with some probability. In Case I. the first shivering occurred on admission, and may have been due to the undressing and slight washing which such patients receive. For in Case IV. the reduction of temperature, whether by phenacetin or sponging, was accompanied by shivering, much as might happen in health, and in Case II. it seems as if the same cause were at work.

In others some abdominal stimulus seems to start a rigor. Thus in Case II. it came on while the patient was at stool. In Case I. it was closely preceded by abdominal pain, and in Case VIII. an enema, given in the post-febrile period, was followed by a rise of temperature, though the rigor did not occur till thirty hours later.

In Case IV. severe rigors accompanied the onset of thrombosis during lysis, and were repeated many times afterwards. The first chills are thus sufficiently explained, but no cause could be discovered for the latter. In Cases V. and VI. rigors occurred during a primary lysis. No anti-pyretics had been given, and no cause could be discovered for the rigors. In Case VII. the rigors occurred during the second lysis, the lysis of the relapse. Again, no cause could be discovered at the time when

the rigors began. It was not till nearly a fortnight later that the small pelvic abscess was palpable. It is possible that thrombosis in vessels which did not cause outward oedema may have caused these rigors, or that in the last case the suppuration may have begun when the rigors first set in. But there is, I think, little doubt that similar causes would not produce symptoms of such severity save under peculiar conditions, and the facts merit close examination.

Every one knows that often in typhoid fever lysis begins by a fall in the morning temperature alone, and that, generally speaking, the extreme daily temperatures are more widely apart in lysis than when the fever is at height. In the chart of my own case, the close character of the chart during acme contrasts well with its open appearance during lysis. In some cases this oscillation is very great, and extends through three or four degrees Fahrenheit, though the "rhythm" of morning fall and evening rise is preserved. In others, as in Case V., the rhythm is reversed, and the morning temperature is higher than that of evening; and lastly, there are many in which there is great fluctuation, and the rhythm is almost or entirely lost. In both health and disease the temperature is believed to be ruled by thermal centres in the brain, and such irregularity as this to which I have referred connotes some irregular action of these centres. They are not the only parts of the brain affected in typhoid; the tremor and delirium which are common in the acme, and may last long after that is past, the insanity which is by no means uncommon in the later periods, and of which a good instance is given in Case IV., are due to paralysis of the motor and of the mental centres. A similar affection of the thermal centres appears to me the best explanation of this irregularity of temperature. It is believed that in fever the thermolytic centre is paralysed by some poison, we know not what, due to the disease. When this is at its height, and the poison in full strength, the pyrexia is comparatively continuous. As the poison, undergoing the process common to all fevers, loses its strength, or is perhaps counteracted by some product of our own tissues, its effect becomes at first irregularly, and then steadily weaker. I am tempted to draw an analogy, which may seem fanciful, between the effect of typhoid upon the thermal centres, and the effect upon the motor centres of epilepsy. The tonic stage of the latter corresponds to the continuous high fever of the acme, the oscillations of the lysis to the clonic stage of epilepsy, when the stimulus that has produced the fit is passing off, and the sub-normal temperature frequently seen after lysis to the weakness left after the epileptic attack. It is true that if fever be due

to the inhibition of the thermolytic centre, the positions in the two cases are reversed, that in fever the thermolytic centre must be held to be at first paralysed, and at last over-active, instead of the reverse as in epilepsy. But our knowledge of the mechanism of either state is too imperfect for any exact conclusion, and all I wish is to suggest, that after the cessation of a morbid stimulus it is the natural tendency of nervous centres to act with great irregularity before they recover their former state of health.

Lastly, there is another class in which the rigors and vagaries of temperature occur as isolated phenomena in the post-febrile state. Cases VIII., IX., and X. are examples of this. In the first of these the symptoms have been already ascribed to the enema, and the fact that on the first night pyrexia alone resulted, while on the next a rigor took place also, is evidence that no real distinction can be drawn between the two rises of temperature. For the other two cases no cause could be discovered. This only means that typhoid patients have the unstable thermotaxia of children, and that slight causes, which do not affect the self-controlled adult brain of health, are sufficient to upset it when exhausted by typhoid. It may be that thrombosis of some small internal vein may sometimes be such a cause.

Let me now pass to the second phenomenon which heads this paper—collapse. By it I mean a rapid, shallow respiration, a rapid and very small pulse, pallor, and lividity. Often these symptoms occur, as in Case III. and Case X., with sudden fall of temperature, but often they accompany a rigor, and in some cases, *e.g.*, Case VII., they occur both with a rigor and alone. It is for these reasons that I consider them akin, and have placed them together on my title. I believe them, like rigors, to originate in the highest nervous centres, and to be similar in nature. They are not so frequent, at least not so frequently noted, as the others, and I have not sufficient material to classify them in detail.

Having shown how often and when rigors and collapse occur, and having suggested a theory of their causation, I will add a few words as to their significance in prognosis and as to their treatment. Briefly, I should say that they cannot be foreseen, that although they appear most serious, they do not affect the prognosis, and that no treatment is of any avail. We have already seen that there are no predisposing causes common to these ten patients. Nor is there any general resemblance in their fever likely to enable the occurrence of rigors to be foretold. Both severe and mild cases have them. The pulse

is no criterion. In Case I. the pulse was very rapid; in Case X. it was 140-120; in Case IV. it was about 120-110; in Case VI. it was rarely so high as 110; and in my own case it was rarely above 100 during the acme.

Secondly, that they do not affect the prognosis is shown by the fact that all but Case I. recovered, and that this patient was from the first evidently in great danger.

Thirdly, no treatment is of any avail. These attacks, being in their nature transitory, pass off when let alone, as well as when drugs are given.

In Case III. quinine was given in eight-grain doses; in Case VII. opium was given constantly for many days, both without effect. In my own case quinine was given before the rigor came on, but did not stop it, while anti-pyretics, such as anti-pyrim, antifebrin, phenacetin, and the like, have been directly accused by Osler¹ of causing the rigors. When once the rigor has begun, one knows with certainty that its duration is limited, and a careful examination of these records entirely forbids me to believe that rigors in which quinine, opium, or other anti-pyretic has been given, differ in the least from others in which they have not. For instance, in my own case, on September 19, two rigors occurred early in the morning and one in the evening. For the former nothing was given, in the last five grains of quinine. All three lasted exactly the same time, viz., three-quarters of an hour. For the collapse occurring at the same time, or with low temperature, stimulants and warmth are the natural treatment. Brandy by the mouth, and a hypodermic injection of strychnia, are usually, and reasonably, given. One may, however, be permitted to hope that, should accident prevent the taking of such precautions, the patient would yet recover within no very long time from his alarming state.

¹ See a very interesting paper on Chills in Typhoid Fever, Johns Hopkins Hospital Report, vol. v. p. 445.

THE RELATION OF GOUT AND RHEUMATISM
TO DUPUYTREN'S CONTRACTION OF
PALMAR FASCIA,

WITH RESULTS OF TREATMENT BY ADAMS' OPERATION ;

BEING A THESIS FOR THE M.B. DEGREE AT
CAMBRIDGE UNIVERSITY.

BY

CHARLES EDWARD HEDGES, M.B.

The deformity known as Dupuytren's contraction of the palmar fascia, noted by Sir Astley Cooper in 1820, and called by Boyer in 1821 "Chrispatura tendinum," was first accurately described and its pathological appearances pointed out by Dupuytren in 1831. In his lectures, 1831-32, he says: "It has been made necessarily depend on a rheumatismal, a gouty affection of tendons, external violence, fracture, on a 'morbific cause induced by metastasis,' such as follows inflammation of the sheaths of tendons, or on a species of ankylosis." It was Dupuytren who, by careful dissection, first proved the deformity to depend on a thickening and contraction of the palmar fascia. As to the cause, he believed it was wholly provoked by repeated injuries of the palmar fascia by pressure and friction from implements habitually used in different mechanical callings (*Gazette Medicale de Paris*, 1835, vol. iii. p. 481). The deformity does not appear to have interested English surgeons until Adams, in 1873, published his "Observations on Contraction of Fingers, commonly called Dupuytren's Contraction," in which he ascribed it partially to a local and

partially to a general constitutional cause. This constitutional condition was, he believed, gout or rheumatism, not so much the acute form of either as that which is called rheumatoid arthritis. A second edition appeared in 1892, expressing the same opinion as previously.

He bases his views of its constitutional origin upon (1) few cases of Dupuytren's contraction amongst the labouring classes, and amongst any particular class of mechanics; (2) in hospitals the majority of patients are butlers or indoor servants, who lead a sedentary life, and thus favour gout; (3) affection is of common occurrence in upper and middle classes of society, in most of which there is a gouty disposition; (4) frequent occurrence in left hand and both hands; (5) its hereditary character.

He says that up to the publication of his book in 1873, he had never seen a case in a woman. In the majority of his cases, which were derived mainly from patients in good circumstances, there were other manifestations of gout, and he was able to trace a well-marked family history of gout, although the patients may not have been afflicted with that disease. He quotes a case of Dupuytren's contraction in both hands associated with contraction of plantar fascia of both feet.

In two instances in which he operated, each patient had an acute attack of gout in the hands soon after the operation, although one of the patients had not previously shown any manifestation of it. The other had suffered from iritis, which he had been told by a physician was of a gouty nature.

About the same time, Cæsar Hawkins had a patient who also suffered from an acute attack of gout previous to the operation for division of the palmar fascia.

Adams says he has seen most cases amongst clergymen, barristers, medical men, and officers, the only condition common to a great majority of which was a disposition to gout co-existing with the finger contraction.

In 1875 Sir James Paget, in a lecture on the minor signs of gout in hands and feet, says: "A number of old people were seen with their fingers drawn down into their palms, especially the little and ring fingers, sometimes in one and sometimes in both hands. This condition is often characteristic of gout, but it must be clearly understood that in a certain number it was due entirely to occupation, and when the cases which were due to injury or occupation had been separated, that the rest were almost always significant of a gouty constitution." He points out the occurrence of abnormal development of fibrous tissue in other parts, as in the sheath of corpus cavernosum of penis, and in plantar fascia associated with Dupuytren's contraction.

In 1876 Madelung of Bonn states the cause to be absorption of the fat of the palm of the hand, which occurs in old people, thus more easily exposing the palmar fascia to pressure and injury. This leads to a chronic inflammatory process, thickening and subsequent contraction of the inflammatory tissue. This theory will not, however, explain those cases which occur before the age of 40, nor will it explain the cases in which the deformity occurs in both hands, and in those who do no manual labour. Nor will it explain those cases, which are not at all uncommon, in which the contraction commences several years after active life has ceased.

In 1877 Post of New York puts down the cause of Dupuytren's contraction to constant irritation of the integument setting up a chronic inflammation, which spreads to the fascia beneath.

In 1880 Fothergill had frequently noticed these contractions of palmar fascia in gouty subjects; and Gilbert Smith quotes, as an indication of its hereditary character, a family, three brothers of which, all the subject of gout, suffered from Dupuytren's contraction.

In 1881 Dr. Myrtle admits its heredity, but does not admit of gout or rheumatism as a cause, for the following reasons:— (1.) It is never met with amongst women, whereas gout is partial to the fair sex; (2.) many, and worst cases, cannot boast of a gouty progenitor, and never exhibited a symptom of gout or rheumatism; (3.) remedies for gout no good for this affliction. Against these three objections of Dr. Myrtle's must be urged:— (1.) That he is totally wrong in stating that it never occurs in women; it is rare in women, and so is gout rare in the fair sex; (2.) statistics show that most of the cases have either a gouty or rheumatic history; (3.) what remedies are there which are known to have any influence over the chronic thickening of the capsule and fibrous structures around joints affected with gout and rheumatoid arthritis?

In the same year Reeves published a paper in the *Lancet* contradicting Dr. Myrtle's statement about its occurrence in women, but at the same time stating that he did not believe in gout or rheumatism as a sole condition predisposing to the disease. He states, as result of his experience, several causes. (1.) Rheumatism and gout, (2.) injury and occupation, (3.) heredity, (4.) neurosis. He had three cases on whom he operated which were followed by an acute attack of gout.

In 1884, Abbé of New York read two papers before the New York Academy of Medicine, advancing a theory of neurotic origin of the disease; he entirely rejected the gouty

origin of it, and sought to establish the theory that it was of a reflex nervous origin, consequent on a traumatism. The sequence of events, he suggests, is as follows:—(1.) Slight traumatism of palm; (2.) spinal impression provoked by the peripheral impression; (3.) reflex influence to part originally injured, producing hyperæmia and new growth; (4.) through the tense contraction, a second series of reflex symptoms, such as neuralgias, &c., and a reflection of the trouble to the opposite side. He brings forward many cases in support of this theory, but such cases, if common in America, are rarely if ever met with in England. Both Adams and Keen state they have never seen the various painful neuralgias and neurotic affections he so graphically describes, and not in a single case I have examined did one complain of any “pain shooting up the arm and down the opposite side,” and the various other neuroses which he enumerated. Besides this, the patients will often tell you that the dimpling of the skin commenced almost simultaneously in both hands. Neither Keen nor Adams will allow of this nervous origin of Dupuytren’s contraction, except in so far as gout and rheumatism are possibly nervous in their remoter origin. Besides, if we are to regard the causation of the deformity as due to this reflex nervous influence, we should expect to see the disease of much greater frequency amongst the labouring classes, whereas we find most cases of Dupuytren’s contraction in those who do no hard manual labour.

By making this statement I am taking it for granted that Adams is correct in stating that the deformity is more common amongst patients who are well-to-do, and consequently have no need to do hard manual labour.

In about 2000 cases of infirmity patients over forty years of age, I found only 24 cases, which is about 1.2 per cent., and at least four-fifths of the cases occur after that age.

Sir Dyce Duckworth and Garrod also state that the disease is much more common amongst those who lead a sedentary life.

Abbé brings forward 10 cases to support his theory; in the first two the contraction started in the left hand, whereas the patients used their right hands in their trade of cloth-cutting; in his eight other cases there was distinct rheumatic history in three cases, and in two others finger contractions existed in members of the same family.

In 1884, Noble Smith read a paper before the Clinical Society, in which he denied gout and rheumatism as a cause, but allowed that there was some constitutional cause at work in addition to some local cause. He regarded the primary cause as some

chronic irritation reflexly causing contraction of the palmaris longus tendon, and subsequently affecting the palmar fascia, for he says that in almost all the cases which he examined he found the tendon stand out prominently from the others at the bend of the wrist; although I carefully looked out for this prominence, I failed to find it so, even in one case.

He also states farther on, that out of 700 patients he examined in some infirmary, he found 70 cases of Dupuytren's contraction. It seems almost impossible to believe this, as out of 2000 patients whom I examined in four different infirmaries, I was only able to find 24 cases, and this including those with only the primary dimpling of the skin. Is it possible that he included several cases of spastic contraction of the tendons in cases of paralysis, of which there were several instances? I also think his percentage of females attacked is far too high, 1 to 4 males, as Keen only gives it as 1 in 15 males out of 187 cases. My own statistics will give it at about 1 to 8 out of 64 cases.

Professor Humphry, speaking on the discussion of this paper of Noble Smith's, suggested that in causation the truth probably lay between the two opinions of a local and constitutional cause. He pointed out that pressure on palm of hand fell more strongly on ring-finger, and hence the reason why this was most often primarily affected; he also pointed out the limited extension of this digit alone.

In 1885, Lockwood read a paper before the Pathological Society "On Contraction of the Digital and Palmar Fascia." He brought forward three cases. (1.) A girl, aged 21, who had had her finger contracted as long as she could remember. Having had occasion to amputate the finger, he found the fascia to be thickened and shortened, but otherwise normal. He says the case was probably one of heredity. (2.) Male, aged 21. No occupation. Six other members of same family similarly affected. Lockwood says he has recently seen other cases which strongly support the view of heredity. (3.) A male, aged 45. On dissection of the hand affected with Dupuytren's contraction, urate of soda was found in the thickened bands of fascia. Although there was no clinical history of the case, it can scarcely be doubted that the thickening and contraction was due to inflammation set up by the urate of soda. At the end of his paper he says: "Without doubt an enumeration of the causes of contraction of the digital and palmar fascia, putting aside the cases of congenital shortening, would be merely a recital of the various affections which are capable of causing chronic inflammation."

In 1882, Keen of New York read two papers on Dupuytren's contraction, which were published in the Philadelphia Medical Times of 1882. He collected 253 cases, including the 70 of Noble Smith already referred to.

Sex affected—1 female to 15 males.

Occupation of 123 cases in which it was noted—74 non-manual; 49 manual.

Hands affected of 184 cases. Both 103 times; right 58 times; left 23 times. Right hand thus involved 161 times; left hand, 126 times.

Out of 214 cases in which it was noted which finger was affected, in only 11 was the thumb affected, and in 24 the forefinger. The ring and little finger bore the brunt of the affection.

Heredity.—Out of 198 cases there were 50 of heredity. Of 95 cases, in 64 distinct personal or hereditary history of gout; in 31 it was excluded.

Dr. Keen maintains the view of a constitutional origin, and this he states to be, as a rule, gout or rheumatism. He says the patients are rarely free from the "minor manifestations of gout," as Sir James Paget calls them. He has seen it follow on acute rheumatism, although Adams excludes this as a cause of the affection.

In 1885 Stevenson quotes a case in which there is a family history of Dupuytren's contraction for three generations with no history of gout.

In 1891, in a series of lectures at the Royal College of Surgeons, William Anderson divides cases of Dupuytren's contraction into two classes. (1.) True Dupuytren's contraction, with no traumatic history, and a tendency to multiplicity of lesion. (2.) Those cases which are the result of a wound and confined to the part in direct relation to the injury.

In his statistics we find the following facts:—

Hand affected.—Bilateral in 24 cases out of 39. Right hand affected in 10 cases out of 39. Left hand affected in 5 cases out of 39.

Of eight patients, six of whom were women, the band was purely palmar, with no contraction of the fingers. In only one case was it associated with disease of the plantar fascia. He points out that the seat of the initial lesion is situated at a spot where the finger-nails come in contact with the palm of the hand.

Out of 2600 adults, 33—*i.e.* 1.27 per cent.—were found suffering from various stages of this affection. Out of 800 children under 15, none were affected.

Sex.—Cases of any degree of severity are rare in women. Of 39 cases, 25 were in men and 14 in women, but in only 8 of these was there any contraction of the fingers.

Age of onset.—Most cases of non-traumatic form begin after fifty years of age.

He does not believe in the influence of occupation as a cause of the disease; he states that it is very rarely found amongst soldiers and sailors. Shoemakers are said to suffer more than others, but this is not borne out by statistics. The disease is more frequently seen in those not employed in manual labour; perhaps the thickening of the skin may in some way prevent the underlying fascia from irritation.

Constitutional condition of his 39 patients:—1 gout; 1 acute rheumatism; 3 rheumatoid arthritis; 6 chronic rheumatism; gouty inheritance in 3 other cases.

Anderson says his own conclusions are based upon patients in hospitals, and hence the remarkable absence of gout or neurotic disposition.

Race and climate.—Very rare in Eastern countries, *cf.* Japan and India and Central Asia.

Inheritance.—Unquestionably a strong predisposition to heredity in the disease.

Cause.—Disease is of no more than average frequency in certain employments in which men are peculiarly and constantly liable to palmar friction. Some source of irritation must be present, and it has been suggested that this may be a gouty deposit (Lockwood). This experience is exceptional, as the majority of patients in this country are not, and have not been, subject to gout or rheumatism.

Anderson says the changes are more suggestive of chronic rheumatism than gout; but even the probability of this origin is not supported by observed facts. He believes strongly in the agency of a specific micro-organism which gains access to the palm of the hand by slight traumatism of the epidermis with the finger-nails.

I don't think any one attached any importance whatever to Anderson's remarks about the causation of the disease being due to a specific micro-organism; the only evidence he brought forward was the growth of a yellow nodule on agar, liquefying the medium in which it was placed and the tube being odourless; sections of it stained with fuchsin and Gram's method showed no organism under $\frac{1}{12}$ immersion. He finishes by saying that he hoped to publish this and other experiments *in extenso* within a short time, but since that time no further statements upon the subject have appeared substantiating his theory.

In Hutchinson's Archives appear some valuable cases in support of the hereditary and gouty origin of the disease. In 1891, vol. ii. p. 52, he points out several cases in support of its hereditary character and its association with fibrous induration and contraction of other parts of the body, such as the penis and plantar fascia. He quotes a case of Gaubius:—A man had contraction of his little finger, caused by a tense band of fascia going to that digit. The eldest of his two sons, on attaining to the same age as that at which the contraction commenced in his father's case, became similarly afflicted. The younger brother became similarly affected at the same age in spite of preventative measures. Hutchinson says we have here a good example of what is not unfrequently seen in cases of finger contractions; the condition of health, or perhaps of tissue, giving tendency to it, is unquestionably sometimes hereditary, although I think but to a slight degree. It is possible that it is connected with an arthritic diathesis.

On page 79 of same volume he quotes a case of Dupuytren's contraction of the little finger in both hands associated with hard fibroid induration and thickening of the plantar fascia. The patient had suffered from chronic rheumatism and his left shoulder was considerably stiffened. Hutchinson says he did not admit a history of gout, but looked a likely subject for it.

In Archives for 1894, p. 176. A male, in which the disease was strictly limited to little finger of both hands, for he could straighten ring-finger completely. The contraction was caused by a strong band of fascia from ulnar border of finger, which passed from its base to its tip. There was a family history of gout, and patient himself had often suffered from that disease. No history of heredity or local cause.

In Archives for 1894, p. 333. Three cases of curved penis in association with Dupuytren's contraction, two of which cases are of rheumatic and one of gouty stock. There was no induration or contraction detected in any way, but on erection the penis became bent with its concavity upwards.

Van Buren in 1888, in his Diseases of the Genito-urinary Organs, describes a circumscribed induration of the erectile tissue of the penis, sometimes occurring in erectile tissue, at other times in fibrous envelope of the corpora cavernosa: it occurs after middle life. Although many patients are noted to have gout or rheumatism, he says that the patients are not uniformly subject to any diathetic disease. He also points out the association of this condition of the penis with the condition known as Dupuytren's contraction.

Kirby in 1849 put down all such cases as of gouty origin.

Amongst my 60 cases will be found two cases of fibroid induration of portions of penis. In one patient, aged 57, a printer, who had suffered from Dupuytren's contraction for some time, and both hands were affected, there was a well-marked history of gout. Along the dorsum of the penis there extended a hard fibroid mass from just behind glans to near its root. On erection of the penis its point was directed against the abdominal wall.

In the second case, that of a bootmaker aged 59, both hands were affected with Dupuytren's contraction. There was a well-marked family history and personal history of gout. Tophi in ears, there were marked fibroid induration of the sheath of corpus spongiosum, causing penis to be bent upwards on erection.

I have also quoted the observations of Sir James Paget in the earlier part of this paper with regard to the occurrence of this phenomenon. The association of the two conditions, and the marked occurrence of one of them in association with gout, makes it very probable that this disease plays not an unimportant part in being a factor in the causation of the other.

Dr. Archibald Garrod, writing in *St. Bartholomew's Hospital Reports* for 1893, reports three cases characterised by the presence of nodules or cushions of fibrous tissue upon the finger-joints; in one of them it was associated with Dupuytren's contraction of palmar fascia.

Case I.—Male, aged 20, clerk, suffered from dyspepsia. He came of a markedly gouty stock, his paternal grandfather being a martyr to the disease, and his father had Dupuytren's contraction of the fingers. His mother was said to be somewhat rheumatic. Nodules about half the size of a hazel-nut projected from the dorsal aspect of several of the joints of the fingers of both hands; skin over them was freely movable. The lumps were first noticed at the age of 13, and they have steadily increased in size. The most remarkable point about the case is that an elder and younger brother of the patient are said to suffer from similar deformities.

Case II.—A woman, aged 50, with no personal or family history of gout, but with typical Heberden's nodes upon the terminal joint of her right index finger. There were no signs of rheumatoid arthritis in any other joint. The nodules were similar in character to those in the first case. Some of them had existed at least thirty years. No complaint of pain made. She ascribes their appearance to resting upon the affected joints whilst scrubbing floors.

Case III.—Male, aged 43. No personal or family history of

gout. No other member of his family was similarly affected. First nodule appeared at age of 13; he attributes its development to scraping his hands against the wall when playing games at school. It is interesting to note that upon the palm of the left hand, beneath the ring and little fingers, there was an induration and some puckering of the skin, indicating the commencement of Dupuytren's contraction. As in the first case, the occupation of the patient involved much writing.

The remarkable family history of the first patient, and the occurrence of the nodules in several members of his family, suggests a constitutional origin for them, and lends some colour to the idea that they may be connected with a gouty tendency; but, on the other hand, no one of the three patients had suffered from acute gout. It is further interesting to note the coincidence in one case of an early stage of Dupuytren's contraction of the fingers, and the history of the same affection in the father of the first patient. The influence of injury in determining their formation was in some instances well marked.

I consider the pathology and causation of these growths as analogous to the growth and contraction of the palmar fascia in Dupuytren's disease, and as a further proof of the tendency to the formation of fibrous tissue in abnormal abundance in situations exposed to slight irritation in persons of a certain constitutional state. Whether this state is always a tendency to gout or rheumatism I am unable to state; but that such is very often the case I feel almost confident.

Summary of Statistics of Sixty-four Cases collected by myself at four Infirmaries and one Hospital.

Age stated in 63 cases—

60 years and over,	22 cases.
Between 50 and 60,	17 „
„ 40 and 50,	11 „
„ 30 and 40,	11 „
Under 30	. . . 2 „

Thus 50 cases began after 40 years of age, and 61 cases began after 30 years of age.

Sex stated in 64 cases—

57 males—7 females.

In all the female cases I saw the condition was slightly marked, and only bilateral in one case; in one instance it was probably congenital.

Hand affected in 62 cases—

Both hands affected in 24 cases.			
Right hand	„	20	„
Left	„	18	„

This makes the right hand affected 44 times, and the left hand 42 times. Thus the right hand is only affected in two more cases than the left in 62 cases.

In nearly all the cases of which I could get any history of its origin, it commenced in either the ring or little finger. Hutchinson quotes one case where it commenced in the middle finger of left hand, and affected that finger alone.

Family or Personal History, 60 cases—

In 39 cases	history of gout or rheumatism.
In 3 „	„ heredity.
In 7 „	„ traumatism.
In 12 „	no history of gout, and in 5 of these cases I could not ascertain any family history.

Of the 12 cases in which there was no history of gout or rheumatism, four had suffered from undoubted syphilis, three were suffering from phthisis, one had cancer of œsophagus.

Thus of 53 cases of true Dupuytren's contraction there is a distinct history of gout, rheumatism, or heredity in 42 cases.

No particular class of occupation is affected more than another, except perhaps in the case of painters and printers, of which there are six instances. This may be significant, as in both instances their occupation puts them much in contact with lead, which, as one knows, is very apt to bring out gout where one is predisposed to it. Of bootmakers, who are popularly supposed to be frequently affected, there is only one solitary example, he being one of the cases with fibroid induration along the dorsum of penis.

Frequency of Occurrence.—Of 2000 patients examined at Kensington, Greenwich, and Marylebone Infirmaries, most of which were over 40 years of age, I only discovered 24 cases, *i.e.*, about 1.2 per cent.

Now, with regard to the various theories which have been set forth to explain the deformity—

1st, That of Dupuytren himself, who stated it to be solely of traumatic origin, I don't think it possible to hold this view, for the following reasons:—(a.) Its frequent occurrence in both hands. (b.) Left hand almost as often affected as the right. (c.) Its more frequent occurrence in the wealthier classes, who have to do no manual labour. (d.) Its frequent development after the period of active life has finished. (e.)

If it were solely of traumatic origin, we should expect it to affect one class of mechanics more than another.

As regards the theory of Abbé, I have partially discussed this in the early part of this paper, and can only add now that only in one sense can I admit of its being nervous in origin, and that is in the same sense in which Charcot's disease and rheumatoid arthritis are said to be due to lesions of the trophic nerves going to the part affected, and are thus said to be nervous in their origin; or in the same sense as the formation of urate of soda in the body is said to be controlled by the central nervous system.

I have already discussed Anderson's theory of a micro-organism being the cause, and also the theory of reflex contraction of the palmaris longus of Noble Smith.

Now to what do the statistics and various authorities which I have quoted lead one to believe to be the cause of this affection? Firstly, that the cases must be divided into two classes:—(1.) True Dupuytren's contraction, in which there is no history of traumatism. (2.) False Dupuytren's contraction, in which there is a distinct traumatic history. In this class of cases it is as common to see the disease in childhood as in old age. The seat of the initial lesion is single, and the affection is confined to the injured hand, and even finger. The contraction in these cases progresses very rapidly to a certain point, and then ceases to get worse.

Of course there is always the difficulty in saying how much of the deformity is due to the traumatism, and how much to the predisposing cause. It is just the same in gout and rheumatism as in tuberculosis; a slight injury to the knee may be just the exciting cause required to set up acute tubercular arthritis in that joint, just as a slight injury may set up acute gout or a gouty inflammation in one predisposed to the disease.

Now with regard to the causation of true Dupuytren's contraction, I believe it to be a constitutional disease for the various reasons:—(1.) Results of statistics. (2.) Marked heredity. (3.) Age of its onset. (4.) Occurrence in women, and yet its rarity in the sex, who are, as a rule, exempt from hard labour and gout. (5.) Occurrence more often in men exempt from manual labour. (6.) Its involving of left hand so frequently, and often before, or even without right. (7.) Involvement of both hands, more often than one alone, point to a general cause. (8.) Occasional appearance as a congenital disease. (9.) Analogy to contraction of plantar fascia and other fibrous structures. I also believe that there is more than one constitutional diathesis which predisposes to the deformity, to enumerate which would be to enumerate all the causes of chronic

inflammation. But that gout and rheumatism play a prominent part in its causation I do not think any one can deny from the statistics and other evidence I have brought forward.

I regard the thickening of the palmar fascia as in every sense analogous to the thickening of the fibrous tissue and capsule around joints affected with osteo-arthritis and gout. It is remarkable to note the many cases of rheumatoid arthritis one meets with in connection with Dupuytren's contraction.

No one can but admit that there is a great tendency to heredity in this deformity after the various cases I have quoted earlier in this paper. One must also take into account the five or six cases of Dupuytren's contraction I have shown associated with fibroid thickening of other parts of the body, which are almost always associated with a gouty tendency, and in many cases with a personal history of acute attacks of gout. Paget even goes so far as to say Dupuytren's contraction of the palmar fascia is one of the many manifestations of gout.

The age of onset is also an important point, as, like gout, it comes on, as a rule, after 40, and often after the period of active life has ceased. The disease rarely attacks women, *i.e.*, it is rare in the sex who are, as a rule, exempt from hard labour and gout.

Adams excludes acute rheumatism from amongst its causation, but Keen has frequently seen it follow that disease; and I have the notes of its occurrence in three or four cases apart from gout.

It is also worthy of note that in four cases in which there was no history of gout there was a distinct history of syphilis; and also in some of the cases with a history of gout or rheumatism there was a specific history as well. In such cases would it not be possible that tertiary syphilis played a prominent part in the causation of the deformity? for it is a well-known fact that the manifestations of tertiary syphilis tend to be in the formation of fibrous tissue. Of course, I have had no personal experience of the occurrence of Dupuytren's contraction in the wealthier classes; but from information I have derived from various sources, there is in most cases a personal or hereditary history of gout.

Various opinions are held as to the result of operation in cases of Dupuytren's contraction, some surgeons alleging that the operation is in no way beneficial, and that the deformity returns in an aggravated form in a short time, others that the operation is a complete success. I thought it would be interesting to find out the result of cases which had been operated on for from two to five years ago.

I collected eighteen cases which had been operated on at the Hospital, and visited the addresses at which the patients lived

at the time of the operation. Unfortunately, I could only find eight out of the eighteen cases. I have reported fully upon the result of the operation, its success or failure. The operation performed in every case was the subcutaneous method of Adams.

The facts to be gathered from these cases are—(1.) That in all the cases the patient's condition was improved, and in two or three cases to a marked degree. (2.) The great tendency to recontraction of the finger. (3.) There is some danger of getting the fingers stiff, with no power of flexion.

One would imagine that, were the patients to take greater care, and wear a splint for some time at night after leaving it off in the daytime, better results would be obtained. The operation is much more successful amongst the upper classes, who are able to take greater pains and spare more time in having their deformity attended to, and are not obliged, directly the splints are removed, to go to hard work and use their hands in a similar way as before the operation.

I have no experience of the results of the operation as performed by Reeves at the Orthopædic Hospital; but from all accounts, and from published statistics, Adams' subcutaneous operation appears to be the more successful of the two.¹

In conclusion, I must thank Mr. Keats of the Greenwich Infirmary, Mr. Lunn of the Kensington Infirmary, Mr. Gibbes of the Marylebone Infirmary, and Mr. Sansom of the Lambeth Infirmary for their kindness in allowing me to examine the patients at the various institutions.

Reports of Nine Cases operated on at St. Bartholomew's Hospital.

1. John M., aged 59, bootmaker, admitted October 20, 1893, complaining of contraction of fingers, which prevents him from earning his livelihood.

Condition before operation.—All fingers of both hands are

¹ There are two points brought out by the results of operation which may be useful. (1.) The earlier the operation is done, the better the result that will be obtained, as when there is a great degree of contraction there is such a degree of force required to get the fingers extended, and subsequently always great loss of power in the flexion of the digits operated on. This appears to be sometimes due to injury to the joints and subsequent fibrous ankylosis, and sometimes due to loss of power in the flexor tendons themselves, as the fingers can easily be flexed by passive movements. Besides, there must ensue some changes in the ligaments and soft structures about the joint; these will become shortened or extended as may be required to adapt themselves to the altered condition of the part, and consequently these will have to be divided before the finger can be straightened. (2.) I think gradual extension of the digits is much more efficacious than forcible and rapid extension in these bad cases, and there is much less danger of splitting up the skin and leaving an ugly wound to granulate up, which not only prevents your applying gradual extension at the time, but also, by the cicatrisation of the scar tissue, leaves the patient in a worse condition than before the operation.

more or less flexed, especially right ring-finger and left little finger; the tips of these two almost touch the palm of hand. Operation on October 28, 1893, by Mr. C. B. Lockwood. Multiple punctures were made between skin and fascia, where they were not very adherent to one another, and the tense bands divided in many places both in the palm and fingers. All the fingers could be then straightened with the exception of right ring-finger, which could not be straightened on account of articular changes. Malleable iron back splints put on in a semi-flexed position, and straightened out day by day. On December 1, 1893, patient left the Hospital; could use all fingers well except right ring-finger, which is over-extended and stiff at the terminal joint.

On May 15, 1895, about one and a half years after the operation, I went to see the patient, and found the following condition:—Patient able to follow his employment perfectly well; no pain or discomfort. He will tell you the operation did him a "lot of good." The right ring-finger is bent at an angle of 90° at the first inter-phalangeal joint, and a dense cord of fibrous tissue can be felt running up to it. Left little finger almost straight; other fingers straight; a tense band of tissue can be felt running from the base of right index finger to the base of index finger.

He wore the back splint for six weeks after the operation.

He says the ring-finger of right hand is gradually getting more flexed.

Condition of penis same as when in the Hospital.

2. J. C., male, 35, iron-fitter, admitted on March 28, 1893, complaining of "bent fingers" of both hands.

Condition previous to operation by Mr. Langton.—Both little fingers flexed at metacarpo-phalangeal and first phalangeal joints, and tips almost touch the palms. Ring-fingers are semi-flexed at same joints, the middle fingers slightly affected. This condition of his fingers prevented him from following his employment.

Operation on April 4, 1893.—The palmar fascia of both hands was divided in numerous situations in both hands with an Adams fascia knife, the knife being passed between the skin and fascia, and then by cutting downwards the bands were divided; both hands were bandaged firmly to a straight back splint.

On April 4 patient was discharged, still wearing his splints. Condition much improved. Little finger of left hand only one not quite straight.

On March 28, 1893, patient writes to me, as a result of inquiries, as he lives far away in the country.

He wore splints two months after operation. Fingers of both hands are better and straighter. He is well able to follow his employment. The fingers are somewhat contracted and are gradually becoming more so. He wears no splints at the present time.

3. S. L., male, 32, letterpress printer, admitted March 21, 1892, complaining of bent fingers, and in consequence being unable to properly follow his employment.

Condition of hand before operation.—The ring and little fingers of the right hand in a state of forcible flexion; the other fingers of both hands are normal.

Operation on March 30, 1892, by Mr. Willett.—Multiple subcutaneous incisions by a small tenotomy knife; in all, nine punctures made between the skin and fascia, and fascia divided by cutting downwards; the fingers were then at once forcibly straightened and bandaged to a hand splint. Splint was removed on April 20, and left off as the skin over the outer side of metacarpal bone, on outer aspect of its palmar surface and on inner side of little finger, had sloughed. The notes say patient left the Hospital improved.

On May 16, 1895, three years after the operation, the state of the hand is as follows:—Both fingers are quite straight; the tense band can still be felt running up to both the ring and little finger of right hand. Patient is unable to flex these two fingers at inter-phalangeal joints, nor can they be forcibly bent to any great degree; the joints are somewhat flattened and thickened. Flexion at the metacarpo-phalangeal joints is perfect. On account of the inability to flex the two joints I have mentioned, he was unable to further follow his employment. So although in his case the fingers were straightened, the fingers became practically useless to him in his occupation. The grasp of the thumb, index, and middle finger is good.

4. E. J. M., 53, porter, was admitted into Hospital on March 16, 1892, suffering from a contracted left ring-finger. It did not much interfere with his work.

Condition of hand before operation.—The ring-finger was bent chiefly at metacarpo-phalangeal joint, so that tip of this finger was bent to within one inch of palm, and little finger also somewhat flexed; all other fingers normal.

Operation on March 18, 1892, by Mr. Thomas Smith.—The contracted band of fascia was divided subcutaneously in several places with a tenotomy knife, finger partially straightened and bandaged on to a back splint, which the patient wore for two weeks after the operation.

On May 13, 1895, more than three years after the operation, I find him a cripple with gout, scarcely able to get about.

The condition of the hand is improved, the second phalanx being bent at an angle of 90° with the proximal phalanx; flexion good and powerful. The patient says the operation improved the condition and usefulness of his hand, but states that the finger is gradually getting more bent; the palm of the hand is much indurated.

5. J. F. W., 56, upholsterer, was admitted into Hospital on October 21, 1892, suffering from Dupuytren's contraction of both hands.

Condition of left hand.—Patient is quite unable to extend fully his fingers or thumb; he has a good grasp if anything can be got into his hand; he is quite unable to pick anything up, and thus cannot feed himself. The left hand is the worse of the two; third and fourth fingers are flexed to such an extent that they all but touch the palm.

Operation by Mr. Walsham, October 26, 1892.—Left hand only operated on. The fascia was divided by many subcutaneous punctures in palm and along fingers and thumb; the fingers could be brought out much straighter, but were still considerably flexed; the skin broke down in one place over middle finger. The hand was dressed with salalembroth gauze and bandaged tightly on a back splint.

Patient left the Hospital with index finger quite straight, and other fingers gradually becoming more so. The note says patient left the Hospital much improved.

On May 14, 1895, I saw the patient, and the condition of his hands was as follows:—All the fingers of the right hand were so flexed that they touched the palm of hand, which in consequence was ulcerated. The thumb is bent over the fingers so as to keep them down. In the left hand the fingers are almost straight, but there is very little power in the grasp, and fingers cannot be well flexed; a dense band of fascia can be seen extending from base of index finger to base of first phalanx of thumb, and binding them tightly together. Patient says his condition is somewhat improved, as he can feed himself and do little jobs, as he has considerable power in the thumb and little fingers.

6. W. C., 43, barman, admitted into Hospital on September 12, 1891, suffering from contraction of palmar fascia in both hands.

Condition of hands before operation.—Right hand—little and ring fingers are much flexed and cannot be extended; middle finger affected to a less degree.

Left hand—corresponding fingers of left hand affected to a less degree.

Operation on right hand by Mr. Walsham on September 15, 1891.—Several punctures were made with a tenotome, in some cases passed between the skin and fascia, and edge directed downwards; in others, the knife was passed beneath the fascia and cut upwards. Fourth and fifth fingers were then brought into good position and extended on a back-splint. Discharged wearing splint.

On May 10, 1895, I saw the patient, and the condition of his hands was as follows:—Left hand—the three inner fingers are flexed on the metacarpo-phalangeal and first phalangeal joints in such a way that their tips are within half an inch of palm of hand; tense bands of fascia can be seen running up to them and binding them down.

In the right hand, which was the one operated on, and was by far the worse of the two at that time, the ring and little fingers are almost straight, but there is great loss of power in these two fingers, and considerable difficulty in flexing them; says the operation did him some good, as he thinks his hand would soon have closed altogether. He now wants Mr. Walsham to operate on the left hand, as it is practically useless to him. He wore the splint eight weeks after operation.

7. H. S., male, 33, costumier, admitted into Hospital on October 21, 1891, suffering from “bent” fingers.

Condition of hand before operation.—Fifth finger of left hand bent so as nearly to touch the palm. Fourth finger of same hand bent to a less degree. Thumb also slightly affected.

Operation by Mr. Walsham, October 22, 1891.—With a sharp tenotome, several subcutaneous incisions were made over the ulnar side of the hand, dividing several bands of fascia binding down the fourth and fifth fingers; puncture also made over the ball of thumb, dividing the fascia holding that down. The fingers were firmly bandaged to a back-splint. Patient wore this for one month.

I saw the patient on May 13, 1895, three and a half years after the operation.

The patient says all the fingers were put quite straight after the operation, now the little finger has gradually gone back to its original contracted condition, whilst the ring-finger is quite straight; movement and power of grasp good. There is a tense band of fascia spreading from base of the index finger of this left hand to the base of the thumb, and drawing them together. In the right hand there are three puckerings of the skin in the

first transverse fold, corresponding to the three inner fingers, which are slightly bent. On the under surface of the left foot is a tough subcutaneous mass, over which the skin is freely movable. It is under the metatarsal bone of third toe, which is in no way contracted. Patient is quite satisfied, and has derived great benefit from the operation.

8. T. E. H., male, 42, Custom-house clerk, admitted into Hospital on April 21, 1890, suffering from contraction of right little finger.

Condition of hand before operation.—The little finger of right hand is bent at metacarpo-phalangeal joint in such a way as to be within an inch of palm. The ring-finger of same hand is affected to a less degree.

Operation by Mr. Walsham, April 21, 1890.—Multiple subcutaneous incision of the tense band; fingers quite straightened and put upon a back-splint. This splint the patient wore for one month after operation.

Patient writes to me on May 10, 1895, five years after operation:—Ring-finger is quite straight, little finger almost so. He is quite satisfied with the operation, and says he can the better follow his occupation. The little finger is gradually becoming more bent.

9. William S., 39, gold lacemaker, admitted into Hospital August 13, 1890, complaining of contraction of left ring-finger, which interfered with his work.

Condition before operation.—The left ring-finger at the first phalangeal joint is bent at an angle of 60° , and a tense band of fascia can be felt running up to the digit, keeping it in its bent condition. Condition of the right hand was natural.

Operation by Mr. Lucas, August 19, 1890.—The tense band of fascia was divided in several places by subcutaneous incisions and the fingers partially straightened. Gradually the finger was more extended, and on August 25, 1890, patient left the Hospital with the finger almost straight, wearing an anterior finger-splint, which he continued to do for six weeks.

In May 1895, four and a half years after the operation, the condition of the hand is as follows:—The second phalanx of left ring-finger is bent on the first at an angle of 90° , and a tense band of fascia can be felt running from the palm to the finger. Patient says contraction does not appear to be progressing, and he is now able to do his work well and finds no inconvenience. He says "the operation did him a lot of good," and there is now slight dimpling of the skin over the metacarpal bone of the ring-finger of the right hand.

No.	Sex.	Age.	Occupation.	Duration.	Hand Affected.	Family History.	Past History.
1	Male	57	Printer	6 months	Both hands. 1st appeared in right	Family phthisis. 1 son acute rheumatism and chronic rheumatism	Pain and swelling big toe 1 year ago; also sub-acute attacks for years; also cramps in legs. Fibrous band along dorsum of penis, described by Paget as a gouty thickening and hardening of fibrous sheath of corpus cavernosum.
2	Female	67	Housework	3 years	Left hand	No family history of gout or rheumatism	7 years ago primary syphilis. No history of gout or rheumatism.
3	Male	57	Printer	6 months	Both hands. 1st in right	Family phthisis. 1 son acute rheumatism and chronic rheumatism	Pain and swelling big toe 1 year ago. Also sub-acute attacks.
4	Male	59	Bootmaker	14 years	Both hands. 1st in right	Father suffered from gout and Dupuytren's contraction	Suffered from gout for years. Tophi in both ears and fibroid induration of corpus spongiosum.
5	Female	53	Housework	No history stated
6	Male	37	Brewer's drayman	...	Right middle finger	No family history in notes	Ran a piece of iron hooping into hand 5 years ago; does not know if it was extracted. No history of gout or rheumatism. Heavy drinker.
7	Male	35	Iron-fitter	3 years	Both. Began in left	No family history stated	Acute rheumatism 8 years ago. Syphilis 9 years ago.
8	Male	40	Bricklayer and sail-maker, using the ulnar side of hand extensively	2½ years	Right hand	No family history known	14 years ago "rheumatic fever." Rheumatic pains at times.

9	Male	32	Letterpress printer	6 years	Right hand	No history given in notes
10	Male	32	Grocer's porter	5 months	Left hand	No history given	5 months ago burnt thumb, suppurated; cellulitis of whole hand. Opened; all fingers stiff.
11	Male	60	Carman	...	Both	No family history given	Potus laceration dorsum right hand, compound fracture 4th metacarpal bone, contraction 4th and 5th fingers right hand, 3rd finger left hand.
12	Male	53	Porter	6 years	Left	Father had a similar finger	"Gout in big toe," and suffers from periodical attacks of pain in joints; 2 fits.
13	Male	56	Upholsterer	5 years	Both hands	Father had gout and "painter's colic." Mother asthma	Gout in left hand for about 8 years; generally suffers from gout in cold weather. No history of rheumatism or syphilis.
14	Female	15	Needlework	7 years	Both hands	Father suffers from rheumatism	No history of gout or rheumatism.
15	Male	43	Barman	8 years	Both. Began in right	Brother suffers from gout	No history of gout or rheumatism.
16	Male	33	Costumier	5 years	Left	No history in the notes	Potus.
17	Male	44	...	9 months	Left	No family history in notes
18	Male	38	Butcher	2 years	Right	Brother died of phthisis	Acute rheumatism and rheumatic pains in joints.
19	Male	42	...	9 months	Right	Family history not given	Chronic rheumatism. Right testicle removed for tubercular testis; writer's cramp lately.
20	Male	39	Carpenter	10 months	Left	No family history known	No history of gout or rheumatism. 3 years ago syphilis. No gout or rheumatism.

No.	Sex.	Age.	Occupation.	Duration.	Hand Affected.	Family History.	Past History.
21	Female	43	Scrubber	6 years.	Right	No history of gout in family history or in past history
22	Male	37	Flute-player	4 years	Right	No family history in notes	4 years ago injured hand with cricket ball; after this contraction started.
23	Male	53	Carpenter	...	Both	No family history in notes	Much employed with William's nail-puller, which requires much pressure with palm of hand; has also carried heavy bag in hand for long distances. No past history in notes.
24	Male	58	Gardener	8 months	Both	No family history in notes	No traumatic history. Has suffered from gout in big toe and rheumatics; acute rheumatism 10 years ago. Urine: abundance of lithates and traces of albumen.
25	Male	56	Labourer	20 years	Both	No family history in notes	1st attack of gout 2 years ago in right metatarso-phalangeal articulation; 14 days after left foot also attacked. Suffers from chronic rheumatism and emphysema.
26	Male	54	Bricklayer	4½ years	Both. Began in left	No family history in notes	Small-pox and typhoid fever. No history of gout or rheumatism.
27	Male	47	Bricklayer	4 years	Both hands	No history of gout	No history of gout or rheumatism or other illness.

28	Male	29	Clerk	10 years	Right hand	No history of gout	Severe burn on right ring-finger when a child; a small hard lump formed 10 years ago in palm right hand, since then ring and little finger contracted. No gout or rheumatism.
29	Male	41	Labourer	4 years	Right	Father suffered from "gout in big toe"	Acute rheumatism 20 years ago. 2 years ago gout in big toe. 3 attacks since.
30	Male	40	Bookbinder	6 years	Both	1 brother similarly affected; does same work	No history of gout or rheumatism. None of 40 employés except brother thus affected.
31	Male	44	Railway guard	5 years	Both. Began in right	No family history in notes	No past history in notes; began right hand 5 years ago; to give this a rest put on brake with left hand, which also became affected.
32	Female	50	Nurse	4 years	Right	Father and mother both suffered from gout	Acute rheumatism 15 years ago. Gout right "big toe" 4 years ago.
33	Male	48	Painter	2 years	Right hand	No family history of gout	Has suffered from rheumatism; has had similar affection of left hand and right foot.
34	Male	54	Boiler-maker	2 years	Both hands. Began in left	No family history stated in notes	Acute rheumatism 12 years ago and rheumatic pains.
35	Male	37	Clerk	7 years	Left hand	Father had rheumatism and lumbago. Mother died of apoplexy; had rheumatism	No history of gout or rheumatism. Wound of left palm 27 years ago. 4th left toe affected in same way.
36	Male	34	Hatter	12 years	Right hand	No family history stated in notes	Gonorrhoea twice and a chancre accompanied by buboes. Patient in very bad health, dyspepsia and great pain in region of kidneys.
37	Male	64	Watch jeweller	6 years	Both. Began in right	1 younger sister dyspeptic. Grandfather had gout	3 attacks. Gout in right metatarsophalangeal articulation. Last attack 7 years ago.

Cases from Kensington Infirmary, by Permission of Dr. Potter.

No.	Sex.	Age.	Occupation.	Duration.	Hand Affected.	Family History.	Past History.
1	Male	62	Gunner in army	6 years	Both. Left first	No family history known	Contraction came on 14 years after leaving army. No history of gout. Suffers from chronic rheumatism in shoulders.
2	Male	77	Labourer and hawker	5 years	Right hand	No family history of gout or rheumatism.	Suffers from chronic rheumatism, which affected right metatarso-phalangeal joint. Tophi in ears. Cataract.
3	Male	79	Plasterer	5 years	Right hand	Mother suffered from gout. Father from chronic rheumatism	Suffers from chronic rheumatism.
4	Male	70	Labourer	?	Both hands	No family history of gout or rheumatism	Has had gout in big toe and has tophi.
5	Male	38	Painter	2 years	Left hand	Mother rheumatic gout. Father none	Has had gout in big toe. Is in infirmary for acute rheumatism.
6	Male	75	Painter	?	Right	No family history of gout or rheumatism	No history of gout or rheumatism. Very slight puckering of skin; no contraction.
7	Male	72	Nobleman's servant	7 years	Left	No family history known	Acute rheumatism at 15, since then chronic rheumatism. Toes much deformed.
8	Male	89	Baker	6 years	Left	No family history known	Has suffered from gout and chronic rheumatism in shoulders.
9	Male	78	Horsekeeper	?	Left	Slight case	Gout 20 years ago; also suffers from chronic rheumatic pains. Tophi in ears.

10	Male	72	Labourer	?	Right	No family history known	Has had rheumatic fever, in bed 7 weeks, and rheumatics on and off. No gout.
11	Male	63	Mason	8 years	Right	No family history known	Has always suffered from chronic rheumatism and gout.
12	Male	93	Tailor	?	Left	No family history known	Chronic rheumatism. No gout.
13	Male	34	Horsekeeper	?	Right	No family history of gout or rheumatism	Slight ease. Is suffering from acute phthisis.
14	Male	54	Baker	2 years	Right	No family history of gout or rheumatism	Severe dog bite before contraction in palm of right hand. No history of gout or rheumatism.
15	Male	65	Clown	10 years	Both hands	Mother had rheumatic fever	No history of gout; had syphilis. Well-marked case.
16	Female		Who could not be found.				

550 patients: 300 males, 250 females, 16 cases of Dupuytren's contraction: 15 male, 1 female.

By kind Permission of Dr. Keats at Greenwich Union Infirmary.

1	Male	78	Joiner	10 years	Both.	Left first	No family history of gout or rheumatism	No history of gout or rheumatism. Cancer of oesophagus.
2	Male	71	Painter	23 years	Both.	Right first	No family history known	Has had several attacks of gout, and also suffers from chronic rheumatism. No female case.
Only 2 cases out of 700 patients examined, amongst whom were many sailors.								
1	Female	76	Housework	10 years	Right hand		Father suffered from gout	Chronic rheumatism for many years. No history of acute gout.
1 female case in 300 patients. Many with hands deformed by chronic rheumatism.								

No.	Sex.	Age.	Occupation.	Duration.	Hand Affected.	Family History.	Past History.
1	Male	50	Engineer	12 years	Left. Little finger	No family history of gout or rheumatism. Mother died of asthma	12 years ago severe crush over hypothenar eminence, since then contraction of little finger. Rheumatic fever 12 years ago.
2	Male	41	Goldsmith	...	Both hands. Began in 1st ring-finger	Brother suffers from gout in big toe	Never had gout or rheumatism.
3	Male	77	Porter	40 years	Left hand little finger.	Brother had gout. Mother had acute rheumatism	Ran a nail into hypothenar eminence about a month before deformity started. It does not get any worse, and has not spread to other fingers or to right hand. Has frequently suffered from gout. Mother had acute rheumatism, brother suffered from "gout in big toe."
4	Male	58	Horsekeeper	5 years	Left hand little finger	No family history known	Suffers from chronic rheumatism. No gout. No history of injury.
5	Male	50	Wheelwright	4 years	Left hand	Mother had acute rheumatism. Father suffered from gout	No past history of gout. Has had syphilis; is suffering from phthisis.
6	Male	65	Porter	30 years	Left hand little and ring finger	Father suffered from gout	No past history of gout or rheumatism.
7	Male	55	Cabinetmaker	3 years	Left hand little finger	None known	Has frequently suffered from gout, and has chronic rheumatism.
8	Male	73	?	14 years	Both little fingers	None known	No gout or rheumatism; always been healthy. Suffers now from bronchitis and emphysema.

ON THE
OCCURRENCE OF THE DIPHTHERIA BACILLUS

IN A SERIES OF CASES OF SORE-THROAT AMONGST
THE FEMALE STAFF OF THE HOSPITAL.

BY

F. W. ANDREWES, M.D.

More than a year ago I had occasion to employ the bacteriological test in the diagnosis of several cases of doubtful sore-throat amongst nurses. The results were in some instances by no means what I had anticipated, and I resolved to undertake a series of observations to determine the frequency with which the diphtheria bacillus was to be found in cases of sore-throat occurring under conditions of hospitalism, and the clinical characters of the sore-throats in which it occurred. Dr. Kanthack very kindly undertook the task of collaboration with me in this inquiry, and to his advice and help I have been throughout much indebted: it was indeed largely at his suggestion that I undertook the matter. It was decided to examine systematically every case of sore-throat, of whatever type, which presented itself to me in my capacity of medical attendant to the nursing staff, and it will be evident that the circumstances were very favourable for such an investigation. Every case of sore-throat amongst the female staff of the Hospital, save such slight ones as were not reported, necessarily came at once under my observation, so that I was able immediately to make cultivations from the throat, usually before any antiseptic local treatment had been adopted. I was, moreover, in a position to follow the cases clinically from first to last, and I have kept accurate notes of them. The importance of such a series of observations cannot be disputed. So far as I am aware, no one has yet

undertaken a systematic inquiry of the sort in a series of consecutive cases of "hospital sore-throat," so that the frequency of the occurrence of the diphtheria bacillus in such cases has not hitherto been estimated, nor has, I believe, a combined clinical and bacteriological inquiry been attempted. Apart, too, from the abstract interest of the question, the immediate practical value of the individual observations cannot well be overrated. Whether one does or does not accept the presence of the diphtheria bacillus as a positive criterion of the nature of any given case—and this is an academic question—no physician can afford to ignore the evidence of bacteriology in deciding matters of treatment, disinfection, and isolation in doubtful cases of sore-throat. Speaking for myself, I will at once say that, having realised the advantages of systematic bacteriological examination in all cases of sore-throat, I should be very sorry to be without it. I confess myself wholly unable to recognise the slighter cases of diphtheria without its aid. I should have made repeated mistakes, both positive and negative, had I trusted in clinical observation alone, and the cases which I record below will, I think, fully bear me out in this statement.

The methods adopted in the investigation were as follows. Every case of sore-throat, whether suspicious or not, was investigated. On first seeing the case, I made two cultivations from the fauces—serum agar-agar, prepared from ascitic fluid, being the medium employed. One of the tubes I handed to Dr. Kanthack, or, in his absence, to Dr. J. W. W. Stephens; the other I retained myself. Our examinations were perfectly independent, and our results in every case unanimous, and I think the value of the evidence is considerably enhanced by the mutual check thus imposed. Whenever necessary, more than one pair of cultures was made from the throat, and in some cases the observations had to be repeated a number of times.

It is needful to state explicitly at the outset what criteria we employed in deciding what was and what was not a diphtheria bacillus. The standard adopted was to all intents and purposes a morphological one. Non-motile bacilli, which showed the characteristic clubbed form, the segregation of the protoplasm and the typical grouping under the microscope, were regarded as diphtheria bacilli. We endeavoured, as far as possible, to obtain the bacilli in pure culture in all cases in which any doubt existed, and in nearly all cases this was achieved. The cultural characters of the organism were of course of weight in arriving at a decision, and the marked clubbing of the bacilli, when grown upon ordinary agar-agar, was to my mind of special value in diagnosis. Further, whenever needful and possible, the

virulence of the bacilli was tested by inoculation in guinea-pigs—this part of the work being undertaken by Dr. Kantback—and valuable confirmatory evidence was thus obtained. We agree in attributing less importance, however, to proofs of virulence than to the morphological and cultural characters of the bacilli. It has been the custom with some to reject as true diphtheria bacilli organisms which fail to show virulence when injected into animals, even though in all other respects they coincide with it in characters. The term “pseudo-diphtheria bacilli” has been employed for such organisms, as it has also been employed in a variety of senses for organisms allied to the diphtheria bacillus. Virulence is, however, so varying a character in this group of bacilli, that too much importance may easily be attached to it. No more conclusive proof of this can be afforded than by one of the cases in this series, in which the cultures, when first isolated from the throat, were non-virulent, but after some months of cultivation became highly virulent. We therefore regarded as diphtheria bacilli all those organisms which we obtained from the fauces, which corresponded morphologically and culturally with the type, even when they failed to produce a fatal effect in animals. Both the large and the small form of the diphtheria bacillus were met with. In one or two cases it was found that the growth of the organisms obtained was exceedingly scanty upon gelatin, resembling that of a streptococcus. But in each case of the kind, on repeated subculture, the growth became more luxuriant, and reverted to the cultural characters typical of the diphtheria bacillus.

The total number of cases examined, including recurrences in the same case, was more than seventy, and of these I have full notes of some sixty-eight, with which I propose to deal in this paper. Two of them proved to be cases of scarlet fever. Four or five were cases of manifest diphtheria, slight, but clinically recognisable as such; three were cases suspiciously like diphtheria, but which I should not have felt justified in certifying without the help of bacteriological examination; one was a case in which bacilli reappeared in the throat during convalescence from diphtheria. Five were cases of quinsy, and in two of these definite membrane was present on the tonsils at the commencement of the attack. Ten were cases of mere catarrhal sore-throat without exudation; I do not doubt that some of these were cases of influenza. The remainder, some forty-three in number, were cases of tonsillitis with exudation, mostly follicular in character, though in some the exudation consisted merely of minute specks, while in others it was so extensive as to spread beyond

the crypts and form a sheet of loose pultaceous exudation more or less covering the tonsil. There was hence no lack of variety in the clinical characters of the cases of sore-throat examined; indeed, almost every form of the affection now occurring under conditions of hospitalism came at one time or another under observation, and all were impartially investigated by the same methods. From the clinical point of view I paid attention to the precise characters of the faucial affection, and the amount, distribution, and consistency of the exudation, to the presence or absence of enlargement of the lymphatic glands at the angle of the jaw, to the height and duration of the fever, and to the pulse frequency. I regret that I made no systematic observations as to the presence or absence of albumin in the urine. I did so in certain cases, but not often enough to make the matter worth recording. From the bacteriological point of view, the one point to which we paid attention was the presence or absence of the diphtheria bacillus; to have pursued an adequate study of the other organisms present would have been the labour of many years.

I will now give an abstract of some of the more important of these cases, dividing them into groups for the sake of clearness and convenience. Each case must necessarily be dealt with very briefly; but I will endeavour to give adequate details of those cases which appear of special importance.

GROUP A.—*Cases of Scarlet Fever.*

Case 1.—E. H. April 27. Sore-throat, malaise, headache, pains all over; no vomiting. April 28. Temperature, 101° ; pulse, 110. Fauces and pharynx red and injected; tonsils not swollen; no exudation; uvula very œdematous. Tenderness at angles of jaw, but no glands felt. Scarlatinal rash in afternoon. Cultures taken from fauces on morning of 28th yielded no bacilli, only streptococci and staphylococci.

Case 2.—A. C. May 16. Slight sore-throat. 17th. Fauces red; no exudation. Temperature, 100.4° at night; vomited. 18th. Temperature, 102° ; pulse, 96. Tongue dry, brownish in centre; fauces red; tonsils swollen and congested, with yellowish follicular exudation. Glands at angles of jaw palpable. Scarlatinal rash on chest and abdomen. Cultures taken from fauces on 18th yielded no bacilli, only a few cocci.

GROUP B.—*Cases of Manifest Diphtheria.*

Case 1.—C. A. October 27. Throat dry and rough in evening. 28th. Backache and slight headache. Temperature, 99° at night. 29th. Temperature, 100° ; pulse, 88. Commencing membrane on tonsils, white and not very adherent, in patches which have not yet coalesced; most on left side. Not much redness or swelling. Some glandular enlargement on left. Temperature, 100.4° at night. 30th. Temperature, 97.8° . Much pain in neck, and aching in back, legs, and head. Patches of exudation clearing up on left tonsil, but increasing on right, in spite of local application of peroxide of hydrogen. Faint trace of albumin in urine. Warded in Radcliffe. Cultures taken from the fauces on the 29th yielded typical diphtheria bacilli in abundance: their virulence was not tested. They had vanished from the throat by November 9. On November 2 and 3 there were doubtful and transitory indications of paralysis of the palate and ciliary muscle, but these vanished in a day or two. No genuine diphtheritic paralysis supervened. The gland enlargement at the left angle of the jaw was still present on December 10.

Case 2.—K. R. March 16 and 17. Throat slightly sore, but still felt well. During night of 17th increased sore-throat, headache, and shivering. 18th. Temperature, 100.2° ; pulse, 100. Headache and backache. Fauces reddened, but not much swollen. Lying obliquely across left tonsil is a patch of greyish-white exudation, sharply outlined, but not tough or adherent; not definite membrane. No glandular enlargement. Temperature, 101.6° at night. 19th. Temperature, 98.8° ; pulse, 96. Headache and aching all over. Exudation on left tonsil more extensive; right tonsil now also covered with exudation, but the latter is confined to the tonsils. There is more redness of the throat, and œdema of the uvula. Small gland felt on right. Warded in Radcliffe. Cultures taken on March 18 yielded typical diphtheria bacilli, and almost nothing else. Subcultures were characteristic, but their virulence was not tested.

Case 3.—H. M'D. April 26. Throat first sore; had suffered from headache for a day or two. 27th. Remained on duty, but felt ill; throat painful; headache. 28th. Temperature, 100° ; pulse, 112. Not evidently ill. Not much redness or swelling of fauces, but much dense white exudation on right tonsil, and several patches in the crypts of the left. Glands at angles of jaw palpable. 29th. Temperature last night, 101.6° ;

this morning, 101.4°; pulse, 96. Still does not look ill. On the right tonsil the membrane is denser and more opaque, and just trespasses on the anterior pillar of the fauces. Patches on left tonsil still discrete, but larger and more opaque. A small white speck on the back of the pharynx. No albuminuria. Cultures taken from the throat on the 28th yielded scanty somewhat clubbed bacilli, which we regarded as the small form of the diphtheria bacillus. Subcultures failed, so that virulence could not be tested. Antitoxin treatment was begun on the morning of the 29th, but on the evening of the same day the temperature was 102.6°. 30th. Temperature has fallen to 99.4°; pulse, 72. Feels much better. No extension of membrane. Throat clearing up with perchloride of mercury gargle (1 in 1000). May 1. Temperature normal. Only a few specks of exudation left. May 2. Temperature subnormal: pulse, 70. Throat now scarcely red; no exudation present after this day. Cultures taken from the throat on May 5 yielded no bacilli.

Case 4.—D. W. Diphtheria at eight years old. Chronic enlargement of tonsils, most on right side. June 1st. Legs ached; throat felt sore at night. 2nd. Temperature, 99°; pulse, 116. Slight brownish fur on tongue. Fauces deep red; tonsils enormous. Abundant exudation in patches on the right, chiefly in crypts. On the left tonsil a thin sheet of definite adherent membrane. The exudation is confined to the tonsils. No distinct glandular enlargement. Warded in Radcliffe. A piece of the membrane was removed, and cover-glass preparations made from it and stained. These showed numerous diphtheria bacilli, morphologically typical. Yet cultures taken at the same time from the throat yielded no growth, or at most a few doubtful bacilli. (This is the only case in which bacilli, undoubtedly present in the membrane, failed to appear in the culture tube; they must have been dead, perhaps killed by some previous local application.)

GROUP C.—*Cases of Diphtheria, clinically doubtful, but in which the Bacilli were found.*

Case 1.—C. M. October 7th. Throat sore; had been shivering slightly for four or five days. Temperature, 99.6°; pulse, 120. Discrete greyish exudation present on both tonsils. Gland palpable at left angle of jaw. Had been on duty in a ward in which several cases of follicular tonsillitis had recently occurred. October 8th. Temperature normal; pulse, 80. Felt better. 9th. Temperature normal. Exudation gone. Throat remained thenceforward clear. Backache. 12th. Temperature remains normal. Cloud of albumin in urine. 14th. Pulse 72, slightly

irregular. Less albumin. Backache persists. The albumin disappeared after the 16th. Cultures taken from the throat on the 7th and again on the 11th, after the exudation had vanished, yielded bacilli morphologically identical with the diphtheria bacillus; virulence was not tested. Cultures taken on the 14th and 18th yielded no bacilli, nor were they present when the patient returned to the Hospital on November 12th. No paralysis ensued.

Case 2.—J. W. November 15th. Ill. Temperature, 100.4° at night. 16th. Temperature, 99.8°; 101.2° at night. A large patch of exudation of suspicious appearance on left tonsil. 17th. Temperature subnormal. Cultures made on 16th from the patch on the left tonsil yielded numerous typical diphtheria bacilli. 19th. Temperature subnormal; pulse, 60. A small patch of definite firm membrane still present low down on the left tonsil: it is rather adherent, but can be lifted with a probe; no bleeding or apparent excoriation of surface beneath it. No glandular enlargement. Throat painted with peroxide of hydrogen. 20th. Feels quite well. Throat not sore. Membrane gone; fauces barely reddened. Cultures made this day yielded no bacilli. 22nd. Temperature subnormal in morning; pulse, 68. Right tonsil now rather red and œdematous. Slight glandular enlargement on right; gland not tender. Temperature, 100° at night. 23rd. Temperature subnormal. Right tonsil shows reddening, with greyish exudation in a long crypt. Left tonsil normal. Cultures made this day from the right tonsil yielded abundant colonies of characteristic large clubbed bacilli. Few other organisms were present. A subculture of the bacilli was made, and on December 2nd was tested on a guinea-pig; it proved highly virulent, killing the animal in twenty-four hours. From the local tumour, which was small, produced in the animal, Dr. Kanthack isolated the diphtheria bacillus in pure culture. The patient's throat rapidly improved after November 23rd, and the exudation disappeared with local antiseptic treatment. The throat was clean and normal in appearance by the 25th, and she was apparently well. She went away for a holiday on the 26th—as it proved, prematurely—for on the 28th patches of exudation again appeared on each tonsil, with sore-throat and malaise. She used a chlorine gargle, but one patch remained till December 3rd. She felt well on December 10th, and remained well. This case appears peculiarly interesting from its relapsing character, which is unusual. Clinically I should certainly not have ventured to diagnose diphtheria. No paralysis ensued.

Case 3.—E. E., a ward-maid in Radcliffe; subject to sore-

throat. December 10th and 11th. Felt faint; vomited; throat sore. 12th. Temperature subnormal; pulse, 96. Fauces slightly reddened, but hardly at all swollen. A patch of loose white exudation on the right tonsil. Glandular enlargement on right. Cultures made this day from the throat yielded numerous colonies of the small form of the diphtheria bacillus. These were readily obtained pure in subculture, and on agar-agar showed a growth of typical large clubbed bacilli. A gelatin subculture injected into a guinea-pig on December 20th proved highly virulent, killing the animal in twenty-four hours. The patient was admitted into Isolation; on the 16th there was still a long strip of exudation on the right tonsil, and the case was regarded as one of slight diphtheria. On this day the temperature reached 99° —its highest point. She was discharged from Isolation on the 27th. Seen on January 8th and 13th; glands still enlarged.

GROUP D.—*Cases of apparently simple Sore-throat in which the Diphtheria bacillus was found.*

In all, 53 cases of apparently simple sore-throat were bacteriologically examined (including recurrences in the same individual). In six of these cases diphtheria bacilli were found, and in two others doubtful bacilli. That is to say, that one out of every nine cases of apparently simple sore-throat, which I should have viewed clinically without the slightest suspicion, was in reality, or at least potentially, of diphtheritic nature, and capable, we may presume, of infecting others. I invite special attention to the notes on these cases, as they illustrate the point to which in this paper I attach most importance.

Case 1.—F. T., a ward-maid. Subject to sore-throats when she gets a cold. History pointing to possible gastric ulcer. October 11. Throat first sore, but had suffered from headache and vomiting for some days previously—the latter probably depending on the chronic gastric trouble. First seen on October 14. Temperature, 99.4° ; pulse, 116. Fauces reddened and swollen, in a catarrhal condition, with one or two small white spots in the crypts at the upper extremity of each tonsil. No glandular enlargement. 15th. Temperature normal. Fauces cleaning with hydrochloric acid gargle; still some specks of exudation at the top of the right tonsil. 16th. Temperature normal. Throat practically well; exudation gone. Having no suspicion as to the nature of her throat, I admitted her to a general ward for her gastric symptoms. Two cultures were made from the throat on October 14; one of these yielded only streptococci,

the other, along with streptococci, yielded several colonies of diphtheria. The bacilli were obtained in pure subculture, and when tested on a guinea-pig proved highly virulent, killing the animal in forty-eight hours, with the production of a typical local tumour at the seat of injection. The patient's throat remained normal after the 16th. Cultures taken on the 21st yielded no bacilli.

Case 2.—A. D. November 14. Throat sore in evening. 15th. Temperature, 99.8° . Left tonsil swollen and red; no exudation. Temperature, 101.4° at night. 16th. Temperature, 98.8° . Slight traces of exudation about the tonsils. 17th. Temperature normal. 18th. Exudation gone. Throat thenceforward normal. In cultures made on the 16th, Dr. Kanthack found bacilli which resembled the diphtheria bacillus; re-examining the same tubes on the 25th, he again found typical colonies. Subcultures failed, so that the matter remained doubtful.

Case 3.—D. K., ward-maid. November 26. Throat sore; backache. 27th. Temperature, 99.6° ; pulse, 100. Headache. Fauces reddened and swollen, with a few small points of exudation on tonsils. No glandular enlargement. Temperature, 100° at night. 28th. Temperature, 99.2° ; pulse, 96. Headache. Fauces red and swollen as on preceding day, but the points of exudation have gone (under treatment with chlorinated soda gargle). Glandular enlargement now present, especially on left side. Warded in Isolation. 29th. Temperature fell to normal and remained so. The throat rapidly got well, and was quite normal on December 2, though on the 4th an enlarged gland could still just be felt at the left angle of the jaw. Cultures made from the throat on November 27 yielded several typical colonies of diphtheria; subcultures on agar-agar, gelatin, and in broth were characteristic. On December 4 a guinea-pig was injected with a gelatin culture; it developed a local tumour, but recovered. On December 21, two more guinea-pigs were injected, one with a broth culture and one with an agar-agar culture; both died on the sixth day, and the diphtheria bacillus was recovered from the local tumours. The bacilli were hence virulent, though not highly so. Cultures made again from the throat on December 2 yielded no bacilli.

Case 4.—M. M., not subject to sore-throat. December 8. Headache. 11th. Backache and sore throat. 12th. Temperature, 101.6° in afternoon and 102.6° in evening; exudation seen on left tonsil. 13th. Temperature, 99° ; pulse, 100. Feels better; less headache. Moderate redness and swelling of fauces. A little loose whitish exudation on left tonsil. No glandular enlargement. Temperature, 99° in evening. 14th. Temperature normal; pulse, 100. Throat better; exudation

has gone, under the influence of chlorinated soda gargle. Slight glandular enlargement on the left. 16th. Temperature normal; pulse, 88. Throat scarcely red. No gland felt. 18th. Throat normal. Feels well. Cultures made from the throat on the 13th; one yielded no growth at all, the other showed innumerable colonies of a bacillus resembling diphtheria, with marked segregation of protoplasm, and often clubbed. Subcultures on agar-agar showed bacilli with immense clubs; subcultures on gelatin grew very scantily, after the manner of a streptococcus, but showed typical clubbed bacilli. On January 6 a guinea-pig was inoculated with a mixed broth and agar-agar culture; it developed a slight local tumour, but remained otherwise well. The subcultures were kept up for some months, and on gelatin always grew scantily, till, in April, Dr. Kanthack got a subculture which suddenly developed a large and rapidly-growing colony. From this subcultures were made which grew well and typically on gelatin, and proved highly virulent, killing a guinea-pig in twenty-four hours. Cultures made from the patient's throat on December 16 yielded no bacilli.

Case 5.—J. S. February 11. Onset during night, with shivering, vomiting, and headache. 12th. Temperature, 102° at night. 13th. Temperature, 100.2° ; pulse, 96. Fauces scarcely at all red or swollen; at the upper part of the left tonsil is some greyish-white soft exudation, not in the crypts, but non-adherent, and not forming definite membrane. No glandular enlargement. Temperature fell to normal by the evening. 14th. Temperature subnormal; pulse, 84. Feels much better. There are now numerous discrete white patches all over the left tonsil, but none on the right. No glandular enlargement. Temperature, 99.4° at night. 15th. Temperature subnormal; pulse, 84. Throat now quite clean, with chlorinated soda gargle. 17th. Throat normal. Cultures taken from the throat on February 13 yielded very numerous colonies of the small form of the diphtheria bacillus. Subcultures were typical, and proved highly virulent, killing a guinea-pig in less than forty-eight hours. Dr. Kanthack recovered the diphtheria bacillus from the local tumour. A culture taken from the patient's throat on February 17 yielded only a few doubtful bacilli.

Case 6.—D. P. Some chronic enlargement of the tonsils. March 16th. Sore-throat, with slight malaise and headache. 17th. No worse; temperature normal. 18th. Temperature, 99.2° ; pulse, 60. Tonsils enlarged, but not very red. Some scanty white specks of exudation on the left tonsil, not easily removed from the crypts. Does not feel really ill. 19th. Temperature subnormal. Feels well. Under chlorinated soda gargle the

throat has cleared. There is now no redness or exudation, and no pain on swallowing. No glandular enlargement at any time. Cultures taken from the throat on March 18th yielded very numerous colonies of a small bacillus, which I at first hesitated to diagnose as diphtheria, though Dr. Kanthack did so. Subcultures on agar-agar, however, showed large clubbed bacilli, with much protoplasm segregation, in all respects typical of diphtheria; but they did not prove very virulent on animals, though local tumours were produced, from which the bacilli were recovered. Cultures taken again from the throat on March 23rd yielded no bacilli. This patient was on two subsequent occasions under treatment for tonsillitis—in April, and again in May. In April there was merely a little catarrhal inflammation of the enlarged tonsils. In May there was follicular tonsillitis, the crypts being stuffed with yellowish exudation, and the condition lasting four days, though with very little fever. No bacilli were found on this occasion.

Case 7.—Z. P. In March 1896 suffered from an attack of sore-throat, with transient specks of exudation on the tonsils. The temperature was never above 100°, but there was much aching of the back and limbs, a flushed face, and a little erythema about the upper arms. The possibility of scarlet fever was considered, but not entertained for long, and no peeling ensued. The sore-throat lasted about a week, and cultures taken from the throat yielded no bacilli. Six weeks later, on May 5th, the throat felt sore on swallowing, but she felt perfectly well. Seen on May 8th, she had a normal temperature, and felt well, with no headache or other constitutional symptoms. The throat felt slightly sore on swallowing, and low down on the left tonsil there was a small white patch of exudation, unaccompanied by redness, swelling, or glandular enlargement. Cultures made from this patch yielded very numerous colonies of bacilli, in all respects resembling the diphtheria bacillus. Subcultures on agar-agar were typical. The throat was painted with peroxide of hydrogen, and by the 10th the patch had vanished. On the 12th the throat was normal, and there was no longer soreness on swallowing. The bacilli, however, persisted in the throat. Cultures taken on the 12th and 16th yielded bacilli in abundance. In cultures taken on the 18th and 25th they were still present, though the throat was treated after the 23rd by brushing with perchloride of mercury solution (1 in 500). In a culture taken on the 27th only one colony of bacilli grew. A guinea-pig was inoculated with a subculture on May 31st. It developed a large local tumour, but recovered; the virulence was therefore slight. The

patient had, of course, to be isolated during this period, though remaining throughout in perfect health. I fear that she formed a low opinion of the methods of bacteriological diagnosis.

Case 8.—E. W. Attending since June 8th on an unsuspected case of diphtheria, which ultimately required tracheotomy, and died on June 11th. On the 10th her throat was slightly sore, and there were slight headache and backache. On the 11th the throat was red, but there was no exudation. Temperature normal. Diphtheria antitoxin was injected as a precaution. 12th. Temperature normal; pulse, 84. Throat red; no exudation. 13th. Temperature normal; pulse, 58, somewhat irregular. Throat still red; no exudation or glandular enlargement. 15th. Pulse, 64, regular. Throat nearly normal. Cultures taken on the 12th yielded a few bacilli, some of which were fairly typical clubbed forms, but they could not be recovered in subcultures, and it was not possible to feel positive about them.

GROUP E.—*Cases of simple Sore-throat, catarrhal and follicular, in which the Diphtheria bacillus was not found.*

These were 45 in number, and it is superfluous and needless to enter into the clinical details of the individual cases. It would clearly be of the greatest possible importance to set forth the clinical characters of these cases, for contrast with those of the cases of apparently simple sore-throat in which the diphtheria bacillus was found, were it possible to do so. I find, however, on analysing the cases, that I am unable to point out any important clinical distinctions between the two groups of cases. I have taken the averages of the highest temperatures observed in each individual case. In the group in which bacilli were found this comes to 100.3°, while in the group in which bacilli were not found it comes to 100.9°. I have done the same with the highest pulse frequencies observed in each case. The average for the bacillary cases is just under 98; that of the non-bacillary cases is just over 99. I do not think that the duration of the fever or of the symptoms was, on the whole, longer in the one set of cases than in the other. The characters of the faucial affection itself yield no distinctions of any note. Bacilli were several times found in cases with a small amount of loose non-adherent exudation, precisely like that of ordinary follicular tonsillitis. I observed no more redness and swelling in one set of cases than in the other. Glandular enlargement was, however, more commonly present in the bacillary than in the non-bacillary cases. In 13 cases of all kinds in which bacilli

were present, the glands at the angles of the jaw were palpable in 9, or in 69.2 per cent. In 36 cases in which bacilli were not present, the glands could be felt in 15, or in 41.6 per cent. The difference is not large enough to render the point one of very material assistance in diagnosis. As I have said, I have unfortunately no data for estimating the relative frequency of albuminuria in the two sets of cases.

GROUP F.—*Cases of Suppurative Tonsillitis.*

Five cases of quinsy occurred in the series, one being a recurrence in the same patient. In two of them definite membrane occurred, and one of these may have been truly diphtheritic, though the evidence was not conclusive. I will give the notes of these two cases.

Case 1.—A. H. Subject to tonsillitis. No attack for two years. October 29. Throat first sore. 30th. Headache and back-ache; evening temperature, 99.8°. 31st. Temperature, 99.6°; pulse, 74. No constitutional symptoms. Very little redness or swelling of the fauces. On the left tonsil is a sheet of membrane, soft and easily detached, but not leaving a clean surface. On the right tonsil a little membrane and some greyish sloughy-looking ulceration. Glandular enlargement on the left. Temperature in evening, 100.2°. November 1. Temperature, 100.2°. Membrane gone from left tonsil (after painting with peroxide of hydrogen); patches of exudation still present on right; faint trace of albumin in urine. November 3. Temperature, 98.8°; pulse, 72; temperature, 99.8° in evening. 4th. Temperature subnormal; pulse, 72. Still a small patch of membrane on right tonsil; left tonsil clean; gland on left side smaller. Still a faint trace of albumin in urine. Evening temperature, 100.8°. 5th. Temperature, 99.8°; pulse, 72. A little pus escaping from left tonsil. 6th. An incision into the left tonsil let out some two drachms of pus. Recovery immediate and uninterrupted. Cultures made from the throat on October 31 showed no sign of any diphtheria bacilli. A piece of membrane removed from the right tonsil on November 4 was examined under the microscope for bacilli, but none resembling diphtheria were found. Cultures made from this fragment yielded negative results.

Case 2.—H. D. Suffered from a rather severe attack of diphtheria eight years ago. Subject to "relaxed throats." An attack of tonsillitis a year ago. January 11. Throat sore for the last three days, slight; no exudation seen. Has not felt ill. Temperature normal; pulse, 96. Left tonsil much swollen,

but very little reddened, with numerous patches of greyish exudation, small and not easily detached. A tender gland palpable on left; right tonsil normal. Temperature in evening, 101.8°. 12th. Temperature in morning, 102.4°; in afternoon, 103°; in evening, 102.6°. 13th. Temperature, 100.2°; pulse, 94. Face flushed; no rash; left tonsil now covered with thick, somewhat tough, yellowish, purulent membrane, not easily detached, but confined to tonsil. A little pus coming away from left tonsil; much glandular enlargement and tenderness, mainly on left side. Swelling of right tonsil now present, but much less than on left side. Temperature, 100.2° at night. 14th. Temperature, 98.8°; pulse, 76. Less swelling and exudation on left side; pus escaping freely; glands enlarged and tender on both sides. 15th. Temperature subnormal; pulse, 78. Left tonsil less red and swollen; right tonsil almost normal, but glandular enlargement and swelling more marked than on left. 17th. Temperature subnormal for three days; no exudation. 21st. Right tonsil painful. 22nd. Right tonsil a little red and swollen, but without exudation; gland on right much more swollen and tender; much headache. Temperature normal. 23rd. Temperature normal; pulse, 72. 24th. Right tonsil subsiding without suppuration; no albuminuria. 27th. Is now much better; enlargement and tenderness of glands nearly gone. Went away for a long holiday. The throat was sore at intervals for several months, with patches of exudation lasting a few days. The patient's health was much broken down, and she was unfit to return to work for many months. No signs of diphtheritic paralysis occurred. Cultures taken from the throat on January 11 yielded many staphylococci and streptococci, but no bacilli at first; later bacilli were found, amongst which were some clubbed forms. Cover-glass preparations of membrane taken from the throat on January 13 showed no distinctive bacilli. Cultivations from the membrane yielded bacilli which were possibly diphtheria; subcultures on gelatin grew scantily, like a streptococcus, but subcultures on agar-agar showed large clubbed forms, which morphologically resembled the diphtheria bacillus. Tested on two separate occasions on guinea-pigs, the bacilli proved absolutely non-virulent, not even producing a local tumour. The conclusion is hence indefinite; but I think that the morphological characters of the bacillus, and still more the serious results upon the patient's health, point to something more than an ordinary quinsy. Should it really have been a diphtheritic case, the occurrence of suppuration is unusual and interesting.

CONCLUSIONS.

In drawing conclusions from the facts which I have set down above, two different positions may be maintained. It is open to any one to argue that the presence of the diphtheria bacillus, in cases of apparently simple sore-throat, does not justify the application to them of the term "diphtheria." This implies that the criterion to be employed in judging the nature of cases should be a clinical one, and not merely the presence or absence of the diphtheria bacillus. It may be urged, too, that in many of the cases I have recorded the organisms which we found were not genuine diphtheria bacilli, but allied and harmless forms. To this I would answer that our results are those of two independent observers in substantial agreement, one at least of whom has had very wide experience in the matter, while I myself have worked at it for some years. I may add that Dr. J. W. W. Stephens, who did some of the work in Dr. Kanthack's absence, has been devoting himself for some time to this very subject, and there are few whose opinion I should receive with greater confidence. Into the question of the "pseudodiphtheria bacillus" I have entered in the introduction to this paper, and I am unable to admit that the mere absence of virulence—a varying character readily lost in the laboratory—is sufficient to disprove the genuineness of a bacillus otherwise corresponding with the type. The morphological characters of the virulent bacillus differ so widely in different cases, and on different culture media, that I regard with the greatest mistrust the statements of those who affect to recognise the "pseudodiphtheria" bacillus by mere microscopic examination. Moreover, in six at least of the cases of doubtful sore-throat which I have recorded above, the bacilli were not only typical morphologically, and in cultural characters, but were very virulent, and indisputably true diphtheria bacilli.

For my own part, I frankly take the alternative position and accept the presence of diphtheria bacilli, even if their virulence is slight or absent, in a case of sore-throat, as a criterion of its diphtheritic nature, and I would modify the definition of diphtheria in this sense. It may, indeed, be possible to recover the bacillus from normal throats. This may be because the bacillus has lost its virulence, or because the host has temporary immunity. Bacilli may persist in the throat for very many weeks after an attack of the disease. I would, therefore, only maintain that their presence in a case of recent sore-throat is to be accepted as evidence of its diphtheritic nature. There are

strong grounds for believing that diphtheria often appears in non-membranous form under the guise of a simple sore-throat. In families attacked by the disease some members often suffer simultaneously from trivial sore-throat. In times of its epidemic prevalence, the number of cases of "simple sore-throat" is usually largely increased. Dr. Thorne Thorne in his Milroy Lectures has shown good ground for believing that the most important way in which diphtheria spreads is by slight cases of sore-throat in elementary schools. I am becoming more and more convinced that the same is true of the spread of diphtheria in hospitals. Again and again does it happen that a nurse or patient is attacked by indubitable diphtheria without the possibility of explanation by contact with a case of the declared disease. I believe that the cases I have above recorded under Group C., and especially under Group D., are the very cases which are demanded by Dr. Thorne Thorne for his hypothesis as to the epidemic spread of diphtheria, and are the ones responsible for many an unexplained case arising in a hospital. I therefore make no apology for presenting a series of cases of this sort in what may appear, from their frequently trivial clinical characters, unwarrantable detail; it is only by detailed narration that conviction can be carried to the minds of those who doubt. The lesson conveyed by this investigation to my own mind is to distrust clinical appearances in the diagnosis of sore-throat, and to rely chiefly on bacteriological examination. I estimate that had I relied on clinical appearances alone in the above series of sore-throats, I should have been right in some eight cases out of nine, and wrong in the ninth, and that would be a percentage of error which one cannot regard with equanimity.

SKIN-GRAFTING BY THIERSCH'S METHOD.

BY

H. J. PATERSON.

The introduction of skin-grafting has, without doubt, conduced more than any other form of treatment suggested within recent years to the rapid and sound healing of granulating wounds. And inasmuch as the treatment of ulceration, from whatsoever cause, forms such a large part of the surgical practice of every practitioner, the record of some cases and observations on this subject may, I trust, prove not altogether devoid of interest to the readers of the Hospital Reports.

The older method of skin-grafting, introduced by Reverdin of Geneva, consists of shaving off small pieces of the superficial layers of the epidermis, and placing them on the ulcer, where they form the starting-point of a circumferentially spreading growth of epithelium. This method greatly hastens the healing of an ulcer, but does not to any great extent diminish the subsequent tendency to contraction. In the more recent method of Thiersch, grafts much larger in size are utilised, and consisting of all the layers of the epidermis, together with just the tips of the papillæ of the cutis vera. These larger grafts are placed on the base of the ulcer in close juxtaposition, so that in this way the whole granulating surface can be completely covered over at one sitting.

The advantages of this method are very great. Healing is greatly expedited, and there is much less after-contraction. Further, in Reverdin's method, the final result is numerous islets of skin, surrounded on all sides by bands of scar-tissue, which forms a half, or even more, of the whole healed area. In Thiersch's method, on the other hand, no intervening granulation tissue being left between the grafts, the whole ulcer becomes covered with sound elastic skin; thus the bands of scar-tissue which traverse the site of the ulcer, when treated by the older method, are absent, and consequently one great cause of the after breaking down of the healed ulcer does not exist.

The operation itself is simple, although tedious; in fact, its only disadvantage is the necessity of having the patient anaesthetised.

During my tenure of office as House Surgeon, Mr. Willett instructed me to skin-graft by this method on several occasions, and I have now to thank him for kindly allowing me to report on some of the cases here.

The first patient¹ was a carman, aged 44, who nine months previously had been severely burnt about his right knee. The resulting ulcer had refused to heal, and was, on admission, about four inches in diameter, there being also some necrosis of the anterior surface of the patella. The necrosed bone was scraped away, leaving a large wound to granulate up. Two weeks later the whole ulcer was covered in by three grafts. On dressing the knee five days later, all the grafts were found to have taken, and ten days later the patient left the Hospital with the ulcer soundly healed. This patient came up to the Surgery about eighteen months later with a dislocated shoulder, and on examining his knee at that time, I found the grafted skin had remained soundly healed, and the use of his limb quite unimpaired.

On two occasions the result of grafting was a complete failure. In both these cases the ulcers were due to large carbuncles. One of the patients was a man aged 67 years,² with a carbuncle on his anterior abdominal wall. This was scraped, and a few days later was grafted, but none of the grafts survived. The other patient,³ aged 41, had a large carbuncle at the back of his neck. As soon as healing had commenced in the extensive ulcer resulting after thoroughly scraping the carbuncle, I completely covered it in with grafts, none of which, however, became vascularised. I do not attribute the failure in these two cases to the fact that the ulcers were of carbuncular origin, or even to the patient's necessarily poor state of health, but rather to the situation of the ulcers in a region where it was quite impossible to maintain firm pressure upon them, or to keep the part at rest. That that is so is, I think, shown by the fact that in the second case just mentioned, a week later I again applied several grafts, at the same time moulding, at Mr. Willett's suggestion, a gutta-percha splint round the patient's neck and over the back of his head. The parts were thus kept more at rest, and firmer pressure could be applied, with the result that on this occasion several of the grafts lived. Mr. Wallis of Charing Cross Hospital, too, tells me that several times he has grafted the ulcers resulting from carbuncles with success.

This leads me to say a word or two about the conditions favourable or unfavourable to successful skin-grafting. For

¹ See Table, Case No. 1. ² See Table, Case No. 2. ³ See Table, Case No. 7.

convenience I will allude to some of the more important points under three headings. As regards—

- (1.) The situation of the ulcer.
- (2.) The condition of the ulcer.
- (3.) The condition of the patient.

(1.) As regards the situation of the ulcer to be grafted.—Firm pressure and perfect rest are essential for success. Hence any ulcer situated in a region where these conditions do not obtain, is not favourably circumstanced for grafting. It is difficult to keep the neck free from movement, impossible to apply firm pressure to it, hence it is difficult to apply grafts there with success. The same holds good for the abdomen. On the other hand, ulcers on the extremities, on the chest, on the scalp and parts of the face, are most favourably situated for good results. Joints, although naturally mobile, are readily fixed by splints, and when this is done, grafting in the neighbourhood, although more tedious, is quite as satisfactory as elsewhere. The following case will illustrate this.

A boy¹ aged 10 was run over by an omnibus, sustaining a severe contused and lacerated wound about the left elbow, involving the joint. Much sloughing of skin and underlying tissues ensued, with the result that, with the exception of a narrow strip of skin in front, the whole area round the elbow became a large ulcer, measuring 5–6 inches long and 6–7 inches round, the joint being opened and the internal condyle carious. Later on the whole ulcer was covered in by grafts, the operation lasting rather more than 1½ hours, the arm being fixed by an angular splint and swung. The grafts were dressed on the sixth day, when all were found to have taken. The boy left the Hospital three weeks later with the ulcer soundly healed. I saw the patient about one year after his accident. The ulcer had remained perfectly healed, and did not in any way impair the movements of the joint, which were, however, very slightly restricted, as the result of the involvement of the joint at the time of the accident.

(2.) The condition of the ulcer.—The ulcer is ready for grafting as soon as it has commenced to heal, as evidenced by the existence of a thin blue epithelial film at any part of its margin. The sooner the ulcer is grafted, the less will be the subsequent contraction.

(3.) The condition of the patient.—Most persons who have extensive ulceration are broken down in health. This is not necessarily a bar to successful grafting. How, then, in a given

¹ See Table, Case No. 6.

case are we to decide whether the patient's general condition be favourable or otherwise? Our most reliable guide is the state of the ulcer. And indeed it may, I think, be stated as a broad rule, that if the ulcer has shown signs of healing, the patient's general condition is such that the grafting will probably be successful.

Tertiary syphilis does not in itself preclude successful skin-grafting. A patient¹ aged 37 was admitted with a large mass of suppurating lymphatic glands, situated on the inner aspect of the right thigh, secondary to several neglected tertiary syphilitic ulcers on the inner and outer aspects of the leg. The abscesses were incised and thoroughly scraped, some of the undermined skin being removed. Three weeks later the granulating wound, measuring 6 inches long by 3½ inches wide, was completely covered by three large and two or three smaller grafts. The result was quite satisfactory; all the grafts lived, and the patient was discharged two weeks later. Some months later this patient had further suppuration, but the grafted area remained quite sound.

The next case² which I record will illustrate the value of Thiersch's method in the treatment of the large ulcerating surfaces which may follow extensive cellulitis. The patient, aged 58 years, was admitted into the Hospital on November 16, 1893, with extensive cellulitis of the left lower limb, the result of an injury about the knee three weeks previously. Incisions were at once made, much pus evacuated, and an abscess cavity found extending upwards on the outer side of the limb to within a hand's-breath of Poupart's ligament, and two or three inches down the outer side of the leg. Notwithstanding the operation, the cellulitis continued to spread, and much sloughing took place within the abscess cavity. On December 4th his condition was so serious that Mr. Willett asked several of his colleagues to see the case in consultation with him, and the question of amputation of the limb was raised. It was decided, however, that such a severe operation would in his condition almost inevitably prove fatal, and that in any case it would be impossible, owing to the extensive undermining and destruction of skin which had ensued, to cover in the necessary operation wound, and it was agreed that it was best not to perform any radical operation at that time. On December 19 a further consultation was held, and the opinion was that probably sooner or later amputation would be

¹ See Table, Case No. 3.

² This case is recorded in the *Clinical Society's Transactions*, vol. xxix. p. 236. See also Table, Case No. 4.

necessary, but that inasmuch as the patient was not now losing ground, and was taking his food well, it would be advisable to wait, in the meantime trying the effect of removing all the undermined skin, so as to allow free vent to all the pus, which tended here and there to be retained in recesses of the abscess cavity. If this succeeded, Mr. Willett suggested Thiersch's grafting later on. Accordingly, under nitrous oxide gas, all the undermined skin was rapidly cut away with scissors, leaving a large wound extending from Poupart's ligament and the iliac crest above, right down the outer aspect of the thigh, and as far as the middle of the leg. Nor was it a mere superficial wound, inasmuch as the femoral vessels could be plainly seen therein. On the outer side of the limb the vastus externus and biceps femoris muscles were also exposed.

On January 10, 1894, Thiersch's grafting was performed; five strips of skin, measuring approximately $5\frac{1}{2}$ by $2\frac{1}{4}$ inches, taken from a patient's upper limbs, were laid at intervals transversely across the ulcer. The ulcer was of such large extent, and the discharge of pus so abundant, that it was considered inadvisable to attempt to cover it in completely. The portions of granulation tissue intervening between the grafts were dressed daily so as to prevent the retention and accumulation of pus. The grafts were dressed on the sixth day, and were all found to be living.

On February 2 the remainder of the ulcer was covered in by grafts in the same manner.

The patient left the Hospital on March 26, 1894. Of course the skin-grafting had nothing to do with the patient's recovery from the serious condition in which at one time he was, but it did greatly hasten his recovery after convalescence was once established, and very considerably lessened the duration of his stay in Hospital. Further, there can be little doubt the skin-grafting greatly improved the after-utility of his limb. Had an ulcer of such large extent been allowed to heal in the ordinary way by scarring, it could—and this is the point on which I wish to lay stress—hardly have healed soundly, and would subsequently have broken down at intervals, to the discomfort of the patient. Whereas, at the present time, the whole area of the ulcer remains soundly healed, the limb is freely movable, and has not caused him any trouble whatsoever; in fact, as he himself says, the limb is as useful as ever it was. The scar now measures about 21 inches by 5–6 inches, and the original grafts can be plainly distinguished, separated from each other by a narrow line of scar-tissue. The skin all over the ulcer is quite elastic, and moves quite readily over the deeper parts, but it is entirely devoid of tactile sensation as

tested by a needle. And it is interesting to note that, with the exception of one or two scars, where too deep a cut was made, there is scarcely any indication on the upper extremities that any skin has been taken from them.

It may be of interest to describe the method of performing skin-grafting, so that I will here give the procedure which I found most satisfactory, after trial of various modifications as to detail. My apology for going into such minutiae is that successful results are essentially dependent on attention to these seemingly small details.

For purposes of description we may consider the procedure under three heads:—

- (1.) Preparatory treatment.
- (2.) The performance of the grafting.
- (3.) The after-treatment.

1. Preparatory treatment.—This we have to consider as regards

- (a.) The ulcer to be grafted.
- (b.) The skin from which the grafts are to be cut.

(a.) Preparatory treatment of the ulcer.—The first step is to get the wound as aseptic as possible. The skin surrounding the ulcer must be shaved, and the whole ulcer and surrounding skin thoroughly washed in soap and water, the skin being also well rubbed with turpentine. For the next forty-eight hours I recommend the use of sanitas fomentations, frequently changed. These are of great value in getting the ulcer clean and sweet. For a further period of forty-eight hours some stimulating application should be applied, such as red lotion. At the end of that time the whole region should be again thoroughly washed with perchloride lotion, and enveloped in sterilised lint or gauze wrung out in hot carbolic lotion (1 in 30), covered with protective and bandaged. This dressing should be put on about eighteen or twenty hours previous to the time fixed for the operation.

(b.) The preparation of the skin.—The skin should be prepared the day previous to the operation by shaving, thoroughly scrubbing it with soap and water, then with ether, and finally with perchloride lotion (1 in 2000). It should then be covered with a dressing similar to that placed on the ulcer.

2. The performance of the grafting.—Here we must consider

- (i.) The final preparation of the ulcer.
- (ii.) The cutting of the grafts.
- (iii.) Placing and maintaining the grafts in position.

(i.) The final preparation of the ulcer.—The patient having been anæsthetised, the whole of the superficial flabby part of

the granulation tissue in the base of the ulcer is scraped away with a Volkmann's spoon. This prepares a level bed for the grafts to be laid upon, and by removing granulation tissue, which would eventually become converted into fibrous tissue, diminishes the subsequent contraction of the ulcer. It is advisable, at the same time, to scrape away the thin blue epithelial film at the healing margins of the ulcer. The whole surface is then vigorously scrubbed with a sterilised nail-brush and soft soap and water. Finally, after free irrigation with hot perchloride lotion, it is sponged dry and enveloped in gutta-percha tissue or protective, which has been kept for some time in carbolic lotion (1 in 20), and dipped before application in saline solution. **Firm pressure is then applied** by means of a bandage. This arrests the hæmorrhage. If this be not effectually done, blood will collect between the graft and the surface of the ulcer, with the result that the graft will die. Gutta-percha tissue is placed in immediate contact with the ulcer, in preference to gauze or sponges, as it does not tend to stick to the granulations, and thus start the bleeding afresh when it is removed. The operator should now again thoroughly disinfect his hands, and for the rest of the operation anti-septic lotions should be avoided, as the chemicals might injure the delicate grafts.

(ii.) **The cutting of the grafts.**—I have found the knife known as Thiersch's knife work admirably. It should be sharpened after each occasion on which it is used. A razor has not sufficient weight to give it the steadiness necessary for cutting large grafts. In addition to the knife, two pairs of dissecting forceps, a pair of scissors and a probe are all the instruments required. The flexor aspects of the forearms are the most suitable regions from which to take the grafts; the skin here is fine, comparatively free from hairs, and easily kept on the stretch while the grafts are being cut. If more grafts than can be obtained from these regions are required, they may be taken from the extensor aspect of the thighs and from the upper arm. The skin must be kept *tightly* on the stretch in the long axis of the limb by an assistant. The operator then stretches and flattens the skin from side to side while cutting the grafts with the knife in his right hand. The grafts should be cut with a sawing motion, the handle of the knife being at the same time alternately slightly depressed and raised, so as to follow the contour of the limb as far as possible. In this way grafts may be cut measuring $2\frac{1}{2}$ inches wide, and if necessary 5 inches or even longer if the limb be a big one. While cutting, the knife should be kept wet with hot saline solution (one

drachm of common salt to a pint of hot water), and each graft as it is cut should be placed in a bowl of the same solution at a temperature of from 98°–100° F.

In cutting the grafts, care must be taken not to cut too deeply, otherwise scarring will ensue. The part removed should consist of the whole of the horny later and the superficial part of the Malpighian layer of the skin, the tops of the papillæ being just removed, so that a surface covered with minute drops of blood is seen, each drop of blood corresponding to a cut papilla. If this is done, there will be absolutely no scarring; in fact, after a time the same place may be utilised for the taking of other grafts.

The part from which the grafts have been taken should be well washed with perchloride lotion and dressed with antiseptic gauze. This may be left on about fourteen days, at the end of which time a covering of plain wool will be all that is necessary. **All the grafts required should be cut, and the part from which they have been taken wrapped up, previous to removing the gutta-percha tissue from the ulcer which is to receive them.** I lay stress on this point because some authorities recommend that the grafts be transferred on the knife direct to the ulcer. If this be done, there is great risk of inoculating the raw surface from which the grafts have been taken with some of the pus from the ulcer, with the result that ulceration may ensue over an area as extensive, or even greater, than the ulcer for the cure of which the grafting has been undertaken. And indeed this is no imaginary danger, as I myself have seen it occur on more than one occasion. Further, by cutting all the grafts at once, owing to the longer time during which pressure is being exerted on the prepared ulcer, the hæmorrhage is more likely to be completely arrested,—a condition most essential for success; and when all the grafts are applied together, there is less likelihood of any of them becoming accidentally shifted during the manipulations incidental to their being cut and transferred to their resting-place. The vitality of the grafts is effectually preserved for an hour or even more, if only the temperature of the saline solution be maintained by the addition of hot water.

Grafts from a freshly amputated and healthy limb may be used instead of grafts from the patient. This is of especial service if the patient's condition be such as to render a long operation under an anæsthetic inadvisable. The scraping of the ulcer may be accomplished while the patient is under the influence of nitrous oxide gas, and the actual placing of the grafts quietly finished without any anæsthetic.

(iii.) **Placing and maintaining the grafts in position.**—The ulcer is surrounded with towels wrung out in hot carbolic. The grafts are then removed one by one from the saline solution by means of a pair of dissecting forceps, and spread out on the ulcer, raw surface undermost. If it be borne in mind that the grafts always curl up towards the raw surface, no difficulty will be experienced in spreading them out correctly.

Round the margin of the ulcer the grafts should overlap the skin for about one-eighth of an inch. Over the remainder of the ulcer the grafts should be placed with the edges in close contact, no intervening islets of granulation tissue being left. The grafts must be completely uncurled and evenly spread out. For this purpose I have found nothing answer so well as two pairs of dissecting forceps, occasionally aided by a probe. The grafts must be handled with the utmost gentleness, and no air or fluid must remain between them and the base of the ulcer. This is the most tedious part of the operation, and requires much care and patience. Any attempt at hurrying will probably result in the operator displacing some of the grafts already placed *in situ*. If the ulcer be large or the discharge profuse, it is advisable to divide it into several smaller ulcers, by placing several broad grafts across it. The intervening ulcers may be covered in at a later period while the first grafts have become vigorous. It has been recommended by some authorities that the grafts be stitched in position. This is quite unnecessary, and would greatly increase the duration of the already prolonged operation.

Next as to the keeping of the grafts in position. As already insisted upon, **very firm pressure must be maintained if success is to be assured.** The grafts should be first covered with strips of isinglass plaster. Each strip is dipped in hot saline solution and then firmly applied over the grafted area. Above each of these strips a thick strip of antiseptic gauze is placed, and kept in position by a strip of Leslie's strapping of sufficient length to get a firm hold on the skin beyond the ulcer. The strapping is best heated in the flame of a spirit-lamp; this makes it adhere better than warming it round a hot-water tin, and at the same time sterilises it. Over the plaster is laid a thick layer of wool, and the whole part firmly bandaged with a Domette bandage. If the part grafted be a limb, it should be slightly raised for the first few days. If the ulcer is near a joint, this must be securely fixed by some splint.

3. **The after-treatment.**—The dressing should be left undisturbed for at least four days. If the part be quite comfortable, if the patient's temperature be not raised, and especially if there

be no discharge through the dressings, and the dressings smell quite sweet, the first dressing may be advantageously postponed until the fifth or sixth day. I prefer to let the gauze and plaster soak off unaided by placing the part, or, if necessary, the patient in a hot bath, having first removed the bandage and wool and loosened the Leslie's strapping. In a successful case the result may at first sight appear to be a failure. The grafts are now pinkish or red in appearance, and the granulation tissue below shows through them, so that their presence is not very evident. If the grafts are conspicuously white, we may be certain that they are no longer living.

In brief, then, the conditions most essential to success are four in number :—

- (1.) A healing ulcer.
- (2.) Strict asepsis.
- (3.) Perfect rest.
- (4.) Firm pressure.

I have already indicated some of the advantages gained by the treatment of an ulcer by Thiersch's method of skin-grafting, and the selection of cases recorded will, I think, afford illustrations better than any further comments of mine.

Briefly recapitulated, the benefits to the patient are :—

- (1.) Permanent healing.
- (2.) Hastened recovery.
- (3.) Lessened contraction, and consequently
- (4.) Less impairment of the part grafted.

In conclusion, I may add that, having had no practical experience of Thiersch's method as applied to the treatment of fresh wounds, due to wide removal of malignant or other growths in the skin, I have confined myself to its use in the treatment of granulating wounds. It has, however, been successfully performed in the Hospital on several occasions during the past two years. The technique is very similar. The grafts are cut in the same manner, and transferred to the fresh surface of the wound, after all hæmorrhage has been completely arrested by firm pressure. The after-treatment differs in no respect from that already described. For this procedure I would suggest the use of the term "Primary grafting." "Secondary grafting" might be used to signify the application of grafts to a granulating wound. Although the methods are very similar, the circumstances under which they are done differ materially, so that the two proceedings should, I think, be clearly distinguished, just as it is customary to draw a distinction between a primary and secondary amputation, although both are performed in precisely the same manner.



LEFT LOWER LIMB EXTENSIVELY GRAFTED BY THIERSCH'S METHOD. (*From a photograph taken three years after operation, by Mr. J. Preston Maxwell.*)

The appearance of contraction in the print is deceptive, and is in reality due to wrinkling of superabundant skin. (See Table, Case No. 4.)

Table of Cases Treated by Thiersch's Method of Skin-Grafting.

No. of Case.	Sex.	Age.	Cause of Ulcer.	Situation.	Approximate Size.	Remarks.	Day of First Dressing.	Fate of Grafts.	Notes.
I.	M.	44.	Burn.	Over knee.	4 x 4 in.	Ulcer of nine months' duration. Some necrosis of patella.	5th day.	All lived.	Seen eighteen months later, ulcer soundly healed.
2.	M.	67.	Carbuncle.	Abdomen.	4th day.	None lived.	Complete failure, probably due to inadequate pressure.
3.	M.	37.	Suppurative lymphadenitis.	Thigh.	6 x 3½ in.	Ulcer completely covered in.	5th day.	All lived.	Patient had tertiary syphilis. Seen subsequently, ulcer soundly healed.
4.	M.	56.	Cellulitis.	Thigh and leg.	24 x 6-7 in.	Five grafts placed at intervals.	5th day.	All lived.	Grafted various separate occasions, because of large size of ulcer.
5.	M.	56.	Same patient.	Thigh and leg.	24 x 6-7 in.	Remainder of ulcer covered.	7th day.	All lived.	Soundly healed up to present time.
6.	M.	10.	Traumatic gangrene.	Elbow.	6 x 7 in.	Completely covered over.	7th day.	All lived.	One year later still soundly healed.
7.	M.	41.	Carbuncle.	Neck.	...	Completely covered over.	5th day.	None lived.	Failure due to inadequate pressure and imperfect rest.
8.	M.	41.	Same patient.	Second attempt made—partially cured.	7th day.	Some of grafts lived.	Gutta-percha splint applied.
9.	M.	53.	Cellulitis.	Scalp.	3 x 4 in.	Completely covered over.	6th day.	All lived except one.	...
10.	F.	38.	Cellulitis.	Leg.	Several large ulcers.	Completely covered over.	6th day.	All lived.	Grafts taken from Case 9. No anæsthetic.
11.	M.	59.	Cellulitis.	Leg.	6 x 4 in.	Completely covered over.	5th day.	All lived.	...

A CASE OF
DISSEMINATED SCLEROSIS WITH PALATAL
AND LARYNGEAL ANÆSTHESIA.

BY

F. E. BATTEN, M.D.,

AND

W. J. HORNE, M.B.

Disseminated sclerosis, apart from its intrinsic gravity, will always command attention on account of its clinical vagaries and remissions being apt to lead to unfortunate conclusions. The opportunities do not often occur for watching these vagaries from the beginning to the end, and then for establishing the diagnosis by a minute pathological examination.

The girl whose case forms the subject of this paper came under observation at the very onset of the disease, during the House-physiciancy of one of us, and, after a fluctuating course, extending but a little over two years, the case terminated during the House-physiciancy of the other. Through the kindness of Dr. Church we are jointly able to present the facts complete.

A single girl, at the age of 17, whilst apparently in good health, and following the occupation of an electric wire-worker, was suddenly seized with fits. There were seventeen in one day, with convulsions, loss of consciousness, and biting of the tongue. She was brought to the Hospital, and detained for two days in the Surgery Ward.

During the following fortnight she felt well, and then complained of numbness in the right hand and arm and weakness of grasp. This was followed in a few days by numbness of the right leg; and when walking, the foot was noticed to be dragged. The mouth also seemed to twitch on the same side as the numbness. There was no urinary trouble; she was rather constipated; she slept well; the appetite was not very good.

The symptoms noticed became worse, and she was admitted to Faith Ward on the 15th February 1892.

The friends stated that she had always been fanciful and irritable. Six months previously there had been an abscess of the left ankle. When six years of age she had typhoid. There was no history of chorea, nor of rheumatic or scarlet fever. The family history obtained was that her father was 54 and living, but a sufferer from sciatica. Her mother, 43, was alive and healthy. There were thirteen brothers and sisters, living and in good health; four had died young, one from "chest complaint," and one from typhoid. A cousin on the father's side had been in an asylum.

On admission the following condition was noted:—

Well nourished, of a somewhat neurotic aspect. Complexion natural, pupils equal, no facial paralysis. Tongue pale, clean, and moist. Speech not affected. Pulse 80, regular and of good volume. Respirations quiet. Temperature 99.4°. Bowels opened morning of admission. Urine 1022, acid, no albumen, no sugar. Chest well shaped. Breasts well developed. Percussion note and breath sounds natural all over. Heart's impulse in the fifth space and inside the nipple-line, sounds natural.

Abdomen.—Nothing abnormal noted.

Arms.—Grasp equal and firm. Some anæsthesia of right hand and forearm; force used under the thumb-nail was only slightly felt. The right arm was rigid, and the sense of its position was completely lost.

Legs.—The skin over the front of both was tight and shiny; there was slight pitting on pressure. The knee-jerk was increased in both legs, markedly so in the right. No "ankle-clonus" was obtained. The right leg was much colder than the left. The plantar reflex was absent on both sides, and the sensation appeared impaired on the right. The right foot was dragged.

The temperature was subnormal throughout. The weight ranged from 7 st. 1 lb. to 7 st. 10 lb.

On February the 17th the extremities were the same temperature; anæsthesia was well marked, for severe pin-pricks in the right hand, forearm, foot, and lower leg apparently were not felt.

March 1.—Anæsthesia of arm and leg disappeared after hypnotic suggestion. The gait had improved, but she still walked stiffly. All the movements of the legs were free.

March 8.—She still walked stiffly with the right leg, the foot of which was held in a position of talipes equinus. When

hypnotised, the foot could be bent up to a right angle, but no farther.

March 15.—Distinct improvement in gait; distinct stretching of the calf muscles.

March 22.—Further improvement; the arm held stiffly.

April 4.—Gait much improved; slight facial weakness noticed on right side.

April 6.—Power of arm and leg greater.

April 12.—Improved. Still loss of sense of position in right arm. Massaged every day.

April 15.—Very marked tremor of right hand, increased on trying to perform movements. Writing very shaky. Nystagmus very slight on lateral deviation.

April 25.—Improved. No anæsthesia, but still tremors of right hand on trying to perform movements. The knee-jerk was increased on the right side. The sense of position had returned in the right arm. General condition good.

On April 27, 1892, she went home.

Between the end of April 1892 and the beginning of April 1894 the girl's health continued sufficiently good to permit of her following the occupation of an electric wire-worker. In 1893 she became pregnant; a full-term child was born December 4, 1893. She was married Christmas, 1893.

On May 8, 1894, on account of recurrence of the paralysis, she became an out-patient under Dr. Brunton.

On May 9, 1894, then 19 years of age, she was re-admitted to Faith Ward.

The history then given was that fourteen days previously, whilst up and about, nursing her child, and feeling in fair health, she had a tingling sensation in the left arm and leg, immediately followed by a sudden loss of power in those limbs, rendering her unable to stand or to continue suckling her child. There was no loss of consciousness, no fit, nor convulsion. Pain followed in back of neck, passing down the back and into the fingers. The loss of power continued. Her mother stated that latterly she had been subject to fits of crying and screaming. The appetite and digestive powers had been good, but there had been costiveness.

Condition at time of readmission.—Complexion fair. Mouth half open; countenance stupid, vacant, stolid expression of obstinate stupidity. Mental activity sluggish, replying to questions slowly, and with a silly, melancholy, indescribable whine; but speech was articulate, there was no scanning. The lips were rather pallid, but the general nutrition was well maintained.

There was no loss of facial symmetry, no impairment of vision, nor of hearing. No evidence of any cranial nerve lesion. No nystagmus, pupils natural, no jerking movement of head or neck, no tremor of tongue or lips.

Muscular System.—The voluntary motor powers of the right arm and leg were complete and natural. Passive movement of left limbs was resisted; the left leg could be raised from the bed and kept raised, likewise the left arm. The finger movements were perfect and free from tremors. The legs were kept extended in bed, and the left foot inverted. The knee-jerk was active in both legs, perhaps more so in the left. There was left “ankle-clonus.” The sense of position was retained.

Subjective sensations.—Numbness and tingling in the left leg, but less than there had been. Pain was referred to the back of the neck.

Objective sensations.—No anaesthesia made out, except in the soft palate, and there universal. Sensation to heat and cold normal.

No vaso-motor or atrophic changes of skin or joints.

Condition of spinal column normal.

Ophthalmoscopic examination detected nothing abnormal.

Thorax.—Heart natural. Pulse 88, regular, good strength. Lungs natural. Respirations 20.

Abdomen.—Nothing abnormal detected. Recto-urinary functions normal. No loss of sphincter control.

Urine sp. gr. 1012, trace of albumen, acid. Temperature 97°.

May 10.—When taken out of bed she was unable to stand without assistance. She could not or would not move either leg; on attempting movement she shot forward on to one side. When prevailed upon to move one leg in front of the other, the toes were dragged, the left more than the right. The left foot was inverted and crossed the right, the knees having a tendency to interlock. Whilst unobserved the movement of the limbs in bed had been noticed to be natural. She complained of headache and was costive. The urine and faeces had been passed under her. The mental condition had changed; at times she was irritable and crying.

May 11.—She was more helpless. The urine contained a trace of albumen; the temperature remaining subnormal.

May 12.—The right arm and leg had become powerless. The urine yielded a cloud of albumen.

By May 13 she was no longer able to feed herself. There was no dysphagia. Mentally she was more depressed. The control of the sphincters was completely lost.

On May 14 the temperature rose to 100°. The palsy of all four limbs was more universal. Towards evening dyspnœa developed, followed by orthopnœa. There were no signs of pulmonary disease; the intercostal muscles and the diaphragm were becoming involved.

The soft palate was void of tactile sensation. The epiglottis stood widely open, and the larynx was viewed in its full extent. The cords were in a state of extreme abduction; on phonation they moved readily and equally, on the same level and with good tension, producing a full, clear, and strong voice. The mucosa was intact, but the pallor extreme. A laryngeal probe, under the guidance of the mirror, was passed even roughly over the entire laryngeal mucosa without eliciting reflex or complaint. The anæsthesia was general and complete, and not a mere impairment of tactile sensation.

By midnight respiration was still further restricted. There was no cyanosis, but greater pallor. The girl, propped up in bed, appeared to be but little distressed, she complained of headache and pain in the neck on attempting to bend the head forward. The head, thrown back on a pillow, was constantly rolling from side to side; momentarily stopping when asked a question, then she stared and replied "yes" or "no" in a sharp, snappy tone, adding, "I want to go home; where's my mother?" It was Whit-Monday, and the wish was occasioned by a memory of the pleasures a year ago.

The respiratory muscles ceased to act, the accessory ones less and less were brought into action; respiration was reduced to a minimum; a jerking of the thyroid up and down alone was apparent. Sufficient air passed to permit of speech; the pulse kept in comparatively good strength; the head still rolling from side to side; finally at 2 A.M. it rolled to the left and there remained, death having taken place in the midst of babbling a still audible but silly reply. The pulse gave another twenty beats, rapidly slowed down, and went out.

A post-mortem examination was made by Dr. Tooth eleven hours after death, and we are indebted to him for the following notes:—

Height, 56½ inches. Fairly well nourished. Breasts rather full. Lineæ albicantes on abdomen well marked. Cranial bones, dura mater, and sinuses, arachnoid, and pia mater natural. Arteries natural. Brain: The cortex, corona radiata, ganglia, and capsules were carefully examined, and nothing abnormal was found. The hemispheres were somewhat adherent along the longitudinal fissure. At about the middle of the pons a transverse section showed that the pyramidal tract on

the left side was distinctly whiter than on the right. This disappeared in the upper pons, but was appreciable in the medulla, but not in the cord.

The spinal cord, apparently natural, was reserved for examination.

The eye and ear were not examined.

Chest: Lungs, right, weighed 14 oz.; left, 10½ oz., both natural.

Heart, 8½ oz., soft and flabby; no valvular disease.

Abdomen: Liver, 49 oz., natural in appearance. Spleen, 7 oz. Kidneys, the two weighed 11 oz., rather engorged; the venæ stellatæ stood out very strongly.

Microscopical examination.—The cord was hardened in Müller's fluid. Portions from the cervical, dorsal, and lumbar regions were embedded in celloidin, and after being stained by Pal's method, were mounted in the usual way. Other pieces from the same regions were placed in Marchi's solution for a week, after which they also were embedded in celloidin, cut and mounted.

On examination without the aid of the microscope, the sections stained according to Pal showed extensive areas of degeneration in the cervical and dorsal regions, involving not only the pyramidal tracts, but extending into the posterior and antero-lateral tracts. In the lumbar region the degenerated area was limited to the crossed pyramidal tract on the one side, and the direct pyramidal tract on the opposite side.

On examination with the microscope, it was found that the degeneration had even a wider distribution than it appeared to have with the naked eye. In the portions of the cord where the degeneration was most extensive, hardly any nerve fibres remained intact, and the posterior column in the dorsal region was seen to be slightly affected.

The sections stained with Marchi's solution were remarkable as showing how few fibres presented any recent degeneration with such an extensive sclerosis.

We have also evidence that the pyramidal tracts were affected in the pons and medulla.

Further, we have evidence of a diffuse change taking place in the spinal cord, most marked in the cervical region, becoming less in the dorsal, and least marked in the lumbar region, where it is no longer a diffuse sclerosis, but a descending degeneration.

The case is instructive, as has already been said, on account of its being under observation from the onset to the end, the diagnosis being verified by a pathological examination.

The sudden onset with epileptiform convulsions is unusual. The occurrence of hemiplegia, hemianæsthesia, and the loss of sense of position, together with recovery from the same, is not unusual; and it is worthy of note that on readmission the hemiplegia was on the opposite side—the left—and before death again involved the right side.

Other points of interest are the nystagmus and the “ankle clonus.” The nystagmus undoubtedly was slight, and that only on lateral deviation, and its absence was noted during the second stay in Hospital.

Upon her readmission, symptoms of a hysterical or emotional character, so frequently met with when the disease occurs in young women, were not absent; in fact, they were more in evidence than any unequivocal sign of organic disease, and a conclusion, apart from the inference established by the records of her previous admission, and consonant with the clinical evidence of May 9, 1894, would have turned upon the significance to be attached to “ankle clonus.”

The history of some moral shock or strain, commonly given as a factor in the ætiology, was not wanting on this occasion, and it would seem that pregnancy had caused a rapid advance of the disease.

A complete anæsthesia, affecting the entire mucous membrane of the larynx, and obliterating reflex action, is rarely observed. The motor fibres to the intrinsic muscles, including the tensors, remained intact. Thus the larynx gave the first evidence of a medullary centre being involved.

The mode of death, affording retention of intellect and memory up to the fatal moment, was presumably consequent upon the respiratory centre being invaded. From the pathological condition of the cord, it is difficult to understand how the girl was able to continue her work, for the amount of sclerosis in the lateral column would lead one to suppose that the path was completely interrupted.

“There is the fact mentioned by Charcot, that an optic nerve which was affected through its whole thickness by a patch of sclerosis was yet capable of performing its function.”¹

It is interesting to note that the cord, when stained by Marchi’s method, showed very few recently-degenerated fibres.

It is improbable that the amount of degeneration which the cord presented had taken place in the few weeks before death, so it would seem that either the fibres passing through the sclerosed regions were still capable of transmitting impulses, or that the impulses had taken up some circuitous course.

¹ Bastian, “Paralyses, Cerebral, Bulbar, and Spinal,” 1886, p. 650.

REPORTS OF
CASES IN THE SURGICAL WARDS.

BEING THE BENTLEY PRIZE REPORTS FOR
THE YEAR 1896.

BY

THOMAS J. HORDER.

CASE I.—“*Elephantiasis*” of one Leg in a Boy.¹

Abstract:—The condition was congenital—Skin and bones chiefly affected—No vascular abnormality—Leg amputated on account of ulceration, pain, and uselessness of limb—Good recovery—No local recurrence of original condition.

Albert David Beckwith, aged 15 years, mechanic, admitted into Darker Ward, under the care of Mr. Butlin, September 16, 1895, on account of a skin eruption on the right leg and inability to walk because of pain in it.

Since birth the right leg had been larger than the left and discoloured. Five years previously patient had attacks of pain in the limb, but since that time had no trouble with it until one year previously, when it again became very painful, and prevented him doing his work; from this time he had also walked lame on account of great tenderness of the sole of the foot and of the ankle. The skin eruption, which had always been present to some degree, had increased considerably during the previous three months. During the previous three weeks the foot had bled from an ulcerated patch over the dorsum of the ankle.

The boy was born in London, and had lived there all his life. When three years old he had an abscess opened in the left thigh, the scar of which, still visible, showed on admission a patch of eruption similar to that on the right leg. No other illnesses. His father was living, aged 48, but had lost the

¹ *Vide* Museum Collection of Photographs, No. 364b.

sight of both eyes when 30 years old, "through weakness of the nerves." His mother died of "hæmorrhage from the lungs." One brother and two sisters were living and healthy. One sister died in infancy. There were no signs of congenital syphilis to be discovered.

Patient was a well-nourished youth, stammered in his speech, but presented no other physical abnormality besides the one described below. Tongue and mouth were quite natural—no thickening or discoloration. The arms were quite natural.

The left leg.—About 6 inches above the knee, internally, was the small patch of dark-coloured, raised, hard tubercles, marking the seat of the old abscess—altogether about the size of a shilling.

The right leg presented abnormalities both above and below the knee. The most conspicuous object was a dark, ash-coloured area of skin, extending from knee to ankle, almost surrounding the limb, but specially marked in front. This area was covered by large patches of desquamating epithelium of various sizes, some as large as shillings. These patches were easily detached, without abrasion of deeper layers, and showed enlarged orifices of sweat-glands. Most of these scabs were placed upon the anterior surface, and were at a maximum number and size about three inches above the ankle. On the sole of the foot, and especially beneath the toes, were several small, quite black, shotty particles, which easily came away and (according to the patient) reappeared shortly afterwards. Microscopically, these particles consisted solely of some form of amorphous pigment. The hairs grew longer and thicker on the right leg than on the left leg, except just where the scales were thickest—here they were absent. A broad band of pigmented skin stretched from the knee upwards and outwards as far as the hip; it was definitely marked off from the natural skin, but there was no desquamation upon it. The whole leg below the knee was in a condition of elephantiasis: the bones were thickened, the toes very large, and there was actual lengthening of an inch as compared with the left leg. The natural curves of the foot and the depressions around the malleoli were obliterated. The foot, ankle, and lower part of the leg pitted on pressure slightly. Small, firm, warty growths were seen at the roots of the toes dorsally, and two inches above these, on the same aspect, was a patch of actual ulceration, an inch long, placed transversely; this discharged freely a thin, dark, blood-stained fluid. The ankle and foot were very painful—the sole of the foot especially so, so that walking caused considerable pain. The limb above the knee

presented marked muscular wasting; half an inch of actual shortening, as compared with the left leg, was also found. The superficial veins were prominent, the two chief radicles of the internal saphenous being clearly traceable for some distance. The right femoral and inguinal glands were very distinctly felt, were hard and knotty, but were not enlarged. The pulse in the right femoral artery was of good volume. The limb was not at all cold; perspiration was natural, except where the branny scales were thickest.

Summary of measurements.—(i.) In the thigh, all measurements were greater on the left than on the right side.

(ii.) In the leg and foot, all measurements were greater on the right than on the left side.

(iii.) There was half an inch real shortening of the right thigh.

(iv.) There was an inch real lengthening of the left leg.

(v.) Circumferential measurements showed the right thigh smaller than the left at all points, and the right leg and foot larger than the left at all points.

Measurements in Inches.

A. Above the knee—	Right.	Left.
Ant. sup. sp. ilium to int. cond. femur . . .	18 $\frac{3}{4}$	19 $\frac{1}{4}$
Round thigh, 1 inch above patella . . .	11	11 $\frac{1}{2}$
Round thigh, 6 inches above patella . . .	12 $\frac{1}{2}$	13 $\frac{1}{2}$
B. Below the knee—		
Int. cond. femur to int. mall. tibia . . .	16	15
Round leg, 1 inch below patella . . .	12	11
Round leg, 1 inch above ankle . . .	7 $\frac{1}{2}$	6 $\frac{1}{2}$
Round heel and malleoli . . .	14	11 $\frac{1}{2}$
Round foot, at middle of dorsum . . .	10	8 $\frac{1}{2}$

The ulcerated patch healed on treatment, leaving a rough scar. The pain also diminished. But a few days later the bleeding recommenced from the old spot, and also from the erosions beneath the toes, without any obvious cause.

Ten days after admission the patient's temperature rose rather suddenly to 102.6°, he vomited, and on examining the leg an erythematous condition was found on the foot and ankle, and there were signs of lymphangitis; the limb became excessively painful to touch.

Three weeks after admission, when all the above signs had quieted down, the limb was amputated just above the knee, by a long anterior and a short posterior flap. The stump was dressed on the tenth day and the stitches removed. Healing went on uninterruptedly, and a month after the operation

patient left the Hospital with no signs of any recurrence of the original condition in the stump.

An examination of the limb after amputation showed no abnormality of the large vessels, and no thickening of the nerve-trunks. The bones, however, were much thickened.

Comment.—I can find no description by authors of a condition similar to the one presented in this case. If it was a case of so-called "Elephantiasis Arabum," it was quite atypical, inasmuch as it was congenital, sporadic, and there was an absence of any folds in the skin, with hypertrophy of subcutaneous tissues. Moreover, here the bones were thickened—a feature said never to be present in Elephantiasis Arabum (Fagge). The case seems to fall more readily into the group classed by Dermatologists as "Ichthyosis," where the condition is often congenital, and the appearance of the skin very much as seen in this case. The hypertrophied bone, however, still remains unexplained, or rather excluded. Perhaps it is best to group the case with a few which have been recorded as "Elephantiasis" merely—using the term in a clinical sense—or again as "Pachydermia," where no parasite has been found in the lymphatics.¹

CASE II.—*Primary Sarcoma of Synovial Membrane of Knee-Joint in a Boy aged 17.*²

Abstract:—Fourteen months' history—Growth removed—Found to be confined to synovial membrane—Recurrence after 2½ years; removal—Recurrence after six months; removal—Recurrence after three months; removal—Good recovery each time—Movements of joint very good after fourth operation—Variability in "malignancy" of growth.

Alfred Batty, aged 17 years, labourer, was first admitted into Coborn Ward, under the care of Mr. Marsh, September 28, 1892, on account of a swelling over the left knee and pain.

Fourteen months before admission the swelling was first noticed, but for sixteen months patient had had "shooting pains," extending from the knee downwards, the knee itself having also been very tender at times during this period. There was no history of syphilis nor of injury; nothing abnormal noticed except the local condition.

¹ *Vide* Pathological Transactions, 1875, p. 15.

² This case is quoted incidentally by Mr. Marsh in his chapter on "New Growths in Joints," in "Diseases of the Joints and Spine," edit. 1895.

The swelling was situated just above the internal condyle of the left femur, was firm, elastic, very tender to touch, about two inches in length, and a little less in breadth—about the size of a billiard-ball. There was an inch of wasting above the knee, and half an inch below, though it is not stated at what levels these measurements were taken. It was treated by lotions and counter-irritants without benefit, and the inflammatory nature of the tumour appearing to be doubtful on account of its persistence, exploration was thought advisable. Three weeks after admission, the tumour having increased slightly in size, an operation was performed. The tumour was found to be growing from the synovial membrane of the joint, and was removed. The bone was not involved. The joint was freely opened and irrigated. The wound suppurated superficially, but the joint remained unaffected. Healing was complete by December 7, and patient was discharged. The joint was slightly stiff, but there were no signs of recurrence of the growth. The pathological report was to the effect that the tumour consisted “chiefly of spindle cells, with a minority of small round cells, and was a sarcoma of moderate malignancy.”

The boy was admitted a second time, into Abernethy Ward, December 10, 1894, complaining of a return of the pain, which was more severe than before. There was found to be marked wasting of the limb, but no shortening. A tender spot was present over the inner side of the head of the tibia. Movements were limited, but no pain was felt when the joint was manipulated. Examined under an anæsthetic, some adhesions were broken down, and the actual cautery was applied for the relief of the pain. No definite recurrence of the growth was made out, and patient left the Hospital, January 31, 1895.

On March 11 of the same year, however, he returned with increased pain, impaired movement, and an obvious recurrence of the growth. There was no shortening of the affected limb, but considerable muscular wasting, amounting to two inches above the patella, and one inch at a level eight inches below that bone. On attempting flexion of the joint, pain soon commenced, and the angle of this movement could not be made less than 150° . The joint felt warmer than its fellow, and the skin over it was very tense, the tissues beneath feeling swollen. On its inner aspect, situated just above the internal condyle of the femur, was a small lump, lying with its long axis in that of the limb, and measuring $1\frac{1}{2} \times \frac{3}{4}$ inches. It was rounded, tense, immovable, and very tender. The pain remained undiminished after a week of rest and treatment. Examination of the lungs and other viscera revealed nothing abnormal. The left femoral

glands were indurated, but not enlarged. On March 29 the capsule of the joint was exposed and opened and the tumour laid bare. This was seen to be encapsuled, a fibrous investment being formed from the ligamentous tissue around. On cutting through this, the growth was seen as a white, homogeneous, soft structure, the size of a marble. It was firmly attached to the periosteum, which was removed, together with a wedge of bone from the internal condyle of the femur. The bone appeared quite healthy. The joint was opened by the operation, being irrigated, and the capsular tissues sutured together. Recovery was uninterrupted, and the patient left the Hospital April 16.

He was admitted a fourth time, to Darker Ward, on September 17, 1895, for symptoms similar to those of previous admissions, and with a well-marked tumour situated now on the *outer* side of the knee. Since the operation in March he had had little movement in the joint. The local wasting was found to have increased, and the patella was more prominent on the affected side. The swelling "fluctuated," and was very tender. Movements were considerably restricted. On September 20, the joint was opened for the third time, and the growth, which was firmly attached to the external condyle, was freely removed, as well as the condyle itself. On section, the growth was pearly white, of gelatinous consistence, and encapsuled. Recovery was again good, and patient was discharged October 4.

In January 1896, the boy was admitted for the fifth time, to Darker Ward, with a third occurrence of the growth, situated internally, close to the seat of the original tumour. It was removed, and microscopic examination showed it to contain more fibrous tissue than previously. The boy made a good recovery. The movements at the knee-joint were limited, but very good, considering the amount of surgical interference to which it had been subjected during the past four years.

Comment.—Cases of primary sarcomatous growth in synovial membrane are probably very rare. A very similar case, however, was in Sitwell Ward during 1895—a young woman from whom the growth was removed by operation.

In the above case diagnosis was practically impossible. If sarcoma was considered at all, it was thought that the tumour sprang from the articular end of the internal condyle of the femur. Three points are noteworthy in the case: (i.) that no secondary growths were found at any time; (ii.) that the intervals between the local recurrences became less and less,—2½ years, then six months, then three months; (iii.) that the

recurrent growth showed no constant increase or decrease in malignancy as judged by microscopic examination. The first recurrence was largely fibrous, the second was almost entirely sarcomatous, and the third was again fibro-sarcomatous.

CASE III.—*Hydatid Disease of Muscles of Arm.*

Abstract:—Painless onset—History apparently dating from sudden rupture of cyst—Difficult diagnosis from hæmatoma—Suppuration of cyst—Evacuation by incisions and drainage—No signs of disease in viscera, &c.—Good recovery.

Charles East, aged 24 years, porter, was admitted into Colston Ward, under the care of Mr. Butlin, July 2, 1895, suffering from a painful swelling at the bend of the left elbow.

Three months previously patient was lifting a heavy piece of wood, when he suddenly felt his left arm "give way." Immediately afterwards, and for the next few days, he was unable to use the arm for any heavy work, on account of its weakness. At this time he had no pain in the arm, and noticed no swelling. Fourteen days after "the accident," the arm began to swell just above the elbow, giving him considerable pain for the first time. The swelling became slowly larger and more painful, and the arm quite useless for work. Since the accident he had never been able to completely straighten the forearm upon the arm. Four days before admission the swelling commenced to decrease in size, and continued to do so up till day of admission. There was no previous illness of consequence, and no history or signs of syphilis.

On admission, the tongue was slightly furred, the temperature 97° , the heart-sounds natural, the pulse 72, regular, good volume and tension, and equal at the two wrists. Nothing abnormal was discovered in the chest or abdomen. Urine was quite natural. The appetite, however, was bad and the bowels confined.

The local condition was as follows:—The left arm presented a swelling on its flexor aspect, immediately above the bend of the elbow. The biceps muscle was pushed forward, and on either side of its lower end was an ovoid swelling filling up the two bicapital grooves. The muscle itself felt natural, and seemed to be in front of the tumour; when the joint was flexed its tendon could be distinctly felt. The brachial artery was displaced forward, but still lay in the middle line. The triceps muscle and the elbow-joint seemed unaffected. There was no local heat or redness, but there was some tenderness

on pressure. The tumour itself felt tense, but fluctuated easily. The arm could not be extended more than 150° , and all movements were performed with difficulty. The patient complained of a continuous, dull, aching pain at the seat of the tumour, which increased when the joint was manipulated.

On July 12 patient was anæsthetised, and an incision was made on the inner side of the arm, along the edge of the biceps muscle. When this was carried to about an inch in depth, about an ounce of yellow, purulent-looking fluid escaped, together with several small hydatid cysts. With pressure-forceps the incision was enlarged, and many cysts, varying in size, were evacuated. A second incision was then made on the outer side, between the biceps and brachialis anticus muscles, and a connection was established between the two incisions. The cyst cavity was then drained.

On July 26 both wounds were healing well, and on August 3 patient was discharged. Seen again on August 10; both wounds were healed, and considerable use was being made of the arm. An examination of the liver and lungs by careful percussion and palpation revealed nothing abnormal. On inquiry, patient stated that he was accustomed to eat water-cresses very frequently, being "particularly partial to them."

Comment.—The case illustrates another of the various conditions where diagnosis becomes very difficult. The cyst was thought by some to be a hæmatoma, and its situation and characters, with the history given by the patient, made this suggestion very reasonable. With the knowledge revealed by the operation, the history was seen to be very characteristic of the various stages in the development of hydatid tumours.

Primary tumours in muscle are rare. A list of 12 consecutive cases, given by Erichsen, comprise five sarcomata, three hydatid cysts, and one each of the following:—fibro-sarcoma, enchondroma, fibro-cystic tumour and simple cystic tumour. These, Erichsen thinks, represent the nature of primary growths in muscle in fairly proportionate frequency. But of 62 cases of primary tumours in muscle collected by Teeran, only five were hydatid cysts, the order being: sarcoma (20), fibroma (16), simple cystic growth (8), hydatid (5), nævoid (5).

As regards the distribution of hydatid cysts in the body, Duraine's large collection of over 1800 cases only contained 30 in muscle and connective tissue.¹ Leuckart states that one hydatid cyst exists elsewhere for every three in the liver. Fagge thinks even this proportion too high. Duraine's figures would give the liver as the seat of disease in just 50 per cent. of cases.

¹ Dictionnaire Encyclopédique des Sciences Médicales, tom. xxxii. 1835.

CASE IV.—*Empyema of Gall-bladder.*

Abstract:—Only six days' history—Opened and freely drained—Recurrence ten months later—Opened again and drained—Gall-stones removed from cystic duct and gall-bladder—Good recoveries after both operations—No jaundice at any time.

Mary Jane Stacey, aged 32 years, housewife, was first admitted into Sitwell Ward, under the care of Mr. Butlin, on January 2, 1895, suffering from pain and tenderness in the right side of the abdomen.

Six days before admission patient had an attack of pain, not severe, between the shoulders. Next day she shivered, had a bad headache, and felt very thirsty. On the third day of her illness she suffered severe pain in the right side of the abdomen; on the fifth and sixth days she vomited all her food. The bowels were open twice after medicine on the fourth day, but not since.

As regards her past history, menstruation began at 16, and had always been natural. As a child she suffered from measles, chicken-pox, and small-pox, which last disease was followed by abscesses in both legs. She had four children living, had lost one from bronchitis, and had had two miscarriages. There was a strong family history of tubercle; her father and one brother died of phthisis, and one sister had curvature of the spine.

Four years before admission the patient suffered from "inflammation of the body," with acute pain, tenderness, and swelling, which condition was treated by fomentations. Off and on for the year before admission she had suffered from painful micturition.

On admission the local condition was as follows:—A tender swelling extended from the right costal arch to a spot three inches below the umbilicus. It moved with respiration, and did not extend backwards into the loins. It was ill-defined, and of no particular shape. The patient's temperature was 101.6°, and she sweated much at night. There was no cough, and there were no signs of lung disease. The urine contained a cloud of albumin.

On January 8, patient having remained in much the same condition, an exploratory incision was made midway between the crest of the ilium and the costal margin on the right side. The kidney, on examination, was found to be natural, but the swelling was discovered to be in front of it. The wound was closed. On January 11, no improvement having taken place, a second operation was undertaken, the incision being made in

the right *linea semilunaris*. A fluctuating swelling was exposed just beneath, and intimately connected with, the liver; pus escaped from this when punctured with a trocar. The wall of the abscess was stitched to the abdominal parietes and the cavity freely drained. Five ounces of pus were let out, but no gall-stones. A probe passed in some ten inches towards the upper part of the swelling seemed to touch something gritty. The cavity was irrigated and drained and the wound dressed. The sinus subsequently discharged mixed pus and mucus, and the cavity later healed completely. Patient was discharged on March 9 with a very slight superficial wound.

After leaving the Hospital the wound completely healed. From this time patient suffered no pain, and did her work as usual. In August she had another miscarriage, and was laid up in consequence. She, however, quite recovered her health until October 1, when severe pain and tenderness reappeared rather suddenly in the old situation, as well as some swelling. She lay up and had medical advice; but getting no better, she came again to the Hospital, and was readmitted on October 10.

At this time patient appeared to be a fairly healthy woman, of cheerful expression and good complexion. There were no signs of jaundice either in body tissues or urine, and no history of it. Head, neck, tongue, and chest were all apparently natural. Respirations were shallow, and interrupted on account of spasmodic pain "right in the old wound," and which seemed to be very severe. The general shape of the abdomen was natural, but the movements were slight on respiration. In the right *linea semilunaris* the old scar was seen, and this marked the centre of a swelling situated beneath the abdominal wall, stretching from the costal arch to the iliac crest, and for two inches each side of the scar. The swelling moved freely with respiration, and the whole region was very tender to palpation. The kidney was not felt. The tissues around the scar were red and swollen. The urine was acid; sp. gr. 1028; a cloud of albumin; a deposit of urates; no blood, pus, casts, crystals, or bile. Four days after admission the abscess "broke" at the seat of the scar, discharging an ounce of white, thick pus. The pain was much relieved; the sinus continued to discharge; no stones were discharged. The colour of the motions was quite natural.

On October 16, under chloroform, the sinus was opened up, but previous to this the abdominal cavity was opened well below the orifice of the sinus. On opening the peritoneal cavity, the intestines were found adherent to the parietal peritoneum in this situation. Through this wound digital

examination of the gall-bladder and ducts was made. A stone was discovered in the cystic duct and others in the gall-bladder. The sinus was then enlarged and carefully probed. It led into what was found to be the altered gall-bladder, from which several pieces of stone were removed by forceps. By means of the finger in the lower wound, a larger, round, non-faceted stone was pushed from the cystic duct into the gall-bladder, and removed through the upper wound. It was the size of a marble. The tissues composing and around the gall-bladder were felt to be indurated and matted together. The lower wound was then sutured by deep stitches; the upper was left to drain into separate dressings.

The following day the patient vomited a large amount of greenish liquid, and this continued on the third day also. Only warm water and a little milk were given *per os*, nutrient enemata being also administered. The urine was throughout free from bile, and the motions were of a natural colour. By the fifth day the lower wound was quite healed; the upper had been discharging a greenish limpid liquid in large amount, but free from pus. Temperature remained normal, and the patient made an uninterrupted recovery. The discharge from the biliary sinus diminished in amount, and on the tenth day the tube was removed. A probe failed to discover any stone present. Patient left the Hospital twenty-one days after the operation, with the sinus quite healed, and with no pain or tenderness in its region.

Comment.—The case is of interest chiefly in showing advances that have been made of late years in the surgery of the gall-bladder. The cause of the trouble was here blocking of the cystic duct by a stone. The absence of jaundice indicated patency of the common duct. The case was therefore one of the most favourable for operation. As the result of the first operation the gall-bladder was reduced to a very small sac, with thickened walls, bound down by adhesions, but it was still able to be the seat of formation of calculi. After the second operation there could not have been much more than a dilatation of the common duct left to represent the gall-bladder. If so, the tendency to the formation of stones must have been permanently counteracted, if it is true, as stated on good authority, that stones never form except in the gall-bladder itself. The suppuration occurring in such cases as this argues against the "antiseptic" properties of bile. The secretion, however, is now known to contain micro-organisms; indeed, these are by some supposed to set up changes which favour the formation of concretions.

CASE V.—*Traumatic Gangrene of Leg in a Man aged 67.*

Abstract:—Injury slight in extent—Signs followed in four days—Rapid development—*In extremis* on admission—Death eighteen hours after admission.

Charles Joseph Foulkes, aged 67, clerk, was brought to the Hospital about 7.30 P.M. on March 10, 1895, on account of a gangrenous condition of the left leg.

The patient was quite well until three weeks previously, when the little toe of his left foot was run over by a perambulator, causing a slight abrasion, which had never healed well, but caused him no great inconvenience. Eighteen days before he was seen at the Hospital he slipped off a railway platform on to the rails, bruising his left ankle and heel and scratching his great toe. For four days after this accident patient got about without difficulty. Then the foot began to swell, becoming also red and tender. A doctor ordered poppy-head fomentations and linseed poultices to be applied; "blisters" formed after these applications, and at the same time "red streaks" were noticed running up the leg. The swelling and redness continued to spread—at first slowly, but during the latter three or four days more rapidly, and black patches appeared on the inner side of the foot and back of the leg. There was great pain in the affected parts. Food was taken fairly well, but patient was very restless at night. On the recommendation of his doctor, patient was brought to the Hospital, and was at once admitted into Casualty Ward, under the care of Mr. Marsh.

Nothing of importance could be made out with regard either to his family or previous history; his mind seemed confused and clouded. He denied habits of alcoholism in any form, and syphilis.

On admission, patient was very pale and thin, looked older than his years, the expression of his face being extremely anxious and worn. The eyeballs were very prominent. The tongue was coated by a thick white fur. The bowels had not been opened for two or three days. The pulse was increased in frequency, of medium volume and tension, and the artery at the wrist did not feel hard or tortuous. Temperature in the mouth was 101°. The urine was acid, contained a heavy cloud of albumin, but no sugar.

The whole of the left leg as far as the knee was much swollen. The skin of the foot was ashen-white in colour, colder than that of the right foot, and presented patches of blue discoloration, chiefly on the inner side and about the heel. There was a very

small wound, fairly clean in appearance, on the little toe. The lower third of the leg was of the same colour as the foot, but in the upper two-thirds the colour gradually became bright red anteriorly, while posteriorly they were covered by patches of a blue colour, looking gangrenous. The redness disappeared slowly on pressure, and reappeared slowly. There was a single blue patch on the inner side of the thigh, about two inches above the knee. Sensation was completely lost in the foot, but the leg was very painful to the touch. No enlarged glands were found in the groin, and there was a total absence of any offensive smell.

Shortly after admission chloroform was administered, and about thirty or forty linear incisions, about two inches long, were made on the dorsum of the foot and back of the leg. A considerable quantity of dark red blood oozed away from these wounds, which were then plugged with iodoform gauze and dressed. The whole leg was then well wrapped in cotton-wool, raised, and kept warm by hot bottles. The anæsthetic was taken very well.

During the night the pulse became very feeble. A strychnine injection was given, with a temporarily good result. Towards morning the breathing became irregular and stertorous. Small doses of brandy were given continuously after admission, and at 10.30 A.M. the strychnine injection was repeated. An hour later the dressings were removed and the leg was placed in a hot bath. This could not be continued, owing to the feeble heart action. The patient sank rapidly, and died at 1.15 P.M. the same day, having been in the Hospital only eighteen hours. No post-mortem examination was obtainable.

Comment.—The case seems to have been one of “true spreading gangrene,” the *gangrene foudroyante* of Maisonneuve. An acute infective process is present in these cases. The gangrene sets in before suppuration has started, and on examination one usually finds colonies of the rod-shaped bacilli found in earth, &c., present in the gangrenous tissues. There is no tendency to limitation of the gangrene. General prostration comes on early, and a fatal result is usual within three or four days after the symptoms have become marked. The usual traumatic gangrene runs a somewhat different course. Diabetes was excluded by examination of the urine; the albuminuria may have indicated a condition of the kidneys associated with arterio-sclerosis. The man’s age (67) was doubtless an important point, though not evidence in favour of the process being of an acute infective nature, since this form of the disease may apparently occur at any age.

CASE VI.—*Tumour of Groin in a Woman aged 61.*

Abstract :—Three months' history—Carcinomatous character of tumour—No sign of any primary growth—Pigmented condition of great toe nail—No operative interference—Discharged *in statu quo*.

Caroline Augusta Ciavarelli, aged 61 years, widow, was admitted into Sitwell Ward, under the care of Mr. Butlin, September 27, 1895, on account of a lump in the left groin.

On June 6 of the same year, patient was lifting a heavy chest, when she felt something "catch" her in the left groin. There was no pain and no sickness. Next day she noticed, for the first time, a lump the size of a horse-bean in the situation occupied by the tumour to be described. It was not tender, but it had gradually increased in size until time of admission. Patient had had no illnesses since June; her bowels had been freely open, and she had done her usual household work without trouble.

Besides an attack of inflammation of the bowels twenty years previously, and pleurisy five years previously, patient had had no diseases. Her husband and one brother had died of phthisis. There was no family history of malignant disease.

Patient looked a healthy woman, was fat, and complained of nothing but the presence of the lump in the groin, which was not painful, but caused anxiety as to its nature. Nothing abnormal was discovered in face, neck, or chest. The abdomen moved well, was nowhere tender, and presented no fulness or resistance. Several small, bright, raised petechial spots were scattered over its surface.

Lower limbs: Right leg was quite natural. The left leg presented two abnormal conditions. (i.) The tumour mentioned above, occupying the groin, and situated half above and half below Poupart's ligament, was a dome-shaped circular swelling about the size (and shape) of a penny bun, very hard in consistence, fairly freely movable—more so laterally than vertically—and well circumscribed below, though not above. Its surface was not uniform. Externally, the examining fingers could limit the growth easily; internally, it seemed to have connections with the contents of the pelvis, as the free edge could not be felt. The skin over it was purple in appearance, and slightly adherent in one or two places. It was only very slightly tender on manipulation. (ii.) The great toe nail was much discoloured. This was injured by a falling body fifteen years previously; the nail came off, and the new one became blue some time after its

appearance. It "swelled up" every now and again, and the colour increased in intensity, then it "went down again." But it had been constantly coloured for fourteen years at least. No other sore or abnormality was discoverable; no enlarged glands in popliteal space. The joints were all freely movable.

Upper limbs: At the distal phalangeal joints of all the fingers were irregular hard nodules projecting from the joints, making movement in them much restricted. They presented all the characters of "Heberden's nodes" seen in osteo-arthritis.

Micturition was performed naturally, and the act was not increased in frequency. No soreness about the genitals. Urine: sp. gr. 1020; acid; a cloud of albumin; a few pus cells. Bowels were rather constipated. A vaginal examination by Dr. Champneys gave the following result:—"Vagina in senile condition, small, presenting two pouches superiorly, which might each contain a pea; the left and more anterior one probably contains the cervix. A condition of senile vaginitis is present, the walls of the vagina easily bleeding. *Per speculum*, nil abnormal. No signs anywhere of malignant disease." The catamenia had commenced at 14, had ceased at 46, and were always natural in rhythm and amount.

Patient was seen at "Consultations."

Mr. Butlin considered the case to be one of those where an apparently secondary carcinomatous growth in the glands was present, and where the primary growth had aborted. Mr. Bowlby considered the condition of the toe nail important, though he confessed that the course of the disease, if really starting from this, was quite atypical. Mr. Lockwood instanced a case, in support of Mr. Butlin's suggestion, where secondary growths had been removed from glands, no primary growth being discovered. All agreed that the extent of the tumour rendered any operative measures impracticable.

A fortnight after admission the patient was discharged, the local condition remaining practically unaltered.

Comment.—Undoubted instances seem to have occurred where a well-marked "secondary" growth has appeared, the "primary" source of which has aborted. The evident carcinomatous nature of the tumour in this case makes such a conception rational. The idea seems to favour Paget's view of the nature of cancerous dissemination,—that in some instances, at least, "secondary" growths may arise from an increase in the "cancerous diathesis," or morbid state of the tissues. If so, it seems not unreasonable to suppose this particular growth one which had increased rapidly, and focussed, as it were, the

morbid principle, thus causing the "primary" neoplasm to remain rudimentary or be absorbed. But there is a second view possible in this case,—that the growth was secondary to a melanotic malignant deposit in the toe; for secondary growths in these cases are not seldom colourless, occur in almost every organ of the body, and grow very rapidly and extensively.

Two points of secondary interest were the number of petechial spots present, and the "Heberden's nodes" upon the fingers. The former condition is said by some authorities to accompany malignant disease not infrequently, and Charcot affirmed that the latter condition was significant of the same tendency. All persons with petechiæ, and all persons with Heberden's nodes, do not develop malignant disease; but the co-existence of all three conditions is perhaps worthy of note at a time when we are so much in the dark as to the relations, causal or otherwise, of new growths.

CASE VII.—*Ruptured Kidney after a Fall.*

Abstract :—Marked collapse after accident—Immediate hæmaturia—Pain in left loin—No signs of fractured pelvis—No pain on micturition—Persistence of hæmaturia for five weeks—Complication: pyuria—Recovery.

James Needham, aged 21 years, butcher, was brought to the Hospital at 4.15 P.M. on October 28, 1895, in a collapsed condition after an accident.

About ten minutes before patient was seen at the Hospital, he was carrying a sack of onions up a ladder to a loft, when the ladder slipped, and he fell forwards—striking the ground with his left hip—a distance of about 12 feet. He felt badly shaken, but was not unconscious, nor did he feel stunned; he rose to his feet without assistance. He then began to stagger, and felt sick, but did not vomit. He found he could not walk alone, so he was assisted by a couple of friends to the Hospital, about 200 yards off, arriving ten minutes after the accident.

On reaching the Hospital, the man was seen to be suffering from shock; his face, ears, and neck were blanched, and sweating profusely. He walked with difficulty, even when supported, and could not lie down without first being lifted off his feet, owing to pain in the region of the pelvis. The pupils were equal, of natural size, and reacted to light. Pulse was small, of low tension, but of increased frequency (120). Respirations were hurried and shallow (58). On examination, no blood was found at the end of the penis, and there were no signs of bruising anywhere. Pressure upon the iliac spines

caused no pain, nor did palpation of any part of the pelvic bones. There was a tender region just above and in front of the left ilium, also extending backwards into the loin; it was too tender here to admit of deep palpation.

For some ten minutes or so the man was unable to "pass water," but on doing so then, he passed four ounces of almost pure blood, bright red and free from clots. The stream was of natural size, and there was no trickling at the end of the act. Subsequently, after being allowed to stand, the greater part of the blood clotted characteristically. The man had passed his water just previous to the accident.

The man was immediately admitted into Charity Ward, under the care of Mr. Butlin, and his temperature was there found to be 96° . There was no pain except on movement, when it was felt in the tender region noted above. There was also a good deal of tenderness over the left pubic bone, but no irregularity was detected in it.

During the night of the 28th patient slept at intervals, but was very restless. At 9 P.M. six ounces of urine were passed containing quite four ounces of blood as well as some clots. At 11 P.M., and again at 3.30 A.M., about five ounces of fluid were passed similarly constituted. The specimens, on standing, showed a layer of dark-coloured urine on the top of a deeper layer of blood. These specimens were passed naturally and without pain. At midday on the 29th patient was still very restless; his face was blanched, the skin cool and moist, the tongue and mouth very dry and parched, and coated with stringy mucus. Patient complained much of thirst. Pulse 128, still small. Respirations, very irregular, 40. The temperature had risen since admission (96°) to 99° . There was no vomiting, and the bowels were not opened. Patient was fed as follows:—After admission, for two hours, hot water was given in one-ounce doses; then during the night hot water and mutton essence in similar quantities; milk and water in the morning.

On the 30th (second day after admission) a general improvement set in. The face was a better colour, and patient had a fairly good night. Locally, the left lumbar region was still very tender and the abdomen held tense, but no tumour was discovered. Throughout the whole of the 29th and 30th the urine was passed at intervals of about three hours, and contained blood in diminishing quantities, but still about one ounce to every six of urine. On the evening of the 30th, however, the urine became rather suddenly freer from blood, and on the morning of the 31st only contained just enough to

be visible to the naked eye. Its reaction was acid, and no crystals or casts were present. At 3 P.M. on the 30th the temperature reached 100.2°.

During the 31st of October (fourth day) the improvement continued, and the pain became less. The temperature remained up (100°). The pulse was 100, volume improved, also tension. On November 1 patient was still better; temperature fell to 99°. The urine contained a fair amount of blood on microscopic examination, the red and white cells being in natural proportions. The pain did not abate much on November 2, and the temperature remained raised (99.8°). No blood in the urine on naked-eye examination, but discovered microscopically. The abdomen was tender, and, for the first time, a hard mass was felt on the left side, in the position of the tenderness, but not distinct in outline.

For four days the urine remained free from blood, but on November 8 it again became port-wine coloured, and deposited a layer of blood cells on standing. The general condition continued to improve; temperature normal. From this date onwards the amount of blood in the urine fluctuated, until November 30, whence it steadily decreased until December 10, on which day the urine was quite free from red-blood corpuscles. Blood had thus been present for forty-four days. Leucocytes (? pus cells) were, however, found in some quantity. The reaction was acid throughout. At this last date pain was quite absent.

There was a deposit of leucocytes on standing, and a cloud of albumin on boiling, up till December 14, when the patient got up. On going to Swanley Convalescent Home on December 24, the condition of the urine was much the same: specific gravity, 1020; very slightly acid in reaction; a cloud of albumin on boiling; a deposit of white cells, with both single and multiple nuclei.

Comment.—This is a fairly typical case of injury to the kidney resulting from a fall. It illustrates most of the common characters found in a rupture of moderate severity. The collapse was very marked; the temperature chart indicates a subnormal temperature after the accident, a raised temperature during the second to the fifth day, and a normal temperature after the fifth day. The persistence of the hæmaturia for a period of five weeks, with intermissions, probably indicates a wound of considerable extent. The pyuria following the hæmaturia is, according to Henry Morris, a not unusual sequela, and is due to a pyonephrosis.¹ It is worthy of notice, in this connection, that the reaction of the urine remained acid throughout.

¹ Surgical Diseases of the Kidneys, edit. 1886

CASE VIII.—*Fractured Pelvis, with Extra-Peritoneal Rupture of Bladder, after a crush.*

Abstract :—Collapse not marked—Immediate hæmaturia, which lasted fourteen days—Increased mobility and pain on manipulation of pelvic bones—Abdominal distension and tenderness, simulating peritonitis, but relieved by rectal tube—Complication : cystitis—Treated by irrigation—Recovery.

Thomas Forwood, aged 29 years, labourer, was brought to the Hospital on September 6, 1895, suffering from the effects of a crush.

About 7 A.M. on that day the man was assisting in the destruction of some hoarding, and was supporting a large piece of it whilst it was being dislodged. Finding it too heavy for him, he left it, but was overtaken in his attempt to escape and knocked down by the falling timber. He was dragged from under it by his arms in a fully conscious condition, but was quite unable to walk owing to pain "in the stomach," which "doubled him up." He sat down for half-an-hour, but finding the pain still present, came to the Hospital in a cab, arriving just an hour after the accident.

On reaching the Hospital, he complained of pain in the lower abdomen and right thigh. The bladder was found to be distended, no urine having been passed for some eight hours. A little blood was seen at the end of the penis. A rubber catheter (No. 7) was passed; a few drops of blood first appeared, then blood-stained urine, and lastly almost clear urine. Altogether nine ounces were drawn off. The abdomen moved well and the man's general condition was fairly good. He was admitted into Paget Ward, under the care of Mr. Butlin. The catheter had been tied in, and shortly after admission a little blood-stained urine was passed through it. On examination, it was found that pressure on the pubes gave some pain, and pressure inwards over the anterior superior iliac spine caused severe pain. There was also increased mobility of the pelvic bones.

Later in the day a soap-and-water enema was given, but without result. About eight ounces of blood-stained urine flowed away through the catheter. The man was very drowsy and slept well.

On the morning of the 7th patient was put upon a water-bed; the temperature was raised slightly. Towards night there was increased pain in the abdomen, which was distended and tympanitic. The possibility of peritonitis being indicated, nutrient enemata were given, but these were rejected with a quantity of

flatus. The patient was sick, vomiting green fluid containing bile. The hæmaturia persisted.

On the 8th patient was better and the temperature normal. Towards evening there was a return of the abdominal distension; a rectal tube was passed, and this produced great relief. No sickness. The urine still contained blood.

By the 11th the patient had considerably improved; the hæmaturia was only slight. The constipation had been overcome by castor-oil. By the 17th the urine had become markedly alkaline, so that the bladder was washed out by a saturated solution of boracic acid. The urine only contained a trace of blood, and there was freedom from pain. The urine was still allowed to drain away through the catheter.

On the 19th (14th day) the urine was free from blood and the catheter was removed; there was no difficulty in micturition.

On October 4th patient was still doing well; his condition improved rapidly until October 18, when he was discharged, after being in the Hospital just four weeks.

Comment.—Here the hæmaturia contrasted markedly with that of Case VII. in only lasting fourteen days. The pyuria, however, was only slight. The urine, too, became early alkaline, and strongly so; bladder affections more commonly give rise to alkalinity of the urine than do kidney diseases. As regards the source of the blood, the diagnosis of extra-peritoneal rupture of the bladder met the facts of the case best; fractured pelvis is far more commonly complicated by extra- than by intra-peritoneal rupture of the bladder. The temporary abdominal distension, vomiting and pain, coming on shortly after admission, gave some semblance to peritonitis being present; but the use of a rectal tube relieved these symptoms: the distension was intestinal.

CASE IX.—*Extensive Tuberculous Disease of the Urinary Tract in a Woman aged 34.*¹

Abstract:—Long history (four years) of increased frequency of micturition—Short history (five weeks) of hæmaturia—Only slight pain—Pyuria well marked—Rapid aggravation of symptoms—Suppression of urine—Death after three weeks in Hospital—Bilateral pyonephritis—?Congenital deformity of ureteral orifices.

Eliza Howard, aged 34 years, housewife, was admitted into Stanley Ward, under the care of Mr. Marsh, November 18, 1895,

¹ *Vide* Museum Collection of Photographs, No. 649b. The ulceration and deformity of the bladder are well shown.

complaining of passing blood with her urine, and of passing it frequently.

For four years the patient had had increased frequency of micturition, and sometimes trouble in holding her water. She had suffered no definite pain, however, and nothing was noticed unnatural about the urine. Five weeks before admission she first noticed she was passing blood in her urine, with which it seemed mixed. At this time patient was passing her water six or seven times in the day, and as often during the night. There was no increase in this frequency up till date of admission; but the amount of blood had much diminished for eleven days previous to admission.

Patient had had no bad illnesses. She had had three children, her last confinement being two years before admission. There was no family history of malignant disease or of tubercle.

On admission, the woman looked thin, but not very ill. Appetite good, bowels open once daily, temperature 98.6° , slept well. The chest showed no signs of disease. *Per hypogastrium*, nothing abnormal was felt. The urine was passed six times in the day, and as often in the night. The specimens were thick and cloudy, sp. gr. 1010, reaction alkaline, blood and pus, both in small quantity.

After resting in bed a week, patient was anæsthetised and the bladder was sounded. No stone was found. The urethra was then dilated, and the finger, on being passed into the bladder, discovered a velvety condition of the mucous membrane, and a small rough (? ulcerated) patch on the left side. The fundus of the bladder was invaginated.

On November 28, patient seemed a little better, but still weak. The urine was drawn off by a catheter, and the bladder was washed out twice daily with boroglyceride solution. On December 1 patient became worse, and continued so until December 6, when she vomited, looked worn and haggard, and was very drowsy. The temperature, which had risen to 99.6° on November 29, now fell below normal. The face became very pinched. From this date the quantity of urine passed became rather suddenly smaller, and consisted largely of blood and pus, which latter constituent gradually increased in amount.

On December 7 patient was much worse, and evidently in a critical condition, lying in a semi-comatose state. A peculiar smell as of decomposing urine came from the bed. No urine was passed for eighteen hours. A catheter being used, only one ounce came away, and this was almost entirely pus. The odour became more marked. Later in the day, pilocarpin gr. $\frac{1}{6}$ was given hypodermically, but with no appreciable benefit.

Hypodermic injections of strychnine were also given, and nutrient enemata. But the patient sank without rallying, and died at 9.30 P.M. Shortly before death the thermometer registered 97° in the axilla.

Post-mortem examination.—Abdomen only was examined. Intestines: three or four inches near the cæcum presented an appearance as of being the seat of old ulceration. Here the calibre was considerably narrowed. Kidneys: the left kidney was a mere bag of pus, no actual kidney substance being found. The right kidney presented extensive ulceration in the pelvis, the cavity of which was filled by pus, and an abscess as large as a walnut in the kidney substance. A quarter of the whole kidney was replaced by tubercle. Neither organ was materially enlarged. Ureters: both were much thickened and ulcerated on their inner surfaces. Bladder: almost wholly covered by ulceration internally. The fundus was invaginated. In many places the muscular fibres were quite bare. There was much recent congestion, and the cavity contained two ounces of thick, ropy pus. In the region of the trigone, and a little to the left side, was a flattened circular tumour, an inch in diameter, and raised a quarter of an inch from the general surface. Its surface was soft and bluish in colour. To the naked eye this looked like a nævus which had undergone fibrous changes. Running from the right upper extremity of this sessile tumour was a band of tissue, $\frac{5}{8}$ inch long and $\frac{1}{4}$ inch broad, attached to the bladder-wall at the orifice of the right ureter, which was actually situated upon this bridge of tissue. When raised, the mucous membrane beneath the band appeared similar to the mucous membrane elsewhere. The appearance suggested congenital deformity. On the left side, also, there was a faint ridge seen running from the sessile growth, looking like a rudimentary condition similar to the more advanced one on the right side.

Comment.—The cardinal symptoms in cases of new growth of the bladder are three—increased frequency of micturition, hæmaturia, and pain. In the above case the sequence of these symptoms was as they are here written. The pain, however, was markedly slight throughout. The history extended over a period of four years, and there was an extremely rapid aggravation of symptoms towards the close. Pus was present in the urine with the blood in fair amount, increasing very considerably later, until the urine seemed to consist almost entirely of pus. The condition of the kidneys (pyronephrosis and pyonephritis) would account for this, and it seemed, from the condition of both these organs, that the condition of the

bladder was most probably secondary to the kidney disease. Such is stated by most authorities to be the more usual course of tuberculous disease in these parts. The ulcerated ureters are of importance in this connection. The abnormality at the ureteral orifices is interesting developmentally, but seems to have been quite independent of the disease.

CASE X.—*Malignant Disease of Bladder in a Woman aged 53.*

Abstract:—Only three months' history of (1) pain; (2) hæmaturia; (3) frequent micturition—Removal of growth *per vaginam*—Complication: cystitis—Good recovery.

Fanny Aubertin, aged 53 years, widow, was admitted into Sitwell Ward, under the care of Mr. Butlin, September 14, 1895, complaining of abdominal pain and of passing blood in her urine.

Three months previously pain had commenced in the lower part of the abdomen, seeming to be situated "in the passages." This continued until seven weeks previously, when blood was first noticed in the urine. Patient consulted her doctor, who kept her in bed until fourteen days previous to admission, when she got up, the symptoms having diminished. Thence, up till date of admission, there had been no aggravation of the condition. A month before admission the doctor had examined the bladder for stone, had found none, but had told the patient she "probably had a cancer." The bladder walls had been found "swollen and tender:" no anæsthetic had been used. At this time blood was being passed at the beginning and end of micturition, and the act was only kept under voluntary control with difficulty. The urine was also passed very frequently.

Patient had had no other illnesses. There was no family history bearing on the case. Catamenia had commenced at 17, were regular and average in amount, and had ceased nine years previously.

Patient was a fairly healthy-looking woman, showing no appreciable wasting. Nothing abnormal was discovered besides the urinary trouble. The umbilical region of the abdomen was rather tender when examined, and the tenderness was confined to the middle line, between the navel and the pubic crests. Neither bladder nor kidneys were felt. *Per vaginam*, the anterior bladder wall was found a little prolapsed; the urethral orifice was very tender, swollen, and red. Urine: 33 ounces were passed during the twenty-four hours following admission, containing a few red blood cells and crystals of triple phos-

phate; reaction alkaline; sp. gr. 1020; no sugar. Two days after admission 49 ounces of urine were passed, which separated, on standing, into a fluid and a solid portion. In the former, blood was visible to the naked eye. In the latter, two layers were distinguished, the upper consisting of amorphous phosphates coloured by blood, with some pus cells; the lower of a viscid substance holding blood and pus cells in its meshes, together with some bright red shreds $\frac{1}{4}$ to $\frac{1}{2}$ inch long. Reaction was alkaline; no casts; some epithelial cells, which seemed to be normal bladder and vaginal cells on examination with the microscope.

Temperature on admission was 97.8° , normal afterwards. The pain was increased during micturition and any movement of the parts. The urine was passed eight times in twenty-four hours.

On September 17th the patient was anaesthetised, and the bladder, after being evacuated, was washed out with boracic acid solution until the washings were clean, and filled with six ounces of the fluid for cystoscopic examination. Looking posteriorly, nothing was seen; looking anteriorly, some form of growth was made out behind the pubes, the nature of which could not be ascertained. On sounding, this mass was again felt. Digital examination of the bladder walls *per urethram*, with the patient in the lithotomy position, was then made. Little finger only could be introduced, so the anterior wall of the urethra was nicked with a curved bistoury, to allow passage of index finger. By this means, just near the urethral orifice, and to the right of middle line, a raised, firm, adherent mass was felt, covered by gritty material, and ulcerated upon the surface. It was about the size of a small walnut. The growth, judged to be malignant, was removed, and found on extraction to be as large as a pigeon's egg. The position of the growth made its removal difficult, scrapers and a Volkmann spoon being used. There was not much bleeding. The parts were irrigated well, and a large rubber catheter was tied into the bladder.

First day after operation: 15 ounces of urine passed through catheter in first twelve hours, containing much blood; dressings also soaked with blood; a good deal of pain, requiring morphia; temperature 98.4° .

Second day: Small quantities of urine passed at a time (one ounce or so), fairly free from blood; urine drawn off at intervals, leaving catheter clipped between the times. Somewhat suddenly on this day about two ounces of pure blood with clots came away, after which the specimens became much clearer; reaction acid; temperature 99.7° .

Third day: Still much pain and tenderness, and some abdo-

minimal distension, relieved by passage of rectal tube. Patient vomited two or three times; morphia necessary to procure sleep; temperature normal; urine again became alkaline, containing blood and pus.

Fourth day: Temperature 100° ; pain less; bowels opened by enema; urine alkaline; a few clots passed; catheter removed; no sickness.

Sixth day and onwards: Gradual improvement; vagina syringed out three times daily with boracic acid solution, as urine was strongly alkaline and contained much pus; temperature still 100° .

Ninth day: Bladder syringed out as well as vagina, the cystitis being still considerable. Condition improved under treatment until eighteenth day, when patient got up. The urine was then acid, and only contained a few blood and pus cells on microscopic examination; no pain. Good progress made until October 19th (thirty-second day), when patient was discharged. The urine was quite free from blood, and contained only a few pus cells; reaction acid. Patient was taught how to wash out her bladder, and was instructed to do this daily if necessary.

Comment.—The history was very short in this case, and the series of symptoms, in order of appearance, was (1) pain; (2) hæmaturia; (3) frequent micturition. The success of the operation probably turned upon the fact that an early diagnosis had been made. The possibility of doing this in the case of the female bladder, without an exploratory incision, is a point of considerable importance. As in many other operations *per vaginam*, the recovery was rapid. No abdominal wound had to be dealt with.

CASE XI.—*Malignant Disease of Bladder in a Man aged 60.*

Abstract:—Admitted three times for hæmaturia, which ceased during first and second stay in Hospital—Frequent micturition—No very bad pain—Suprapubic cystotomy $2\frac{3}{4}$ years after appearance of hæmaturia—Extensive malignant growth, not removed—Discharged *in statu quo*.

John Mitchell, aged 60 years, navy, was admitted into Colston Ward, under the care of Mr. Butlin, complaining of passing blood in his urine,

First in February 1893. He had then only noticed the blood three days. There had been some aching in the back, but no definite pain. After being in the Hospital a week, the hæmaturia ceased, and patient was discharged.

He was admitted a second time in May 1894, when he was passing urine at times dark with blood, at other times nearly clear. At times small clots were passed. There was still very little pain. Patient was sounded for stone with negative result. Cystoscopic examination revealed nothing. The urine again became free from blood, and he was discharged a second time.

He was admitted (into Darker Ward) a third time on September 2, 1895, his condition since the second discharge having varied; sometimes he passed a good deal of blood, sometimes none at all. Two months previous to this admission, however, he commenced to have pain at the root of the penis after micturition, lasting for five minutes. When the blood passed was most this pain was worst. There was no difficulty with the act itself. Fourteen months previously he had noticed he was unable to contain his water for longer than two hours together. The blood usually came with the last few drops of urine. On admission there was still no very bad pain.

On admission the man looked strong and well nourished, presenting no signs of wasting. He gave no family history of malignant disease or of urinary trouble. The abdomen was fat; no viscus was felt, and there was no tenderness. Micturition: patient passed his water every two hours; the stream was feeble and small; at first the urine was almost clear, the last few drops were mixed with much blood. After the act there was a little pain at the root of the penis, lasting a couple of minutes, and some pain over the sacrum. The urine: specific gravity, 1020; acid; blood and blood-clot; some shreds of tissue bearing the appearance of belonging to a firm villous growth; oxalate crystals. Examination *per rectum* discovered the prostate hard and enlarged, but no tumour was felt.

On September 10 patient was anæsthetised, the bladder was emptied and washed out with boracic lotion, and a cystoscopic examination made. Nothing definite could be made out. A rectal bag was introduced, the bladder filled with boracic lotion, the penis ligatured by rubber-tubing, and suprapubic cystotomy performed. The bladder being opened, examination was made of its interior digitally, and visually by means of a laryngoscopic mirror. A growth was found on the anterior wall, extending from the orifice of the urethra almost to the angle of the incision, about $2\frac{1}{4} \times 1\frac{3}{4}$ inches in extent, with raised and indurated edges and uneven surface. This was evidently malignant in nature, and no attempt was made to remove it. No smaller growths were found. A soft rubber catheter was tied into the bladder through the penis. The edges of the bladder were sutured by silk, and the skin incision by a continuous

catgut suture. The lower angle of the wound was drained and the whole dressed.

September 11. Dressed; wound healing well; the urine contained blood. Temperature normal. September 12. Temperature 99.6° (highest point reached); urine still bloody, but patient doing well. September 21. Tube removed from wound; No. 10 catheter tied into urethra, but removed three days later. September 28. Patient got up and went into Hospital Square, eighteenth day after operation, having recovered very rapidly. He could not hold more than two ounces of urine at any time. October 5. Patient left Hospital; urine was acid, contained a little pus and a very little blood. Sent to Swanley Convalescent Home.

Comment.—This case contrasts well with Case X., inasmuch as removal of the growth was impossible on account of its extent, the condition having been diagnosed after two and a half years' history. Here again the pain was not marked; hæmaturia was the first symptom noticed. The large extent of the growth was not suspected prior to the operation, and it is worth noting that an exactly similar case (as regards the result of exploration) was present in the ward at the same time. Both cases were found to be beyond surgical help. They illustrate a point borne out in the consideration of all the cases of bladder tumours in the Hospital for the past six years, that removal of carcinomatous growths is rarely undertaken, because they are usually detected at so advanced a stage (*vide* Tables).

CASE XII.—*Compound Villous Tumour of Bladder in a Man aged 52.*

Abstract:—Long history (five years) of hæmaturia—Intervals of relief from this—Frequency of micturition later—Severe pain.—First operation: growth removed by suprapubic cystotomy—Discharged free from symptoms, which did not return for twelve months—Second operation, three years after first: growth removed as before—Local pelvic peritonitis—Rather sudden death.

Isaac Harbottle, aged 52, wireworker, was admitted into Rahere Ward, under the care of Mr. Marsh, complaining of passing blood in his urine.

This he did first on April 19, 1892. Five years before this he had first noticed blood in his urine, which became clear in a few days, the condition recurring at fairly long intervals. One year before admission patient had been in the Middlesex Hospital with the same complaint. He left (without any

operation) with the symptom relieved, and remained well until February 1892. At this time, two months before admission, he began to pass his water more frequently, getting up several times in the night for this purpose, and passing very little at one time. There was also severe pain after making water.

On admission, this condition of things was still present. Pain, fairly severe, was also present over the left lumbar region. The urine was of a chocolate colour; sp. gr. 1030; acid; about a half of albumin on boiling (probably from the blood only).

Examined cystoscopically, a growth was seen on the floor of the bladder. On April 29, suprapubic cystotomy was performed, when a red, tufted, villous growth was exposed, which bled freely on being cut into. It was clamped, and six days later the clamp came away. The instrument was re-applied the next day, and again came away after four days, carrying the growth with it. Recovery was uninterrupted, and the patient was discharged on June 13.

The patient was admitted a second time on June 27, 1895, three years after the operation, suffering from the old trouble. For twelve months following the operation he had remained quite well, when suddenly he passed urine quite dark red in colour, and continued to pass similar urine, on and off, for two years. During the six months preceding admission he had passed this dark red urine every day, but it had been lighter in colour during the month previous to admission. The urine was passed every hour during the day, and almost as often during the night. At the end of micturition patient had very severe pain, lasting about a minute, starting in the hypogastric region and passing down the penis. He noticed at this time that after drinking more alcohol than usual the amount of blood in the urine seemed increased.

On admission the man was pale and thin. His temperature was normal. The abdomen presented nothing abnormal save the scar of the old operation, pressure upon which, however, caused pain which shot down to the end of the penis. There was also pain on pressing over the left lumbar region behind. Near the end of micturition the stream sometimes stopped quite suddenly (? as a result of the pain), and then went on again. Urine, 1008; slightly alkaline; some blood, visible to naked eye. Microscopically, besides the blood cells, crystals of urates were seen, some pus cells, but no casts or pieces of growth. Examination *per rectum* revealed an enlarged prostate, but no growth. Only slight pain on making this examination. During the following few days the pus increased in amount, the

blood remaining much the same. The pain also became worse, and the urine markedly alkaline.

On July 8 the bladder was sounded; no stone was felt, but the inside of the bladder seemed very rough, so that the instrument could not be turned with equal ease in all directions. After using the sound, almost pure blood was passed.

The patient's condition seeming to get worse, an operation was decided upon, but the same day patient had two rigors, the temperature rising to 103.2° ; it fell the next day, however, and remained normal.

On August 9 a second suprapubic cystotomy was performed in the line of the scar of the first operation. The peritoneum had become adherent to the bladder wall, and overlay this, so as to make the ordinary extra-peritoneal operation almost impossible. The peritoneal sac was opened during the exposure of the bladder cavity, and a bit of omentum appeared through the opening; this was pushed back, and the peritoneal wound was sewn up by three silk sutures. When the bladder was opened, a mass of blood-clot and loosely hanging growth were seen and removed. The growth was of considerable extent, stretching from the roof of the bladder beneath the pubes as far as the trigone. It was removed by the finger. There was much bleeding, controlled by irrigation with hot water, and application of the actual cautery to the parts seen to be bleeding most freely when the cavity was illumined by the electric lamp. A rubber tube was sewn into the bladder through the wound, part of which was closed by sutures.

The night following the operation the temperature rose to 100° , but fell to normal on the second day, during which a fair quantity of urine was passed through the abdominal wound into the dressings. The general condition was good, but there was superficial suppuration of the wound. On the third day there was a marked change for the worse; very little urine passed through the tube, and patient seemed very ill. There was some bleeding into the dressings, but not great in amount. On the fourth day, the temperature having remained subnormal, the patient died rather suddenly. No signs of peritonitis had been manifest.

Post-mortem examination of abdomen.—Around the wound, on the anterior abdominal wall was some infiltration of purulent material, and a tag of the lower end of the great omentum was adherent at the upper part of the wound. There was some purulent peritonitis, but this was localised to the pelvis and to some coils of small intestine in close proximity to the wound. The bladder was small, thick-walled, and hypertrophied, open

above, and empty. The mucous membrane was injected; there was ulceration in places, and here and there shaggy remnants of a compound papillomatous growth. The ureters were natural in calibre, but were filled with purulent urine. Both kidneys contained purulent urine in the pelves. The cortex of each organ was contracted and a good deal congested.

Comment.—The case presents several interesting points, especially when compared with the foregoing cases.

(i.) The sequence of symptoms was—hæmaturia; frequent micturition; pain.

(ii.) The history was very long—five years before first admission to the Hospital, and eight years before second admission.

(iii.) For a long time the hæmaturia was markedly intermittent.

(iv.) The pain was very severe, and situated chiefly in the penis. There was worse pain than in either of the cases of malignant disease or of tubercle.

(v.) The hæmaturia was at times very profuse.

(vi.) There was complete absence of symptoms after the first removal of the growth.

(vii.) The growth, though “innocent,” had a considerable local recurrence, more or less characteristic of papillomata of the bladder.

(viii.) There was much hæmorrhage during the operation; this, too, is characteristic.

A NOTE UPON SIX UNSELECTED CASES OF HÆMATURIA.

The consideration of Cases VII. to XII. inclusive leads to a few important deductions, which, if any deductions are legitimate from such a small number of cases, may be worth notice.

(i.) Hæmaturia, in surgical cases, has its source more often in the bladder than in the kidney. Of the six cases, only one had the source of bleeding in the latter organ.

(ii.) Hæmaturia accompanying “malignant” disease of the bladder is usually more persistent in character, but less profuse, than where the growth is “innocent.”

(iii.) The duration of the hæmaturia is liable to be very long in cases of “innocent” growth, and is likely to be the first symptom noticed. In “malignant” disease its duration is shorter, and it is usually not the first symptom.

(iv.) Pain may be as severe (it is sometimes more severe) in cases of “innocent” growths as in cases of “malignant” disease.

(v.) Hæmaturia is an extremely unsatisfactory symptom for purposes of diagnosis of the nature of a bladder growth, but it is of more value than either of the other two great symptoms

of this condition—pain and frequent micturition. *Ceteris paribus*, the hæmaturia is more profuse and more intermittent in papillomatous growths than in either “malignant” growths or tubercle.

(vi.) There is no aggravation of the hæmaturia (or other symptoms) proportional to the advance of a “malignant” growth of the bladder. Hence exploratory incisions often discover an extent of growth quite unsuspected.

(vii.) More carcinomatous growths are liable to be mistaken for “innocent” ones than the reverse.

(viii.) The prognosis after removal of a papilloma of the bladder is favourable; but a large growth would seem to possess as great ultimate liability to prove fatal as “malignant” ones.

TABLE I.—*Showing all the Cases of Neoplasm of the Bladder admitted to the Hospital during the six years 1889–1894 inclusive.*

	1889.	1890.	1891.	1892.	1893.	1894.	Total.
Carcinoma	2	5	9	6	3	1	26
Sarcoma	2	2	...	4
Papilloma	4	2	2	4	3	15
Tubercle	2	...	6	3	3	1	15
							60

There were thus 60 cases in all. The order of frequency given by this group of cases is, carcinoma, 26; papilloma and tubercle, each 15; sarcoma, 4. This agrees with the result quoted by Morris from a collection of new growths of the bladder.¹ The entire absence of fibromata from the above list is significant. Perhaps (i.) its frequent occurrence in children, and (ii.) the occasional absence of symptoms, may account for this fact. But it would seem as if this particular kind of growth were really less common than is usually stated.

TABLE II.—*Showing the Ages of Patients at time of admission, in decades.*

	First.	Second.	Third.	Fourth	Fifth.	Sixth.	Seventh.	Age Limits.
Carcinoma	6	8	12	40-70
Sarcoma	1	1	...	2	...	20-60
Papilloma	3	3	6	3	30-70
Tubercle	2	...	6	4	1	1-70

¹ Diseases of Genito-Urinary Organs, ed. 1895.

The cases of carcinoma are thus seen to have occurred in the last three, and those of papilloma in the last four decades of life. The sarcomata occupied the middle three decades, with a tendency towards the later ones. The cases of tubercle occupied a significant position at both extremes, but especially in the middle decades. A greater number of cases would doubtless have shown some cases of tubercle in each decade, but there is an undoubted rise of the curve of age-frequency during the two middle decades, 10 cases out of 15 having occurred in patients between 20 and 40 years of age. The table gives the "average age" (if the term may be used) for the particular growths as follows:—Carcinoma, 58; papilloma, 54; sarcoma, 46; tubercle, 27.

TABLE III.—*Showing all the Operations undertaken for the Removal of the New Growths.*

	Operations.
Carcinoma	4
Papilloma	3
Sarcoma	1
Tubercle	1
(Unclassified)	3
	} Total, 12.

Out of 60 cases, therefore, only 12 were subjected to operation,—a rather striking fact. The reasons for this small minority of operations seem to have been (i.) want of patients' consent; (ii.) evidence leading indirectly to the diagnosis of a condition not adapted to a radical treatment of the growth; or (iii.) direct evidence of the same kind from exploration. Proportionately fewer carcinomatous growths were interfered with than any other kind. The single operation for tubercle consisted in local treatment of the ulcerated mucous membrane. The "unclassified" operations were recorded as being done upon "tumours," the nature of which was not specified; they were performed in 1892.

I am desirous of expressing my thanks to Mr. Marsh and to Mr. Butlin for the ready permission they granted me to publish the foregoing cases.

A CASE OF
DOUBLE EMPYEMA, WITH RECOVERY.

REPORTED BY PERMISSION OF DR. HENSLEY.

BY

W. E. N. DUNN.

As cases of double empyema are rare, and recovery from them still more uncommon, it is important that each case should be fully reported.

The following case was under Dr. Hensley's care, and has recently gone to Swanley quite well.

Frederick Trower, æt. $4\frac{1}{2}$, admitted into Colston Ward, September 29.

History.—This was difficult to obtain, as the boy's mother was dead, and he had no one to look after him when his father was at work. Five weeks ago sore throat and languid. Three weeks ago seemed to be better. Two weeks ago began to get worse again, and had a cough; was losing flesh; very thirsty; breathing very bad at times.

Past history.—Healthy; no previous illness.

Family history.—Mother died in confinement; no phthisis in the family.

Condition on admission.—Extremely wasted; cheeks hollowed; the bones almost protruding through the skin all over the body; skin sallow, dry as parchment, and covered everywhere with filth and flea-bites. Eyes: pupils widely dilated; tongue furred; alæ nasi actively dilating and contracting. Chest: lungs, movement on either side equal. Percussion note over both sides in front hyper-resonant. Behind, below the angle of the scapula on the left side and a little lower on the right, wooden dulness to the base. Auscultation: breath sounds in front exaggerated; behind, very weak from the angles of the scapulæ

downwards. Crepitations at the upper limits of the dulness on both sides. Both axillæ dull; breath sounds weak. Heart: apex beat not localised; cardiac dulness not defined; sounds clear. Pulse, rapid but regular, 136; temperature, 99.5°; respiration, 72. Abdomen: spleen felt. Liver an inch and a half below costal margin. Arms and legs extremely wasted.

October 1.—Patient more lively. Signs the same, except that dulness has extended a little higher on both sides. Pulse, 156; respiration, 64; temperature, 99.2°.

To-day an exploring needle was inserted into the chest through the eighth interspaces in the post-axillary line, first on the left and then on the right side. In each case pus was withdrawn.

Dr. Hensley decided to wait till next day before operating, as from the cough, which had been dry and irritable, but was now looser, he thought one effusion might possibly be going to discharge itself through the lung, in which case he would have opened the other externally. Pulse, 148; respiration, 60; temperature, 99°.

October 2.—No sign of any pus in the sputum; the patient had a few whiffs of chloroform, and an *incision* was quickly made into the tenth left interspace behind the post-axillary border, and a drainage tube inserted.

10 P.M.—Good deal of cough; fairly comfortable. Respiration, 72; pulse, 138. About eight ounces of pus as result of operation.

October 3.—Child worse; pulse, 170; respiration, 82; temperature, 102°. Fair night on the whole; takes well. Left side: dressings soaked; good air entry on left side, some friction in axilla. Heart's apex beat not made out. Right side: dulness has increased, but breath sounds are heard feebly to the base.

October 4.—Temperature has gone up steadily since operation; now 104°. Breath very foul. Pulse uncountable, respiration, 96; child's condition desperate. Right side aspirated in seventh space just below the angle of scapula, and eight ounces of pus withdrawn. Slight relief, but condition very serious. Pulse, 175; respiration, 60; temperature, 102°.

October 5.—Respiration, 80; pulse, 160; temperature, 99.6° last night; a trifle better this morning; scarcely any discharge from left side; right not perceptibly filled up; good air entry on both sides in front.

October 6.—Respiration, 76; pulse, 168; temperature, 103.8° again; patient more distressed; breath extremely foul. An *incision* was made into chest in the eighth space below the angle

of the right scapula. One or two ounces of pus withdrawn; patient collapsed; rallied a couple of hours later.

October 7.—Respiration, 60; pulse, 150; temperature, 98.6° earlier, now 101°; very weak, but taking well. Right dressings soaked; left practically nil.

October 9.—Respiration and pulse on the whole less frequent; temperature lower (100° highest point); takes well; is a bit stronger; bowels opened once or twice every day.

October 13.—Temperature has been subnormal for four days; patient is getting stronger; only slight discharge from either side now. Pulse, 130; respiration, 44. Has had an iodine bath; stood it well and liked it.

October 14.—Temperature, 101.8°; otherwise seems well; bath omitted.

October 15.—Temperature normal; respiration, 48; pulse, 144; both tubes left out to-day; no bath.

October 16.—Temperature, 101°; respiration, 56; pulse, 144. No bath; does not seem to be worse.

October 18.—Temperature normal now, 101° yesterday; respiration, 44; pulse, 136; looks better; another iodine bath given.

October 22.—Temperature, 100.6° last night; pulse, 135; respiration, 44; still free discharge from both sides of chest; both wounds healthy; air entry better; no signs of accumulation of fluid on either side.

October 23.—Temperature slightly unsettled; free discharge; lungs resonant all over; having iodine bath daily; quite comfortable.

October 29.—Temperature subnormal; respiration, 40; pulse, 120; much better.

November 6.—Temperature has been up and down since last note (reaching 102° several times); there is a good deal of discharge from the right side to-day, and as there has been complete absence for some days, it suggests some accumulation.

November 8.—Right side probed; profuse escape of pus and a long sinus; temperature, 101°; very slight discharge from left side all along.

November 10.—Temperature normal since last note. Left side, no discharge; right, slight discharge; probed again; very little pus; sinus shorter. Patient to pull himself up while holding on to a bar, in hope of helping the sinus to close up.

November 16.—Temperature normal; both sinuses healed; excellent percussion note and breath sounds all over lungs; got up yesterday.

December 9.—Went to Swanley. Since last note he has been

quite well. The child took everything so well that the treatment by diet and drugs was rendered easy; there was nothing special in either, except that stimulants of various kinds were freely used at first.

As to the operative interference in children a few words may not be out of place. Two of the three available methods—aspiration, simple incision, and resection—were used in this case, viz.: (1.) Aspiration; (2.) Incision.

As to the first, it gives in most cases only a temporary relief, and cannot replace either of the other two as a radical operation. It is useful where, as in this case, relief is urgently needed, but where, at the same time, the patient's condition deters one from disturbing him more than is absolutely necessary; thus the patient may be given temporary benefit for a day or two, and may be enabled to gain a little more strength to prepare him for the more radical operation.

As to the second, incision is undoubtedly the operation which should be done in cases where the patient's general condition is bad. Firstly, it is scarcely an operation at all if performed by a man who recognises that every minute he has the patient kept under an anæsthetic needlessly means another weight thrown into the descending scale. Secondly, the abscess generally drains quite well, and nothing further need be done; or at any rate, should it be necessary, resection of a rib may come in later, by which time the patient will probably be in a fit state to stand it.

Given that the patient's general condition be *good* in empyema, the choice between incision and resection is not so important; but as incision is generally successful and is much more simple, it should surely be given the preference.

NOTE ON THE
VALUE OF EHRLICH'S REACTION IN THE
DIAGNOSIS OF ENTERIC FEVER.

BY

HERBERT J. PATERSON, M.B.

The signs of enteric fever may be so uncertain and unreliable, that from time to time efforts have been made to discover some sign, constant and reliable, which might settle the diagnosis of a doubtful case. In 1882 Ehrlich announced that he had discovered such a sign, depending on the fact that the urine of patients with enteric fever gives a reaction with one of the aniline derivatives, different from that of normal urine, and the urine of patients suffering from other diseases.

More recently the serum method of diagnosis has been introduced. Which of the two is the more reliable, time alone can show. Ehrlich's test has the advantage of being the easier to carry out clinically, although even this method, like so many aids to clinical medicine, unfortunately requires considerable practice before any reliable conclusions can be drawn from the results obtained. At any rate, I trust these notes will show that Ehrlich's reaction is a valuable aid, and one not lightly to be cast aside and forgotten, until such time as the newer method has been shown superior to it.

The test solutions required are—

(A.) A saturated solution of sulphanilic acid in dilute (1 in 20) hydrochloric acid.

(B.) A .5 per cent. solution of sodium nitrite in water.

It is important that both the solutions should be fresh, especially the latter, which must not be more than a week old at most.

For use, 1 part of the sodium nitrite solution is added to 25 parts of the sulphanilic acid solution. The resulting mixture consists of sulphanilic acid containing free nitrous acid. It is of the utmost importance that the quantities should be most accurately measured. I cannot help thinking that the failure of some observers to obtain good results has been due to neglect of this simple precaution. The slightest excess of sodium nitrite will often prevent the development of the reaction in a typical case.

The urine to be tested is rendered alkaline with strong ammonia and an equal bulk of the testing mixture is added. In normal urines only a slight deepening of colour ensues. In some diseases attended by pyrexia, the urine becomes of a dark sherry tint, but the urine of a patient suffering from enteric fever turns immediately to a beautiful rich port-wine colour, and on shaking the test-tube a delicate pink froth is produced.

Unfortunately this test does undoubtedly occur, however rarely, in diseases other than enteric fever. To a certain extent this does detract from its value. I have scarcely had sufficient experience to state definitely what is the precise positive or negative significance of the test, neither have I tried it in as many cases of enteric fever as I should have liked. I have careful notes of thirty cases; but I have tried it in so large a number of doubtful cases, and acute and chronic cases other than enteric fever, that I think I can form fair conclusions as to the question of the frequency of its occurrence in diseases other than enteric.

First of all, I will record two cases in which this test was clearly of great help—almost the only help—in diagnosis.

CASE I.—C. H., 37, married, Elizabeth Ward. September 9, 1892, admitted for pain in the epigastrium and cough.

History of present condition.—September 1.—Rather suddenly ill, became deaf, giddy headache, pain across middle of abdomen, sleepless, loss of appetite, no vomiting.

September 3.—Much cough, much sputum, no hæmoptysis, bowels regular.

On admission.—Thin, dusky, sallow, drowsy, sweating about the face; per ophthalmoscope, discs quite clear; tongue almost quite clean and moist; pulse 120, and very weak and soft; respiration 84.

Examination of chest.—Percussion note against right apex, especially over and below right clavicle; no decided dulness; air entry best at left apex; a few crepitations there. Many

fine crepitations at right apex, also large crepitations and rhonchi.

Back.—Percussion note less good just internal to external border of right scapula; scattered crepitations all over back, more on right side; no œdema of loins.

Abdomen.—Some general tenderness. Liver, dulness up to sixth rib. Spleen felt in ordinary respiration three-quarter inch below ribs. No spots; hands tremulous; knee-jerks absent. Very slight pitting on pressure over spines.

Cough short and frequent; much frothy, purulent sputum.

September 11.—Slept badly; respiration laboured, 48 per minute. Abundant muco-purulent sputum; no tubercle bacilli found; sharper crepitations at right apex. Urine no Ehrlich's reaction.

September 12, about 2 A.M.—Patient had several attacks of rapid breathing; heart acting fairly; no added sounds; chest and abdomen as before. Ehrlich's test not tried.

September 13.—Spleen larger; crepitations all over chest. Ehrlich's reaction highly marked; two or three very doubtful spots. Bowels open five times during day, six times since 8 P.M.; very loose.

September 14.—Motions loose and soupy; sweats much.

September 15.—Better colour; some more faint spots on abdomen; typical "pea-soup" motion.

The history of the diagnosis of this case is very interesting. On admission the diagnosis seemed to lie between enteric fever and acute phthisis. The question of enterica was carefully considered, but Dr. West was inclined to the view that it would prove to be a case of acute pulmonary phthisis. The urine, it will have been observed, did not give Ehrlich's reaction. Two days after admission the lung symptoms had become so marked, that a diagnosis of acute phthisis was confidently made. There was still no Ehrlich's reaction. Unfortunately I did not again test the urine until two days afterwards, but in so doing found Ehrlich's reaction highly marked. On examining the abdomen, I found two or three very faint and doubtful spots, which were not, however, considered sufficiently definite to be of any importance. The next two days cleared up the diagnosis, the disease being clearly shown to be enterica by the occurrence of a succession of crops of faint spots and typical "pea-soup" motions. This case will illustrate the difficulty in diagnosis in the early stage of the disease, the prominence of the pulmonary symptoms leading to a diagnosis of acute phthisis, which, as already remarked, so often simulates enterica. As Dr. Gee observes: "In typhoid fever we meet with pulmonary signs

due to simple temporary collapse, but which, when discovered in a patient for the first time, may be thought due to phthisis."¹ But although the case illustrates this difficulty, at the same time it shows how such a difficulty may be overcome by the use of Ehrlich's test, which, when obtained on the eighth day of the disease, led to the diagnosis of the case.

CASE II.—C. D., 13, doctor's surgery-boy, Matthew Ward, October 14, 1892.

History.—Quite well until October 3, then headaches and fever; his temperature taken several times, and found higher in the evenings than in the mornings.

Present condition.—Earthy complexion; tongue flabby, dorsal fur. Lungs, slight impairment at bases; coarse pleural friction left base. Abdomen, rather full; no spots. Spleen felt. Gurgling in right iliac fossa. Liver not palpable. Legs natural. Pulse 100, regular. Temperature 100°. Occasional cough. Urine 1018; no albumin. No Ehrlich's reaction.

October 15.—Slept well. Temperature last night 102°, this morning 99°. Sordes on lips; tongue furred. Abdomen less distended. Friction at left base less. No dulness.

October 17.—Spleen still felt. No friction heard at left base. Bowels open.

October 18.—Temperature still 99°. Spleen not felt.

October 20.—Dulness right base to angle of scapula. Breath sounds very feeble; no friction.

October 21.—Spleen felt again. Bowels open once; solid motion.

October 22.—Coarse pleural friction right base.

October 25.—Temperature last night 102.2°. Slept fairly; feels better this morning. Lungs, nil, abnormal in front; behind, vocal vibration nearly lost at both bases, especially right. Slightly impaired percussion note both bases; no adventitious sounds at left base; friction heard at right base. Patient rapidly improved after this, although temperature remained between 99° and 100°.

On admission this patient was thought to have enteric fever. There had been, according to the doctor who sent him in, a temperature during the previous week suggestive of enterica, and on admission he was found to have a temperature of 100°, one-sided pleurisy, distended abdomen, gurgling in right iliac fossa, easily felt spleen. The urine never gave Ehrlich, and as he had been ill quite a week, I think this was almost conclusive against enterica. The subsequent progress of the case in no way

¹ St. Bartholomew's Hospital Reports, x. p. 13.

pointed to enterica. The occurrence of pleurisy, first on one side and then on the other, was also much against such a supposition. Such cases are always very suggestive of acute tuberculosis, and the enlarged spleen and persistent slight rise of temperature would tally with such an opinion. On admission, and for the first seven days after admission, Ehrlich's test was here the only sign by which to exclude enterica, and so to come to a correct diagnosis.

Two important questions must now be considered.

(1.) Does the reaction come off in every case of enteric fever?

(2.) Is the reaction of such frequent occurrence in other diseases—especially diseases which may simulate enteric fever—as to seriously impair its utility?

To answer the first question definitely I have scarcely had sufficient experience. I can only state that out of 30 cases of undoubted enterica, the reaction was present in 29. That the reaction was absent in the thirtieth case was to be expected, inasmuch as the duration of the patient's illness was very indefinite, probably four weeks at least, and the reaction usually ceases long before that period of the disease; in fact, I have found it usually stops before the twentieth day, even although the pyrexia continue. Sometimes the reaction ceases earlier, as in Case I., in which no reaction was present on the fourteenth day of the disease. According to Taylor,¹ the reaction is not always given until the latter part of the first week. I have only had an opportunity of trying it so early in one case, and in that it was not given on the fourth or fifth days, but was present on the eighth day, the urine not having been tested on the intervening days. Conclusions based on thirty cases cannot be very dogmatically expressed, but the fact that the reaction was present in every case—I exclude the thirtieth case for reasons given above—is sufficient, I think, to justify us in assuming that the reaction is present in a large majority of the cases.

To the second question I would answer an emphatic No. During rather more than three months of last year² I tried the test on the urines of every case admitted into Sir Dyce Duckworth's wards in St. Bartholmew's Hospital, and only obtained the reaction three times altogether out of a large number of cases, and one of these cases was, as I shall endeavour to show later, quite possibly enteric fever. In the remaining two cases which gave the reaction, the diagnosis was quite obvious, there being no suspicion of enteric fever; one of the

¹ Medical Annual for 1890, art. Typhoid Fever

² 1892.

cases, a fatal instance of rheumatic fever and morbus cordis, gave the reaction beautifully; the other was a case of advanced pulmonary phthisis, in which, however, the reaction was not well marked. In advanced cases of chronic phthisis the reaction is, according to Taylor, present in a few out of many cases, but in acute tuberculosis, the disease most nearly simulating enterica, the reaction has never been obtained by Taylor, and in the few cases I have tried I have obtained the same negative result. The third case in which I obtained the reaction was that of a little girl, aged 4, admitted into Elizabeth Ward on September 18, 1892, with anuria. She had been ailing about a week, with headache, loss of appetite, and during the first few days of her illness had vomited a good deal.

On September 13 she was seen as an out-patient; temperature 99.8° . Tongue furred.

September 16.—Temperature 101.4° ; bronchi in lungs; slight tonsillitis. Cough getting worse; slept badly; screamed occasionally; bowels constipated.

September 18.—Worse. Would not take food; passed some urine in morning; none before that for three days.

On admission.—Flushed face. Temperature 102.4° ; pulse 160° ; respiration 45 (in sleep); cough severe. Patch of consolidation or collapse at right base behind. Later on same day: short irritative cough; right back, resonance impaired below sixth rib; distinct bronchial breathing below scapula; fine crepitations all over. Breathing sounds harsh, especially at left base and in axilla.

September 20.—Bronchial breathing over half right back behind, some fine crepitations; distinct bronchial breathing in left axilla. Passed much urine last night, which gave Ehrlich's reaction. Still drowsy. Respiration, 52; pulse, 150. Less cough. Urine not passed from 7 P.M. on 19th until 11 P.M. to-night, and then about 8 oz. Ehrlich's reaction.

September 23.—Not taking well; no spots; spleen not felt. Ehrlich's reaction given.

September 27.—No Ehrlich's reaction.

October 7.—Takes well; up on couch.

This case then gave Ehrlich's reaction on the first occasion the urine was obtained, that was on the eleventh day of the disease, the reaction not being given on the eighteenth day. Although this was thought to be a case of pneumonia, I think no one could possibly deny that it may possibly have really been an attack of enteric fever complicated with broncho-pneumonia. In the first place, it could scarcely have been ordinary croupous pneumonia, which is rare in children under six years

old, and the breathing, although rapid, was scarcely laboured enough: the *alæ nasi* were not acting; the fever was too prolonged, did not fall by crisis, but by lysis, and there was no dyspnoea or lividity. Secondly, if it were purely catarrhal pneumonia, why did it begin so suddenly, and in a child apparently so well nourished? and why was there retention of urine? I cannot find such a condition mentioned as occurring in either croupous or catarrhal pneumonia by any authority in children's diseases. On the other hand, the supposition that it was enterica is at least supported by its sudden onset in a previously healthy and well-nourished child, and by the great depression and weakness which ensued. Retention of urine is known to be an occasional symptom of this disease in children, and the temperature chart after admission would do well for the end of a mild attack of enterica. I do not mean to assert that because the urine gave Ehrlich's reaction that therefore this was enteric fever. I merely say it was possibly that disease, and, in the absence of an autopsy proving that it was not, I do not think that any one could reasonably adduce such a case as proving the occurrence of Ehrlich's test in cases other than enterica. I consider, therefore, that, out of a large number of cases, only two undoubtedly not enterica gave the reaction, and in both of these the real nature of the disease was perfectly apparent.

In addition to these cases, I have tried the test in forty-eight cases of scarlet fever at the Western Hospital, and not in one of them, even in cases complicated by acute nephritis, was the reaction present. This fact may sometimes be of service in diagnosis, inasmuch as sometimes erythematous rashes occur in enterica, which may be puzzling, and have indeed in some cases led to an erroneous diagnosis. This scarlatiniform erythema most usually occurs at the end of the first week, or possibly, according to J. W. Moore, in the third week. The presence of Ehrlich's reaction would point to enterica to the exclusion of scarlet fever. Out of four cases of diphtheria the reaction was present in one, and that not well marked. I have notes of one interesting case in which diphtheria occurred in a patient convalescent from enterica, and in which the reaction reappeared. The patient, who was quite convalescent, suddenly developed a temperature of 104.4° , and Ehrlich's reaction reappeared. The cause of the pyrexia was not obvious, and at first it was thought to be a sudden relapse, but in two days typical diphtheria membrane appeared on the tonsils.

As to the occurrence of Ehrlich's reaction in infective endocarditis, influenza, and pyæmia, I have had no experience.

In conclusion, I think we may fully assume, from the above

results, that the absence of the reaction is a strong presumption against the disease being enteric fever, provided it has lasted seven days, and not more than eighteen days, and that the temperature is not normal. The presence of the reaction points strongly to enteric fever, provided there be no other recognisable disease—*i.e.*, acute rheumatism or chronic pulmonary phthisis—to account for the pyrexia. The probability is greater the deeper the tint of ruby red produced. This of course, like any other colour test, is a matter of degree, and so requires practice. I myself think more reliance is to be placed on the very characteristic pink froth, and this I have only seen in one case other than enterica, in which it is always highly marked. I would therefore venture to amend Murchison's rule as to the diagnosis of doubtful cases of enterica, and make it read thus:—A fever which in this country (aguish districts excepted) persists beyond seven days, and is unattended by any cutaneous eruption, or by signs of local disease in the head, chest, or elsewhere, and in which the urine gives Ehrlich's reaction, is *certainly* enteric fever, even although there be no symptoms of the intestinal lesion. I put these opinions forward with reserve, based as they are on such limited observations, bearing in mind the possibility that a greater experience may alter the conclusion here given; but I feel convinced that in careful and judicious hands this test is of the highest value, and that in the future it will be a recognised and legitimate aid to the diagnosis of enteric fever.

THE LEUCOCYTOSIS OF SCARLET FEVER.

FROM THE

WESTERN FEVER HOSPITAL AND THE PATHOLOGICAL
LABORATORY OF ST. BARTHOLOMEW'S HOSPITAL.

BY

R. SEVESTRE, M.D.

INTRODUCTION.

The importance of making an examination of the blood in diseases such as anæmia is fully recognised; in fact, an absolute diagnosis between the various forms of leukaemia can be made only after an examination of the blood has been carried out.

Within more recent years attention has also been paid to the condition of the blood in infective fevers,¹ especially to the behaviour of the white cells, and many authorities hold that a prognosis, if not diagnosis, can be made from these blood examinations.² In pneumonia, for instance, it is held by some that, within certain limits, the prognosis depends directly upon the number of leucocytes; furthermore, that from the presence or absence of leucocytosis a differential diagnosis can be made between this disease and enteric fever in cases in which a doubt has arisen, the number of leucocytes in typhoid fever being unchanged, or even diminished.

There is, however, only partial agreement amongst the various observers. In diphtheria, for instance, some associate a high leucocytosis with a bad prognosis, while others consider it to mean a good reaction.

In many of the specific fevers, such as measles and scarlet fever, the behaviour of the leucocytes has been very incompletely worked out, and on this account Dr. Kanthack sug-

¹ Von Limbeck, *Klinische Pathologie des Blutes*.

² See Grawitz, *Klinische Pathologie des Blutes*.

gested to me, in order to assist him in his hæmatological studies, to carry out an examination of the white blood corpuscles in scarlet fever in a more or less systematic manner.

In making a complete examination of the blood corpuscles, it is necessary—(1) to enumerate the red and white corpuscles; (2) to estimate the amount of hæmoglobin present, and (3) to make a differential or a relative count of the varieties of white cells. I have not thought it necessary in these investigations to do more than to take into consideration the white corpuscles, for the observations of others have shown that the number of the red corpuscles in scarlet fever is practically not affected.¹

Several propositions had to be taken into consideration:—

First, what relationship, if any, exists between the leucocytosis and some of the most important symptoms; for instance, the temperature and the rash.

Secondly, the effect that complications have on the leucocytosis.

Thirdly, I had to consider to what degree the various forms of leucocytes are affected.

Lastly, whether a good or bad prognosis can be deduced from the total number of leucocytes present, or from the preponderance of one form or other of white cell.

PART I.

Methods of Investigation.

The hæmocytometer that has been used for counting the absolute number of white corpuscles is the one that bears the name of Thoma-Zeiss; for description of the apparatus and method of using it, reference may be made to Von Kahliden's book of Pathological Histology, edited by Dr. Morley Fletcher.²

The blood was taken from the tip of the finger or the lobe of the ear—in most of the cases from the latter position, as less pain was caused and a greater quantity more readily obtained. It was diluted with a mixture after the formula given by Toison. Two separate counts, in some cases three, were made of each specimen of blood, so that a fair average might be obtained.

In preparing specimens of blood for future examination, it is necessary first to fix and then to stain them. Ehrlich³ suggested that the films should be kept on a hot metal plate for

¹ Grawitz, *op. cit.*, p. 258.

² Von Kahliden's *Pathol. Histology*, p. 123.

³ Ehrlich, *Farbenanalytische Untersuchungen zur Klinik des Blutes*.

some hours before staining. This, however, Kanthack and Hardy, as well as other observers, have found to be unnecessary. The following method, which can be carried out quickly and gives good results, is one used by Dr. Kanthack. The films are obtained in the usual way, and are at once held for about thirty seconds over formalin, or a mixture of osmic acid and Müller's fluid (Marchi's solution). When dry, they are passed through the flame, and held in a solution of eosine (.5 grm. eosine, 100 cc. of 50 to 70 per cent. alcohol) for about thirty seconds, then taken out, washed in water, and dried carefully between folds of blotting-paper. The films are then placed for about half to one minute in Löffler's methylene blue, after which they are washed in water, dried, and mounted in Canada balsam. In this way the nuclei of the cells stain a deep blue, with the protoplasm around of a lighter shade of the same colour. Some of the leucocytes, examined under higher power, appear granular.¹ The granules of the finely and coarsely granular cells take up the eosine, and present a reddish appearance. The specimens prepared in the above way were examined with Zeiss D. and Oc. 4; many also with $\frac{1}{12}$ oil immersion. The percentage of the different forms was calculated by means of a mechanical stage from a number not less than 300 of each specimen of blood. I may add that the blood for these dried specimens was obtained at the same time as that for estimating the total number of leucocytes.

It is not my intention to enter into an account of the morphology and physiology of the wandering cells, since this has been so fully done by others.² Of late years it has been recognised that they play a most important part in health and in disease, and many different views have been put forward to explain their functions, which are yet but dimly understood.

The Varieties of White Corpuscles.

I may be forgiven if I give a brief account of the various forms of leucocytes, for which I am indebted to a large extent to the work of Kanthack and Hardy. In examining blood, it is at once seen that there are several varieties of white cells. Wharton Jones in 1846,³ describing the blood of vertebrate animals, speaks of two varieties, viz., a granular and a nucleated blood cell. It was not, however, till 1865 that Max

¹ For a classification of the granular and other leucocytes, see papers by Kanthack and Hardy in the *Philosophical Transactions and Journal of Physiology*.

² See Adami's article on *Inflammation* in *Allbutt's System of Medicine*.

³ *Proceedings of Royal Society*, vol. v. p. 550, 1846.

Schultze carried our knowledge further, and described four varieties:—

- (1.) A small round nucleated cell with a thin film of protoplasm around it.
- (2.) A cell like the above, but larger, with more protoplasm round the nuclei.
- (3 and 4.) A finely and a coarsely granular cell.

This remains still the basis on which the present classifications are made.

In 1878 Ehrlich¹ made the important discovery that there is a relationship between certain stains and the chemical constitution of the cells, and on this he based his classification.

Since then Uskew and Tschistowitsch, quoted by Thayer,² have made elaborate classifications, consisting chiefly of subdivisions of the lymphocytes and multinuclear cells. The following classification, used by Kanthack and Hardy,³ and almost identical with that originally made by Max Schultze, must appear to most to be the most simple and at the same time the best suited for clinical purposes.

(1.) The most common form of corpuscle is one the cell substance of which is finely granular, with a nucleus irregular in shape, often semicircular, or lobed with two, three, or more parts united by narrow or fine threads. This irregularity in the shape of the nucleus and the difficulty of detecting the connecting threads has led to the erroneous names of "polynuclear" and "multinuclear" being applied to these cells. Ehrlich described them as "neutrophile" cells, on the supposition that they stain only with the so-called neutral dyes, and not with the acid or basic dyes alone. This, however, Kanthack and Hardy and later observers have found not to be strictly correct, for the granules will stain with eosin, which is an "acid" dye. Hence they prefer to call them finely granular eosinophile or oxyphile cells, and this is the name which has been used to designate them in the following pages. In normal human blood they constitute from 70 to 75 per cent. of the total number of leucocytes present.

(2.) Less common than the above, and forming about 1 to 5 per cent. of the total number of leucocytes, is a cell with an irregular nucleus and a cell substance containing a large number of coarse granules, which stain with acid dyes, and especially well with eosine. On account of this reaction, they

¹ *Op. cit.*

² *Boston Med. and Surg. Journal*, vol. cxxviii., No. 7.

³ *Med. Chronicle*, *Blood Changes in Diseased Condition*, N.S., vols. i. and ii.

are termed by Kanthack and Hardy coarsely granular eosinophile cells (the eosinophile cells of other writers).

(3.) Another form met with is a small cell of varying size, with a deeply-staining nucleus surrounded by a small rim of cell substance. These are generally termed lymphocytes, and in number vary from 15 to 25 per cent.

(4.) Somewhat similar to these, but much larger and fewer—about 5 per cent.—are nucleated cells surrounded by a quantity of hyaline cell substance. They are called large hyaline or uninuclear cells or mononuclear cells.

(5.) Lastly, a very infrequent cell is met with, resembling the eosinophile cells in possessing discrete granules, but differing from them in the granules showing an affinity for certain basic dyes, such as methylene blue, and called accordingly basophile cells.

The above seems to me the most simple classification. Osler,¹ following Thayer and others, describes another variety which he terms transitional cells, resembling the uninuclear cells, but differing from them in irregularities and indentations of the nucleus. I have included them under the heads of lymphocytes, or finely granular oxyphile cells, as the case may be.

It is difficult to say what is the origin of these various forms of cells and the relation they bear to each other. That they are different stages in the life history of one particular form of cell is improbable; their behaviour under pathological conditions, their difference in function, some being phagocytic in action and others not, the want of evidence of true transition, their difference in behaviour to chemical stains, are reasons for negating this view.

The origin of the various forms of cells is therefore still a matter of considerable obscurity; it is generally acknowledged that the lymphocytes arise from the lymphatic glandular system, and there are reasons for thinking that the uninuclear cells may develop from these lymphocytes.

The coarsely granular eosinophile cells present many points of interest: morphologically they are of great antiquity, for they are found in most classes of animals possessed of a vascular system. Kanthack and Hardy² showed that in animals these cells are especially numerous in the fluid of the cœlomic cavities; also that they are found in the spleen and the marrow of the bones, and in the connective tissue, their habitat being in the interstitial tissues, while the finely granular cells are confined almost entirely to the blood. Some authorities consider that

¹ Text-Book of Medicine, 2nd edit., p. 738.

² Journal of Physiology, vol. xvii. p. 81, 1894.

they originate in the bone-marrow, but the observations of the authors quoted above tend to show that the interstitial tissue spaces play an important part in their history.

With regard to the finely granular cells, very little is known besides the fact that they are essentially blood cells; many hold the view that they arise from the lymphocytes, but many difficulties have to be explained before this can be definitely accepted.

Number of White Corpuscles normally present.

The number of white corpuscles in the blood is subject to wide variations, even within the limits of health; the proportion of white to red in normal blood ranging from 1 in 300 to 1 in 700.¹

Osler,² in his Text-book of Medicine, gives the average of white cells per cubic millimetre as 7000. This seems a small estimate. Muir,³ from his results, considered that a pathological condition was present when the number exceeded 10,000 per c.m. Whatever number be taken, it must be remembered that considerable allowance has to be made for errors in observation, as well as for physiological processes, which alter the number from time to time. Inasmuch as no fixed number can be given, I have taken from 8000 to 9000 per c.m. as representing the normal average.

The Meaning of Leucocytosis.

Some confusion exists regarding the meaning of the term leucocytosis, some authors giving a definition too limited in its scope. For instance, Cabot⁴ speaks of it as an increase in the number of white cells of the same morphological variety as that most commonly present in normal blood, the increase being of the polynuclear variety. This conveys the idea that an increase in any other varieties of cell—for instance in leukæmia—is not really a leucocytosis.

Von Jaksch⁵ defines it as a condition in which the number of white corpuscles in the blood is temporarily much increased. This, however, brings in a question of time, and, as I shall show later on, in scarlet fever the leucocytes are increased in numbers for many weeks. This I consider more than a temporary increase.

¹ Foster's Physiology, Book i. chap. i. p. 38.

² Principles and Practice of Medicine, 2nd edit., p. 730.

³ Journal of Anatomy and Physiology, vol. xxv. p. 361.

⁴ Boston Med. and Surg. Journ., vol. 130, No. 12.

⁵ Clinical Diagnosis, p. 28.

Kanthack and Hardy¹ define the term as meaning an increase in the number of white cells in the blood, the increase being in a restricted area of the body or in the body as a whole. This is the most inclusive definition I have come across. Under certain conditions in the healthy body, the number of leucocytes may be greatly increased, so that one can speak of a *physiological leucocytosis* as compared with the increase in the number of white cells due to a pathological condition, a *pathological leucocytosis*.

I may be allowed briefly to state some of the conditions in which a physiological leucocytosis exists, for in making clinical investigations in blood, and in estimating the results obtained, some of these conditions must be borne in mind. It is agreed by all that during the process of digestion the number of leucocytes for the time being is considerably increased, but the numbers given vary a great deal. Cabot² states that two and a half hours after a meal, the white cells may vary from 1000 to 7000 in excess of the normal; other authorities that he quotes place the number even higher than this. I have tried to avoid this source of error by taking most of my counts before the principal meal, in the middle of the day.

In children, especially during the first few years of life, the white cells are increased in number. During the first year of life 12,000 per c.m. is not considered an abnormal number,³ and it is said that the normal is not reached till the sixth year. Many of my cases have been taken among children, and this fact has to be borne in mind. Rieder further points out that in new-born infants the leucocytosis affects especially the lymphocytes, not the multinuclear (fine granular eosinophile) cells.

Thayer⁴ finds that when a bath at 70° F. has been administered for twenty minutes to a healthy person, himself in this instance, and in cases of enteric fever, the number of leucocytes is increased; he inclines to the idea that further observation may show that the increase is local rather than general. He is unable to state definitely whether the local application of cold has any effect or not. This small point may be important, and lead to a source of error, by taking a count of the blood immediately after the person has washed in cold water.

Drugs, as quoted by Dr. Kanthack,⁵ may prove to be another source of mistake, for many lead to an increase in the number of white cells; for instance, pilocarpin has been recommended

¹ Journal of Physiology, vol. xvii., 1894.

² Op. cit., p. 278.

³ Von Limbeck, op. cit.

⁴ Bulletin of the John Hopkins Hospital, April 1893, vol. xxx.

⁵ Med. Chronicle, N.S. i.

by Waldstein in diphtheria, with the idea that the white cells may act beneficially. Many tonics are supposed to act in this way. I have been fortunate enough to obtain a series of cases in which but few drugs were administered.

Other conditions of physiological leucocytosis may be mentioned, such as the change of position, pregnancy, and the puerperal state; these, however, need not be considered here.

PART II.

Leucocytes in Scarlet Fever.

It has been through the kindness of Dr. Bruce, the Medical Superintendent of the Western Fever Hospital, that the cases upon which these observations are based were obtained. In taking cases, I selected as far as possible those that were suffering from scarlet fever only, and in whom no concurrent affection or infection was detected.

It is obvious that it must be difficult to obtain cases *ab initio*, and I have been able to obtain only one case from the commencement of the disease; this case, however, presented curious features, which will be commented upon later.

(a.) In all cases there was a well-marked leucocytosis, varying in extent but present in all.

I have not been able to ascertain the time at which the leucocytosis commences. Kotschetkoff,¹ who examined the blood in twenty cases of scarlet fever, states that the leucocytosis commences two to three days before the eruption, and lasts for five to six weeks. Rieder also found leucocytosis, which often was only moderate. Felsonthat insists upon a marked leucocytosis, while Von Limbeck, Pick, and Halla detected no leucocytosis in uncomplicated cases.²

In Case 9 (see Appendix) the blood was examined a few hours after the first symptoms had developed, before the appearance of the rash, and *a well-marked leucocytosis was present*. What occurs during the period of incubation is uncertain. Experimentally, it has been shown that in effective and non-effective processes there is, first of all, a diminution in the number of white cells, a "leucocytopenic phase,"³ which lasts for a few hours, and precedes immediately the leucocytotic phase. It is possible, if not probable, that the same occurs in

¹ Original article in Russian. Summary in *Centralblatt für Pathologie*, 1892, vol. iii. p. 468.

² Grawitz, *op. cit.*, p. 259.

³ Sherrington, *Proc. Roy. Soc.*, vol. lv. p. 195; Kanthack, *Brit. Med. Jour.*, 1892, and others.

scarlet fever, but whether this diminution in the number of leucocytes occupies the whole period of incubation or only a portion of it, I had no opportunity of determining.

(b.) The severity of the attack has an important effect upon the number of leucocytes present.

(1.) In mild cases, for instance, Nos. 5, 6, 7 (see Appendix), there was a low leucocytosis which reaches its maximum in the early part of the disease, and when no complications arise, as in No. 5, there is a gradual fall during the period of convalescence.

(2.) In cases of moderate severity, Nos. 1, 2, 3, 4 (see Appendix), the leucocytosis reached its maximum in the early part of the disease; it was also greater in extent than in mild cases, reaching as high as 40,000 to 50,000 per cubic millimetre, and in convalescence a somewhat high level was maintained.

(3.) What the conditions are in fatal cases I have not been able to ascertain. In one case which ended fatally on the sixth day, I found a rising leucocytosis towards the end; but as the patient had diphtheria, the diagnosis being made bacteriologically as well as clinically, I have decided not to include it in my series. Kotschetkoff¹ states that in the most severe cases the number of leucocytes rises above 40,000 per cubic millimetre, and he implies that the leucocytosis is greater as the severity of the case is more marked. My results in some measure agree with this, for in Nos. 1, 2, 3, which are all severe cases, the number of leucocytes kept at a higher level than in the others; but in No. 6 a low leucocytosis is seen, though the symptoms were grave during the first four days.

The time at which the leucocytosis ceases is a matter of great interest and will be considered later.

(c.) I propose now to consider the relationship between the number of leucocytes and some of the most prominent symptoms of the disease. For the sake of clearness, the rise and fall of the leucocytes has been expressed graphically (see Charts) in the same way as that of the temperature. Before any comparison can be made between the curve of the leucocytes and that of the temperature, it must be remembered that they are not productive the one of the other, but both are the effects of the same cause, namely, that which produces the characteristic set of symptoms which we designate by the name of scarlet fever.

¹ *Op. cit.*

Comparison between Temperature and Leucocytic Curves.

In most of the cases, *e.g.*, Nos. 1, 2, 3, 4, 5, 7, 9, 12 (in this series No. 13 is not included, for the patient had pneumonia), two facts are noticeable: first, that the number of leucocytes is greater during the time when the temperature is raised; secondly, with the fall of the temperature there is a tendency for the leucocytes to diminish in number. Here, however, the comparison ends, for on looking more closely into the charts, it is seen that with each variation in temperature there is not a corresponding alteration in the number of white cells. Moreover, one would not expect this to be the case, for in experiments on animals it has been shown that the two curves are not identical, although their course is often similar.¹

Nos. 2, 3, 5 show well the tendency of the two curves to fall in the same way, and the first two show the great difference between the height of the temperature and that of the leucocytosis. Another point to be noted is that the fall of the leucocytic curve is slower and later than that of the temperature, and is not usually so complete.

In No. 10 the leucocytic curve is seen to be rising in spite of the falling temperature; but this case was complicated by the joints being affected on the fifth day (rheumatism).

In No. 8, although no comparison can be made during the early part of the illness, yet after the fifth day there is a tendency for the number of leucocytes to diminish at the same time as the temperature gradually falls.

In No. 11 I attribute the difference between the two curves to be caused by the complications that were present; but in No. 6 I see no reason for the great difference that exists between the two curves.

I think that there is sufficient evidence to show that, although no very close connection exists between the two curves in scarlet fever, yet, generally speaking, without being identical, they tend to run a similar course.

It will not be out of place if a short comparison be made between the temperature and leucocytosis in some other processes, especially those of an infective nature, for though the infective nature of scarlet fever has never been proved, yet the probability of its being so is great.

Sherrington² has shown that, as a result of a simple, non-infective inflammation of an acute local character, produced by mechanical means, there is a well-marked leucocytosis, and

¹ See Kanthack, *Brit. Med. Jour.*, 1892.

² *Proc. Roy. Soc.*, vol. lv. p. 161.

he states that he "did not detect any very constant relation between it and body temperature."

Turning now to infective processes, Kanthack¹ has shown that on intravenous injection of bacterial products there is in the first few hours a fall in the number of leucocytes, a "leucocytopenic phase," which lasts but a short time—a few hours—and is followed by a marked increase in the number of leucocytes, a "leucocytotic phase."

He has found that the stronger the febrile reaction has been, the more marked is the leucocytosis. On comparing the curves, he found that they were not identical, but certainly analogous, the most rapid rise of temperature taking place during the leucocytopenic phase, and the more gradual fall occurring before the leucocytosis had returned to normal; though not identical, the course of the two curves was similar.

In diseased conditions such as pneumonia² there is a distinct resemblance between the leucocytic and temperature curves, the former falling within a few hours of the latter in cases which have a favourable termination; no absolute correspondence, however, can be traced between the daily variations of the two.

Kikodse³ and others, however, have observed that the temperature crisis is followed by a leucocytic crisis, the white corpuscles, like the temperature, sinking below the normal line. Von Jaksch maintains that an absence of leucocytosis implies a bad prognosis.

In diphtheria there is, according to Bonchut and Dubrisay,⁴ always a pronounced leucocytosis, greatest at the climax of the disease, and steadily declining during convalescence.

Ewing⁵ found that the degree of the leucocytosis often varies with the fever, but more frequently corresponds to the extent of the local lesion.

An exception to the rule that an infective fever is accompanied by leucocytosis is found in typhoid fever, for Thayer,⁶ like Halla,⁷ Von Limbeck,⁸ and Rieder,⁹ found that during the febrile period, when no complications are present, the number of leucocytes is slightly, or even considerably decreased, and that in the post-febrile condition there is a slight increase.

¹ Brit. Med. Jour., 1892, vol. i. p. 1300.

² J. S. Billing, Bulletin of the John Hopkins Hospital, Nov. 1894.

³ Grawitz, op. cit., p. 229.

⁴ Compt. Rend., 1877, vol. lxxxv. p. 158.

⁵ New York Med. Jour., vol. lxxii. p. 196.

⁶ John Hopkins Hospital Reports, vol. iv. p. 30.

⁷ Zeitschr. f. Heilkunde, vol. iv. p. 198.

⁸ Ibid., vol. x. (1890), p. 392.

⁹ Leukocytose, 1892.

Comparison between Leucocytosis and Throat Symptoms.

The throat lesions, not including a subsequent adenitis, which I shall regard as a complication, have no constant effect on the number of leucocytes. In No. 8, during the first few days of the disease the throat symptoms were severe, yet the leucocytosis was not high. The reverse, however, is seen in Nos. 2, 3. In the other cases no definite connection can be traced between the two conditions. I am inclined to think that the severity of the throat symptoms has no effect upon the leucocytosis except in those cases in which much ulceration and sloughing takes place.

Comparison between Leucocytosis and the Rash.

The rash of scarlet fever may be said to be the most prominent and characteristic symptom of the disease, the intensity of the exanthem often proving a rough guide to the severity of the attack. This is not, however, an invariable rule, for, as Fagge¹ states, some of the most severe cases are accompanied by a rash that is but ill developed.

Affections of the skin—for instance, many forms of erythema—are generally known to be associated with an increased number of leucocytes, so that one would expect that some connection exists between the leucocytosis and the rash and desquamation. What influence the onset of the eruption has on the number of white cells I have not been able to determine definitely. In No. 9 the rash appeared on the second day, the number of leucocytes remaining about the same as on the first day. In the other cases I was not able to obtain a count sufficiently early to settle this point.

It is by no means an easy matter to make a comparison between the leucocytosis and the rash, for complications, such as rhinorrhœa, otorrhœa, and adenitis, may arise quite early in a case, and are apt of themselves to increase the number of leucocytes, as I shall show later. In Nos. 1, 2, 4, the rash assumed a severe character, whereas in Nos. 5, 10, 8, 6, it was slight, and lasted a short time—in the last-mentioned case it amounted to no more than an erythema. With this difference in the intensity of the rash there is a marked difference in the number of leucocytes; in the severe cases it will be seen that they are far more numerous.

To facilitate the comparison I have marked on the charts (see Appendix) the day on which the rash reached its maximum intensity, also the day of its disappearance. I have not been

¹ Principles and Practice of Medicine, 3rd edit., vol. i. p. 187.

able to decide whether in an uncomplicated case the maximum intensity of the eruption coincides with the greatest number of leucocytes. In No. 4 the eruption reached its maximum on the fifth day, on the same day the highest leucocytosis was noted; the other cases do not bear this out. In No. 1 the rash was most marked, and one of the most severe I have seen; it reached its maximum on the fifth day and had faded by the eleventh. On the sixth day the greatest number of leucocytes were counted per cubic millimetre, but on that day, although the rash had commenced to fade, the skin was covered with innumerable vesicles, the contents of which were quite opaque, and this possibly may have accounted for the great number present. In looking more closely at the chart, it is seen that the fall in the leucocytic curve coincides somewhat with the fading of the rash. This is seen better in No. 7, where there is a low leucocytosis on the last day on which the eruption was present. Many other cases show this fall, for instance, Nos. 4, 5, 6, 2, 12. In many of these cases the leucocytic curve rises again before the complete disappearance of the rash, but I am inclined to attribute this to some complication.

An apparent exception to the rule that the leucocytic curve falls with the fading of the rash is seen in No. 8.

Nos. 10, 11, 13, 3, cannot be taken as exceptions to this, for other conditions were present in all which were quite sufficient to profoundly alter the number of leucocytes. I consider, therefore, that the leucocytosis varies with the rash, and shows a tendency to fall with the fading of the same.

The Connection between the Leucocytosis and Desquamation.

Desquamation commences sometimes before, sometimes after the disappearance of the rash. Its onset is not marked in any definite way on the leucocytic curve; in some cases a fall is observed, in others a rise. Kotschetkoff¹ carried out the investigation on his cases for a period of six weeks, and found the leucocytosis present during that time.

In many of my cases the examination of the blood was carried out over a larger period of time, in a few as long as any desquamation was present. In these, Nos. 12, 2, 7, 8, a leucocytosis was observed during the whole time; in the others it was present as long as I had the opportunity of examining the blood. This is not more than one would expect from the blood examination of some chronic skin diseases. In one case, No. 9, a peculiarity was observed. During the third week of

¹ Op. cit.

the disease the leucocytes fell in number to below normal, but rose again after a few days. I am quite unable to offer any explanation of this.

During the period of desquamation there is a gradual fall in the leucocytic curve. The curve does not always show an even descent; irregularities are seen, some of which can be accounted for by the clinical condition of the patient; for others I can find no explanation.

Comparison between the Leucocytosis and Complications.

I shall now pass on to consider what effect complications have on the leucocytic curve. Kotschetkoff states that they had no direct effect on it. With this statement I am unable to agree. In No. 11 there was considerable induration and glandular enlargement at the angles of the jaws; rhinorrhœa and otorrhœa were present in addition. It is seen that with these the curve is raised considerably.

In No. 10 arthritic pains were felt on the sixth day; at the same time there was an increase in the leucocytes which lasted for days after all joint pains had disappeared. In No. 7 a weak and anæmic-looking child, a high leucocytosis was observed at the same time as there was glandular enlargement at the angles of the jaw. The same was observed in No. 3, and here I attributed it to the ragged ulcerated condition of the tonsils.

These instances tend to show that complications have a considerable effect upon the leucocytic curve.

Analysis of the Differential Count.

In nine of my cases stained specimens of blood were examined from time to time, and the percentage of the various forms of white cells estimated. The time taken up in examining these few cases has been considerable, and I have not been able to examine all the cases systematically in this way.

In some of the specimens the granules of the finely granular cells were more deeply stained than in others. In estimating the amount of the leucocytosis I diluted the blood with Toison's fluid, which stains the white cells and makes their recognition easier. During the first few days of the disease it was noticed how large and refractive the cells looked. Very soon a change was observed, the cells being not so large, their outline more regular, and they look more deeply stained, and many irregular forms are present.

Instead of taking the cases separately, I think it will be best

to describe the changes in the percentages in each form of cell during the disease as illustrated by the cases.

No. 1 shows well the changes in the early part of the disease. During the first few days of the illness the chief increase is in the finely granular cells, which in this case reached to 88 per cent. In Nos. 8, 5, 2, 4, the same is observed. Towards the end of the first week there is a gradual fall in the number of finely granular cells, till they go below the normal, where they remain for some considerable time. The large uninuclear cells with the lymphocytes are diminished in the early part of the disease; the former return to the normal within a short time and continue there. They may, however, continue to go above the normal, reaching in some cases to over 40 per cent. Examples are seen in Nos. 8, 2, 3.

The coarsely granular cells in my cases show a somewhat diminished percentage during the whole period of the disease; they were always present, but rarely more than 1 to 2 per cent. In No. 3 they reached to 6 per cent.

These results differ very much from those obtained by Kotschetkoff.¹ He found that so far as the coarsely granular cells are concerned, after two to three days the percentage increased, to reach its maximum in two to three weeks, the percentage then being 8 to 18; after this the number fell, returning to the normal in about six weeks. In some cases he stated they were absent.

Very little is known with reference to the functions of these coarsely granular cells. Kanthack² and Hardy³ have shown that when bacilli or their products are injected into animals, the increase in the number of leucocytes in the neighbourhood of the irritant is due to the arrival first of the coarsely granular cells.

It is generally acknowledged that a patient suffering from scarlet fever is able to communicate the disease to another quite early in the attack, as well as during the period of desquamation. It has been shown that during the disease a well-marked leucocytosis is present, but I am convinced that this cannot be taken as a measure of the period of infection.

Prognosis.

I have left till now the consideration whether or not a prognosis can be made from the leucocytosis. It has already been shown that the severity of a case can be only partly

¹ Loc. cit.

² Journal of Physiology, vol. xvii. p. 102.

³ Kanthack and Hardy, Phil. Trans. Roy. Soc., 1892.

ganged by the extent of the leucocytosis. Kotschetkoff goes as far as stating that when 30,000 leucocytes per cubic millimetre are found, a fatal termination is not unlikely. With this I cannot agree, for in many of my own cases a much higher leucocytosis was reached, and at no time in the case was there any cause for anxiety. Possibly a more accurate prognosis may be arrived at by estimating the percentages of the different varieties of white cells, and the author quoted above, as far as I can understand, considers that in cases ending fatally the percentage of the eosinophile cells continues to decrease rapidly. I have not had an opportunity of verifying these statements from want of time.

In Nos. 2, 8, the condition of the patients may be described as serious for the first few days; in both it will be noticed that the percentage of the finely granular eosinophilous cells is above 90.

No. 1 is an example of a moderately severe type of the disease; in this case the percentage of these cells reached to 88. In cases assuming a milder form, the percentage of these cells is not increased to any great extent above the normal. These few instances are not sufficient to draw any definite conclusions, but they indicate that the number of finely granular eosinophilous cells may prove to be of some value in forming a prognosis.

SUMMARY.

The following conclusions may be drawn from the examination of the blood in thirteen cases of scarlet fever.

1. There is a leucocytosis in scarlet fever which reaches its maximum in the first few days of the disease, but the return to normal is gradual.

2. The actual time of onset of the leucocytosis is uncertain: it is found to be present on the first day of the disease, and it continues throughout the period of desquamation.

3. No absolute relationship can be traced between the curve of the temperature and that of the leucocytes, but they tend to run a similar course.

4. A closer relationship has been found to exist between the leucocytosis and the rash: the former varies with the severity of the latter, and with the fading of the same the leucocytes show a marked diminution in number.

5. Complications such as otorrhœa, rhinorrhœa, adenitis, tend to increase the number of white cells.

6. In nine cases the percentage of the various forms of white cells to the total number of white cells present was esti-

mated. It was found that the percentage of the finely granular eosinophilous cells was greatly increased during the first few days of the disease; but within the next few days the percentage falls below the normal, and continues so for some considerable time. The large uninuclear cells and lymphocytes are diminished in number in the early part of the disease: after a short time the percentages of both forms gradually increase, the large uninuclear cells remaining about normal, but the lymphocytes increase, and this increase exists for some time.

In the majority of cases the percentage of the coarsely granular eosinophilous cells was found to be diminished during the whole period of the disease. In no cases were they found to be absent.

7. A slight leucocytosis usually accompanied a mild infection.

A high leucocytosis indicates a pronounced reaction against a severe infection, and is not a sign of an unfavourable prognosis.

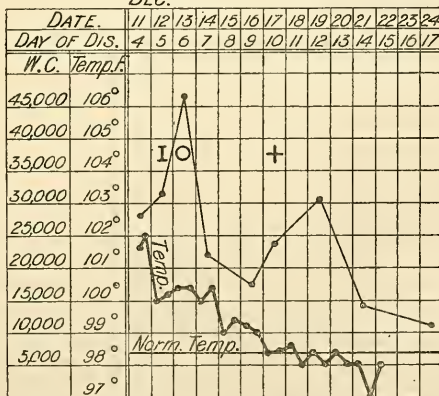
In severe cases it was found that the percentage of the finely granular eosinophilous was always high.

APPENDIX OF CASES.

I.—Kate G., aged 8, admitted on December 8, 1895.

On December 8 she vomited and complained of sore throat.

NAME. *Kate G.* AGE. *8.* No. *1.*
DEC.



I = Day on which rash was most marked.
 + = " " " " " last seen.
 O = " " " " desquamation commenced.

On December 9 rash observed.

When admitted on December 11, the patient was covered

with a bright punctate erythema. Tongue furred in places. The fauces were injected, and the tonsils were slightly enlarged. The patient had scarlet fever four years ago. Leucocytes, 28,000 per c.m.

December 12.—Rash accompanied with much vesiculation. Leucocytes, 31,500 per c.m.

December 13.—Rash fading; the vesicles contain an opaque fluid. Desquamation has commenced about the neck. Throat still inflamed. Leucocytes, 46,500 per c.m.

December 14.—Vesicles drying up, leaving the skin hard and leather-like. Leucocytes, 22,000 per c.m.

December 16.—Rash faded much; punctate condition about the legs; skin coming off in huge flakes. Leucocytes, 17,500 per c.m.

December 17.—Rash just visible. Leucocytes, 24,000 per c.m.

December 19.—Leucocytes, 30,500 per c.m.

December 21.—Leucocytes, 14,500 per c.m.

December 24.—Out of bed for the first time yesterday; rather pale and weak. Leucocytes, 11,000 per c.m.

December 26.—Sent to a Convalescent Home.

Differential count.—December 11, fourth day of disease.—Lymphocytes, 6.9 per cent.; large mononuclear, 3.1 per cent.; finely granular, 88.4 per cent.; coarsely granular, 1.4 per cent.

December 13, fifth day of disease.—Lymphocytes, 8.7 per cent.; large mononuclear 5.4 per cent.; finely granular, 83.5 per cent.; coarsely granular, 2.1 per cent.

December 17, fifteenth day of disease.—Lymphocytes, 19.1 per cent.; large mononuclear, 12.9 per cent.; finely granular, 66.6 per cent.; coarsely granular, 1.5 per cent.

II.—Henry R., aged 13.

On November 25 the patient vomited and complained of headache. The following day his throat was sore and a rash was observed.

On November 28 he was admitted with a dusky, scarlatini-form rash, severe in character; the fauces were much inflamed, the tonsils were prominent with rugged surfaces, which were covered with secretion; the cervical glands were swollen and tender to the touch. Leucocytes, 54,500 per c.m.

November 29.—Leucocytes, 30,500 per c.m.

November 30.—The rash is not so bright, the throat is not so inflamed, and the secretion is less; the tonsils still present a very ragged appearance. Leucocytes, 55,000 per c.m.

December 2.—Rash is fading rapidly, leaving behind a

January 10.—Leucocytes, 10,300 per c.m.

January 13.—Leucocytes, 15,000 per c.m.

January 15.—Leucocytes, 21,000 per c.m.

January 17.—Leucocytes, 13,000 per c.m.

January 20.—Leucocytes, 14,000 per c.m.

January 25.—Leucocytes, 13,500 per c.m.

January 27.—Leucocytes, 13,500 per c.m.

February 5.—Leucocytes, 5000 per c.m.

February 12.—Leucocytes, 9000 per c.m. Desquamation ceased.

Differential count.—November 28, fourth day of disease.—Lymphocytes, 4.4 per cent.; large mononuclear, 3.6 per cent.; finely granular, 90.8 per cent.; coarsely granular, 1 per cent.

December 18, twenty-fourth day of disease.—Lymphocytes, 33.7 per cent.; large mononuclear, 10.7 per cent.; finely granular, 53.6 per cent.; coarsely granular, 1.7 per cent.

January 13, fifty-first day of disease.—Lymphocytes, 27.88 per cent.; large mononuclear, 10.17 per cent.; finely granular, 58.30 per cent.; coarsely granular, 1.13 per cent.

January 27, sixty-fifth day of disease.—Lymphocytes, 32.8 per cent.; large mononuclear, 7.1 per cent.; finely granular, 57.6 per cent.; coarsely granular, 2.4 per cent.

III.—Louisa S., aged 10.

On December 3 the patient vomited and complained of sore-throat. The following day she complained of feeling generally ill, and on December 5 was admitted into the Hospital. On admittance there was a vivid general punctate rash. The fauces were injected, the tonsils prominent, and on the right was a continuous white covering of secretion. The glands at the angles of the jaws were enlarged.

December 6.—The rash very bright and in parts vesicular.

December 7.—Rash brighter on the lower half of the body than elsewhere. Vesicular in parts. Tongue described as "strawberry." Throat much inflamed, with quantity of secretion. Glands enlarged, mostly on the left side. Leucocytes, 22,800 per c.m.

December 9.—Rash still very bright on lower half of the body. Throat symptoms more pronounced. Child seems very ill. Leucocytes, 39,500 per c.m.

December 10.—Rash fading; much discoloration left. Desquamation is well marked. Throat still much injected. Right tonsil large and very ragged. Leucocytes, 33,000 per c.m.

December 11.—Erythematous flush is seen about the body

and legs. Throat looks more inflamed again. Glands felt at angles of jaws, but they are not markedly enlarged. Leucocytes, 42,000 per c.m.

December 12.—Leucocytes, 28,000 per c.m.

December 13.—Rash has gone, leaving some discoloration. Peeling profusely. General condition has greatly improved. Leucocytes, 45,000 per c.m.

December 14.—Leucocytes, 18,500 per c.m.

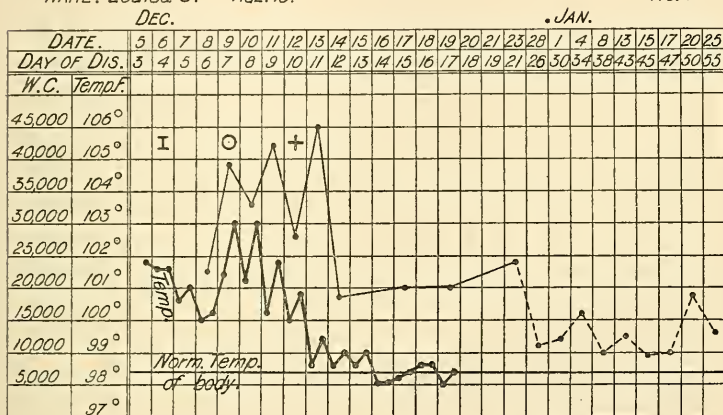
December 16.—Baths commenced; two given every week.

December 17.—Leucocytes, 20,000 per c.m.

December 19.—Leucocytes, 20,000 per c.m.

NAME. *Louisa S.* AGE. *10.*

No. *3.*



I = Day on which rash was most marked.
 + = " " " " " last seen.
 O = " " " desquamation commenced.

December 23.—Leucocytes, 24,500 per c.m.

December 28.—Allowed to be out of bed. Pale and thin. Tonsils still large. Glands in the neck palpable. Leucocytes, 10,500 per c.m.

January 1.—Leucocytes, 12,000 per c.m.

January 4.—Leucocytes, 16,000 per c.m.

January 8.—Leucocytes, 10,000 per c.m.

January 13.—Leucocytes, 12,500 per c.m.

January 15.—Peeling confined to the feet. Leucocytes, 9600 per c.m.

January 17.—Some roughness about feet. Leucocytes, 10,000 per c.m.

January 20.—Skin on feet still rough. Leucocytes, 19,500 per c.m.

January 25.—Leucocytes, 13,000 per c.m.

January 27.—Sent to a Convalescent Home.

Differential count.—December 7, fifth day of disease.—Lymphocytes, 14.1 per cent.; large mononuclear, 13.8 per cent.; finely granular, 65.7 per cent.; coarsely granular, 6.6 per cent.

December 13, eleventh day of disease.—Lymphocytes, 10.7 per cent.; large mononuclear, 7.4 per cent.; finely granular, 79.9 per cent.; coarsely granular, 1.8 per cent.

January 18, forty-seventh day of disease.—Lymphocytes, 24.5 per cent.; large mononuclear, 9.7 per cent.; finely granular, 64.2 per cent.; coarsely granular, 1.4 per cent.

IV.—Bessie C., aged 5.

On November 11 she complained of a sore throat.

On November 13 the patient was admitted into the Hospital with a general punctate rash, the tongue was furred, the tonsils were swollen, and the fauces red. Nothing abnormal found on examination of heart and lungs.

On November 14 the rash in places was vesicular, in other places blotchy; in character it was severe. A muco-purulent discharge from the nose was seen for the first time. The glands at the angles of the jaw were slightly enlarged. Leucocytes, 27,000 per c.m.

November 15.—The rash is more dusky in appearance. Leucocytes, 34,400 per c.m.

November 16.—Rash shows signs of fading. The rhinorrhœa is less; the enlarged glands are situated chiefly at the left angle of the jaw. Leucocytes, 17,900 per c.m.

November 17.—Leucocytes, 22,800 per c.m.

November 18.—Leucocytes, 21,200 per c.m.

November 19.—Desquamation starting about the neck. The rash has faded very much; it is still present about the legs. Leucocytes, 32,700 per c.m.

November 20.—Rhinorrhœa has ceased. Leucocytes, 27,700 per c.m.

November 21.—Leucocytes, 20,000 per c.m.

November 22.—The general condition of the patient has greatly improved. The rash has disappeared. Desquamation is well marked. The glands at the angles of the jaws are still palpable. Leucocytes, 33,700 per c.m.

November 23.—Leucocytes, 29,900 per c.m.

November 24.—Leucocytes, 25,000 per c.m.

November 25.—Leucocytes, 25,600 per c.m. The urine contains no albumin. Baths started and given twice a week.

November 27.—Leucocytes, 13,800 per c.m.

November 28.—Leucocytes, 12,500 per c.m.

November 29.—Leucocytes, 15,200 per c.m.

December 2.—The urine contains no albumin.

December 3.—Leucocytes, 11,400 per c.m.

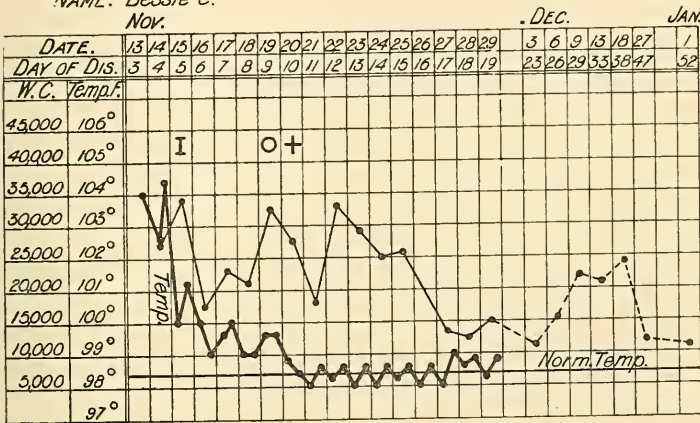
December 6.—Leucocytes, 15,200 per c.m. Allowed to get out of bed.

December 9.—Leucocytes, 22,300 per c.m.

December 13.—Leucocytes, 21,500 per c.m. Faint trace of albumin found in the urine.

NAME. *Bessie C.*

No. 4.



I = Day on which rash was most marked.
 + = " " " " " last seen.
 O = " " " desquamation commenced.

December 18.—Leucocytes, 24,000 per c.m.

December 27.—Leucocytes, 12,000 per c.m.

January 1.—Leucocytes, 11,000 per c.m. The skin is rough, and there is some peeling about the legs and feet. The patient has improved very much of late.

January 3.—Sent to a Convalescent Home.

Differential count.—November 15, fifth day of disease.—Lymphocytes, 25.3 per cent.; large mononuclear, 5.1 per cent.; finely granular, 71.07 per cent.; coarsely granular, .3 per cent.

December 2, twenty-third day of disease.—Lymphocytes, 27.6 per cent.; large mononuclear, 12.3 per cent.; finely granular, 59.1 per cent.; coarsely granular, .8 per cent.

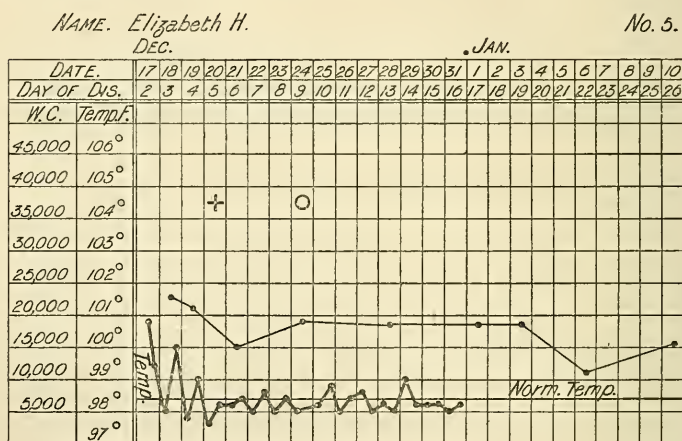
V.—Elizabeth H., aged $2\frac{1}{2}$ years, admitted into Hospital on December 17, 1895.

On December 16 the patient vomited and complained of sore throat.

On December 17 a faint but distinct punctiform rash was visible on the trunk; it was bright on the thighs, and on the buttocks and legs was in patches.

December 18.—The rash still bright. The tongue was red and the throat slightly injected. Leucocytes, 22,500 per c.m.

December 19.—Rash fading; some blotches present on the legs. Leucocytes, 21,000 per c.m.



December 21.—Rash disappeared. Throat still slightly injected. Glands just palpable. Leucocytes, 15,000 per c.m.

December 23.—Baths commenced.

December 24.—Powdery desquamation about forearms. Leucocytes, 19,000 per c.m.

December 28.—Looks well. Desquamation very slight. Leucocytes, 18,500 per c.m.

January 1.—Leucocytes, 18,500 per c.m.

January 3.—Leucocytes, 18,500 per c.m.

January 6.—Allowed to get up. Some roughness about the skin on the arms. Leucocytes, 11,000 per c.m.

January 10.—Leucocytes, 16,500. Patient sent to a Convalescent Home.

Differential count.—December 18, third day of disease.—Lymphocytes, 15.3 per cent. ; large mononuclear, 6.4 per cent. ; finely granular, 72.7 per cent. ; coarsely granular, 4.7 per cent.

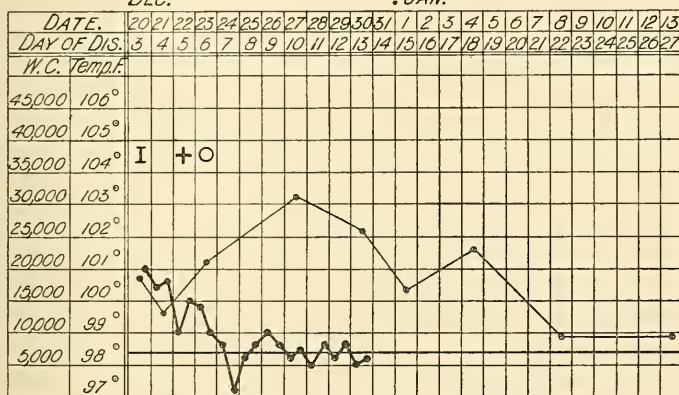
December 28, thirteenth day of disease.—Lymphocytes, 21.1 per cent. ; large mononuclear, 8.2 per cent. ; finely granular, 69.1 per cent. ; coarsely granular, 1.5 per cent.

VI.—Cora L., aged 14, admitted into Ward 8 on December 20. On December 18 the patient vomited and complained of sore throat.

On December 19 a rash was observed.

NAME. Cora L.
DEC.

No. 6.



I = Day on which rash was most marked.
+ = " " " " " last seen.
O = " " " desquamation commenced.

On December 20, when admitted into the Hospital, there was an erythematous rash over the body, punctate in character. The tongue was furred and the fauces inflamed. The lymphatic glands at the angles of the jaws were enlarged. Leucocytes, 18,500 per c.m.

On December 21 the rash was fading. The tongue was clean, strawberry. Tonsils large, fairly clean, but still red. Leucocytes, 13,000 per c.m.

December 23.—No rash visible now. Desquamation commencing, powdery in character. Throat not so injected. Tongue strawberry in character. Leucocytes, 21,000 per c.m.

December 27.—Desquamation, powdery in character. Leucocytes, 32,500 per c.m.

December 30.—Leucocytes, 25,500 per c.m.

December 31.—Allowed out of bed for the first time.

January 1.—Pale in appearance. Leucocytes, 16,500 per c.m.

January 4.—Leucocytes, 24,000 per c.m.

January 8.—Leucocytes, 9500 per c.m.

January 11.—Specimen of urine examined; contained no albumin.

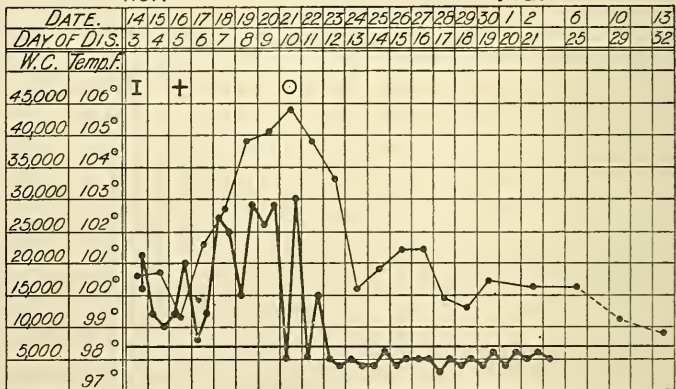
January 13.—Patient peeling still. Leucocytes, 10,000 per c.m.

January 17.—Patient has been sent to a Convalescent Home.

VII.—Edith M., aged 3½.

On November 11 the patient complained of headache. The

NAME. *Edith M.* AGE. *3½.* No. *7.*
Nov. DEC.



I = Day on which rash was most marked.
+ = " " " " " last seen.
O = " " " desquamation commenced.

following day she vomited, her throat was sore, and a rash was noticed.

On November 14 she was admitted into the Hospital with a slight punctate rash; the tongue was furred, and the throat slightly injected. A systolic murmur could be heard over the cardiac area. Leucocytes, 18,000 per c.m.

November 15.—Leucocytes, 18,500 per c.m.

November 16.—A strawberry tongue. Glands at angles of jaw slightly enlarged. The rash is fading rapidly. Leucocytes, 11,400 per c.m.

November 17.—The rash has disappeared. Leucocytes, 23,600 per c.m.

November 18.—Child very irritable. An enlarged and tender gland can be felt at the left angle of the jaw. Leucocytes, 28,800 per c.m.

November 19.—Leucocytes, 39,200 per c.m.

November 20.—Glands at left angle of jaw enlarged. Leucocytes, 40,800 per c.m.

November 21.—Desquamation commenced. Leucocytes, 44,600 per c.m.

November 22.—General condition improved. Leucocytes, 39,200 per c.m.

November 23.—Leucocytes, 33,200 per c.m.

November 24.—Leucocytes, 16,800 per c.m. Baths directed to be given twice a week.

November 25.—Leucocytes, 19,600 per c.m.

November 26.—Leucocytes, 22,300 per c.m.

November 27.—Leucocytes, 22,300 per c.m.

November 28.—Leucocytes, 14,700 per c.m.

November 29.—Leucocytes, 13,600 per c.m.

November 30.—Leucocytes, 17,400 per c.m.

December 2.—Leucocytes, 16,800 per c.m.

December 6.—Leucocytes, 16,300 per c.m.

December 10.—Leucocytes, 11,000 per c.m.

December 13.—Leucocytes, 9300 per c.m. Desquamation ceased.

VIII.—John M., aged 17. Admitted December 20, 1895.

December 19.—Patient vomited and complained of sore throat.

December 20.—Throat much inflamed, with much secretion about the tonsils. An indistinct rash about the arms.

December 21.—Tongue is coated with thick white fur. Fauces much inflamed, and there is a quantity of thick viscid secretion about the back of the throat. An indistinct erythema is seen about the body. Leucocytes, 14,500 per c.m.

December 23.—Much improved. Rash just visible; papillae of skin prominent. Leucocytes, 21,000 per c.m.

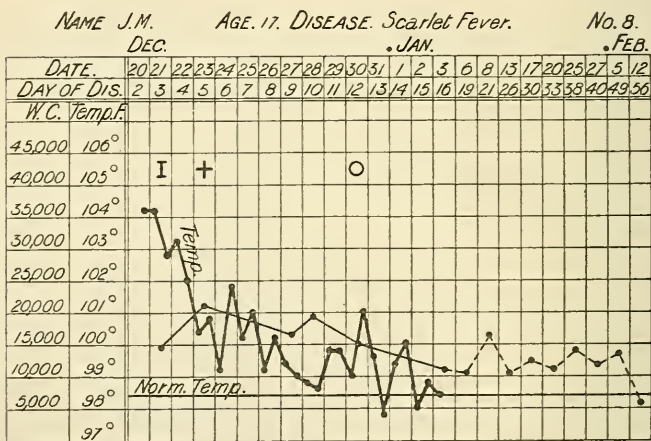
December 27.—Throat much cleaner. Leucocytes, 16,500 per c.m.

December 28.—Leucocytes, 19,500 per c.m.

December 30.—Desquamation seen about the arms. Leucocytes, 15,000 per c.m.

January 3.—Allowed to get out of bed. Baths given twice a week. Leucocytes, 11,000 per c.m.

January 6.—Leucocytes, 10,500 per c.m.
 January 8.—Leucocytes, 16,500 per c.m.
 January 13.—Leucocytes, 10,600 per c.m.
 January 17.—Leucocytes, 12,500 per c.m.
 January 20.—Leucocytes, 11,500 per c.m.
 January 25.—Leucocytes, 14,000 per c.m.
 January 27.—Leucocytes, 11,500 per c.m.
 February 5.—Leucocytes, 13,500 per c.m.
 February 12.—Leucocytes, 5600 per c.m. Some roughness
 about heels.
 February 13.—Sent to a Convalescent Home.



I = Day on which rash was most marked.
 + = " " " " " last seen.
 O = " " " desquamation commenced.

Differential count.—December 21, third day.—Lymphocytes, 4.21 per cent.; large mononuclear, 3.5 per cent.; finely granular, 90.9 per cent.; coarsely granular, 1.3 per cent.

December 28, fifteenth day.—Lymphocytes, 26.06 per cent.; large mononuclear, 14.21 per cent.; finely granular, 57.3 per cent.; coarsely granular, 2.6 per cent.

January 25, thirty-fourth day.—Lymphocytes, 31.45 per cent.; large mononuclear, 7.27 per cent.; finely granular, 59.09 per cent.; coarsely granular, 2.18 per cent.

January 27, forty-first day.—Lymphocytes, 32.11 per cent.; large mononuclear, 7.11 per cent.; finely granular, 59.34 per cent.; coarsely granular, 1.42 per cent.

December 13.—Rash faded; desquamation has commenced; throat has improved very much. Leucocytes, 24,000 per c.m.

December 14.—Leucocytes, 15,000 per c.m.

December 16.—Leucocytes, 11,300 per c.m.

December 17.—Leucocytes, 9300 per c.m.

December 18.—Leucocytes, 9500 per c.m.

December 20.—Leucocytes, 9500 per c.m. No precise reason can be given for the raised temperature of the last few days.

December 23.—Leucocytes, 5500 per c.m. I am not able to account for the low leucocytosis of the last week.

December 28.—Leucocytes, 11,500 per c.m. Allowed to get out of bed.

December 30.—Leucocytes, 15,500 per c.m.

January 3.—Leucocytes, 11,000 per c.m.

January 6.—Leucocytes, 11,500 per c.m.

January 10.—Leucocytes, 13,500 per c.m.

January 14.—Sent to a Convalescent Home.

Differential count.—December 4, first day of disease.—Lymphocytes, 9.5 per cent.; large mononuclear, 5.4 per cent.; finely granular, 84 per cent.; coarsely granular, .9 per cent.

December 7, fourth day of disease.—Lymphocytes, 8.2 per cent.; large mononuclear, 6.6 per cent.; finely granular, 84.1 per cent.; coarsely granular, .9 per cent.

December 18, tenth day of disease.—Lymphocytes, 40 per cent.; large mononuclear, 13.8 per cent.; finely granular, 45.1 per cent.; coarsely granular, 1 per cent.

December 30, twenty-seventh day of disease.—Lymphocytes, 35.2 per cent.; large mononuclear, 11.39 per cent.; finely granular, 49.6 per cent.; coarsely granular, 3.6 per cent.

X.—Percy D., aged 7.

On December 12 complained of sore throat and was said to have been feverish.

On December 13 admitted into Western Fever Hospital with an erythematous rash, punctiform in character, seen best on the arms. Tongue papillated, fairly clean. The posterior cervical glands on the left side swollen.

December 14.—Rash fading, pink condition on the trunk, punctiform in character. Leucocytes, 22,000 per c.m.

December 16.—Patches of discoloration about the body; slight redness still present about the legs. The patient complained this evening of pain in the wrist joints and in some of the phalangeal joints of both hands. The affected joints were swollen and tender to the touch, and the skin over them reddened. Leucocytes, 24,500 per c.m.

much less. There is still a good deal of induration at the angles of the jaws. The patient's general condition has much improved. Leucocytes, 38,100 per c.m.

November 23.—Leucocytes, 37,600 per c.m.

November 24.—Leucocytes, 26,700 per c.m.

November 25.—Bath has been directed to be given twice a week. Leucocytes, 21,200 per c.m.

November 26.—Leucocytes, 26,100 per c.m.

November 27.—Leucocytes, 13,000 per c.m.

November 28.—Leucocytes, 21,200 per c.m.

November 29.—Discharge from the nose and right ear almost ceased. Glands at the angles of the jaw still palpable. Leucocytes, 16,300 per c.m.

November 30.—Leucocytes, 29,900 per c.m.

December 2.—Leucocytes, 15,800 per c.m.

December 4.—Allowed to get up. Leucocytes, 17,400 per c.m.

December 9.—Leucocytes, 14,700 per c.m. Desquamation confined to the legs.

December 11.—Sent to a Convalescent Home.

XII.—Thomas E., aged 11.

On November 13 the patient complained of sore throat and headache. The next day a rash was observed, and on November 15 he was admitted into the Hospital with a faint general punctate rash. The fauces were inflamed, and a bacteriological examination of the secretion did not reveal the presence of the bacillus of diphtheria.

November 16.—Leucocytes, 60,000 per c.m.

November 17.—Leucocytes, 40,300 per c.m.

November 18.—Rash disappeared. Leucocytes, 20,100 per c.m.

November 19.—Baths started and given twice a week. Desquamation commencing. Leucocytes, 38,100 per c.m.

November 20.—Leucocytes, 22,800 per c.m.

November 21.—Leucocytes, 17,900 per c.m.

November 22.—Leucocytes, 17,900 per c.m.

November 23.—Leucocytes, 27,200 per c.m.

November 24.—Leucocytes, 25,000 per c.m.

November 25.—Leucocytes, 26,700 per c.m.

November 26.—Leucocytes, 16,800 per c.m.

November 27.—Leucocytes, 20,100 per c.m.

November 28.—Leucocytes, 14,700 per c.m.

November 29.—Leucocytes, 16,800 per c.m.

November 30.—Leucocytes, 12,000 per c.m.

December 2.—Leucocytes, 18,500 per c.m.

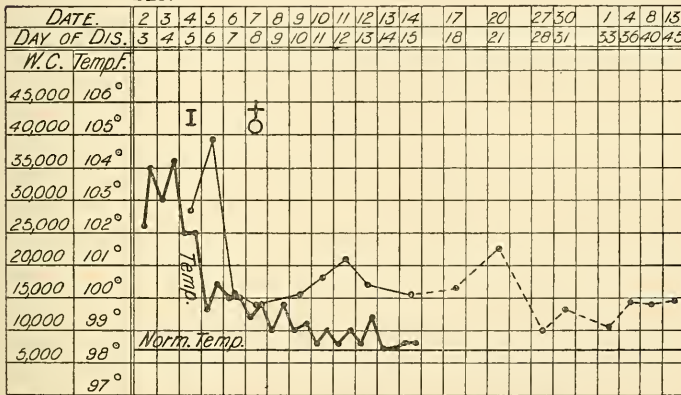
fauces were a dusky red, and the tonsils were large and covered with secretion.

December 4.—A bright punctate rash was visible all over the body. The throat was much inflamed, the tongue was dry and cracked, the pulse was regular but poor in volume and tension. The inspiration was hurried. The cough was accompanied with the expectoration of rusty sputum, and there were signs of consolidation at the base of the right lung—pneumonia. Leucocytes, 28,300 per c.m.

December 5.—Leucocytes, 39,800 per c.m.

December 6.—Crisis in the pneumonia yesterday. The

NAME. Florence M. AGE. 14. NO. 13.
DEC. JAN.



I = Day on which rash was most marked.
 † = " " " " " last seen
 O = " " " desquamation commenced.

physical signs in the lung clearing up. Rash fading. Throat not so dusky, cleaner; on the uvula and fauces small patches of superficial ulceration are visible. Leucocytes, 15,200 per c.m.

December 7.—Desquamation commenced. Leucocytes, 14,100 per c.m.

December 8.—Rash not visible.

December 9.—Leucocytes, 15,500 per c.m. A simple expectorant mixture given.

December 10.—Leucocytes, 18,500 per c.m.

December 11.—Leucocytes, 21,000 per c.m.

December 12.—Leucocytes, 17,000 per c.m.

December 14.—Leucocytes, 15,500 per c.m.

December 17.—Leucocytes, 16,500 per c.m. Throat cleaner. Tonsils have a ragged appearance.

December 20.—Leucocytes, 22,500 per c.m. Allowed to get up. Pale in appearance.

December 27.—Leucocytes, 10,000 per c.m.

December 30.—Leucocytes, 13,500 per c.m.

January 1.—Leucocytes, 10,500 per c.m.

January 4.—Leucocytes, 14,500 per c.m.

January 8.—Leucocytes, 14,000 per c.m.

January 13.—Leucocytes, 14,500 per c.m. Desquamation on the feet.

December 17.—Sent to a Convalescent Home.

Differential count.—December 4, fifth day of disease.—Lymphocytes, 5.6 per cent.; large mononuclear, 3.8 per cent.; finely granular, 89.1 per cent.; coarsely granular, 1.3 per cent.

January 13, forty-third day of disease.—Lymphocytes, 21.34 per cent.; large mononuclear, 10.38 per cent.; finely granular, 65.19 per cent.; coarsely granular, 3.07 per cent.

ON THE RELATIONSHIP OF
CARDIAC HYPERTROPHY AND ŒDEMA TO
CHRONIC RENAL DISEASE.

BY

H. HOLMES, M.B., AND A. A. KANTHACK, M.D.

Since Dr. Richard Bright first drew the attention of the medical profession to the disease now called after his name, the frequent association of cardiac hypertrophy and œdema therewith has become well known.

With respect to cardiac changes Dr. Bright himself remarks: "The deviations from health in the heart are well worthy of observation; they have been so frequent as to show a most important and intimate connection with the disease of which we are treating."

Speaking of cardio-vascular changes that occur in renal diseases, the late Dr. Fagge in his Text-book of the Principles and Practice of Medicine observed: "Hypertrophy of the heart occurs in acute as well as chronic cases of morbus Brightii. It may also develop when the kidneys have become atrophied as the result of hydronephrosis, or of some other affection of the renal pelvis, as in cases recorded by Cohnheim. In association with the lardaceous change, however, it is not seen unless the renal cortex has become also affected with advanced tubal nephritis. It is comparatively slight when complicated by phthisis, cancer, or some other wasting disease, and in those who are very old."

He also says: "The extent to which the heart becomes enlarged differs in different cases—partly according to their duration, and it is far greater in renal cirrhosis than in any other form. Thus whereas in the earlier stages of tubal nephritis its weight may attain fifteen or sixteen ounces, and in the latest granular and atrophic stage of that affection seventeen, or possibly twenty-one ounces, there are some instances of (primary) renal cirrhosis in which it reaches twenty-three,

twenty-four, twenty-five, or even twenty-eight ounces. The chamber chiefly affected is the left ventricle, the walls of which (and also the papillary columns of the mitral valve) become extraordinarily thick and fleshy, their substance being made up of muscular fibres of perfectly normal appearance. Sometimes the cavity is of normal size, sometimes more or less dilated. This generally indicates that the left ventricle has not been able to maintain the circulation efficiently, or that pulmonary obstruction has arisen from bronchitis, œdema of lung, or some other cause. But to some extent it is inevitable that the right ventricle should share in the process of enlargement, especially when the left ventricle becomes very greatly increased in size."

Speaking of the dropsy that occurs in Bright's disease, Dr. Fagge, after pointing out that it is of two kinds, says: "One is identical in its characters with that seen in heart disease, and depends upon obstruction of the systemic veins. When it appears in the course of morbus Brightii, it is only an indirect effect of the primary malady, its immediate cause being failure of the heart to maintain the needful activity of the circulation. It is always more marked in the dependent parts of the body than elsewhere, especially in the lower limbs; and it is associated with dyspnœa, with orthopnœa, and often with lividity. It occurs only in the most chronic forms of Bright's disease, usually when the kidneys are contracted, red, and granular, *i.e.*, cirrhotic."

"Widely different from this is the kind of dropsy which, although perhaps not absolutely more frequent, has been always justly associated with Bright's disease as its characteristic symptom. This kind of dropsy often begins in the face, about the eyelids, even before it affects the ankles. Its distribution is not independent of the influence of gravitation; for one may often notice that whereas the face is œdematous when the patient rises in the morning, this subsides towards the latter part of the day, and the ankles are swollen when he goes to bed. But it is not limited to the dependent regions of the body, like the other form of dropsy, and it is not accompanied by dyspnœa or lividity. Its favourite seats are the eyelids, conjunctiva, the penis and scrotum (or the labia in women), and the loins, where it forms what Bright called 'the renal cushion.' We may explain the two former seats as due to the fact that the skin of the eyelids and genital organs has no subcutaneous fat. Often, however, the whole of the body and limbs swell at the same time, and acquire a peculiar white, waxy appearance, which is very characteristic. The occurrence of such general dropsy is frequently the earliest symptom of

Bright's disease, and first draws the patient's attention to the fact that he is unwell."

It was long thought that hypertrophy of the left ventricle was chiefly confined to cases of interstitial nephritis, the cirrhotic kidney of Dr. Fagge; but Drs. Galabin and Goodhart have shown from analysis of the Guy's Hospital post-mortem records that it occurred in a far greater percentage of cases of tubal nephritis than was generally believed.

Dr. Goodhart analysed all the cases of Bright's disease that died in a period of ten years, 1873-82. He obtained notes of 344 cases of granular kidneys, and 196 cases of parenchymatous nephritis. After deducting all cases of children and those who died with wasting disease, he found hypertrophy of the heart in 226 out of 251 cases of granular kidneys, and in 109 out of 134 cases of tubal nephritis—90 per cent. in the one case, 81.3 per cent. in the other. He took the average weight of the heart to be twelve ounces, which is generally held to be rather high.

We have thought that an analysis of the post-mortem records of St. Bartholomew's Hospital might be of interest and of some importance. Accordingly we have selected a period of five years, 1891-95, a sufficiently lengthy period to make the figures obtained of some little value. At the same time we have made an attempt to trace the incidence of cardiac and renal oedema in interstitial and parenchymatous nephritis respectively.

We have obtained notes of nearly 380 cases of renal disease, variously described as granular kidney or interstitial nephritis, tubal or parenchymatous nephritis, mixed tubal and interstitial nephritis, amyloid or lardaceous disease, consecutive nephritis and acute nephritis. Though these divisions are not altogether satisfactory in some respects, we have thought it better to keep to the same, and not to try to re-classify those cases described as mixed nephritis under the groups interstitial and tubal nephritis, according to the account given in the post-mortem books.

In Table I. a list of all the cases collected from the St. Bartholomew's Hospital records will be found, classified under the above-mentioned headings.

TABLE I. (A.).—*Chronic Interstitial Nephritis.*

No.	Sex.	Age.	Stage.	Weight and Condition of Heart.		Arteries.	Other Diseases.	Œdema.	Remarks.
4.	Female.	55.	Slight.	...	Much enlarged, L. V. hypertrophied. R. side dilated.	Advanced atheroma.
5.	Male.	63.	Slight.	11¼ ozs.	Normal.	Atheroma.	Thin and grey.
6.	Female.	57.	L. V. very thick.
8.	Male.	39.	Slight.	...	Natural.	Normal.	Cirrhosis, pneumonia.
9.	Male.	?	L. V. hypertrophied.
10.	Female.	60.	L. V. somewhat hypertrophied; dilation of all the cavities.	...	Emphysema.	Dropical.	...
11.	Male.	66.	...	12 ozs.	R. side dilated.	...	Gout.
12.	Female.	68.	...	16 ozs.	...	Considerable atheroma.
14.	Male.	58.	Pale, granular.	16 ozs.	L. V. hypertrophied.	Much atheroma.	Gout.	Some oedema of legs.	...
16.	Female.	32.	...	14 ozs.	L. V. much hypertrophied.
18.	Male.	54.	...	15½ ozs.	L. V. much hypertrophied.	No atheroma in big vessels.
19.	Female.	?	R. heart widely dilated.	Much, specially of legs.	...
20.	Male.	55.	Normal.	Aorta very atheromatous.

21.	Female.	62.	Atheroma.
22.	?	?	...	L. V. thick.	Extreme atheroma.
24.	Male.	62.	...	L. V. hypertrophied.	Atheroma.
25.	Female.	44.	...	11½ ozs.	No atheroma to speak of.	Pneumonia.	...
27.	Male.	58.	Not much atheroma.
28.	Male.	32.	No atheroma to speak of.	Gout.	...
29.	Female.	66.	Dilatation of R. side. L. V. hypertrophied. Aortic disease; malformation of valves. Some hypertrophy of L. and dilatation of R.; both slight.	Atheroma of P. A.	...
30.	Male.	?	...	22 ozs.	R. side widely dilated; L. side dilated and hypertrophied.	Much atheroma.	Anasarca of legs.
31.	Male.	65.	Normal, though rather small.	Slight atheroma.	Pneumonia.
32.	Female.	54.	Slight.	...	L. V. thick, and cavity dilated.
33.	Male.	45.	...	23½ ozs.	L. V. hypertrophied and dilated.	...	Gout, cirrhosis.
35.	Male.	44.	Slight.	...	Normal; not hypertrophied.	...	Gout.
36.	Male.	67.	Not hypertrophied. Weight rather under than above normal.	Atheroma.	Cirrhosis.
37.	Female.	34.	R. auricle and R. V. a little hypertrophied; cavities dilated.	...	Infective endocarditis.
38.	Male.	66.	L. V. hypertrophied.	Marked atheroma of aorta and P. A.	Sarcoma of tonsil.

TABLE I. (A.)—Continued.

No.	Sex.	Age.	Stage.	Weight and Condition of Heart.	Arteries.	Other Diseases.	Edema.	Remarks.
39.	Female.	46.	Slight.	Mitral orifice narrowed.
41.	Male.	20.	...	L. V. hypertrophied; all cavities dilated.	Slight atheroma.	...	Nil.	...
42.	Male.	67.	...	Heart rather large.	Slight atheroma.
43.	Male.	45.	...	Considerable hypertrophy of L. V.	...	Gout.
44.	Male.	50.	?	L. V. enlarged, some hypertrophy.	Slight atheroma; extensive in coronaries.	Doubtful, whether chronic congestion or interstitial.
45.	Female.	50.	...	Slight dilation of R. V.	Some atheroma.	Fibroid lung.
48.	Female.	43.	...	General hypertrophy; dilation.	Very little atheroma.	Mitral stenosis.
49.	Male.	55.	...	General hypertrophy and dilation.	Extensive atheroma in aorta.	Aortic disease.
52.	Female.	44.	...	Considerable hypertrophy of L. V. General dilation of R. side.
53.	Female.	33.	...	L. V. much hypertrophied.	Some atheroma.	...	Very slight.	...
54.	Female.	42.	...	R. side dilated.	Slight atheroma.	Stenosis of pylorus.	Slight.	...
55.	Male.	45.	...	Great dilation of all cavities; L. V. hypertrophied, though dilated in excess.	Dropsy, ascites.	...

56.	Male.	? 40.	Slight.	12 ozs.	Some dilatation of orifices.	Normal.
57.	Male.	49.	...	21 ozs.	General hypertrophy and dilatation; L. V. specially large.	Aorta sacculated.
58.	Female.	41.	Slight.	...	Normal.	Some atheroma.	Cirrhosis.
59.	Male.	38.	...	23 ozs.	All cavities dilated.	Normal.
61.	Female.	62.	...	14½ ozs.	...	Atheroma.
62.	Female.	23.	...	15 ozs.	Some dilatation of aorta, and mitral disease.	Slight atheroma.	M. C.	Of legs.	...
63.	Male.	46.	Considerable hypertrophy of L. V.	Marked atheroma.	Gout.
65.	Male.	49.	...	14 ozs.	Some hypertrophy of L. V. Some dilatation of R. side.	Scarcely any atheroma.	Disseminated tubercle.
68.	Female.	56.	...	14 ozs.	L. V. hypertrophied, though not largely.	High degree of atheroma.
71.	Female.	34.	...	14½ ozs.	L. V. thick, hypertrophied.
72.	Male.	46.	...	34 ozs.	Much hypertrophy.	Slight atheroma.	...	Much œdema of lower parts.	...
73.	Female.	40.	Slight.	...	L. V. hypertrophied; R. side much dilated.
74.	Male.	65.	Slight.	...	L. V. dilated and a little thickened.	Atheroma.	Emaciated.
75.	Male.	37.	...	18 ozs.	Considerable hypertrophy of L. V.	A little atheroma.
76.	Female.	38.	...	18 ozs.	Considerable hypertrophy of L. V., which forms bulk of heart.	Marked atheroma.
77.	Female.	57.	...	21 ozs.	L. V. much hypertrophied.	Atheroma.

TABLE I. (A.)—Continued.

No.	Sex	Age.	Stage.	Weight and Condition of Heart.	Arteries.	Other Diseases.	Œdema.	Remarks.
80.	Female.	23.	?	15 ozs. L. V. hypertrophied.	Some atheroma.	? Simple atrophy of kidney. ...
81.	Female.	36.	...	10 ozs. Normal.	Normal.	Phthisis of lungs and larynx. Slight atheroma.
82.	Male.	43.	?	23½ ozs. Enlargement, chiefly due to hypertrophy of L. V.	? Chronic congestion. ...
83.	Female.	48.	Slight.	... L. V. showed some hypertrophy.	Atheroma.
84.	Female.	65.	...	12 ozs.
85.	Male.	38.	Slight.	... Normal; not enlarged.	Aneurysm of descending arch of aorta. Normal.
87.	Male.	35.	...	14 ozs. Some general dilatation.	Normal.
88.	Male.	56.	?	16 ozs. R. V. very thin.	Normal.	Adherent pericardium. ...	Slight in legs. General anasarca.	? Chronic congestion. ...
89.	Male.	49.	...	18 ozs. L. V. thick and dilated; R. side very large, dilated.	Marked atheroma.
90.	Female.	64.	...	22 ozs. Considerable hypertrophy of L. V.	Some atheroma.	Adherent pericardium.
92.	Male.	38.	Slight.	13 ozs. L. V. somewhat thick.	A little atheroma in aorta. Normal.
93.	Male.	31.	...	6½ ozs. ...	Normal.	Chronic phthisis. Leukæmia splenica.
94.	Male.	32.	..	10 ozs. L. V. somewhat thick.	Normal.

96.	Female.	20.	L. V. formed bulk of heart.	Normal.	Pneumonia.	...
97.	Male.	30.	L. V. dilated and hypertrophied; R. side dilated.	Atheroma of aorta; aneurysm of arch.	...	Of feet and legs.
98.	Male.	49.	All cavities dilated, and very thin-walled.	Slight.	Cirrhosis.	...
99.	Male.	24.	...	15 ozs.	Somewhat enlarged.	Some atheroma.
100.	Male.	40.	...	Nearly 20 ozs.	R. side dilated; L. V. hypertrophied.	No atheroma to speak of.
102.	Female.	52.	..	9½ ozs.	No dilatation or hypertrophy.	Normal.	Tubercle of lung and pleura.	...
103.	Male.	66.	...	24 ozs.	Much hypertrophy of L. V.; R. V. dilated.	Atheroma.
105.	Female.	41.	Not enlarged.	Some atheroma.	Fibroid phthisis. Glandular enlargement, pigmentation, and caseation in many glands of body.	...
106.	Female.	49.	...	12 ozs.	L. V. somewhat globular in shape; not hypertrophied to any degree. Looked small in comparison with rest of heart.	No atheroma to speak of.	Mitral stenosis.	...
110.	Female.	53.	Slight.	...	Slight hypertrophy.	A little atheroma.	Carcinoma of pancreas.	...
111.	Female.	26.	...	12 ozs.	L. auricle much thickened.	..	Advanced mitral stenosis.	...
112.	Female.	57.	A little hypertrophy of L. V.	...	Malignant disease of ovaries, &c.	Of lower extremities.
113.	Female.	63.	...	7 ozs.	Mitral stenosis.	...	Malignant disease of ovaries, &c.	...

TABLE I. (A.)—Continued.

No.	Sex.	Age.	Stage.	Weight and Condition of Heart.	Arteries.	Other Diseases.	Edema.	Remarks.
114.	Male.	32.	...	Cardiac hypertrophy ; L. V. dilated.	Atheroma of aorta.
115.	Male.	62.	...	Normal.	Atheroma.
116.	Male.	?	?	Much hypertrophy of L. V. ; R. V. dilated. No valvular disease.	Atheroma of cere- brals.
117.	Male.	51.	...	Normal.	...	Pneumonia.
119.	Female.	52.	...	Slight enlargement of all cavities.	Normal.
120.	Female.	57.	...	Normal.	Normal.	Phthisis.
122.	Female.	28.	Slight.	Some hypertrophy of L. side.	...	Growth in pel- vis connecti- with cervix.
125.	Male.	53.	...	Some hypertrophy of L. V.	Aorta highly athe- romatous.	Gout.
127.	Female.	55.	...	No hypertrophy.	Slight atheroma.	Tetanus.
128.	Male.	69.	Slight atheroma.
134.	Male.	67.	...	No hypertrophy.	Much atheroma.	Stenosis of py- lorus.
135.	Female.	39.	...	Normal, except for slight increase.	Normal.	Malignant dis- ease of uterus.
137.	Male.	74.	...	No hypertrophy to speak of.	Atheroma.	Gout.
138.	Female.	55.	...	Much enlargement; both ventricles dilated.	Atheroma of aorta and P. A.	Aortic disease.	..	Dropsy of legs.

TABLE I. (A.)—Continued.

No.	Sex.	Age.	Stage.	Weight and Condition of Heart.	Arteries.	Other Diseases.	Edema.	Remarks.
162.	Male.	63.	...	Normal.	Slight dilatation; some atheroma of aorta.	Cirrhosis, old caseous masses in lung.
163.	Female.	35.	Slight.	L. V. decidedly small in size. R. heart dilated.	A few patches of atheroma in aorta.	Mitral stenosis.
164.	Male.	60.	...	Much hypertrophy of walls of L. V. R. V. dilated.	Much atheroma.	Mitral stenosis. Gout.	Of legs and penis.	...
165.	Female.	62.	...	L. V. not notably thickened. R. side dilated; walls thin.	...	Mitral stenosis. Adherent pericardium.	Very dropsical.	...
166.	Female.	48.	...	Much hypertrophy of L. V.	A good deal of atheroma.	...	Slight of legs.	...
167.	Female.	45.	...	L. V. normal size. R. V. dilated.	A little atheroma of aorta.
168.	Male.	53.	Slight.	R. side somewhat dilated.	Slight atheroma.	Cerebral tumour. Sarcoma. Gout.
169.	Male (?).	L. V. hypertrophied; R. V. somewhat dilated.	Extreme atheroma.
171.	Male.	27.	...	L. V. hypertrophied.
172.	Female.	59.	...	R. side dilated.	But little atheroma.	Pericardium adherent.
173.	Male.	40.	Slight.	R. V. dilated, but L. V. extremely so; of enormous size.	Singularly free from atheroma; a little in P. A.	Aortic and mitral disease.	Nil.	...
174.	Male.	50.	...	Small. No dilatation.	Normal.	Chronic phthisis.

175.	Female.	53.	...	13½ ozs.	Walls of L. V. thickened.	Some atheroma.
176.	Female.	61.	Small.	Normal.	...	Wasted.
177.	Male.	39.	Slight.	...	Some dilatation of L. V.
178.	Female.	11.	...	6 ozs.	Walls of L. V. appeared somewhat thick in proportion to size.	Slight atheroma.
179.	Male.	60.	Typical.	8 ozs.
181.	Male.	66.	...	21 ozs.	Hypertrophy of L. V.	Some atheroma.
182.	Male.	23.	...	14½ ozs.	L. V. somewhat hypertrophied; dilated.	Normal.
183.	Male.	48.	...	22 ozs.	Enlarged; L. V. dilated and hypertrophied.	Atheromatous.
184.	Male.	53.	...	16 ozs.	Some hypertrophy of L. V.
185.	Male.	41.	Small, otherwise natural.
186.	Female.	34.	L. V. hypertrophied to some extent; R. V. thin.	Some atheroma.
188.	Male.	64.	Flabby, otherwise natural.	Atheroma of aorta, R. radial; splenic, renals.
189.	Male.	59.	Normal.	Normal.
194.	Female.	35.	Rather large; flabby.
196.	Male.	?	...	20 ozs.	L. V. hypertrophied.	Aorta highly atheromatous.

TABLE I. (A.)—Continued.

No.	Sex.	Age.	Stage.	Weight and Condition of Heart.	Arteries.	Other Diseases.	Œdema.	Remarks.
197.	Male.	54.	Typical.	... Not enlarged.	...	Cirrhosis.
199.	Female.	21.	...	17 ozs. Dilated; some hypertrophy of L. V. Very small.	Atheroma of aorta.	...	Great, general anasarca.	...
200.	Female.	71. L. V. wall thick.	Normal.	Cancer of pylorus.
201.	Male.	46.	...	14 ozs. L. V. wall thick.	Natural.
202.	Male.	58.	...	16 ozs. Hypertrophy of L. V.	Normal.
203.	Female.	41.	...	14½ ozs. R. V. thin in places.	Very slight atheroma.
204.	Female.	56.	...	18 ozs. L. V. and R. side much dilated.	Slight atheroma.	Mitral stenosis.
205.	Male.	66.	?	13 ozs. Dilated, flabby, specially R. side.	Patches of atheroma.	? Atrophy.
206.	Male.	51.	...	11 ozs. Dilated R. V.	Nil.	...	Œdematous.	...
209.	Female.	39.	...	8½ ozs. Epicardial fat in excess, else natural.	Nil.
210.	Male.	34.	Slight.	13½ ozs. L. V. hypertrophied and a little dilated; wall fully one inch thick, rounded.	Nil.
211.	Male.	36.	...	24 ozs. Heart generally hypertrophied, but more so on L. side. L. V. thick and dilated.	Natural.

213.	Female.	35.	...	6 ozs.	Walls soft and flabby.	...	Emaciated.
214.	Female.	45.	Moderate.	11 ozs.	Muscular papillæ stand out, suggesting an early degree of hypertrophy.	Some atheroma of aorta.	...
215.	Male.	57.	...	7½ ozs.	...	Some atheroma.	Emaciated.
216.	Male.	40.	Slight.	12 ozs.	Generally dilated and flabby.	Slight thickening of intima above valves.	...
217.	Female.	59.	...	17 ozs.	L. V. hypertrophied; cavity rather dilated.	Some atheroma.	Ruptured L. V.
218.	Female.	40.	...	12 ozs.	L. V. dilated and hypertrophied; walls much thickened.	...	Aortic disease.
219.	Male.	42.	Slight.	11 ozs.	L. side generally dilated, no hypertrophy.	Natural.	...
220.	Female.	60.	...	11 ozs.	L. V. dilated and hypertrophied, but not extremely so.	Some thickening of intima of aorta.	Epithelioma of vulva (removed).
221.	Male.	72.	...	12½ ozs.	L. V. hypertrophied and not much dilated.	Aorta atheromatous.	Cirrhosis. New growth of gall bladder.
222.	Male.	53.	L. V. hypertrophied and rather dilated.	Atheroma.	Aneurism of ascending arch of aorta.
223.	Male.	60.	...	17 ozs.	L. V. hypertrophied; cavity somewhat dilated.	Aorta highly atheromatous.	...
225.	Male.	50.	Both sides dilated and hypertrophied. L. V. very large.	Aorta generally dilated and atheromatous.	...
226.	Male.	52.	...	12 ozs.	L. side rather hypertrophied.	Aorta atheromatous.	Aneurysm of descending arch.

TABLE I. (A.)—Continued.

No.	Sex.	Age.	Stage.	Weight and Condition of Heart.		Arteries.	Other Diseases.	CE lema.	Remarks.
228.	Female.	24.	...	16 ozs.	Ventricles both somewhat dilated and hypertrophied. Natural.	...	Mitral stenosis and regurgitation.	General anasarca.	...
229.	Male.	48.	...	8 ozs.	Natural.	Early atheroma of aorta.	Cancer of oesophagus.
230.	Male.	57.	...	8 ozs.	Mitral orifice slightly constricted.	...	New growth of osmentum.	...	Rather wasted.
232.	Male.	44.	...	11 ozs.	Soft and flabby.	Slight atheroma.	Pneumonia.
233.	Female.	32.	...	10 ozs.	L. V. rather thick.	Natural.	Chrrhosis.
234.	Male.	42.	...	12 ozs.	Flabby.	Natural.	Pneumonia.
235.	Male.	42.	...	32 ozs.	Enlargement practically confined to L. V. Cavity not dilated to any extent. Aortic valves natural.	Slight atheroma of aorta. Radials very hard.	Gout.
236.	Male.	41.	...	11 ozs.	No enlargement of L. V.	Nil.	Bacterial fever.
237.	Male.	55.	...	10 ozs.	...	Atheroma.	Aneurysm of ascending arch.
239.	Male.	29.	Cyanache cellularis.
240.	Male.	49.	...	7 ozs.	Apex pointed.	Remarkably free from atheroma.	Phthisis.
241.	Male.	16.	...	8 ozs.	Natural.	Natural.	Pyo-pneumothorax.

242.	Male.	60.	...	17 ozs.	L. V. remarkably hypertrophied.	Few patches of atheroma.
243.	Male.	49.	Slight.	10 ozs.	Flabby; somewhat dilated.	Patches of atheroma.	Cirrhosis.	...	Anasarca of legs and scrotum.
244.	Male.	71.	...	18 ozs.	General hypertrophy and dilatation. L. V. wall rather thick.	Very slight atheroma.	Cirrhosis.
245.	Female.	55.	...	11 ozs.	L. V. hypertrophied, but not extremely.	Some atheroma. Aneurysm of branch of Sylvian.
246.	Male.	69.	...	14½ ozs.	Dilatation of ventricles, with hypertrophy of L. V.	General atheroma.
247.	Female.	?	...	14 ozs.	R. heart distended. L. V. hypertrophied.	Natural.	Tricuspid incompetence.
248.	Female.	59.	...	11 ozs.	Flabby, epicardial obesity.	Atheroma of abdominal aorta.	New growth of pylorus.
249.	Female.	33.	Slight.	10 ozs.	Ventricles cut a little tough.	Natural.	Pneumonia.
250.	Female.	69.	...	12 ozs.	L. V. rather dilated and hypertrophied. Much epicardial fat.	Extreme atheroma of aorta.
251.	Male.	47.	...	19 ozs.	L. V. hypertrophied and dilated. Substance of wall flabby.	Some atheroma.
252.	Male.	71.	...	14 ozs.	L. V. hypertrophied.	Radials hard; cerebrials atheromatous.
253.	Male.	40.	...	12 ozs.	L. V. hypertrophied. Cavity not much dilated. Of natural shape.	Slight atheroma.	Tubercle of lung and intestine.
254.	Male.	75.	...	16 ozs.	L. V. hypertrophied.	Patches of atheroma. Aneurysm of anterior communicating.

TABLE I. (A.)—Continued.

No.	Sex.	Age.	Stage	Weight and Condition of Heart.	Arteries.	Other Diseases.	Edema.	Remarks.
255.	Male.	30.	...	L. V. rather thick and tough.	Natural.	Pneumonia.
256.	Male.	61.	...	R. side dilated. L. V. hypertrophied.	Not much atheroma.	Emphysema.
257.	Male.	75.	...	Both sides of heart enlarged.	Endoarteritis obliterans of brachial, &c. Slight atheroma.	Emphysema.	Some about ankles.	...
258.	Female.	65.	...	R. side greatly dilated.	Atheroma not extensive.	Mitral stenosis. Tricuspid incompetence.	Legs anasarcaous.	...
259.	Male.	47.	...	R. side generally dilated. L. V. extremely so. Wall thin (no hypertrophy).	General rather extensive atheroma.
260.	Male.	38.	...	No hypertrophy or dilation.	Nil.	New growth of pylorus.
261.	Female.	23.	...	Much hypertrophy, chiefly L. V.; also R. V. to a less extent.	...	Intestinal ulceration. Tubercle of lung.
263.	Male.	20.	...	L. V. cuts toughly, hard, muscular papillares short, stringy, and tough.	Natural.	Psoas abscess. Hip disease.
265.	Male.	46.	...	L. V. much hypertrophied; shape retained perfectly.	Some atheroma.
266.	Male.	51.	...	Nothing abnormal, but small size.	Natural.	New growth of liver.	...	Emaciated.

267.	Male.	27.	...	12 ozs.	Natural in shape, flabby.	L. V.	Natural.	Plumbism.	...
270.	Female.	44.	...	8½ ozs.	Natural.	Alcoholic neuritis. Cirrhosis.	...
271.	Male.	53.	...	21 ozs.	L. V. hypertrophied, but not excessively.	...	Natural, but coronaries patent.	Aortic disease; adherent pericardium.	...
273.	Male.	63.	...	14 ozs.	L. V. hypertrophied. No dilatation of cavity.	...	Cerebrals atheromatous.
274.	Female.	54.	...	11 ozs.	L. V. rather thick-walled.	...	Natural.	Pneumonia.	...
275.	Male.	45.	...	26 ozs.	R. side dilated; L. V. greatly thickened. Though dilated, is not altered in shape.	...	Atheroma, Aorta very atheromatous.	Empysema.	...
276.	Male.	69.	...	15 ozs.	L. side hypertrophied, but not excessively.	...	Atheroma of aorta and iliacs.
278.	Male.	58.	...	12 ozs.	Aorta highly atheromatous; aneurysm of aorta.
282.	Female.	71.	...	7 ozs.	Mitral slightly stenosed.	New growth of uterus and bladder.	...
283.	Female.	40.	...	12 ozs.	R. side much dilated and hypertrophied; L. A. dilated; mitral stenosed and incompetent.	...	Slight atheroma.	General syphilis.	General anasarca.
284.	Male.	57.	...	20 ozs.	L. V. moderately dilated and hypertrophied; mitral much constricted.	...	Slight atheroma of aorta and pulmonary artery.	Cirrhosis.	Legs pit slightly.
285.	Male.	About 70.	...	16 ozs.	Slight dilatation and hypertrophy.	...	Aorta and cerebral atheroma.	Fibroid induration of lung.	...
288.	Female.	58.	...	8 ozs.	Small.	...	Slight atheroma.	New growth of peritoneum.	...

TABLE I. (A.)—Continued.

No.	Sex.	Age.	Stage.	Weight and Condition of Heart.	Arteries.	Other Diseases.	Edema.	Remarks.
289.	Female.	36.	...	L. V. hypertrophied.	Aorta slightly atheromatous.	Spinal curvature.
290.	Female.	25.	...	L. V. hypertrophied and dilated; R. V. dilated.	Natural.	Mitral stenosis and incompetence.	Some of legs.	...
291.	Male.	68.	...	R. V. much dilated; L. V. hypertrophied and dilated, also R. auricle.	Atheroma not extensive.	Aortic disease.	Legs slightly edematous; penis much swollen.	...
292.	Female.	52.	...	L. V. somewhat hypertrophied.	Aorta atheromatous.
296.	Male.	47.	...	Soft and flabby.	...	New growth of pylorus, head of pancreas.
299.	Male.	61.	...	L. V. wall thick; ? hypertrophied, not dilated.	Ascending arch atheromatous and dilated.	Fibroid induration of lung; caseating tubercle.	Edema of both legs.	...
300.	Male.	71.	...	Hypertrophy of L. V.	Extensive atheroma.
302.	Male.	49.	...	Great hypertrophy and much dilatation of L. V.	Considerable atheroma.	Aortic incompetence.	Edema.	...
303.	Male.	63.	...	Chord. tendin. thickened.	Nil.	Cirrhosis.	Pitting about ankles.	...
304.	Male.	40.	...	L. V. not materially hypertrophied.	Aorta atheromatous. Aneurysm of transverse arch.

305.	Male.	53.	...	10½ ozs.	Generally enlarged, but perhaps more on L. side.	Atheromatous.	Slight mitral incompetence.
306.	Female.	63.	...	10 ozs.	No marked hypertrophy.	Very little atheroma.	...	Thin, edentulous.	...
307.	Female.	35.	...	13 ozs.	L. V. externally rounded, on section; wall thick, but cavity natural size.	All thickened.	Alcoholism.
308.	Male.	34.	...	24 ozs.	Both ventricles greatly hypertrophied and somewhat dilated.	Nil.	...	Of lower extremities.	...
312.	Male.	56.	...	26 ozs.	L. V. hypertrophied and considerably dilated; R. side slightly dilated.	All atheromatous.	...	Of lower extremities.	...
313.	Male.	50.	...	15 ozs.	L. V. hypertrophied; substance friable.	Slight atheroma of aorta.
314.	Male.	54.	...	16 ozs.	General dilatation; wall of L. V. much thinned, and somewhat bulged along septum. Thinning involves apical region, and forms commencing aneurysm.	Some atheroma.	...	Of legs.	...
315.	Male.	60.	...	14 ozs.	L. V. hypertrophied.	Thorax and abdominal aorta very atheromatous.	Aneurysm of common iliac.
317.	Male.	65.	...	12 ozs.	L. V. hypertrophied, but not dilated.	Atheromatous.
318.	Female.	40.	...	12 ozs.	L. V. a little dilated; R. also; slight hypertrophy; mitral valve stenosed.	Natural.	Thin.
320.	Female.	45.	...	12 ozs.	L. V. hypertrophied.	Aorta slightly atheromatous.
321.	Female.	62.	...	13 ozs.	L. V. much hypertrophied, and R. side dilated.	Abdominal aorta very atheromatous.	Mitral incompetence.

TABLE I. (A.)—Continued.

No.	Sex.	Age.	Stage.	Weight and Condition of Heart.	Arteries.	Other Diseases.	Edema.	Remarks.
322.	Male.	?	...	15 ozs. L. V. thickened, strongly contracted. Patent foramen ovale protected by valve.	No atheroma; cerebral tortuous.
324.	Male.	19.	...	12½ ozs. L. V. a little hypertrophied.	Aorta not atheromatous.	Aneurysm of middle cerebral.	...	Kidneys very cystic.
325.	Male.	?	...	16 ozs. Not markedly hypertrophied; wall of L. V. soft on section.	Aorta very atheromatous.
327.	Male.	57.	...	12 ozs. ...	Much atheroma and some dilatation of aorta.	Cascous tubercle of lung.
328.	Male.	75.	...	13 ozs. Very soft and flabby.	Aorta atheromatous
330.	Male.	66.	...	10 ozs. Flabby.	Considerable atheroma.	New growth and obstruction of bile ducts.	...	Wasted.
332.	Female.	36.	...	17 ozs. L. V. much hypertrophied; shape well retained.	Arteries all thickened and gape; aorta atheromatous.	Arterio-sclerosis.
335.	Female.	43.	...	12 ozs. Considerable coating of epicardial fat.	Aorta slightly but generally atheromatous.	Sarcoma of L. ovary (alcoholism).	...	Well-nourished.
338.	Female.	42.	Slight.	14 ozs. L. V. considerably hypertrophied and slightly dilated.	Specks of atheroma.	Abortion, 7th month.

339.	Male.	49.	...	12 ozs.	L. V. not hypertrophied nor dilated.	Extreme atheroma.
340.	Male.	54.	...	33 ozs.	Both sides dilated and hypertrophied; valves compressed; R. V. much dilated; L. V. much hypertrophied; somewhat dilated.	Extremeatheroma, with calcareous patches.	Chronic pleurisy; membranous.	...	Dilatation of R. heart not explained.
341.	Female.	49.	...	14 ozs.	No hypertrophy of L. V. though weight indicates some increase of substance.	Atheroma only slight.
345.	Male.	45.	...	16 ozs.	Flabby and dilated; L. V. dilated.	Slight atheroma.
346.	Female.	47.	...	16 ozs.	L. V. dilated and very flabby; large amount of epicardial fat.	Slight atheroma of aorta and cerebials.	Generally adherent pericardium.
347.	Female.	68.	...	17 ozs.	L. V. hypertrophied; dilated, epicardial obesity.	Much atheroma.
348.	Female.	61.	...	16 ozs.	L. V. hypertrophied; cavity a little dilated.	Considerable atheroma of aorta.
349.	Male.	49.	...	11 ozs.	Flabby, putrid.	Slight atheroma	Cirrhosis,	Legs pit slightly.	...
351.	Male.	42.	...	10 ozs.	Putrid; L. V. small, thin.	Nil	Gangrene of lung; symmetrical abscess of brain.
352.	Male.	66.	...	19½ ozs.	Large, very flabby; aortic valves incompetent.	Much atheroma.
354.	Male.	38.	...	11 ozs.	Natural.	Natural.	Otitis media; abscess of brain.
355.	Male.	51.	...	18 ozs.	L. V. considerably hypertrophied.	Not much atheroma.

TABLE I. (A.)—Concluded.

No.	Sex.	Age.	Stage.	Weight and Condition of Heart.	Arteries.	Other Diseases.	Edema.	Remarks.
356.	Female.	44.	...	15 ozs. L. V. hypertrophied and dilated; mitral and tricuspid stenosis.	Nil.	M. C.
357.	Male.	34.	...	18 ozs. Much hypertrophy of L. V.	Nil.	...	Of legs.	...
358.	Male.	65.	...	8 ozs. Brown atrophy.	Some atheroma.	Carcinoma of pylorus, &c.
359.	Female.	49.	...	15 ozs. Much epicardial fat.	Aorta very atheromatous.	Aneurysm of first part of arch.
360.	Female.	36.	...	13 ozs. L. V. somewhat hypertrophied; fatty infiltration.	Some atheroma.	Uræmia.	Some of legs.	...
361.	Male.	75.	...	21 ozs. L. V. much hypertrophied; R. V. wall infiltrated with fat.	Extreme atheroma.
362.	Male.	50.	...	17 ozs. Aortic valve stenosed and incompetent.	Calcareous patches in aorta.	M. C.	Of ankles.	...
363.	Male.	71.	Slight.	... Hypertrophy of L. V.	Aorta atheromatous.	New growth of pancreas.
364.	Male.	47.	...	12 ozs. L. V. dilated and relaxed, hypertrophy. Mitral valves incompetent and stenosed.	Slight atheroma of aorta.	Plumbism, gout.
366.	Male.	42.	Slight.	13 ozs. Natural as to shape and size; fatty degeneration.	Generally healthy; specks in aorta.
368.	Male.	70. L. V. a little enlarged.	General extreme atheroma.	Aorta ruptured.

369.	Male.	43.	...	11 ozs.	Very slight if any hypertrophy of L. V.; mitral slightly stenosed.	Generally highly atheromatous; radicals not at all.
371.	Female.	50.	...	11 ozs.	L. V. hypertrophied.	Slight atheroma of aorta.
373.	Male.	44.	...	16½ ozs.	L. V. considerably hypertrophied; very hard and tough.	Slight atheroma.
375.	Male.	41.	...	10 ozs.	Flabby, rather pale.	Natural.	Pneumonia.	...
376.	Male.	52.	...	11½ ozs.	L. V. a little hypertrophied.	Generally atheromatous.
377.	Male.	61.	...	21 ozs.	L. V. much hypertrophied; not much dilatation; no alteration in shape.	...	Patent ductus arteriosus.	...
379.	Male.	67.	...	16 ozs.	L. V. much hypertrophied; with very little dilatation; aortic valve incompetent.	Aorta generally atheromatous.	Aneurysm of descending arch.	...
380.	Male.	42.	...	11 ozs.	No hypertrophy; L. V. a little dilated; flabby.	Very little atheroma.	Uremia.	...
381.	Male.	52.	?	20 ozs.	Both sides dilated, but L. more so; L. V. flabby, but much hypertrophied.	Aorta very atheromatous and calcareous.	...	Of legs.
384.	Male.	42.	...	16 ozs.	L. V. much hypertrophied.	No conspicuous atheroma, a little in cerebrals.

TABLE I. (B).—Chronic Tubal Nephritis.

No.	Sex.	Age.	Stage.	Weight and Condition of Heart.	Arteries.	Other Diseases.	Edema.	Remarks.
1.	Male.	28.	...	R. side dilated; L. V. firmly contracted.	...	Abscess of liver.
3.	Male.	53.	(?)	L. V. hypertrophied.	Some atheroma.
7.	Female.	31.	(?)	A. little under 12 ozs., 10½ ozs.	Normal.	Abortion induced for uremia.
13.	Male.	17.	...	Normal.	Face puffy.	...
15.	Male.	38.	...	Muscular substance soft and pale.	Normal.
17.	Male.	40.	...	9½ ozs.	Slight atheroma of aorta.	Gout.
26.	Male.	19.	...	Normal.	Normal.	...	Much edema.	...
40.	Female.	31.	...	Normal.	...	Eclampsia.
51.	Male.	34.	...	Normal.	Very little atheroma.	Pneumonia.
60.	Female.	28.	...	11 ozs.	Slight atheroma.	...	Considerable.	...
69.	Male.	25.	...	12 ozs.	Normal.
78.	Male.	33.	...	8½ ozs.	Nil.	Disease of liver, (?) new growth.	Of feet and lower parts of legs.	...
86.	Female.	25.	...	Small.	Normal.	Phthisis, tubercle, peritonitis.
91.	Female.	38.	Slight.	Very pale, Epicardial fat.	Normal.	Carcinoma mammae.
104.	Male.	14.	...	Somewhat enlarged.	Spots of atheroma on abdominal aorta.
107.	Male.	25.	...	Flabby.	...	Tuberculous kidney.	Highly dropsical.	...

108.	Female.	3.	...	Normal.	Normal.
118.	Female.	23.	...	Normal.	Normal.
126.	Male.	18.	...	Dilatation of all cavities; fibrous increase in walls; endocardium very thick on L. side.	General anasarca, ascites.	...
136.	Male.	18.	11½ ozs.	Pleurisy with effusion.	...
146.	Female.	24.	15 ozs.	Large, Mitral stenosis, L. A. dilated; walls thickened.	Normal.	Recent abortion.	Much of legs.	...
147.	Male.	41.	...	Pericarditis.	...	Pneumonia and pleurisy.
157.	Female.	11.	...	Not enlarged.	Nil.	...	Puffy-looking.	...
170.	Female.	15.	...	Normal.	Patches of atheroma near valves.	...	Much of legs.	...
187.	Male.	37.	...	Small.
190.	Male.	27.	12 ozs.	Normal.	No atheroma to speak of.	No atheroma to speak of.	Anasarca.	...
191.	Female.	14.	...	Both ventricles dilated.	...	Chronic peritonitis, M. C.
195.	Male.	47.	24 ozs.	Aortic regurgitation; mitral stenosis; dilatation of R. side. L. side slight dilatation.	No atheroma to speak of.	Stout, dropsical.
198.	Male.	32.	14 ozs.	L. V. somewhat thick.	...	Emphysema.
207.	Female.	34.	...	Endocarditis.	Normal.
262.	Male.	32.	14 ozs.	L. side dilated and hypertrophied.	Normal.	...	General anasarca.	...
264.	Female.	24.	9½ ozs.	L. V. cuts a little tough and is pale.	Normal.	Septicemia.

TABLE I. (B.)—Concluded.

No.	Sex.	Age.	Stage	Weight and Condition of Heart.	Arteries.	Other Diseases.	Edema.	Remarks.
268.	Male.	8.	...	4 ozs. L. V. cavity rather dilated and walls thickened.	Normal.	Pneumonia.	Eyes puffy; looks anasarcaous, but does not pit.	...
269.	Female.	3½.	...	2½ ozs. Natural.	Normal.	Diphtheria.	...	"Nephritis evidently of an earlier date than the diphtheria." ...
272.	Female.	15.	...	10 ozs. Some dilatation and hypertrophy, but not much of L. V.	Normal.	Ulcerative endocarditis.
286.	Male.	39.	...	16 ozs. General dilatation; not great hypertrophy; mitral incompetence; aneurysm of aortic cusps.	Normal.	...	Legs pit, but not deeply.	...
293.	Male.	24.	...	19 ozs. L. V. dilated; walls also much thickened; mitral = 3 fingers.	Normal.	...	Considerable edema.	...
298.	Male.	3.	...	3 ozs. Ulcerative endocarditis.	Normal.	Emphysema.
301.	Male.	28.	...	16 ozs. L. V. considerably hypertrophied; cavity dilated.	Normal.	Tubercle disease of L. kidney.	None.	...
309.	Male.	19.	...	19 ozs. Considerable hypertrophy and dilatation of both ventricles; mitral = 3½ fingers; aortic calcareous; two cusps fused together.	...	Aortic disease.

316.	Male.	32.	...	12 ozs.	Natural. No enlargement of R. heart. L. V. somewhat dilated and hypertrophied. Large L. V. thick, mot-tled.	Nil.
319.	Male.	13.	...	7 ozs.		Normal.	Pneumonia.	Legs œdema-tous.	...
323.	Male.	42.	...	18 ozs.		Slight atheroma.
329.	Male.	7.	...	3 ozs.	No hypertrophy of L. V.; some slight dilatation.	Many specks of atheroma.	Chronic peritoni-tis.	General ana-sarca.	...
334.	Male.	2.	...	4 ozs.	Dry exudative pericar-ditis.	Normal.	Septicæmia.
336.	Male.	34.	...	16 ozs.	L. V. cavity rather dilated; contour pre-served; walls thick.	General ana-sarca.	...
337.	Male.	15.	...	8 ozs.	L. V. rather thick; aortic valves thicker than natural.	Specks of atheroma.	...	General ana-sarca.	...
342.	Female.	32.	...	8 ozs.	Small and flabby.	Natural.	Diabetes.	Of L. hand.	Transfusion wounds at bend of elbows.
343.	Male.	25.	(?)	16 ozs.	L. V. hypertrophied and moderately dilated; mitral stenosed; tri-cuspid dilated; granu-lations on aortic and mitral valves.	Normal.	Infective endo-carditis.
353.	Male.	39.	...	11 ozs.	No hypertrophy.	Normal.	Pneumonia.	None.	...
365.	Female.	21.	...	7½ ozs.	Quite natural; no hyper-trophy nor dilatation.	Some quite calcareous.	Tubercle of lungs and pleura.	Legs œdema-tous.	...
378.	Male.	4.	...	2 ozs.	Natural.	...	Purulent pleurisy.
383.	Female.	23.	...	9½ ozs.	Natural.	Natural.	Septicendometritis.

TABLE I. (C.).—*Mixed Tubal and Interstitial.*

No.	Sex.	Age.	Stage.	Weight and Condition of Heart.	Arteries.	Other Diseases.	Edema.	Remarks.
34.	Male.	45.	...	Normal. Not enlarged.	Aorta very atheromatous.	Aneurysm.
67.	Female.	30.	...	Much dilatation of heart; mitral tricuspid stenosis; aortic disease.	Normal.	M. C.
79.	Male.	58.	...	L. V. slightly hypertrophied.	Atheroma.	Tubercle of lungs.
124.	Female.	54.	...	12½ ozs.	Very atheromatous.	Diabetes (fat).
154.	Male.	46.	...	11½ ozs.	...	New growth of anterior medi.	Legs drop-sical.	...
158.	Male.	39.	...	Mitral valve stenosed.	Aorta dilated; atheromatous.	M. C.
180.	Male.	54.	...	Great dilatation of L. V.; aortic valves thick and everted; mitral very narrow.	Some atheroma of aorta.
192.	Male.	37.	...	26½ ozs.	...	Emphysema.	Very drop-sical.	...
208.	Female.	34.	...	11 ozs.	Normal.	Recent abortion.	Legs edematous.	...
				15½ ozs.				
				20 ozs.				

277.	Female.	42.	...	13 ozs.	Both sides dilated (R. most) and hypertrophied; mitral thickened; anterior cusp of tricuspid ruptured.	Normal.	...	None.	...
279.	Male.	23.	...	11 ozs.	...	Atheroma.	Hæmorrhage into cavity of lung.
281.	Female.	39.	...	8½ ozs.	Flabby, decomposed.	Natural.	Suppression of urine.	None.	...
287.	Female.	30.	...	16 ozs.	Pale and flabby; chord tendin. ruptured; "Thrush breast."	Normal.	Pyæmia.
294.	Male.	57.	Acute.	16 ozs.	Large generally. L. V. walls thickened; no alteration of shape of cavity.	Normal.	Hydronephrosis.	None.	...
297.	Female.	56.	...	12 ozs.	Much epicardial fat; no dilatation; R. V. almost entirely fat.	Normal.
326.	Male.	70.	...	16 ozs.	L. V. flabby; valves natural.	Specks about aorta and pulmonary artery.	Emphysema.
331.	Female.	21.	? interstitial.	18 ozs.	L. V. greatly hypertrophied; R. V. dilated; valves natural.	Atheroma of aorta and iliaes.	Tubercle of lungs.
344.	Male.	60.	?	20 ozs.	Large on left side.	Cerebrals highly atheromatous; aorta free.
367.	Female.	55.	?	11 ozs.	Natural.	Slight atheroma of aorta.	Early cirrhosis.
370.	Female.	55.	...	14 ozs.	No valve defect.	Slight atheroma.	...	Of legs.	...
374.	Male.	35.	...	10½ ozs.	Rather flabby.	Normal.

TABLE I. (D.).—*Amyloid Disease.*

No.	Sex.	Age.	Stage.	Weight and Condition of Heart.	Arteries.	Other Diseases.	Edema.	Remarks.
23.	Female.	23.	...	11 ozs. L. V. perhaps rather thicker than natural.	Normal.	Old hip disease.	...	No amyloid disease elsewhere.
50.	Female.	35. Flabby and under-sized.	Localised patches of atheroma.	Syphilis, empyema.
70.	Female.	44. Soft and flabby.	...	Carcinoma of cervix.
130.	Female.	29. Normal.	...	Pyosalpinx.	...	Amyloid spleen and intestine.
310.	Female.	20.	...	6 ozs. Pale.	Normal.	Phthisis.	...	Liver amyloid.
350.	Male.	14.	...	5 ozs. Natural.	Normal.	Phthisis.	Edema of ankles and dorsum of foot.	Liver, spleen, and intestine also.
382.	Male.	22.	...	6 ozs. Small.	Normal.	Phthisis.	...	Spleen ditto.

TABLE I. (E.).—*Consecutive Nephritis.*

47.	Female.	12.	...	9 ozs. Very considerable hypertrophy of L. V.	Atheroma of cerebral.	Inflammation of ureters and bladder.
123.	Male.	48.	...	12½ ozs. Normal.	Normal.
193.	Male.	54.	...	18 ozs. L. V. hypertrophied, R. V. thin.	Slight atheroma.	Inflammation of ureters and bladder.
372.	Female.	62.	...	12 ozs. Natural.	Normal.
385.	Female.	44.	...	8 ozs. ...	Patches of atheroma in aorta.	Chronic cystitis.

TABLE I. (F.)—Acute Nephritis.

No.	Sex.	Age.	Stage.	Weight and Condition of Heart.	Arteries.	Other Diseases.	Edema.	Duration.
46.	Male.	4. Normal.	Normal.	Pneumonia, pleuritic effusion.	...	4 days.
64.	Male.	3½.	?	... Normal.	Normal.	Pleurisy and pneumonia.
66.	Male.	4.	?
109.	Male.	2.	?	... Normal.	Normal.	Diphtheria.
129.	Female.	5.	Probably acute nephritis.	Diphtheria.
131.	Male.	3. Normal.	...	Diphtheria and pneumonia.
132.	Male.	5.	? Chronic.	5½ ozs. L. V. somewhat globular.	Normal.	Post-scarlatinal.
133.	Male.	1½. Normal.	Normal.	Ulceration of fauces and esophagus.
156.	Male.	3. Normal.	Normal.	Diphtheria.
161.	Female.	6.	? Chronic.	... Normal.	Normal.	Cirrhosis.
224.	Female.	6.	?	5 ozs. ...	Normal.	...	Face puffy.	...
227.	Female.	1¾.	Sub-acute.	1½ ozs. Natural.	Normal.	...	General anasarca; slight.	...
231.	Male.	2½.	Sub-acute.	3 ozs. Natural.	Normal.	Diphtheria.	Body generally rather puffy.	...
238.	Male.	4.	Sub-acute.	4 ozs. Natural.	Normal.	...	General anasarca.	...
280.	Female.	3.	...	2 ozs. Natural.	Normal.	Diphtheria.
295.	Male.	3.	...	3 ozs. Natural.	Normal.	Diphtheria.
311.	Male.	26.	...	20 ozs. L. V. hypertrophied, flabby. Mitral valve incompetent. Aortic valve thickened and incompetent.	Normal.	Ulcerative endocarditis.
333.	Male.	1¼.	...	3 ozs. ...	Normal.	Diphtheria.

From Table I. it appears that there were—

	Males.	Females.
275 cases of chronic interstitial nephritis in	169	105 ¹
53 " " " tubal nephritis	35	18
21 " " " so-called mixed nephritis	11	10
7 " " " amyloid disease	2	5
5 " " " consecutive nephritis	2	5
18 " " " acute nephritis	13	5

Analysis shows the age incidence in the various affections to be as follows:—

TABLE II.

(A.) <i>Interstitial Nephritis.</i>		(B.) <i>Chronic Tubal Nephritis.</i>	
1-10 years	0 cases.	1-5 years	5 cases } 7
10-20 "	4 "	5-10 "	2 " }
20-30 "	16 "	10-15 "	4 " } 12
30-40 "	36 "	15-20 "	8 " }
40-50 "	73 "	20-25 "	6 " } 14
50-60 "	67 "	25-30 "	8 " }
60-70 "	55 "	30-35 "	10 " } 15
70-75 "	14 "	35-40 "	5 " }
Over 75 years	0 "	40-50 "	4 " }
Age not stated in	10 "	Over 50 years	1 "
Average age = 47.69 years.		Average age = 24.1 years.	
(C.) <i>Mixed Nephritis.</i>		(D.) <i>Amyloid Disease.</i>	
Below 20 years	0 cases.	Under 20 years	1 case.
20-30 years	3 "	20-30 years	4 "
30-40 "	7 "	30-40 "	3 "
40-50 "	2 "	40-50 "
50-60 "	7 "		
60-70 "	1 "		
70 years	1 "		
Average age = 43.6 years.			
(E.) <i>Consecutive Nephritis.</i>		(F.) <i>Acute Nephritis.</i>	
12 years	1 case.	Under 5 years	13 cases.
4 others from 44-62 years.		5-10 years	4 "
		26 "	1 "

These tables emphasise the fact that interstitial nephritis is a disease of middle age and advanced life, whereas chronic tubal nephritis occurs chiefly in children and young adults, the average ages respectively being 47.69 years and 24.1 years. The average age of the cases described as mixed nephritis (43.6 years), as would be expected, much more nearly corresponds to that of interstitial than to that of tubal nephritis.

¹ In one sex not stated.

Henceforth we shall leave out of consideration the cases of amyloid disease, consecutive nephritis, and acute nephritis, inasmuch as the number of cases of the two former diseases is too small, and many of those described as acute nephritis were of doubtful nature, so that figures obtained from an analysis of them would be probably only misleading.

Changes in the Heart.

The first point which merits our attention is the condition of the heart in the various forms of Bright's disease, *i.e.*, whether hypertrophy or enlargement of the heart is more especially associated with one form of nephritis than with another. To settle this point, we may first consider the weight of the heart as the index of the presence or absence of hypertrophy, and secondly we may accept the description in the post-mortem records as the most satisfactory evidence, for it is certain that the weight of the heart is not always an absolute measure of an existing hypertrophy. Arranging the cases according to the weight-measure of the heart we obtain the following analysis:—

TABLE III.—*Tables showing Weight of Heart.*

(A.) *Interstitial Nephritis.*

Weight.	No. of Cases.
6-7 ozs.	3
7-8 "	5
8-9 "	9
9-10 "	2
10-11 "	15
11-12 "	22
12-13 "	30
13-14 "	11
14-15 "	21
15-16 "	15
16-17 "	19
17-18 "	10
18-19 "	12
19-20 "	4
20-21 "	9
21-22 "	8
22-23 "	4
23-24 "	4
24-25 "	4
25-30 "	7
30-34 "	3

Weight not stated in 58.

If we single out all the cases between 30 and 70 years of age (the period of life at which the disease is most common), and calculate the average weight of the heart, we obtain a quantity of 14.78 ozs.

(B.) *Chronic Tubal Nephritis.*

Weight.	No. of Cases.
Under 7 ozs.	6
7- 8 ozs.	3
8- 9 "	3
9-10 "	4
10-11 "	2
11-12 "	5
12-13 "	3
13-14 "	0
14-15 "	3
15-16 "	1
16-17 "	4
18 ozs.	1
19 "	2
24 "	1

Weight not stated in 15.

The average weight of the heart for all the cases between 30 and 70 years amounts to 13.5 ozs., and the average weight for the cases between 15 and 35 years (the most common period) is 12.1 ozs.; which shows that with advancing age, or as the disease becomes more chronic, the weight of the heart increases.

(C.) *Mixed Nephritis.*

Weight.	No. of Cases.
$8\frac{1}{2}$ ozs.	1
$10\frac{1}{2}$ "	1
11-12 ozs.	4
12-13 "	2
13-14 "	1
14-15 "	1
15-16 "	1
16-17 "	3
18 ozs.	1
20 "	2
$26\frac{1}{2}$ "	1

Weight not stated in 3.

The average weight of the heart for all cases of mixed nephritis = 14.6 ozs.

Analysing these lists more fully, we find that of the 58 cases of granular kidneys in which the weight of the heart is not given—

- (a.) In 25 the heart is said to be enlarged as a whole, or the left ventricle hypertrophied.
- (b.) In 18 the heart is described as normal.
- (c.) In 6 the heart is described as small.
- (d.) In 9 neither size nor weight are given.

First taking the six cases under (c), in which the hearts are small, we find that they showed the following intercurrent diseases:—

Phthisis	1 instance.
Carcinoma	2 „
Cirrhosis	1 „
Hour-glass contraction of the stomach following cicatrisation of an old ulcer, with wasting	1 „
Pneumonia	1 „
(Omit 5 cases.)	

It may be assumed that with wasting disease no hypertrophy could take place, and therefore on the principle applied by Dr. Goodhart we must omit such cases. Pneumonia being an acute disease, could hardly be accused of inhibiting a compensatory hypertrophy of the heart.

Of these six cases, therefore, we must omit five.

Of the 18 cases under (b) described as normal, we find the following complications:—

Cirrhosis of the liver	4 instances.
Pneumonia	4 „
Aneurysm	2 „
Phthisis	2 „
Malignant disease	2 „
Chronic inflammation of intestine (colotomy had been performed)	1 „
Uterine hæmorrhage (fibroid)	1 „
(Omit 12 cases.)	

In the other two cases no cause sufficient to inhibit the cardiac hypertrophy was mentioned; though one was described as thin, the other as fat.

We think, therefore, that we may fairly omit 12 of these 18 cases.

In the nine cases under (d), in which neither the size nor

the weight of the heart are given, intercurrent disease was present in five instances, as follows:—

Cirrhosis of liver	1 example.
Malignant disease	1 "
Ulcer near pylorus (body much emaciated)	1 "
Ovarian cyst	1 "
Mitral stenosis	1 "

(Omit 5 cases.)

We shall omit all these five cases, as stenosis of the mitral orifice may be said to prevent hypertrophy of the left ventricle.

Hence of these 58 cases we are justified in omitting 22, so that of 36 cases where weight was not given 25 showed hypertrophy.

There are 19 cases in which the weight is below 10 ounces. In two of these the left ventricle is described as hypertrophied. The remaining 17 cases showed the following complications:—

Malignant disease	10 instances.
Phthisis	4 "
Cirrhosis of liver	1 "
Psoas abscess	1 "
Acute mania (body very thin)	1 "

(Omit 17 cases.)

All of these may be omitted. Therefore, of two cases below 10 ozs. both showed hypertrophy.

Let us now analyse the 37 cases in which the heart weighed between 10 and 12 ozs. in similar manner. We find that the left ventricle is described as thick in 11 instances. In the other 26 cases there were the following intercurrent diseases:—

Malignant disease	4 instances.
Stenosis of pylorus (with wasting)	2 "
Phthisis	2 "
Pneumonia	4 "
Cirrhosis	2 "
Enteric fever	1 "
Aneurysm	1 "
Abscess of brain	2 "
Mitral stenosis	1 "
Tetanus	1 "
Wasting, old age	2 "

(Omit 14 cases.)

We may fairly omit 14 of these, leaving the cases with pneumonia, enteric fever, tetanus, and brain abscess. Thus of 36 cases below 12 ozs. we must omit $17 + 14 = 31$. We are then left with 25, of which 13 ($2 + 11$) showed some hypertrophy of the heart. Adding these to the cases where the

weight was not given, our figures show hypertrophy of the heart in 38 out of 61 cases. Taking now the cases where the heart weighed more than 12 oz., we may consider all these as instances of cardiac hypertrophy. Of course it is possible, even probable, that in some of these cases the cardiac enlargement may have been due to other causes, and that the renal condition appeared as a complication or as an exacerbating condition. This point, however, has been disregarded, because the fallacy introduced is but small, as a perusal of the post-mortem records will show.

Hence, summing up after complete analysis, we find that hypertrophy of the heart existed in 199 out of 222 cases of interstitial nephritis, *i.e.*, in 89.6 per cent. We thus arrive at practically the same result as Dr. Goodhart, 90 per cent. of whose cases showed cardiac hypertrophy.

We shall now proceed to compare these results with those obtained from a consideration of the cases of chronic tubal nephritis. Of the 53 examples of this disease, in no less than 15 the weight of the heart is not given. Of these only five showed important complications:—

Phthisis	2 cases.
Pneumonia	2 „
Carcinoma	1 „

(Of these we omit 3.)

In none was there hypertrophy; therefore our figures at present are 0 out of 12 cases. Of the 38 cases in which the weight was stated, 14 were under 20 years of age. Hypertrophy of the heart is noted in six of these 14 cases. Inter-current disease was present in four of the others, thus:—

Pneumonia	2 cases.
Empyema	1 „
Diphtheria	1 „

(Omit 1 case.)

Of these, the one with empyema perhaps ought to be omitted. We thus have six with cardiac hypertrophy, out of 13 under 20 years of age.

Of the 24 cases over 20 years of age, 14 weighed over 12 ozs., which we take to represent the average weight of the heart. Of the remaining 10, four presented important complications:—

Diabetes	1 case.
New growth of liver	1 „
Phthisis	1 „
Pneumonia	1 „

(Omit 3 cases.)

Of these, we omit the first three, there being sufficient causes for wasting. Therefore of those over 20 years of age, there was hypertrophy in 14 out of 21 cases.

On summing up, we find that of 46 cases of chronic tubal nephritis, 20 showed cardiac hypertrophy, which gives us a percentage of 43.47.

Comparing this with our previous results, we see, if we be allowed to judge from such a small number as 46, that cardiac hypertrophy is certainly considerably less common in chronic tubal than in chronic interstitial nephritis.

Contrasting these figures with those obtained by Dr. Goodhart, we obtain the following result:—

<i>Dr. Goodhart's figures.</i>	<i>Our own figures.</i>
109 cases of hypertrophy out of 134	20 out of 46
= 81.3 per cent.	= 43.47

The discrepancy may be accounted for partly by the fact that he examined far more cases, but chiefly by the fact that the macroscopic post-mortem diagnosis is frequently a very uncertain factor, which is capable of introducing a large amount of fallacy. It should also be mentioned that Dr Goodhart expressly states that in his list of parenchymatous nephritis were included "all cases in which the kidneys have been described as white or mottled, whether large or contracted, except such as were lardaceous." We suspect that nearly all our cases of so-called "mixed" nephritis, and even some of our cases of interstitial nephritis, would have been found under his list of parenchymatous nephritis.

To resume our analysis. Of the 21 cases of "mixed" tubal and interstitial nephritis, there were only six under 12 ozs. in weight. Of these, three showed intercurrent disease:—

Diabetes (fat)	1
Phthisis	1
Cirrhosis (early)	1

We omit two of these cases.

We also leave out all three cases in which the weight of the heart was not stated, for they showed the following complications:—(1) morbus cordis; (2) aneurysm; (3) new growth.

We must therefore omit five cases in all, which leaves us 16. Now 12 weighed over 12 oz. Hence we have cardiac hypertrophy in 12 out of 16 cases of mixed nephritis = 75 per cent. Now it would seem that in many of these mixed cases the interstitial changes are preponderant. This may be judged from the age (average age being 43.6 years), which corresponds closely to

that of interstitial nephritis (47.69), and from the co-existence of atheroma. Therefore it is not surprising to find such a high percentage of cardiac hypertrophy. We must, however, be cautious in our deductions from these so-called mixed cases, because it is not always easy to understand the post-mortem account; and to make matters worse, as we shall see, there are awkward discrepancies between them and the Ward notes.

Conclusions as to Cardiac Hypertrophy.—(1.) Cardiac hypertrophy is most marked in chronic interstitial nephritis. The average weight of all cases between 30 and 70 years of age being 14.7 ozs., and the percentage incidence of hypertrophy = 89.6 per cent. (2.) Cardiac hypertrophy is much less marked in so-called chronic tubal or parenchymatous nephritis. The average weight of cases between 15 and 35 years of age = 12.1 ozs. The average weight of cases between 30 and 70 years of age = 13.5 ozs. The percentage incidence of hypertrophy = 43.47 per cent. (3.) Cardiac hypertrophy is common in so-called mixed nephritis. The average weight of all cases examined = 14.6 ozs. And the percentage incidence of hypertrophy = 75 per cent.

Œdema in Chronic Renal Disease.

Let us now consider this other branch of our subject. Of the 275 cases of chronic interstitial nephritis we find œdema noted in 41 instances. Looking through the notes of these 41 cases, we come across the following statements in 12:—

No.	Weight of Heart.	Condition of Heart.	Œdema.
19.	...	R. heart widely dilated.	Much œdema, especially of legs.
30.	22 ozs.	R. side widely dilated. L. side dilated and hypertrophied.	Anasarca of legs.
54.	10 ozs.	R. side dilated.	Slight.
55.	28 ozs.	Great dilatation of all cavities. L. V. hypertrophied, though dilatation in excess.	Dropsy, ascites.
89.	18 ozs.	L. V. thick and dilated. R. side very large, dilated.	General anasarca.
97.	...	L. V. dilated and hypertrophied. R. side dilated.	Of feet and legs.
199.	17 ozs.	Dilated. Some hypertrophy of L. V.	Great general anasarca.
206.	11 ozs.	Dilated R. V.	Œdematous.
308.	24 ozs.	Both ventricles greatly hypertrophied, with some dilatation.	Of lower extremities.
312.	26 ozs.	L. V. hypertrophied, with considerable dilatation. R. side slight dilatation.	Of lower extremities.
314.	16 ozs.	General dilatation.	Œdema of legs.
381.	20 ozs.	Both sides dilated, but L. more so.	Of legs.

It is evident that an œdema appearing in such cases cannot be put down to the renal changes, but is really cardiac, although, of course, the cardiac condition in most, if not in all, instances may have been, and was, the result of the renal lesion.

Now amongst the remaining 29 cases of œdema we find 13 complicated by an independent or intercurrent cardiac lesion, as shown from the following notes:—

No.	Weight of Heart.	Condition of Heart.	œdema.
62.	15 ozs.	Some dilatation. Aortic and mitral disease.	Of legs.
138.	20 ozs.	Much enlargement. Both ventricles dilated. Aortic disease.	Dropsy of legs.
139.	23½ ozs.	Great and general dilatation. Aortic incompetence.	Legs highly dropsical.
164.	20 ozs.	R. V. dilated. Mitral stenosis.	Of legs and penis.
165.	16 ozs.	R. side dilated. Walls thin. Mitral stenosis.	Very dropsical.
228.	16 ozs.	Ventricles both somewhat dilated and hypertrophied. Mitral stenosis and regurgitation.	General anasarca.
258.	16 ozs.	R. side greatly dilated. Mitral orifice stenosed. Tricuspid incompetent.	Legs anasarcons.
283.	12 ozs.	R. side much hypertrophied and dilated. L. auricle dilated. Mitral stenosed and incompetent.	General anasarca.
290.	21 ozs.	L. V. hypertrophied and dilated. R. V. dilated. Mitral valves stenosed and incompetent.	Some of legs.
291.	17 ozs.	R. V. much dilated. L. V. hypertrophied and dilated; also R. auricle. Aortic disease.	Legs slightly œdematous. Penis much swollen.
302.	24 ozs.	Great hypertrophy and much dilatation of L. V. Aortic valves incompetent.	œdema.
318.	12 ozs.	L. V. a little dilated. R. also slight hypertrophy. Mitral valve stenosed.	Of vulva.
362.	17 ozs.	Aortic valves stenosed and incompetent.	Of ankles.

It is evident that, excepting perhaps Nos. 302 and 362, the œdema in these cases was cardiac and other than renal; therefore these 11 cases should also be excluded.

Amongst the remaining 16 there were two cases of emphysema, four of cirrhosis, and one in which the heart weighed 34 oz., but no description is found in the post-mortem notes beyond the statement that it was hypertrophied. These seven cases should also be excluded. Hence there remain nine cases which reasonably might be considered to be renal œdema. If we add the two more doubtful cases Nos. 302 and 362, we have œdema of probable renal origin occurring only 11 times in 245 cases of interstitial nephritis = 4.89 per cent.

It is perhaps advisable to take the cases of "mixed"

nephritis next, because many of them are certainly interstitial rather than tubal. We find that in 21 cases, according to the post-mortem notes, there was œdema in four, but amongst these four are—

No.	Weight.	Heart.	Other Disease.	Œdema.
154. 192.	... 15½ ozs.	Mitral valve stenosed. R. V. dilated. L. V. some slight dilatation and hypertrophy.	... Emphysema.	Legs dropsical. Very dropsical.
208.	20 ozs.	R. side somewhat hypertrophied and dilated, &c.	Recent abortion.	Legs œdematous.

These three cases, or certainly two of them, might reasonably be omitted as cardiac œdema. Hence, according to the post-mortem notes, œdema occurred in one out of 18, or two out of 19 cases. This statement, however, requires considerable modification, as we shall see later.

Of the 53 cases of chronic tubal nephritis, œdema occurred in 20. If we examine these 20 cases, we find that in four there was morbus cordis or considerable cardiac dilatation. We give the notes of these four cases:—

No.	Weight.	Condition of Heart.	Œdema.
125.	...	Dilatation of all cavities, fibrous increase in walls, endocardium very thick on L. side.	General anasarca, ascites.
146.	15 ozs.	Mitral stenosis. L. A. dilated and walls thickened.	Much of legs.
195.	24 ozs.	Aortic regurgitation. Mitral stenosis, dilatation of R. side. L. V. slightly dilated.	Dropsical.
286.	16 ozs.	General dilatation, not great hypertrophy. Mitral incompetent. Aneurysm of aortic cusps.	Legs pit, but not deeply.

These four cases it would be safer to exclude. Hence œdema occurred in 16 out of 49 = 32.6 per cent. This number, which is based on the post-mortem accounts alone, is much too small, as we shall see presently on checking the records of the dead-house with those of the wards.

If we glance over the cases where there was no œdema, we are struck by a few suggestive points:—

(a.) In some, Nos. 3, 7, 323 and 343, interstitial changes are recorded.

(b.) In a few the heart was considerably hypertrophied,

without accompanying dilatation of the right side, as in Nos. 3, 198, 323.

(*c.*) Of the 49 cases mentioned above, in no instance did œdema occur in any one after the age of 40 years.

(*d.*) Of the 16 cases showing œdema, six occurred under the age of 16, and ten between 16 and 35, *i.e.*, under the age of 16 in six out of 13 cases, and between 16 and 35 in ten out of 28 cases.

This seems to show that as tubal nephritis becomes more and more chronic, and the kidney more and more indurated, the incidence of œdema diminishes markedly.

The conclusions with regard to œdema, in so far as they are founded on the post-mortem records, are evident:—

(1.) Œdema is common in the earlier or younger forms of parenchymatous or tubal nephritis, occurring in 16 out of 41 cases below 35 years of age = 39 per cent.

(2.) Œdema is uncommon in interstitial nephritis, or in parenchymatous nephritis of some duration.

It appeared to us, however, that the post-mortem records regarding the absence or presence of œdema were hardly accurate enough, and we have therefore studied the Ward notes of the cases of mixed nephritis and chronic tubal nephritis. They are instructive for further reasons, for they show (1) that with œdema there is almost always oliguria and albuminuria; and (2) with oliguria and albuminuria there is almost always œdema. As far as the cases of mixed nephritis are concerned, we find also that the cases with polyuria were not complicated by œdema. All this proves that œdema appears only so long as the tubal type prevails, and that with increasing interstitial changes the œdema disappears.

Re-tabulating the cases of mixed nephritis with additions from the Ward notes, we learn the following facts:—

Mixed Nephritis.

No.	Heart.	Arteries.	Complications.	Quantity of Urine.	Albumin.	Edema.
34. 67.	Not enlarged. Dilated.	Very atheromatous. ...	Aneurysm. Serious heart disease (also aortic dis- ease).	Faint cloud. Trace.
79. 124. 154.	Slight hypertrophy. ... Mitral stenosis.	Atheroma. Very atheromatous. ...	Tubercle of lungs. Diabetes. Malignant disease of mediastinum. Morbus cordis. ...	Oliguria. Polyuria. Oliguria.	Three-fourths. Cloud. Much.	Slight. ... Legs.
158. 180.	Dilated. ...	Atheroma of aorta. Some atheroma of aorta.	...	Polyuria. ...	Much. Cloud.	(Edema. ...)
192. 208.	Dilated. Hypertrophied and dilated.	Emphysema. Recent abortion.	Oliguria. Oliguria.	Much. Much.	Much. Legs.
277. 279. 281. 287. 294. 297.	Dilated. ... Pale and flabby. Enlarged.	Atheroma. ... Normal. Normal.	Morbus cordis. ... Suppression of urine. Pyæmia. Hydronephrosis. ...	Oliguria. No notes. Oliguria. ...	Much. One-sixth. ... No notes. Much.	(Edema. No œdema. ... No notes. General. ...)
326. 331.	Hypertrophy and di- latation. Enlarged. Natural.	Atheroma. ... Atheroma. Slight atheroma of aorta. Slight atheroma. Tubercle of lungs. ...	Oliguria.	One-half. ... Trace. Trace.	Edema. ... Feet. ...
344. 367.	Natural.	Atheroma. Slight atheroma of aorta. Slight atheroma. ...	Early cirrhosis.	Oliguria. Oliguria.	Heavy. Heavy.	Legs. Face.
370. 374.	Flabby.

It is somewhat distressing to find discrepancies between the post-mortem accounts and the Ward notes. Thus we see that whilst in the post-mortem records œdema was mentioned only four times, that, according to the clinical notes, it had occurred actually eleven times. Such disagreement must throw some doubt upon our previous conclusions, and will necessitate a future control of all the post-mortem notes of the cases of interstitial nephritis.

If we examine the above table carefully, we find that (1) in all cases but one (367), where there is œdema the urine is heavy with albumin; (2) in almost all cases of œdema there is oliguria; (3) in all cases but one where there is but a trace of albumin œdema is absent.

In some of the œdematous cases lesions exist outside the kidney sufficient to explain the dropsy. Thus there is serious cardiac disease in 154, 158, 192, 208, 277, 331, an anterior mediastinal growth in 154, emphysema in 192, cirrhosis in 367; so that of the eleven cases only four remain where we must look for a renal origin of the œdema, viz., 79, 294, 370, 374. In these four cases there was much albuminuria and oliguria, and in three of them comparatively slight cardiac hypertrophy, and also slight or no atheroma; so that we may fairly believe that in these four cases the renal lesion was more of the tubal variety than the interstitial form. The one fact that these cases of mixed nephritis do show is that the œdema of renal or cardiac disease goes together with marked albuminuria and oliguria. Otherwise it seems to us but little can be argued from these cases, because all sorts of conditions apparently have been classified under mixed nephritis. If we regard the kidney lesions as the primary condition, then we find that in most of the cases where œdema appeared this did not occur until the heart was considerably diseased.

Turning now to the clinical notes of the cases of chronic tubal nephritis, we found again marked discrepancies between the Ward notes and the post-mortem records. Thus, according to the clinical accounts, there was œdema in the following cases:—Nos. 1, 15, 17, 69, 86, 108, 118, 136, 187, 301, 309, 316, 378; and hence if we include puffiness under œdema, we find that œdematous changes have occurred in 34 out of 53 cases. Besides the four cases previously mentioned, viz., 126, 146, 195, 286, we must further exclude 309, because in all these five the œdema may have been of cardiac rather than of renal origin. We see, therefore, that œdema occurred in 29 of 48 cases, *i.e.*, in 60.4 per cent. instead of 32.6 per cent., the figure previously given, which was based upon post-mortem data.

In almost all the cases where œdema was present there was during life albuminuria and some oliguria. This is best shown in a tabular form:—

Chronic Tubal Nephritis and Œdema.

No.	Quantity of Urine.	Albuminuria.	Œdema.
1.	Fair.	Solid.	Legs and feet.
13.	"	$\frac{1}{3}-\frac{11}{16}$.	Face puffy.
15.	"	$\frac{1}{4}$.	Slight.
17.	Little.	$\frac{1}{6}-\frac{1}{4}$.	"
26.	Fair.	Solid.	Much.
60.	Very little.	"	"
69.	Scanty.	Thick cloud.	"
78.	Little.	$\frac{5}{8}$.	Feet and legs.
86.	Fair.	Heavy.	Slight.
107.	Scanty.	Solid.	Much.
108.	"	Much.	"
118.	"	"	"
136.	Diminished.	"	"
157.	Little.	"	Little.
170.	Scanty.	No note.	Much.
187.	"	$\frac{7}{8}$.	"
190.	"	Much.	"
262.	Diminished.	"	"
268.	?	$\frac{1}{5}$.	Little.
293.	Good quantity.	Much.	Much.
301.	Scanty.	$\frac{1}{4}-\frac{1}{2}$.	"
316.	"	Trace.	Slight.
319.	"	Dense.	Legs.
329.	"	$\frac{7}{8}$.	Much.
336.	?	Solid.	"
337.	Diminished.	?	"
365.	Scanty.	$\frac{3}{4}$.	"
378.	"	Heavy.	Face.
383.	Little.	Solid.	Vulva (not general).

We see at a glance that in all these cases, with one or two exceptions, albuminuria and oliguria were also present, and it may be mentioned that in the five cases which we have excluded on account of their complications, these two phenomena were also present. Thus:—

No.	Urine.	Albumin.	Œdema.
126.	Diminished.	Solid.	Much.
146.	Little.	Much.	"
195.	Fair.	Cloud.	Dropsical.
286.	Scanty.	One-third.	Little.
309.	Diminished.	One-fourth.	Fair.

We must agree, therefore, that the correlation between œdema on the one hand and albuminuria and oliguria on the other, but especially albuminuria, is firmly established, so far at least as renal or cardiac dropsy is concerned. It requires no further explanations on our part, for reference to these tables is quite sufficient.

On examining the cases of chronic tubal nephritis without œdema, we find that in some death was due to acute infective diseases, such as septicæmia, malignant endocarditis, diphtheria and pneumonia (Nos. 51, 264, 269, 272, 298, 334, 343, 353), and we have been able only to find three cases (191, 323, and 353) where there was considerable albuminuria present but no œdema. That, however, need not surprise us, because we know very well that albuminuria may and does exist without œdema. What we are arguing is a different matter altogether, viz., that with renal and cardiac œdema we find albuminuria and a diminished flow of urine, although the oliguria may be but slight. And we believe that as the albuminuria vanishes and the secretion of urine is re-established the œdema of chronic renal disease disappears. Albuminuria, oliguria, and œdema are phenomena which are closely linked together.

With regard to age, we find that of the 29 cases of œdema 11 occurred between 20 and 30 years, and 11 under 20, so that arranging these cases according to decennia we have—

Between	1-10 years.	.	.	.	4 cases.
„	10-20 „	.	.	.	7 „
„	20-30 „	.	.	.	11 „
„	30-40 „	.	.	.	7 „
					29

Arranging now the cases without œdema, we find that there were—

Between	1-10 years.	.	.	.	3 cases.
„	10-20 „	.	.	.	3 „
„	20-30 „	.	.	.	2 „
„	30-40 „	.	.	.	7 „
Over	40 „	.	.	.	3 „
					18

This points distinctly to our belief that renal œdema is more commonly met with in the earlier forms of parenchymatous or tubal nephritis, and that as the lesion becomes chronic and more interstitial tissue develops, the œdema disappears, while at the same time also the albuminuria and oliguria gradually lessen, until they almost or completely vanish.

ON
SECONDARY INFECTIVE LESIONS IN FATAL
CASES OF LOBAR PNEUMONIA.

BY

H. W. LANCE, M.B., AND A. A. KANTHACK, M.D.

It is well known that in many fatal cases of croupous pneumonia secondary infective lesions of an inflammatory nature, often suppurative, œdematous, or fibrinous in type, are observed in various parts of the body, either in close anatomical relation with the diseased lung, or in more remote situations. This is a matter of some importance, because it shows that in some cases at least the infection has become general, or was in process of generalisation. Anyhow, if pneumonia be actually due to the pneumo-coccus, as seems to be the case, there must have been an escape of the micro-organism from the seat of infection. This escape may take place, (1.) into the circulation, bringing about either a metastatic focus, or even an hæmic infection; or (2.) into the tissues, possibly along the lymph channels.

In this connection it is of some importance that some observers state that in pneumonia the blood should be examined for the presence of pneumo-cocci, since this implies a hæmic infection and a bad prognosis.

Infective endocarditis is by no means a rare complication of fatal pneumonia according to Dr. Osler, who describes it as occurring in 16 per cent. of his cases, and it is invariably a bad omen.

An infective endocarditis must almost necessarily be followed by a general blood infection, and may lead to a true septicæmia, or even a pyæmic state.

The secondary infections in pneumonia are generally, if not always, due to the pneumo-coccus, and therefore form a strong argument in favour of the causal relation between this micro-organism and croupous pneumonia. Anyhow, the frequency with which these secondary inflammatory lesions are found in fatal cases is a point of great pathological importance.

TABLE I.—*Fatal Cases of Pneumonia, 1891-95.*

No.	Age.	Sex.	Lobe Affected.	Complications.
1.	25.	Male.	Upper lobe.	Pleurisy.
2.	39.	Male.	Right upper and middle.
3.	58.	Male.	Left, whole.
4.	39.	Male.	Right lower.	Perihepatitis, perisplenitis.
5.	35.	Male.	Right.
6.	60.	Male.	Right lower.
7.	31.	Male.	Left lower.	Pericarditis.
8.	27.	Male.	Right.
9.	26.	Male.	Right middle.	Pericarditis.
10.	29.	Male.	Right upper, left lower.
11.	39.	Male.	Right middle and lower.
12.	54.	Male.	Right and left bases.	Granulations, ¹ pericarditis.
13.	44.	Male.	Left lower.
14.	44.	Male.	Left base.
15.	28.	Male.	Left base.	Infective endocarditis, meningitis.
16.	39.	Male.	Right and left.	Pleurisy.
17.	45.	Female.	Right and left.
18.	31.	Male.	Right.	Pericarditis.
19.	39.	Male.	Right lower.
20.	51.	Male.	Right upper.
21.	34.	Male.	Left lower.	Bronchiectasis, parenchymatous nephritis.
22.	32.	Male.	Right upper.	Endocarditis.
23.	46.	Male.	Left upper.
24.	37.	Male.	Right upper.
25.	24.	Male.	Left.	Pleurisy (most marked on right).
26.	55.	Male.	Right.
27.	29.	Female.	Right upper.	Morbus cordis, pericarditis.
28.	6 m.	Male.	Right lower.
29.	27.	Male.	Right.
30.	44.	Female.	Right lower.	Pericarditis, granular kidneys.
31.	55.	Male.	Right.
32.	34.	Male.	Left lower.	Hyperæmia of brain.
33.	48.	Female.	Right upper.
34.	32.	Male.	Right apex.
35.	53.	Female.	Right lower.
36.	66.	Female.	Right lower.	Granular kidneys.
37.	42.	Male.	Left lower.
38.	65.	Male.	Right upper.
39.	54.	Female.	Left lower.	Granular kidneys.
40.	19.	Male.	Right and left.
41.	40.	Male.	Right and left.
42.	36.	Male.	Right.	Pleurisy.
43.	15.	Female.	Right and left.
44.	25.	Male.	Right and left.	Pleurisy, pericarditis.
45.	51.	Male.	Right lower.
46.	49.	Male.	Right upper.
47.	46.	Male.	Left lower.
48.	39.	Male.	Right.
49.	37.	Male.	Left lower.
50.	26.	Male.	Left upper.	Pericarditis, peritonitis.
51.	26.	Female.	Right.
52.	16.	Male.	Left lower.	Pleurisy.
53.	24.	Male.	Right upper.	Granular kidneys.
54.	27.	Male.	Right upper.	Hyperæmia of brain.
55.	34.	Male.	Right upper.
56.	46.	Male.	Left lower.	Pericarditis, empyema.
57.	30.	Male.
58.	23.	Female.	Right middle.	Vegetations, pericarditis.
59.	32.	Male.	Left apex.	Empyema.

¹ Granulations on the cusps of some cardiac valve or other.

TABLE I.—*Continued.*

No.	Age.	Sex.	Lobe Affected.	Complications.
60.	30.	Female.	Right.	Septic.
61.	37.	Male.	Right.	Pericarditis.
62.	15.	Female.	Right upper.	Morbus cordis, vegetations.
63.	29.	Male.	Right and left.
64.	41.	Female.	Right and left.
65.	37.	Male.	Right.	Fibrous kidney.
66.	4.	Male.	Left.
67.	21.	Male.	Right lower.	Pleurisy (left), pericarditis.
68.	59.	Male.	Right upper.
69.	27.	Male.	Right lower.
70.	36.	Male.	Right.	Pleurisy, pericarditis.
71.	38.	Male.	Right.
72.	23.	Male.	Right.
73.	28.	Male.	Right upper.	Pericarditis.
74.	41.	Male.	Right.	Pleurisy (left), pericarditis.
75.	32.	Male.	Right and left.
76.	45.	Male.	Right upper.	Pericarditis, synovitis of knee (semi-purulent fluid).
77.	19.	Male.	Right.	Pericarditis.
78.	9.	Female.	Left.	Pleurisy (left), typhoid.
79.	28.	Male.	Right and left.
80.	41.	Male.	Right upper and middle.
81.	5.	Male.	Left lower.	Pericarditis.
82.	2.	Male.	Left.	Pleurisy (right), pericarditis, peritonitis.
83.	34.	Male.	Left, lower two-thirds.
84.	44.	Male.	Left upper.	Kidney large and coarse.
85.	51.	Male.	Left lower.	Granular kidney.
86.	41.	Female.	Right and left.	Ulcerative endocarditis, in- farcts in spleen and kidney.
87.	35.	Male.	Left.	Pericarditis, meningitis.
88.	69.	Male.	Left.	Granular kidney.
89.	14.	Male.	Left.	Morbus cordis.
90.	34.	Female.	Right lower.	Pericarditis.
91.	24.	Male.	Right and left.	Pleurisy.
92.	16.	Female.	Right and left.	Pericarditis.
93.	25.	Male.	Left lower.	Pleurisy (right and left).
94.	37.	Male.	Right.
95.	36.	Male.	Right.
96.	38.	Male.	Left.
97.	32.	Female.	Right upper.
98.	38.	Male.	Right.	Pleurisy (left).
99.	53.	Male.	Right upper.	Kidney interstitial changes.
100.	46.	Female.	Right.
101.	64.	Male.	Left.	Granular kidney.
102.	54.	Male.	Left.	Pericarditis.
103.	29.	Male.	Right.
104.	60.	Male.	Right upper.	Kidneys swollen.
105.	68.	Male.	Right and left.
106.	?	Male.	Left.
107.	50.	Male.	Left.
108.	Unknown.		Left.	Pericarditis.
109.	33.	Male.	Left.
110.	18.	Male.	Right upper.
111.	30.	Male.	Left.	Granulations. ¹
112.	19.	Male.	Right upper.	Morbus cordis, old and recent; ulcerative endocarditis, in- farct of spleen.
113.	38.	Female.	Right.	Vegetations, infarcts.
114.	26.	Male.	Right lower.	Typhoid.
115.	54.	Male.	Left upper.	Granular kidneys.
116.	39.	Male.	Right upper.
117.	22.	Male.	Left.

¹ Granulations on the cusps of some cardiac valve or other.

TABLE I.—*Concluded.*

No.	Age.	Sex.	Lobe Affected.	Complications.
118.	33.	Male.	Right base.	Pericarditis, pleurisy (right and left).
119.	41.	Male.	Right upper.
120.	17.	Male.	Left base.	Morbus cordis.
121.	44.	Male.	Right upper.	Granular kidney.
122.	42.	Male.	Right base.	Pericarditis, peritonitis, pleurisy.
123.	49.	Male.	Right upper.
124.	19.	Female.	Right.	Pericarditis, morbus cordis.
125.	33.	Female.	Left lower.	Pericarditis.
126.	30.	Male.	Right.
127.	45.	Male.	Left upper.	Morbus cordis.
128.	59.	Male.	Right lower.	Endocarditis.
129.	38.	Male.	Left.	Pleurisy (right), pericarditis.
130.	16.	Male.	Purulent pericarditis, empyema (right).
131.	50.	Male.	Right.
132.	31.	Male.	Right and left.
133.	50.	Male.	Right and left.
134.	30.	Male.	Right lower.
135.	45.	Male.	Right upper.
136.	18.	Male.	Right upper.	Pericarditis, peritonitis, meningitis.
137.	28.	Male.	Right and left.	Pericarditis, old morbus cordis, infective endocarditis.
138.	37.	Male.	Right and left.	Large white kidney.
139.	35.	Male.	Left.
140.	40.	Male.	Right and left.	Typhoid.
141.	52.	Male.	Right lower.
142.	25.	Male.	Left upper.
143.	20.	Female.	Right lower.	Endocarditis, pericarditis, pleurisy.
144.	52.	Male.	Right.
145.	19.	Male.	Right upper.	Pericarditis, morbus cordis.
146.	14.	Male.	Right upper.
147.	20.	Male.	Right lower.
148.	62.	Female.	Right, lower two-thirds.	Endocarditis, granular kidney.
149.	40.	Male.	Left.
150.	22.	Female.	Right upper.	Typhoid.
151.	63.	Male.	Right.	Granular kidney.
152.	54.	Female.	Left lower.	Granular kidney.
153.	20.	Male.	Left lower.	Ulcerative endocarditis, granulations ¹ and morbus cordis, meningitis, perihepatitis.
154.	17.	Male.	Right and left.
155.	33.	Female.	Right.	Morbus cordis.
156.	53.	Male.	Left lower.
157.	47.	Male.	Right lower.	Granulations, old aortic disease.
158.	24.	Female.	Right lower.	Pleurisy (left), pyæmic infarction of left lung.
159.	46.	Male.	Left upper.	Pericarditis.
160.	45.	Male.	Left lower.
161.	39.	Male.	Right.	Pericarditis, tubal nephritis.
162.	38.	Male.	Left lower.
163.	35.	Female.	Left upper.
164.	59.	Male.	Right base.	Morbus cordis,
165.	24.	Male.	Right lower.
166.	23.	Male.	Right.
167.	37.	Male.	Right upper.	Pleurisy (right).
168.	55.	Male.	Right lower.	Pleurisy, pericarditis.
169.	45.	Male.	Right lower,
170.	41.	Male.	Left upper.

¹ Granulations on the cusps of some cardiac valve or other.

For this reason we have collected the fatal cases from the post-mortem records of St. Bartholomew's Hospital during the years 1891, 1892, 1893, 1894, and 1895, and analysed them, in order to obtain some sort of idea as to how often secondary infections occur in fatal cases, and in what situations of the body they are commonest. We shall begin by giving a tabular statement of the various cases collected in every year (see Table I.).

From this table it will be seen that there are 170 cases of fatal pneumonia, of which 141 occurred in males and 28 in females (in one case the sex was not given).

Analysing the cases still further, according to the ages and complications, we arrive at the following results:—

TABLE II.

Age.	No. of Case.		Secondary Infection.	
	Males.	Females.	Males.	Females.
1-10	4	2	3=75.0 per cent.	2=100.0 per cent.
10-20	15	4	7=46.6 "	4=100.0 "
20-30	31	6	12=38.7 "	4=66.6 "
30-40	42	7	14=33.3 "	3=42.8 "
40-50	24	4	5=20.8 "	1=25.0 "
50-60	19	3	4=21.0 "	0=0.0 "
Over 70	5	2	0=0.0 "	1=50.0 "
No age given	1	...	0=0.0 "	0=0.0 "
No age or sex	141	28	45=31.8 "	15=28.0 "
Total . . .	170		61=35.7 per cent.	

We have altogether, including the cases where either age or sex were not given, 170 cases with secondary inflammatory or infective complications in 61 cases = 35.7 per cent. Table II. shows that most deaths occur between the ages of 20 and 40, and that also between the same ages secondary infections are relatively less common.

We may here mention that we have only included those cases where it has been definitely stated that there was a fresh inflammatory process, and have excluded all those cases where descriptions had been used, such as morbus cordis, hyperæmia, perisplenitis, perihepatitis, and the like. We have done this in order not to use any but sound and convincing observations.

It is curious, if we may judge from a limited number of cases, how commonly inflammatory complications are seen in young people. Thus, of 25 cases under 21 years of age, 16 or 64 per cent. died with secondary infections—a percentage almost twice as high as that of all the cases taken

together. With advancing age the tendency towards secondary infections seems to disappear. We speak, however, with some reservation, because the numbers are comparatively small, and more extensive statistics might show different results.

In the next table (Table III.) we have analysed all the cases with secondary inflammatory processes according to age and sex.

TABLE III.—*Males.*

No. of Case.	Age.	Nature of Secondary Infection.	No. of Case.	Age.	Nature of Secondary Infection.
59	1-10	Empyema.	22	30-40	Endocarditis.
81	...	Pericarditis.	42	...	Pleurisy.
82	...	Pleurisy, peritonitis, pericarditis.	61	...	Pericarditis.
52	10-20	Pleurisy.	70	...	Pleurisy, pericarditis.
77	...	Pericarditis.	87	...	Pericarditis, meningitis.
112	...	Endocarditis (and M. C.), splenic infarct.	98	...	Pleurisy (L.).
130	...	Pericarditis, empyema.	118	...	Pleurisy and pericarditis.
136	...	Pericarditis, peritonitis, meningitis.	129	...	Pleurisy (R.), pericarditis.
145	...	Pericarditis.	159	...	Pericarditis.
153	...	Endocarditis (and M. C.), meningitis.	161	...	Pericarditis.
1	20-30	Pleurisy.	167	...	Pleurisy (R.).
9	...	Pericarditis.	56	40-50	Pericarditis, empyema.
15	...	Endocarditis, meningitis.	74	...	Pleurisy (L.), pericarditis.
25	...	Pleurisy (R. and L.).	76	...	Pericarditis, synovitis.
44	...	Pleurisy, pericarditis.	122	...	Pericarditis, pleurisy, peritonitis.
50	...	Pericarditis, peritonitis.	157	...	Endocarditis (and M. C.).
67	...	Pericarditis.	12	50-60	Endocarditis, pericarditis.
73	...	Pericarditis.	102	...	Pericarditis.
91	...	Pleurisy.	128	...	Endocarditis.
93	...	Pleurisy (R. and L.).	168	...	Pleurisy, pericarditis.
111	...	Endocarditis.	108	Age and sex not known.	Pericarditis.
137	...	Pericarditis, endocarditis (and M. C.).			
7	30-40	Pericarditis.			
16	...	Pleurisy.			
18	...	Pericarditis.			
Total = 46.					

Females.

No. of Case.	Age.	Nature of Infection.	No. of Case.	Age.	Nature of Infection.
78	1-10	Pleurisy (L.).	60	...	Septic.
86	...	Endocarditis, splenic infarcts.	158	...	Pleurisy (L.), pulmonary infarct (L.).
62	10-20	Endocarditis (and M. C.).	90	30-40	Pericarditis.
92	...	Pericarditis.	113	...	Endocarditis, infarcts.
124	...	Pericarditis.	125	...	Pericarditis.
143	...	Pericarditis, endocarditis, pleurisy.	30	40-50	Pericarditis.
27	20-30	Pericarditis, endocarditis (and M. C.).	148	60-70	Endocarditis.
58	...	Pericarditis, endocarditis.			
Total = 15.					

Great care has been taken not to include any cases where there is the slightest doubt as to the acuteness of the secondary processes. Thus, perhaps, a few cases of infective endocarditis have been omitted, and the rarity of the complication in our cases in comparison to those of other authors may thereby be in part explained, Dr. Osler finding 16 cases of infective endocarditis out of 100 cases, a percentage twice as high as that recorded by us. On analysing the fatal cases of infective endocarditis, diagnosed as such, which had been under observation between the years 1890 and 1895, it was found that pneumonia occurred six times in 41 cases, which gives an incidence of 14.6 per cent.

On analysing Table III. we find that in the 61 cases which are complicated with secondary infective lesions—

- (1.) Pericarditis occurs 37 times = 60.6 per cent., or 21.7 per cent. of all the fatal cases.
- (2.) Pleurisy occurs 20 times = 32.8 per cent., or 11.7 per cent. of all the fatal cases.
Empyema occurs 3 times = 4.9 per cent., or 1.7 per cent. of all the fatal cases.
- (3.) Endocarditis occurs 16 times = 26.2 per cent., or 8 per cent. of all the fatal cases.
- (4.) Meningitis occurs 4 times = 6.5 per cent., or 2.3 per cent. of all the fatal cases.
- (5.) Peritonitis occurs 4 times = 6.5 per cent., or 2.3 per cent. of all the fatal cases.
- (6.) Synovitis occurs once = 1.6 per cent., or 0.4 per cent. of all the fatal cases.
- (7.) Infarcts (septic) occur 4 times = 6.5 per cent., or 2.3 per cent. of all the fatal cases.

We see thus that *pericarditis* occurs oftenest; that eighteen times it occurred alone and seventeen times in combination with other lesions, viz. :—

Four times with endocarditis.
Eight times with pleurisy or empyema.
Once with peritonitis.
Once with meningitis.
Once with synovitis.
Twice with pleurisy and peritonitis.
Once with meningitis and peritonitis.
Once with pleurisy and endocarditis.

Pleurisy and empyema, taken together, occurred twenty-three

times, eleven times alone and twelve times in combination with other processes :—

Eight times with pericarditis.
Twice with pericarditis and peritonitis.
Once with pericarditis and endocarditis.
Once with pulmonary infarcts.

Endocarditis occurred sixteen times :—

Alone six times.
With pericarditis four times.
With pleurisy and pericarditis once.
With infarcts three times.
With meningitis twice.

It is curious that meningitis, peritonitis, infarcts, and synovitis never occurred alone. Infarcts, as a matter of fact, almost invariably occurred together with endocarditis.

Arranging some of the forms of inflammation according to the sex and age at which they occurred, the following results are obtained :—

Endocarditis.

Age.	Males.	Females.	Total.
1 to 10 years	0	1	1
10 to 20 "	2	2	4
20 to 30 "	3	2	5
30 to 40 "	1	1	2
40 to 50 "	1	0	1
50 to 60 "	2	0	2
60 to 70 "	0	1	1
Total	9	7	16

From this we learn that, if we may argue from our rather small numbers, endocarditis was much more frequent in females than in males; for, taking all cases together, it occurred seven times among the 28 female cases, *i.e.*, 25 per cent.; while the percentage among males amounts to 6.4 per cent.

For *pericarditis* we obtain the following results:—

Age.	Males.	Females.	Total.
1 to 10 years	2	0	2
10 to 20 "	4	3	7
20 to 30 "	6	2	8
30 to 40 "	9	2	11
40 to 50 "	4	1	5
50 to 60 "	3	0	3
60 to 70 "	0	0	0
Total	28	8	36

From this we see that *pericarditis* occurred amongst females in 28.5 per cent. ; amongst males in almost 20 per cent. of the cases. For *pleurisy and empyema* the results are as follows:—

Age.	Males.	Females.	Total.
1 to 10 years	2	1	3
10 to 20 "	2	1	3
20 to 30 "	5	1	6
30 to 40 "	7	0	7
40 to 50 "	3	0	3
50 to 60 "	1	0	1
60 to 70 "	0	0	0
Total	20	3	23

Here the percentage for males and females respectively are 14.1 and 14.2 per cent.

Again we find that according to ages the percentages work out in the following manner:—

Ages.	Endocarditis.			Pericarditis.			Pleurisy and Empyema.		
	Males.	Females.	Total.	Males.	Females.	Total.	Males.	Females.	Total.
	Per Cent.	Per Cent.	Per Cent.	Per Cent.	Per Cent.	Per Cent.	Per Cent.	Per Cent.	Per Cent.
1 to 10	0.0	50.0	16.6	50.0	0.0	30.3	50.0	0.0	50.0
10 to 20	13.3	50.0	21.0	26.6	75.0	37.6	13.3	75.0	15.7
20 to 30	9.6	33.3	13.5	19.3	33.3	21.6	18.2	33.3	16.2
30 to 40	2.3	14.2	4.0	21.4	28.5	22.3	16.6	28.5	14.2
40 to 50	4.1	0.0	3.5	16.6	25.0	17.8	12.5	25.0	10.6
50 to 60	10.5	0.0	9.0	15.7	0.0	13.6	5.2	0.0	4.5
60 to 70	0.0	50.0	14.2	0.0	0.0	0.0	0.0	0.0	0.0

From this table we learn that—

(i.) Endocarditis is commonest amongst females, and occurs in them most frequently between the ages of 1–10, 10–20, and 20–30 years.

(ii.) Pericarditis is commoner amongst females, and in them more frequently during the ages of 10–20 and 20–30 years, the ages 30–40 and 40–50 coming next.

(iii.) Pleurisy (and empyema) occurs also more frequently in women, especially during the second decade, the third and fourth decades coming next.

(iv.) Secondary infections both in males and females are most common during the first four decades.

Seeing that pleurisy and pericarditis are common complications of pneumonia, it might be asked whether these inflammations are not merely due to the extension of the inflammatory process from the lungs rather than true secondary or remote infections.

We hardly think that they can be accounted for by assuming direct extension to neighbouring parts for the following reasons:—

(a.) Pericarditis occurs pretty frequently without there being any pleurisy or any inflammatory tract between the diseased pulmonary area and the pericardium.

(b.) Other forms of remote inflammations, such as peritonitis and meningitis, are by no means very rare complications.

(c.) As far as pleurisy is concerned, it often occurs on the side opposite to that which presents the pulmonary changes. This is an important point, and is well demonstrated by the following cases:—

No. of Case.	Lung Affected.	Pleurisy.	Complication.
25 ¹	Left.	Especially right.	...
74	Right.	Left.	Pericarditis.
82	Left.	Right.	Pericarditis.
93	Left.	Right and left.	...
98	Right.	Left.	...
118	Right (base).	Right and left.	Pericarditis.
130	Left.	Right.	Pericarditis.
158	Right.	Left.	Pyæmic infarct in left lung.

In some of these cases (in four) there was also pericarditis, and it might be objected that the process spread from one lung through the pericardium to the other lung; but such an expla-

¹ These numbers refer to Table I.

nation is stilted and more forced than real; besides, it has no actual support.

We believe, therefore, that pleurisy does often occur as a true secondary inflammation, produced most probably by the pneumo-coccus.

Lastly, we may mention that in four cases (viz., Nos. 78, 114, 140, and 150) typhoid fever co-existed with pneumonia. Some writers consider such pneumonia to be typhogenous, *i.e.*, produced by the bacillus typhosus, but there is hardly sufficient evidence for this assumption; indeed, in such cases one of us has invariably discovered the pneumo-coccus, but never the typhoid bacillus; so that we are inclined to consider such cases as double or mixed infections, pneumonia being not an uncommon complication in fatal cases of typhoid fever.

There are many points we have not alluded to in this paper, notably the incidence of nephritis, acute or chronic. Here we were concerned chiefly with the actually proved secondary infections, especially of the serous membranes.

We have shown that such secondary infections are not rare, and that they occur oftenest in the pericardial, pleural, and endocardial membranes. The tables show that pericarditis is somewhat commoner than endocarditis, and therein we disagree from Dr. Osler. We have paid some attention to the question whether morbus cordis is a prominent predisposing factor, although we have not previously alluded to it. We find (see

Relation between Chronic Heart-Disease and Infective Endocarditis in Pneumonia.

Endocarditis and Morbus Cordis.	Endocarditis without Morbus Cordis.	Morbus Cordis without Endocarditis.
No. of Case.	No. of Case.	No. of Case.
27	12	89
62	15	120
112	22	124
137	58	127 ¹
153	86	145
157	111	155
...	113	164
...	128	...
...	143	...
...	148	...
Total, 6	10	6

¹ This case is so doubtful that it should be excluded.

table) that in a number of cases there had existed morbus cordis before infective endocarditis appeared; in fact, in six cases of infective endocarditis out of a total of 16, there has been valvular disease of old standing (*i.e.*, in 37.5 per cent.); but, on the other hand, there were six cases of morbus cordis which were not followed by infective endocarditis, and ten cases of endocarditis without previous morbus cordis. At present, therefore, we are not prepared to decide definitely, although the fact that of twelve cases of valvular disease six also had endocarditis is very strongly in favour of Dr. Osler's statement that pre-existing valvular lesions are markedly predisposing factors.

ON THE TREATMENT OF ANÆMIA BY OXYGEN INHALATIONS.

BY

F. M. BURNETT, Esq., M.B.

In view of the benefit said to be derived from treatment by inhalation of oxygen in cases of leukæmia and other forms of anæmia, it seemed to me to be of interest to try by exact experiment how this gas affects the blood, not only in leukæmia, but also in other blood diseases. The subject of this paper, therefore, deals with the results of that treatment. It is, however, impossible to judge the effect of oxygen in anæmia without understanding its action on normal blood, and therefore I have first studied the changes produced in normal blood, *i.e.*, in my own blood, by oxygen inhalation, and, secondly, those produced in the blood of anæmic patients admitted into St. Bartholomew's Hospital. Dr. Hensley has kindly allowed me to make use of his cases.

I. *Method of Investigation.*

In each case the same treatment was employed, *viz.*, oxygen inhalations three times a day, for ten minutes at a time. Oxygen was allowed to issue gently from a cylinder through an ordinary rubber tube and a large funnel, which was held near the mouth. In the absence of exact measurement, it was computed that one cubic foot of oxygen was administered during each inhalation.

In the case of my own blood, further experiments were made in order to discover what effect, if any, single inhalations would produce on the number of white corpuscles present.

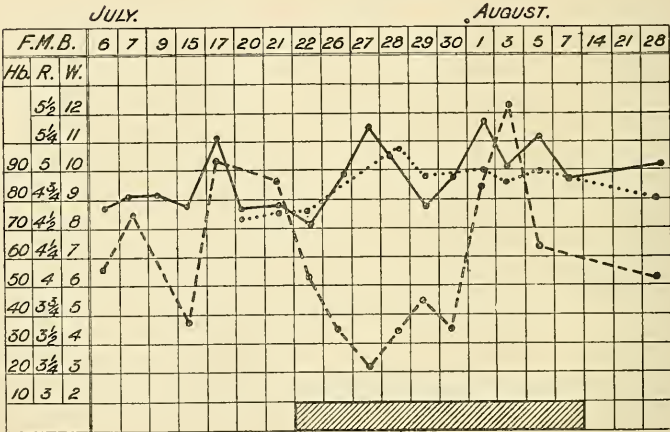
Blood-counts were in every case made with a Thoma-Zeiss hæmocytometer, and hæmoglobin estimations with a Fleischl hæmoglobinometer. These were all made by the same individual. As far as possible, for the purpose of comparison, the counts were made at the same time of day, and at the same period after ingestion of food.

II. Action of Oxygen on Normal Blood.

(See Chart I., A. and B.)

Chart I. illustrates experiments made with my own blood, which I have taken to represent "normal" blood, although

CHART I. NORMAL



R.C.
4,712,000
4,804,000
4,820,000
4,744,000
5,268,000
4,684,000
4,782,000
4,552,000
4,960,000
5,380,000
5,112,000
4,748,000
4,936,000
5,496,000
5,000,000
5,284,000
4,912,000
5,068,000

W.C.
6,770
8,600
4,680
10,000
8,800
6,250
4,680
3,125
4,680
5,077
4,680
9,375
12,490
7,030
6,250

Hb.
72.5%
75%
75%
95%
87.5%
90%
85%
90%
80%

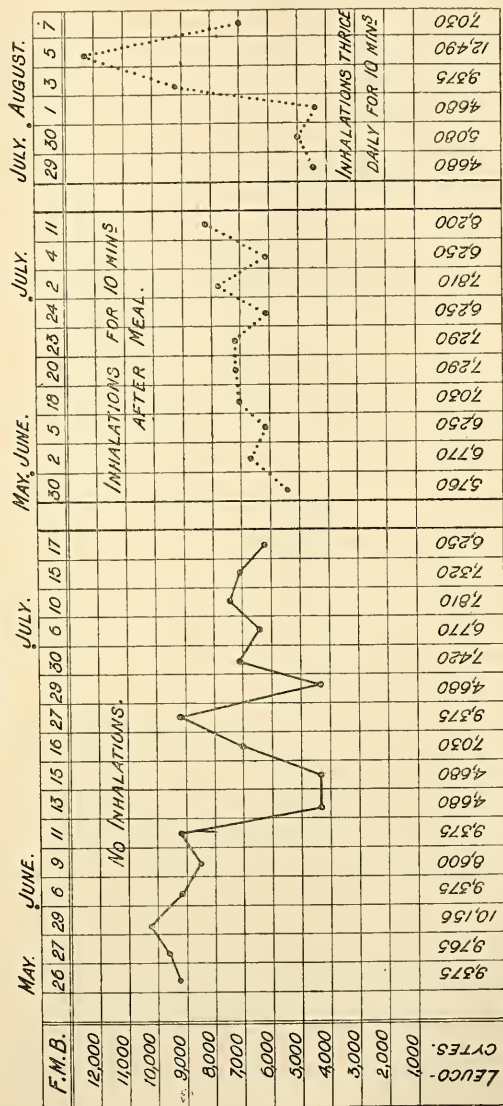
----- = W.C. in thousands. ——— = R.C. in millions. = lb.
 = Oxygen.

perhaps it shows slight anæmia. The life led while under observation was that of House-Physician, and this implies much

work and little outdoor exercise. It was taken as "normal,"

CHART A. NORMAL.

NUMBER OF LEUCOCYTES ONE HOUR AFTER DINNER WITH AND WITHOUT OXYGEN INHALATIONS.



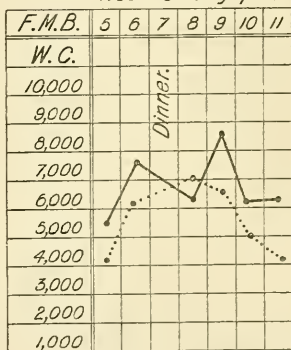
as it was found to compare favourably with that of other healthy individuals living under the same circumstances.

The examination of my blood before the inhalations yielded the following result:—

Red corpuscles $4\frac{3}{4}$ millions.
 White corpuscles = 6000.
 Hæmoglobin = 75 per cent.
 Oxygen was inhaled for a fortnight.

During this period it will be chiefly noticed, on studying the chart, that there was a marked improvement in the percentage

CHART B. NORMAL.
 LEUCOCYTIC CURVE.
 AVERAGE DAILY CHART.
 Hour of day p.m..



no O.	5,750	7,812	6,250	8,600	6,250	6,250
-------	-------	-------	-------	-------	-------	-------

with O.	4,65	6,250	7,000	6,600	5,000	4,100
---------	------	-------	-------	-------	-------	-------

———— = without oxygen.
 = with oxygen.

of hæmoglobin, which is maintained throughout, though not at its original height. An increase is noticed in the number of red corpuscles, but this is not very marked.

No constant change was observed in the number of the white corpuscles, the diminution observed at first being followed by higher readings. It may be remarked that no ill effects were observed to follow each inhalation—no giddiness or other disagreeable sensations. At the end of a few days' oxygen treatment, one's feeling of good health and spirits was unusual.

Turning now to *Charts A. and B.*, the former shows a series of counts of the white corpuscles present in my blood one hour after dinner, both without and with inhalations. The conditions under which the counts were made were as far as possible identical, and in almost every case four separate slides were counted, and the average struck from these, in order to make the result more trustworthy.

Chart A. gives estimations (1) one hour after dinner; (2) one hour after dinner, dinner being followed immediately by an oxygen inhalation; (3) one hour after dinner during a continuous course of inhalations.

Though the figures in the second division perhaps range rather lower than those in the first, no definite effect can be demonstrated.

It may be noted that these observations spread over three or four months, and were not consecutive.

Chart B. again gives two curves, the upper representing readings taken, without inhalations, at various noted intervals after dinner, the lower representing the same when dinner was followed by ten minutes' inhalation of oxygen.

These are hardly more worthy of notice. It will be seen, however, that the leucocytic curve during the oxygen treatment is uniformly below the normal curve where corresponding readings are given. Oxygen apparently has no definite action on the quantity of the white corpuscles.

III. *Action of Oxygen on Anæmic Blood.*

(Charts II., III., IV., V.)

(*a.*) *Chlorosis.*—Charts II., III., IV., and V. are those obtained from four cases of chlorosis of varying severity admitted into the Hospital. In all of them reduced iron was given during the periods indicated on the chart, alternating with periods of oxygen treatment.

With the exception of Chart II., there is in these cases a notable and sustained rise in the percentage of hæmoglobin during oxygen treatment, and this even as compared with the effect of treatment by iron immediately preceding.

Not excepting Chart II., the rise in the number of red corpuscles during inhalation is very marked.

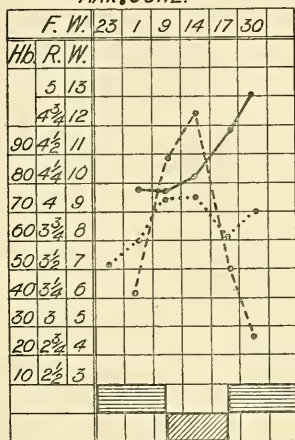
There is certainly no diminution in the number of white corpuscles; rather the opposite is noticed. In all these cases the change in the patient as observed clinically was very marked. Shortly after the oxygen treatment was commenced the patient

would rapidly gain colour and spirits, and change from a flabby, toneless woman into a rosy and animated girl.

(b.) *Secondary Anæmia.*—Chart VI. represents a case of anæmia appearing in the course of perihepatitis. Here oxygen was

CHART II. CHLOROSIS.

MAY. JUNE.



R.C.
4,200,000
4,200,000
4,264,000
4,764,000
5,000,000

W.C.
6,250
10,940
12,500
7,030
4,685

Hb.
50.0%
60.0%
75.0%
75.0%
60.0%
75.0%

----- = W.C. in thousands.

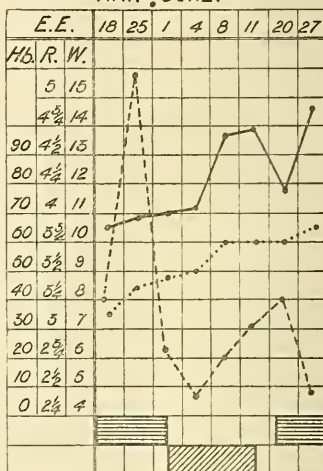
———— = R.C. in millions.

..... = Hb.

▨ = Iron. ▩ = Oxygen.

CHART III. CHLOROSIS.

MAY. JUNE.



R.C.
3,836,000
3,940,000
4,000,000
4,050,000
4,640,000
4,712,000
4,180,000
4,860,000

W.C.
7810
13,825
6,250
4,680
6,250
7,240
8,156
4,680

Hb.
55%
45%
47.5%
50%
60%
60%
60%
65%

----- = W.C. in thousands.

———— = R.C. in millions = Hb.

▨ = Iron. ▩ = Oxygen.

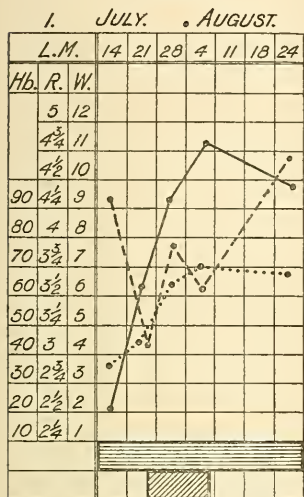
alone employed. While the hæmoglobin and white corpuscles are scarcely affected, a marked rise in the number of red corpuscles is obtained.

(c.) *Pernicious Anæmia.*—Chart VII. is that of a man of 58, which was regarded as a case of pernicious anæmia. On admis-

sion, July 1, 1896, the patient was described as a square, well-made man, not at all wasted, but presenting the typical waxy complexion.

He gave a history of two years' duration, and stated that

CHART IV. CHLOROSIS.



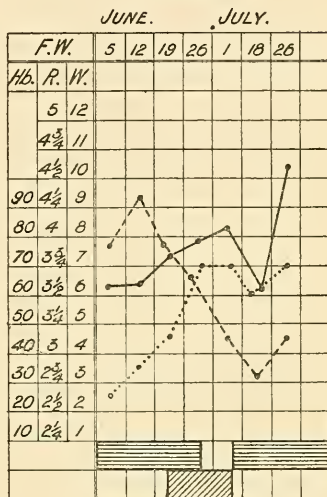
R.C.
2,496,000
3,624,000
4,308,000
4,760,000
4,452,000

W.C.
9,375
9,680
7,810
6,250
10,935

Hb.
35%
45%
65%
70%
67.5%

----- = W.C. in thousands.
 ————— = R.C. in millions. = Hb.
 = Iron. = Oxygen.

CHART V. CHLOROSIS.



R.C.
3,304,000
3,552,000
3,856,000
3,984,000
4,120,000
5,584,000
4,568,000

W.C.
7,812
9,375
7,812
6,650
4,700
3,145
4,685

Hb.
25%
35%
45%
70%
70%
60%
70%

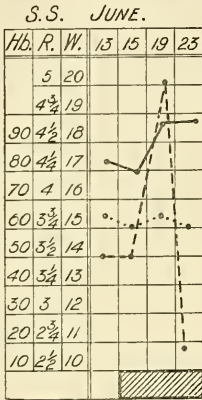
----- = W.C. in thousands.
 ————— = R.C. in millions. = Hb.
 = Iron. = Oxygen.

during that time he was getting weaker and shorter of breath. He had been badly fed during that period, and for nearly six months confined to the house "looking after a child." For about three weeks he had had slight bleeding from the gums

“after washing,” but no other hæmorrhages. A fresh hæmorrhage was observed in the left fundus, and old hæmorrhages in both eyes, and also markedly pulsating vessels.

On August 12 a note was made that the spleen was felt. He

CHART VI. PERIHEPATITIS.



R.C.
4,250,000
4,204,000
4,686,000
4,688,000

W.C.
14,000
14,000
20,500
10,937

Hb.
65%
60%
65%
60%

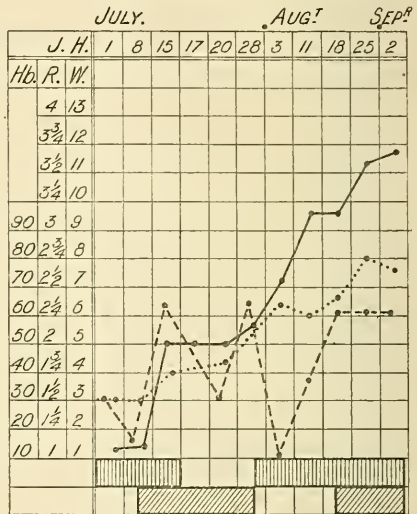
----- = W.C. in thousands.

————— = R.C. in millions.

..... = Hb.

▨ = Oxygen.

CHART VII. PERNICIOUS ANÆMIA.



R.C.
1,006,250
1,118,695
2,076,000
2,076,000
2,168,000
2,320,000
3,196,000
3,320,000
3,624,000
3,624,000

W.C.
3,125
1,500
6,250
3,125
6,250
1,100
3,900
6,250
6,250
6,250

Hb.
30.0%
30.0%
40.0%
45.0%
65.0%
60.0%
67.5%
80.0%
75.0%

----- = W.C. in thousands.

————— = R.C. in millions. = Hb.

▨ = Arsenic.

▨ = Oxygen.

had gained 4 lbs. since admission. His gums bled occasionally, but he rapidly improved in strength and colour.

From his chart it will be seen that he was treated by courses of oxygen and arsenic.

The white corpuscles may be dismissed with the remark that under oxygen they came up from about 3000 to the normal.

The *hæmoglobin* is here found to rise continuously during oxygen, and to remain stationary or rise slightly under arsenic.

On the other hand, the *red corpuscles* under oxygen alone, although rising in number, do not show the improvement manifested under combined oxygen and arsenic treatment, or even arsenic alone. It will be observed that after a fortnight under oxygen alone the rise is not continued, but that when the oxygen treatment was again combined with that by arsenic, the red corpuscles again rise in number.

(*d.*) *Leukæmia* (Charts VIII. and IX.).—Two cases of leukæmia came under observation. One (Chart VIII.) was treated alternately by oxygen and arsenic; the other (Chart IX.), not admitted into Hospital, was treated by courses of arsenic, and is inserted here for the purpose of comparison.

A. K., aged 18 years, was admitted May 5 with an enlarged spleen.

History.—Felt well till three weeks ago, then complained of weakness, shortness of breath, headache, and diarrhœa. No hæmorrhages. For one week attention has been drawn to “stomach getting larger” and swelling of legs. Eyesight getting bad.

On admission.—Anæmic, with red lips. Apical systolic murmur. No enlarged glands. Spleen visibly prominent; two notches felt; reaches midway between umbilicus and pubes; crosses mid-line by three-quarters of an inch; movable and rather tender. Liver not felt. Œdema of legs. Urine 1023; abundant urates; cloud of albumin; no blood. Eyes marked retinitis leukæmica; hæmorrhages in both eyes; discs not much swollen.

May 15.—No œdema. No albumin in urine.

May 22.—Spleen feels much less prominent. Vertical measurement lessened by one inch.

June 11.—Mr. Jessop examined the eyes again, and reported that the changes had almost all cleared up. Spleen less prominent.

June 20.—Spleen more prominent again. Measurement the same as on admission, but reaches a little farther across mid-line.

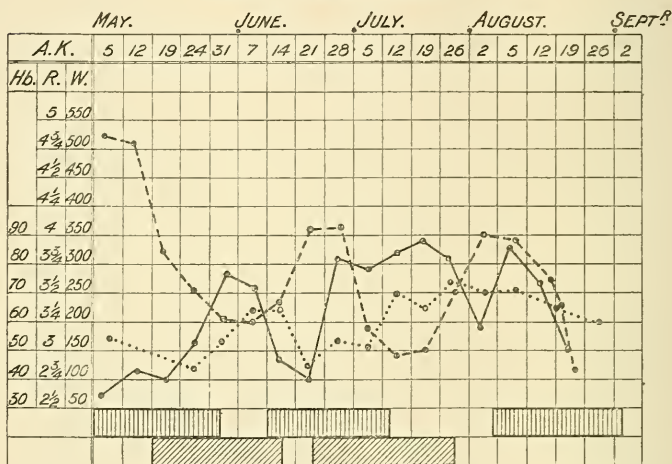
July 20.—Discharged himself. Henceforth treated as out-patient.

August 26.—Glands of neck slightly enlarged.

Clinically the course of the disease during his stay in the

Hospital was one of marked improvement. After about three weeks' treatment with oxygen and arsenic he felt well, was less anæmic, and could walk about naturally. He discharged himself from the Hospital, and was lost sight of for over a

CHART VII. LEUKÆMIA.



R.C.
2,624,000
2,856,000
2,748,000
3,112,000
3,708,000
3,368,000
2,840,000
2,660,000
3,788,000
3,688,000
3,816,000
3,896,000
3,800,000
3,225,000
3,882,000
3,544,000
2,744,000

W.C.
530,000
506,000
528,125
256,250
207,800
200,000
230,000
356,250
357,250
137,450
142,140
150,000
266,570
358,000
340,620
279,630
125,000

Hb.
45%
55%
65%
69%
47.5%
57.5%
52.5%
70%
65%
79%
70%
70%
56%
50%

----- = W.C. in thousands. ————— = R.C. in millions. = Hb

▨ = Arsenic. ▨ = Oxygen.

week. When seen again, his health had certainly deteriorated, and there was increasing shortness of breath.

Examination of his blood on admission gave the following result:—

Red corpuscles	2,625,000
Hæmoglobin	56 per cent.
White corpuscles	530,000
or approximately 1 white to 5 red.	

It will be seen by the chart that he had, in all, three courses of arsenic and two of oxygen.

Taking the latter first, it will be noticed that during each of the oxygen periods there is a marked rise in the number of red corpuscles and in the percentage of hæmoglobin; and at first I felt convinced that the diminution in the number of white corpuscles was due to the same remedy; but this was continued only till the number 200,000 was reached, and then commenced a rise in the last week of the first course; and in the second course this is even more marked. A rise from 140,000 to 260,000 takes place during the last fortnight of it.

Can then any influence on the number of white corpuscles be seen during the arsenic periods?

The first period is coincident with a marked fall; when the arsenic is taken off there is scarcely any further fall, but almost immediately a rise in the number of white corpuscles takes place. Again arsenic is put on in increasing doses, and as soon as the patient gets into the larger doses a rapid fall again takes place.

The arsenic is omitted, another rise follows, and after an interval of three weeks the last course of arsenic is again coincident with a rapid fall in the number of white corpuscles.

It is interesting to observe in this chart how a rise in the number of red corpuscles nearly always accompanies a fall in the white.

I. M., aged 34. Leukæmia splenica (Chart IX.).

History.—June 20.—Four months ago “hardness” in abdomen noticed. Getting weak since. Palpitation. No hæmorrhage. Condition when first seen, very sallow and anæmic. Spleen, notch felt; reaches $2\frac{3}{4}$ inches below umbilical level; keeps to left of mid-line by two inches. No enlarged glands, œdema, or retinitis.

June 30.—Spleen rather more prominent.

This patient was admitted, but had to go home after two days, and so was treated as an out-patient.

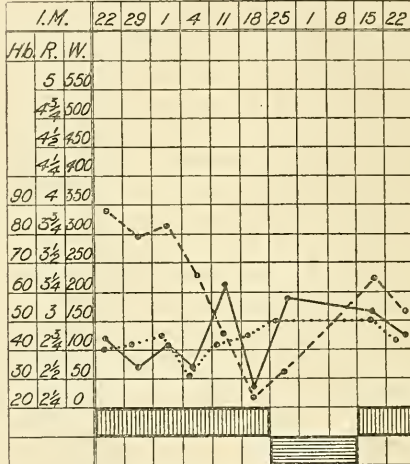
On July 18, when taking liq. arsenicalis three times a day, he showed marked symptoms of poisoning, general pains and tremors, diarrhœa, sickness, anæmia, and dryness of the throat.

He was then put on iron, and these symptoms quickly disappeared.

In this case the behaviour of the red corpuscles and hæmoglobin is less important, but the white corpuscles show very

CHART IX. LEUKEMIA.

JUNE. JULY. AUGUST.



R.C.
2,876,000
2,600,000
2,780,000
2,628,000
3,320,000
2,408,000
3,228,000
3,064,000
2,840,000

W.C.
345,200
293,700
312,940
247,660
128,125
40,625
70,310
224,000
166,700

Hb.
42.5%
42.5%
45.0%
35.0%
42.5%
45.0%
50.0%
50.0%
45.0%

----- = W.C. in thousands.
 ————— = R.C. in millions. = Hb.
 = Arsenic. = Iron.

interesting changes. Coincident with the commencement of the arsenic treatment there is a slight fall in their number, which becomes very rapid after the first fortnight, stops immediately the arsenic is dropped, but reappears promptly after

the first week of a second course. After August 22 the patient could not attend regularly, and so no examinations were made.

In these two cases what struck me most was that the improvement in the patient's general condition was never commensurate with the fall in the number of white corpuscles. On paper the result of the arsenical treatment appears good. As a matter of fact, with the exception of some initial improvement, till last seen, the second patient's condition seemed to become steadily and gradually worse.

In the former case, on the other hand, the patient appeared, both to himself and all who saw him, to make great improvement when under the oxygen treatment. When the report on his blood was "unfavourable," he himself would notice no change in his health.

Conclusions.—From these observations I think it may be granted that:—

- (1.) Oxygen influences the quality of the blood.
- (2.) Oxygen improves the quality of the blood.
- (3.) This improvement is in the direction of an increase in the number of red corpuscles with a rise in the percentage of hæmoglobin.
- (4.) It is *not* proved that it influences the number of white corpuscles present: indeed, it seems that it does not influence them at all.
- (5.) Clinically it effects marked improvement in the patient.
- (6.) Oxygen is therefore a valuable adjunct to other remedies in cases of the various anæmias, and especially perhaps in leukæmia.

It has been claimed that oxygen renders the size of the spleen smaller in leukæmia. Certainly at the end of the first course of oxygen in the case of A. K. the spleen was considerably smaller. But it must be remembered that this period corresponded with his first prolonged rest in bed. It was found that when the oxygen was left off the spleen increased in size; and felt as it might do if it had suddenly become engorged with blood. After this, however, the changes in size were so variable, and did not correspond to the treatment, so that notes on the actual measurements were not continued, but the net result was that the spleen seemed little altered in the end.

It remains to make observations on the effect of oxygen on the various forms of leucocytes. Lack of time unfortunately prevented me from doing this, and I must leave this to

others who may find suitable opportunity of continuing this work.

In conclusion, I must express my indebtedness to Dr. Kanthack for the kind interest he has taken in this research and for the copious advice he has given me at all times. It was his suggestion which induced me to take up this work, and I feel it to be my pleasant duty to acknowledge the readiness with which he watched the progress of my observations.

THE
PROGNOSIS AND CAUSES OF DEATH IN CASES
OF DIPHTHERIA TREATED BY ANTITOXIN
AT ST. BARTHOLOMEW'S HOSPITAL.

BY

H. J. MAY, M.B.

AND

A. A. KANTHACK, M.D.

In discussing the prognosis and causes of death in cases of diphtheria, treated by antitoxin, the cases at St. Bartholomew's Hospital have been particularly considered. For this purpose the notes recorded of sixty-three cases, which have been under the treatment there, since those cases which Dr. Herringham reported in the St. Bartholomew's Hospital Reports for 1895, have been carefully examined.

All these cases have been treated with some form or other of antitoxin, and are taken in order from the Ward notes. All were clinically diphtheria, and all, with three exceptions, were also shown to be bacteriologically diphtheria. Of these three no notes could be found in the records of the Pathological Laboratory.

The number of deaths among the 63 cases was 31, but of these five died within twenty-four hours of admission, in which space of time it may be considered that little or nothing can be expected from the antitoxin; they have therefore been omitted.

We have thus a total of 58 cases with 26 deaths; but of these deaths three occurred so late after admission that we are justified in assuming that they were not deaths from diphtheria, although in none of them an autopsy was allowed to confirm this opinion. These cases were the following:—

(1.) A child, aged 3 years, admitted on the eighth day of the disease, died fifty-nine days after admission. Signs of paralysis appeared six days after admission, which caused the prolonged detention within the Hospital. The patient, being

considered to have entirely recovered, developed measles thirty-four days after admission, followed in fifteen days by bronchopneumonia, which terminated in death.

(2.) A child, aged $2\frac{1}{2}$ years, admitted on the second day of the disease, died thirty-one days after admission. No membrane was seen later than the eighth day after admission, nor were bacilli found after that date. Severe paralysis occurred in this case, the first signs of which were noticed twenty-four days after admission; two days later respiratory difficulty occurred, due apparently to paralysis of the intercostal muscles and the diaphragm. Great prostration and weak cardiac action supervened.

(3.) A child, aged $1\frac{1}{2}$ years, admitted on the fourth day of the disease, died thirty-nine days after admission. This case closely resembled No. 1. No bacteriological examination was made. Ten days after admission signs of paralysis appeared, which caused prolonged detention within the Hospital. On the thirtieth day after admission, when it was considered to have recovered from diphtheria, the child contracted measles from a case in the ward then under treatment for diphtheria, and bronchopneumonia also appeared, death following nine days later.

We have thus 58 cases treated with antitoxin with a fatal result in 23 instances, disregarding the three cases cited above because active diphtheria was not the immediate cause of death, although in one case a sequela of it certainly was; but in this paper we are not considering fatal sequelæ, unless at the same time diphtheria bacilli were found on examining the tissues post-mortem, or death had followed within a few days from the last occasion on which the bacilli had been found in the larynx or pharynx, or other tissues, or unless any membrane had been present.

The bacteriological report on these cases will be found in the records of the Pathological Laboratory at St. Bartholomew's Hospital. They show that out of the total number of 58 cases the bacteriological examination was made in 55 during life. In all of the 55 cases bacilli were found on one or more occasions, which prevents any doubt being entertained of the correctness of the clinical diagnosis in these cases. The examination, as a rule, was made not merely once in each case, the number of such examinations for each case varying from one to five. Out of these 55 an autopsy was performed in 15, and further bacteriological examination of the lungs and other tissues was made post-mortem in 12 out of the 15; of the remaining three no records of post-mortem bacteriological examination were obtainable. Short notes on these fatal cases will be found in the following table:—

(A.) Cases in which an Autopsy was Performed (15).

No.	Bacteriological Examination made during Life.	Appearances Post-mortem.	Bacteriological Examination made Post-mortem.	Bronchopneumonia.
1.	Two examinations during life. (1.) Numerous bacilli diphtheriae in nearly pure cultures (large clubbed form). (2.) One day later. Cast of bronchi examined; bacilli diphtheriae exceedingly numerous.	Membrane on soft palate, naso-pharynx, uvula, larynx, trachea, and bronchi. No appearance of broncho-pneumonia.	Bacilli diphtheriae in lungs and spleen.	Absent. No ante-mortem. Tracheotomy.
2.	One examination during life. Colonies of bacilli diphtheriae and numerous diplococci.	Membrane in larynx and trachea; broncho-pneumonia; both lower lobes solid.	No examination recorded.	Present.
3.	No bacteriological examination made.	Larynx coated with broken-down membrane; lungs "stuffed with broncho-pneumonia."	No examination recorded.	Present.
4.	One examination during life. Bacilli diphtheriae and staphylococci.	Membrane above tracheotomy wound. L. lung retracted out of sight; R. lung, patches of collapse.	Bacilli diphtheriae in lungs and spleen.	Absent. No ante-mortem. Tracheotomy.
5.	One examination during life. Membrane examined showed long threads of bacilli diphtheriae. Culture tubes thickly overgrown with bacilli diphtheriae.	Membrane extends all down the larynx, trachea, and larger bronchi. Lungs, left lower lobe and posterior part of right upper solid with broncho-pneumonia.	Cultivations made from lungs and spleen. In lung, bacilli diphtheriae found; in spleen, doubtful.	Present. Tracheotomy.

(A.) Cases in which an Autopsy was performed (15)—continued.

No.	Bacteriological Examination made during Life.	Appearances Post-mortem.	Bacteriological Examination made Post-mortem.	Broncho-Pneumonia.
6.	One examination during life. Membrane examined; found bacilli diphtheriæ, long and short mixed.	Membrane extends to intra-pulmonary bronchi. (Broncho-pneumonia not evident to naked eye.)	Bacilli seen three days after P.M. Lungs (microscopic examination), broncho-pneumonia.	Present. Tracheotomy.
7.	One examination during life. Bacilli diphtheriæ very numerous.	Much membrane above tracheotomy wound, less below. No appearance of broncho-pneumonia.	Cultures of spleen showed bacilli diphtheriæ and bacilli coli. Cultures of lung showed bacilli diphtheriæ.	Absent. No signs ante-mortem. Tracheotomy.
8.	One examination during life. No bacilli diphtheriæ found.	Broken-down membrane in trachea and bronchi. Lungs, in both typical broncho-pneumonia, "apparently laryngeal diphtheria."	Specimens from larynx, trachea, and lungs showed bacilli diphtheriæ. Cultures of—1. Larynx=numerous bacilli diphtheriæ. 2. Lungs=numerous bacilli in pure cultures. 3. Spleen=bacilli diphtheriæ.	Present. Tracheotomy.
9.	One examination during life. Membrane examined showed bacilli diphtheriæ (medium sized).	Membrane found all through larynx and trachea as far as intra-pulmonary bronchi. Lungs, no broncho-pneumonia.	Bacilli diphtheriæ in lungs.	Absent. No signs ante-mortem. Tracheotomy.
10.	One examination during life. A few small colonies of typical bacilli diphtheriæ found.	No membrane in larynx or trachea. Lungs, in both many patches of broncho-pneumonia; R. lung, practically solid; L. lung, apex free.	Bacilli diphtheriæ found in lungs, larynx, and trachea.	Present. Tracheotomy.

11.	One examination during life. Numerous bacilli diphtheriæ.	Larynx and trachea filled with diphtheritic membrane and greenish pus; this penetrates into large and medium-sized bronchi. Lungs, emphysematous, with areas of collapse. No broncho-pneumonia. Kidneys, slightly granular, "early interstitial nephritis."	Lungs and bronchial glands showed numerous colonies of bacilli diphtheriæ. Spleen and kidneys, nil.	Absent. No signs ante-mortem.
12.	One examination during life. Membrane examined showed bacilli diphtheriæ. Cultures showed bacilli diphtheriæ and streptococci.	Mucous membrane of trachea and bronchi ulcerated throughout; tubes contained much yellowish pus; bronchial glands much enlarged. Lungs, broncho-pneumonic patches throughout.	Lungs, numerous bacilli diphtheriæ. Spleen, streptococci. No tubercle bacilli found.	Present. Tracheotomy.
13.	One examination during life. Bacilli diphtheriæ found.	Membrane on edges of tracheotomy wound; on the cords, epiglottis, and sides of trachea. Lungs, broncho-pneumonia, caseous nodules, and caseous bronchial glands.	Trachea, many bacilli diphtheriæ. Lungs and kidney, bacilli diphtheriæ. Spleen, doubtful.	Present. Tracheotomy.
14.	One examination during life. Nose and pharynx examined; both showed numerous bacilli diphtheriæ.	Membrane on posterior nares, soft palate, and walls of pharynx; in larynx and trachea, but more scanty, there being practically none below tracheotomy wound. Lungs, no evidence of broncho-pneumonia in either, but considerable collapse.	Lungs and spleen showed no growth (over-heated). Trachea showed bacilli diphtheriæ in small numbers.	Absent. No signs ante-mortem. Tracheotomy.
15.	One examination during life. Culture from blood coughed up showed bacilli diphtheriæ.	No membrane on pharynx or naso-pharynx; below tracheotomy wound, the trachea and bronchi contain yellow membranous matter, readily separated from mucous membrane. Lungs, patches of collapse throughout both; in R. upper and lower lobes are extensive areas of broncho-pneumonic consolidations, which assume an almost lobular distribution.	No examination recorded.	Present. Tracheotomy.

(B.) Cases in which no Autopsy was performed (8).

No.	Bacteriological Examination during Life.	Broncho-Pneumonia.
1.	Two examinations made. (1.) In one tube no bacilli diphtheriæ were found, but streptococci, diplococci, and staphylococcus aureus. (2.) Day later: Cast of bronchus examined showed exceedingly numerous bacilli diphtheriæ, and a large number of vibrios.	No note on condition of the lungs was recorded. Tracheotomy.
2.	Two examinations made. (1.) Bacilli diphtheriæ, staphylococci, and sarcinæ. (2.) Six days later: Diphtheria bacilli.	No note on condition of the lungs was recorded.
3.	One examination made. Bacilli diphtheriæ were found.	Broncho - pneumonia (signs of) recorded. Tracheotomy.
4.	One examination made. Large numbers of the large clubbed forms of bacilli diphtheriæ found.	Broncho - pneumonia (signs of) recorded. Tracheotomy.
5.	One examination made. Diphtheria bacilli found.	Broncho - pneumonia (signs of) recorded. Tracheotomy.
6.	Two examinations made. (1.) No diphtheria bacilli found. (2.) Four days later: Trachea showed numerous large colonies of the diphtheria bacillus (small variety).	Broncho - pneumonia (signs of) recorded. Tracheotomy.
7.	One examination made. Diphtheria bacilli in the membrane examined.	Broncho - pneumonia (signs of) recorded. Tracheotomy.
8.	No record of any bacteriological examination.	No note on condition of the lungs was recorded. Tracheotomy.

It is striking that of 23 fatal cases tracheotomy had been performed in 19, or in 82.6 per cent.

When the observations were made, attention was paid merely to the absence or presence of diphtheria bacilli in the lungs or spleen, and unfortunately the presence of pyogenetic organisms was not systematically recorded, but it may be mentioned that streptococci were frequently found in the spleen.

In calculating the day on which the disease began, we have taken the day on which symptoms were first complained of by the patient, or noticed by the parents, as the first day of the disease. Such symptoms have been various, viz., headache, drowsiness, sore throat, croupy cough, a feeling of suffocation, difficulty of swallowing, &c.

This method of fixing the date of onset is open to objection, but seems to us to be the only possible one of approximate accuracy. Cases of diphtheria admitted at general hospitals are almost always severe forms, often *in extremis* at the time of admission, and those at St. Bartholomew's are no exception to this rule. Therefore we have in them the best and soundest criterion regarding the value of the antitoxin treatment; for if a remedy is capable of curing otherwise hopeless cases, the value of such remedy must be assured. Assuming that the records of general hospitals give us the results of treatment in the severer cases, we have considered—

(I.) The fifty cases reported by Dr. Herringham.¹

(II.) Seventy-five cases which Dr. Sidney Martin has published of the results of the antitoxin treatment in diphtheria at the University College Hospital; ² in which Hospital the cases are presumably of a similar type.

(III.) The fifty-eight cases admitted into St. Bartholomew's since Dr. Herringham's report, the notes of which we have ourselves investigated.³

For the purpose of more general statistics, we have consulted the exhaustive report published this year (1896) by the Metropolitan Asylums Board.⁴

Passing now to an analytical consideration of the cases, it is necessary to arrange them in several groups. It is important, to begin with, to classify (*a*) according to age, and (*b*) according to the date on which the treatment was begun. For it is generally recognised that in children under five years of age the prognosis is more serious than in children over five, and also that the effect of the antitoxin depends to a great extent on the promptitude with which it has been administered. The earlier a case comes up for treatment the better the prognosis.

The cases have therefore been arranged, as far as possible, in three chief groups, viz. :—

(A.) The total number irrespective of age.

(B.) The number of cases of five years of age and upwards.

(C.) The number of cases under five years of age.

These groups have been further rearranged, where the notes

¹ St. Bartholomew's Hospital Reports, 1895. These cases extended over the period 8th July 1894 to 29th July 1895.

² British Medical Journal, 25th January 1896.

³ These cases are continued from Series I. up to 17th May 1896, which is the date of the last admission included in this paper.

⁴ Report of the Medical Superintendents upon the use of the antitoxic serum in the treatment of diphtheria in the Hospitals of the Board during the year 1895. This report we shall in future refer to as the "M.A.B.," or the "M.A.B. Report."

allowed, according to the day of the disease upon which the treatment was begun, as follows:—

(1.) The cases admitted up to and including the third day of the disease.

(2.) The cases admitted after the third day of the disease.

In the 58 cases of Series III. there were:—

(A.) *For all ages*:—58 cases with 23 deaths, giving a mortality of 39.6 per cent.

Admitted up to the Third Day.			Admitted after the Third Day.		
No. of Cases.	Deaths.	Mortality.	No. of Cases.	Deaths.	Mortality.
25	6	Per Cent. 24	33	17	Per Cent. 51.51

(B.) *Five years of age and upwards*:—11 cases with 4 deaths, giving a mortality of 36.3 per cent.

Admitted up to the Third Day.			Admitted after the Third Day.		
No. of Cases.	Deaths.	Mortality.	No. of Cases.	Deaths.	Mortality.
3	0	Per Cent. 0	8	4	Per Cent. 50

(C.) *Under five years of age*:—47 cases with 19 deaths, giving a mortality of 40.4 per cent.

Admitted up to the Third Day.			Admitted after the Third Day.		
No. of Cases.	Deaths.	Mortality.	No. of Cases.	Deaths.	Mortality.
22	6	Per Cent. 27.27	25	13	Per Cent. 52

Comparing these results with those recorded by Dr. Herringham in Series I., we find that there were:—

(A.) *For all ages*:—50 cases with 8 deaths, giving a mortality of 16 per cent.

Admitted up to the Third Day.			Admitted after the Third Day.		
No. of Cases.	Deaths.	Mortality.	No. of Cases.	Deaths.	Mortality.
27	3	Per Cent. 11.1	23	5	Per Cent. 21.73

(B.) *Five years of age and upwards*:—7 cases with no death, giving a recovery of 100 per cent.

(C.) *Under five years of age*:—43 cases with 8 deaths, giving a mortality of 18.6 per cent.

Admitted up to the Third Day.			Admitted after the Third Day.		
No. of Cases.	Deaths.	Mortality.	No. of Cases.	Deaths.	Mortality.
23	3	Per Cent. 13.04	20	5	Per Cent. 25

If we add the cases of Series III. to those of Series I., then we obtain the following figures:—

(A.) *For all ages*:—108 cases with 31 deaths, giving a mortality of 28.7 per cent.

Admitted up to the Third Day.			Admitted after the Third Day.		
No. of Cases.	Deaths.	Mortality.	No. of Cases.	Deaths.	Mortality.
52	9	Per Cent. 17.26	56	22	Per Cent. 39.28

(B.) *Five years of age and upwards*:—18 cases with 4 deaths, giving a mortality of 22.2 per cent.

Admitted up to the Third Day.			Admitted after the Third Day.		
No. of Cases.	Deaths.	Mortality.	No. of Cases.	Deaths.	Mortality.
7	0	Per Cent. 20	11	4	Per Cent. 36.36

(C.) *Under five years of age*:—90 cases with 27 deaths, giving a mortality of 30 per cent.

Admitted up to the Third Day.			Admitted after the Third Day.		
No. of Cases.	Deaths.	Mortality.	No. of Cases.	Deaths.	Mortality.
45	9	Per Cent. 20	45	18	Per Cent. 40

In the above tables the most noticeable feature is the influence which promptitude of treatment seems to have on the rate of mortality per cent. To this feature reference will be made at greater length in a later portion of this paper. This is still more emphasised by the Report of the American Pediatric Society, published in the *Medical Record* 1896, July 4. Three groups of cases have been analysed in that report, and they may be tabulated in the following manner:—

Admitted up to the Third Day.			Admitted after the Third Day.		
	Deaths.	Mortality.		Deaths.	Mortality.
Series I. 2449 Cases.	206	Per Cent. 8.4	Series I. 726 Cases.	229	Per Cent. 31.5
Series II. 569 Cases.	74	Per Cent. 13.0	Series II. 356 Cases.	91	Per Cent. 25.5
Series III. 1102 Cases.	23	Per Cent. 2.0	Series III. 366 Cases.	71	Per Cent. 19.4

In such statistics of cases allowances must be made for the errors which must always accompany any large collective investigation. Nevertheless, these figures prove that prompt treatment reduces the mortality in a most striking manner.

In order to estimate the value of the antitoxin treatment, the rate of mortality may be compared with that for six previous years (1888–1893) at St. Bartholomew's Hospital, which comparison is a necessary one to make; for although at

this Hospital the cases may justly be considered to be as nearly as possible of the same severity in each year, nevertheless the rate of mortality varies from year to year. Thus, on consulting this period (1888-1893), the following results have been obtained:—

(A.) *For all ages*:—533 cases with 271 deaths, giving a mortality at the rate of 50.8 per cent.

(B.) *For five years of age and upwards*:—233 cases with 65 deaths, giving a mortality at the rate of 27.8 per cent.

(C.) *Under five years of age*:—300 cases with 206 deaths, giving a mortality at the rate of 68.6 per cent.

Arranging these cases year by year, the following figures are obtained:—

(A.) *For all Ages.*

Year.	Cases.	Deaths.	Mortality.
			Per Cent.
1888	82	50	60.9
1889	78	45	57.6
1890	148	57	38.5
1891	67	36	53.7
1892	60	27	45.0
1893	98	56	57.1
For 6 years	533	271	50.8

(B.) *Five Years of Age and Upwards.*

Year.	Cases.	Deaths.	Mortality.
			Per Cent.
1888	42	17	40.4
1889	34	12	32.3
1890	78	14	17.9
1891	33	11	33.3
1892	24	6	25.0
1893	22	5	22.7
For 6 years	233	65	27.8

(C.) Under Five Years of Age.

Year.	Cases.	Deaths.	Mortality.
			Per Cent.
1888	40	33	82.5
1889	44	33	75.0
1890	70	43	61.4
1891	34	25	73.5
1892	36	21	58.3
1893	76	51	67.1
For 6 years	300	206	68.6

Unfortunately the statistical arrangement according to the day of the disease could not be made.

Dr. Sidney Martin has published a similar table for the University College Hospital for the four previous years (1891-1894), which shows:—

(a.) *For all ages*:—291 cases with 111 deaths, giving a mortality at the rate of 38.1 per cent., which is lower than the rate of mortality at St. Bartholomew's Hospital (1888-1893).

(b.) *Five years of age and upwards*:—127 cases with 26 deaths, giving a mortality at the rate of 20.4 per cent.

(c.) *Under five years of age*:—164 cases with 85 deaths, giving a mortality at the rate of 51.8 per cent.

Arranging Dr. S. Martin's cases year by year, the following figures are obtained:—

(A.) All Ages.

Year.	Cases.	Deaths.	Mortality.
			Per Cent.
1891	62	27	43.5
1892	60	20	33.3
1893	105	39	37.1
1894	64	25	39.0
For 4 years	291	111	38.1

(B.) *Five Years of Age and Upwards.*

Year.	Cases.	Deaths.	Mortality.
			Per Cent.
1891	23	5	21.7
1892	27	5	18.5
1893	47	9	19.1
1894	30	7	23.3
For 4 years	127	26	20.4

(C.) *Under Five Years of Age.*

Year.	Cases.	Deaths.	Mortality.
			Per Cent.
1891	39	22	56.4
1892	33	15	42.3
1893	58	30	51.7
1894	34	18	52.9
For 4 years	164	85	51.8

These figures compare with his antitoxin cases as follows:—

(a.) *For all ages*:—74 cases with 21 deaths, giving a mortality at the rate of 28.3 per cent.

(b.) *Five years of age and upwards*:—26 cases with 4 deaths, giving a mortality of 15.3 per cent.

(c.) *Under five years of age*:—48 cases with 17 deaths, giving a mortality at the rate of 35.4 per cent.

DR. SIDNEY MARTIN'S ANTITOXIN CASES.

(a.) *For all ages*:—74 cases with 21 deaths, giving a mortality of 28.3 per cent.:—

Admitted up to the Fourth Day.			Admitted after the Fourth Day.		
No. of Cases.	Deaths.	Mortality.	No. of Cases.	Deaths.	Mortality.
		Per Cent.			Per Cent.
46	8	17.3	28	13	46.4 ¹

¹ These cases are given according to the fourth day of disease, and the table only accounts for 74 of the total 75 (see *British Medical Journal*, January 25, 1895, p. 198, Column II. Table II.).

(b.) *Five years of age and upwards*:—26 cases with 4 deaths, giving a mortality of 15.3 per cent. :—

Admitted up to the Fourth Day.			Admitted after the Fourth Day.		
No. of Cases.	Deaths.	Mortality.	No. of Cases.	Deaths.	Mortality.
21	2	Per Cent. 9.5	5	2	Per Cent. 40

(c.) *Under five years of age*:—48 cases with 17 deaths, giving a mortality of 35.4 per cent. :—

Admitted up to the Fourth Day.			Admitted after the Fourth Day.		
No. of Cases.	Deaths.	Mortality.	No. of Cases.	Deaths.	Mortality.
25	6	Per Cent. 24	23	11	Per Cent. 47.8

These figures represent (1.) the average percentage rate of mortality in diphtheria previous to the treatment with anti-toxin, and (2.) the percentage rate of mortality, since its introduction, among the more serious cases, *i.e.*, those cases which are so urgent as to preclude any possibility or justifiable reason for delay, such as is necessarily occasioned in transferring them to the fever hospitals.

Let us now consider the change in the mortality rate per cent. at St. Bartholomew's and University College Hospital.

In comparing the rates of mortality before and after the new treatment, we must recognise that it is hardly fair to compare the results of 1896 with those of any one previous year or any arbitrary period of years; for Table A. on page 345 shows how much the rate of mortality may vary from year to year. In fact, as far as our own Hospital is concerned, in 1890, without antitoxin, it was lower than in the antitoxin year 1895 to 1896. We cannot, therefore, say that, taking the average mortality of the period 1888 to 1893 as representing the mortality before the days of antitoxin, that the mortality of diphtheria cases of all ages has dropped from 50.8 per cent. to 28.7 per cent. But we think that a comparison of the mortality of the cases under five years of age before and after the antitoxin days is less fallacious, because there has been considerably less variation in the rate of mortality in the period 1888 to 1893 amongst these

cases. We may say, then, that considering the cases under five years, the mortality has dropped from 68.6 per cent. to 40.4 per cent. = 28.2 difference, if we compare the period 1895 to 1896 with the period 1888 to 1893, or from 68.6 per cent. to 30 per cent. = 38.6 difference, if we compare the period of 1894 to 1896 with that of 1888 to 1893. If, however, objection be made to this method of comparison, we may emphatically maintain that in the period 1894 to 1896 the mortality amongst the cases under five years of age at St. Bartholomew's Hospital was considerably lower than in any year of the period 1888 to 1893.

(a.) *At St. Bartholomew's.*—Cases under 5 years: the decrease from 68.6 per cent. (1888 to 1893) to 30.0 per cent. means an improvement in the death-rate, as compared with the average death-rate formerly, of 56.26 per cent. for cases under 5 years.

(b.) *At University College Hospital.*—Cases under 5 years of age: the decrease from 51.8 per cent. to 35.4 per cent. (= 16.4 difference) means an improvement in the death-rate, as compared with the average death-rate formerly (1891 to 1894), of 31.66 per cent. for cases under 5 years.

For the more mixed cases we must examine the M.A.B. Report, from which we obtain the following figures:—

I. Previous to the Introduction of the Antitoxin, 1894.

(A.) *For all ages:*—3012 cases with 902 deaths, giving a mortality at the rate of 29.6 per cent.:—

Admitted up to the Third Day.			Admitted after the Third Day.		
No. of Cases.	Deaths.	Mortality.	No. of Cases.	Deaths.	Mortality.
1324	368	Per Cent. 27.84	1718	534	Per Cent. 31.08

(B.) *Five years of age and upwards:*—1871 cases with 346 deaths, giving a mortality at the rate of 18.4 per cent.:—

Admitted up to the Third Day.			Admitted after the Third Day.		
No. of Cases.	Deaths.	Mortality.	No. of Cases.	Deaths.	Mortality.
807	128	Per Cent. 15.8	1064	218	Per Cent. 20.4

(C.) *Under five years of age*:—1171 cases with 556 deaths, giving a mortality at the rate of 47.4 per cent.:—

Admitted up to the Third Day.			Admitted after the Third Day.		
No. of Cases.	Deaths.	Mortality.	No. of Cases.	Deaths.	Mortality.
517	240	Per Cent. 46.42	654	316	Per Cent. 48.31

II. *Under the Antitoxin Treatment in 1895.*

(A.) *For all ages*:—2182 cases with 615 deaths, giving a mortality at the rate of 28.1 per cent.:—

Admitted up to the Third Day.			Admitted after the Third Day.		
No. of Cases.	Deaths.	Mortality.	No. of Cases.	Deaths.	Mortality.
938	182	Per Cent. 19.34	1244	433	Per Cent. 34.80

(B.) *For five years of age and upwards*:—1169 cases with 256 deaths, giving a mortality at the rate of 20.1 per cent.:—

Admitted up to the Third Day.			Admitted after the Third Day.		
No. of Cases.	Deaths.	Mortality.	No. of Cases.	Deaths.	Mortality.
503	68	Per Cent. 13.5	666	168	Per Cent. 25.2

(C.) *Under five years of age*:—1013 cases with 379 deaths, giving a mortality at the rate of 37.4 per cent.:—

Admitted up to the Third Day.			Admitted after the Third Day.		
No. of Cases.	Deaths.	Mortality.	No. of Cases.	Deaths.	Mortality.
435	114	Per Cent. 26.2	578	265	Per Cent. 45.86

The total decrease in the mortality rate per cent. in these hospitals is therefore:—

(1.) *For all ages*: from 29.6 per cent. to 28.1 per cent. = 1.5 difference.

(2.) *Under 5 years of age*: from 47.4 to 37.4 per cent. = 10 difference.

This means an improvement in the death-rate, as compared with the year previous to the use of the antitoxin, of 5.06 per cent. for all ages, and of 21.09 per cent. for cases under five years.

We must, however, be cautious in our deductions, because we are simply comparing one year with another year,—an unsafe procedure. It will disarm all criticism if we allow that, taking all ages into consideration, it is not quite easy to come to a conclusion, but if we consider the cases under five years of age there is a noticeable drop from 1894 to 1895. But what is most striking is this, that whereas in 1894 amongst the cases under five years of age there is no appreciable difference in the mortality of cases treated promptly and those treated less promptly, in 1895 the difference is truly remarkable. And this is borne out by a comparison of the mortality for all ages in 1894 with that in 1895, for we find in 1894 a comparatively small difference in the mortality according to the promptitude of treatment, but in 1895 a considerable difference in favour of the more promptly treated cases. It would seem, therefore, that the antitoxin has diminished the mortality in the cases under five years of age where treatment has been promptly applied, but that it has not materially affected the mortality of cases under five years where the treatment has been delayed.

The above tables, therefore, show, both for the cases treated at two general hospitals and for those treated at the hospitals of the M.A.B., that the mortality per cent. has evidently decreased in the cases under five years of age; that is to say, the most marked improvement has occurred at the age when the disease is known to be most dangerous to life.

We must now proceed to examine whether this statement applies also to the most serious forms, not only in respect to age, but also according to the situation of the disease. It is usually assumed that the laryngeal form of the disease is more serious than the pharyngeal or faucial form. In a general way it may be said that laryngeal cases are more dangerous to life than purely pharyngeal ones, but, as we shall show, it is not strictly true that the former cases are *per se* more serious.

Their severity depends on the smallness of the larynx in young children and the extension of the bacilli into the lungs. Before, however, discussing this matter, let us see whether the antitoxin has bettered the prognosis in the laryngeal cases.

To investigate this matter thoroughly, we ought to consider the results according to the recognised clinical forms of diphtheria, viz., laryngeal, pharyngeal, nasal, and other forms of diphtheria, before and since the use of antitoxin. Unfortunately this cannot be done in all cases, for the notes in previous years on this point are not as complete as might be wished.

We shall, therefore, divide all the cases at St. Bartholomew's and the M.A.B. hospitals into two groups:—

(1.) Those in which tracheotomy was performed.

(2.) Those in which tracheotomy was not required.¹

Before giving these figures, we shall first consider a table which Dr. S. Martin has published of the results of the antitoxin in the different clinical forms of diphtheria (*Brit. Med. Jour.*, Jan. 25, 1896, p. 198).

(a.) *Pharyngeal cases.*—(i.) For the four previous years (1891-94 inclusive), 161 cases with 27 deaths = mortality, 16.77 per cent.; (ii.) *in the antitoxin cases* (1895), 45 cases with 11 deaths = mortality, 24.4 per cent.

(b.) *Laryngeal cases.*—(i.) For the four previous years (1891-1894 inclusive), 130 cases with 84 deaths = mortality, 64.61 per cent.; (ii.) *in the antitoxin cases* (1895), 30 cases with 10 deaths = mortality, 33.33 per cent.

At one general hospital, therefore, according to the above table, the following results have been obtained:—

(a.) *Pharyngeal Diphtheria.*—Under the antitoxin treatment in this variety, as compared with the average of the four years previous to its use, the rate of mortality has *increased* 44.9 per cent.

(b.) *Laryngeal Diphtheria.*—Under the antitoxin treatment in this variety, as compared with the average of the four years previous to its use, the rate of mortality has *decreased* 48.41 per cent. Dr. Martin has classified all tracheotomy cases under the heading "laryngeal," and all those in which tracheotomy was not performed under the heading "pharyngeal," a confusion which must lead to error.

These figures, although they add further support to the

¹ It must, however, be remembered that from the fact whether a tracheotomy has been performed or not, we cannot possibly judge whether a case was laryngeal or pharyngeal, for tracheotomy may have been necessary in purely pharyngeal cases, or in mixed pharyngeal and laryngeal cases.

great improvement in the dangerous cases, curiously enough appear to point to a markedly increased mortality in the cases generally thought to be less dangerous. We do not say *severe* cases, but designedly use the attribute *dangerous*, because we hope to show that, if we single out all the severe or otherwise dangerous cases, *i.e.*, the cases admitted into general hospitals, apart from the physical danger to life which the presence of a membrane in a small larynx implies, the pharyngeal cases are sometimes perhaps actually more toxic, or *per se* severer, than the laryngeal ones. If, as some observers are inclined to do, we considered the laryngeal cases as always necessarily severer than the pharyngeal ones, then from Dr. S. Martin's tables it would appear as if in this instance, under the antitoxin treatment, the mortality had increased in the less severe cases, and decreased in the more severe cases.

This is a result which we cannot accept, and therefore, before endorsing such a verdict as a true representation of fact, we must see—(1) whether, from a further analysis of other cases, as tracheotomy was or was not required, the same result is obtained; (2) whether it is really true that laryngeal cases are always severer *per se* than pharyngeal cases.

From the Medical Registrar's tables in our Hospital Reports, we find that at St. Bartholomew's Hospital for five previous years (1888-92 inclusive), tracheotomy was performed in 42.52 per cent. of all cases of diphtheria, and the rate of mortality in these tracheotomy cases was 76.75 per cent., as shown by the accompanying table.

*Cases of Diphtheria in which Tracheotomy was required at
St. Bartholomew's Hospital.*

Year.	Number of Cases.	Tracheotomies.	Percentage of Tracheotomies.	Deaths in Tracheotomies.	Mortality of Tracheotomies.
			Per Cent.		Per Cent.
1888	82	27	32.9	26	96.29
1889	78	47	60.2	37	78.29
1890	148	52	35.1	38	73.07
1891	67	30	44.7	22	73.33
1892	60	29	48.3	19	65.51
Total for } 5 years }	435	185	42.52	142	76.75

The cases in which tracheotomy was not performed for the same period were 250 in number with 73 deaths = mortality of

29.2 per cent., as shown in the following table. We cannot say that in all these cases tracheotomy was not necessary, because opinion differs on this point and is often against a tracheotomy, and also because occasionally a child dies before the required operation.

Non-Tracheotomy Cases.

Year.	No. of Cases.	Deaths.	Mortality.
			Per Cent.
1888	55	24	43.6
1889	31	8	25.8
1890	96	19	19.7
1891	37	14	37.8
1892	31	8	25.8
For 5 years	250	73	29.2

It will be seen that the mortality from year to year varies much more in this group of cases than it does in the group of tracheotomy cases. We find the same variability in Dr. Martin's table of pharyngeal cases.

During the antitoxin period, tracheotomy was performed more frequently at St. Bartholomew's Hospital, but with a much decreased mortality, as the accompanying table shows:—

*Tracheotomy Cases under Antitoxin Treatment
(St. Bartholomew's Hospital).*

Cases.	Total Cases.	Tracheo- tomies.	Percentage of Tracheo- tomies.	Deaths in Tracheo- tomies.	Mortality of Tracheo- tomies.
			Per Cent.		Per Cent.
Series I.	50	32	64.00	6	18.75
Series III.	58	36	62.05	19	52.77
Total St. Bart. Hosp. 1894-1896	108	68	62.96	25	36.76

The cases in which tracheotomy was not performed in the antitoxin period were, combining Series I. and III., 40 cases with 6 deaths = mortality, 15 per cent.

*Non-Tracheotomy Cases under Antitoxin Treatment
(St. Bartholomew's Hospital).*

Cases.	No. of Cases.	Deaths.	Mortality.
Series I.	18	2	Per Cent. 11.1
Series III.	22	4	18.2
Total St. Bart. Hosp. 1894-96	40	6	15

These figures show, (1) in tracheotomy cases,¹ that the mortality has fallen from 76.75 per cent. to 36.76 per cent. = a difference of 39.99, or an improvement, as compared with the previous rate of mortality, of 52.10 per cent., and that, in spite of the incidence having risen from 45.52 per cent. to 62.96 per cent. = increased 32.46 per cent.; (2) in non-tracheotomy cases, that the mortality has fallen from 29.2 per cent. to 15 per cent. = a difference of 14.2, or an improvement, as compared with the previous rate of mortality, of 48.63 per cent.

It may not be quite fair to compare a period of five years with one of two years, but at any rate we see that the highest mortality amongst tracheotomy cases is lower than the lowest mortality amongst similar cases previous to the introduction of the antitoxin treatment, and this is also true if we compare the non-tracheotomy cases.

At the Metropolitan Asylums Board Hospitals we find that in 1894 (Table XXXIX. p. 18), out of 3042 cases treated during the year preceding the antitoxin treatment, the rate of mortality for tracheotomy cases was 70.4 per cent. out of 261 tracheotomies, thus giving a tracheotomy incidence for all cases of 8.57 per cent.

By deduction we find that of 2781 cases in which tracheotomy was not required, 718 died, which yields a mortality at the rate of 25.81 per cent.

During the antitoxin year at these hospitals, we find (Table XXXI. p. 15) that out of a total number of 2182 cases, tracheotomy was performed 225 times, this being a tracheotomy incidence of 10.31 per cent. for all cases, and the mortality in these tracheotomy cases was at the rate of 50.22 per cent.

¹ In the tracheotomy cases at St. Bartholomew's Hospital we have combined Series I. and Series III. (see p. 343), to give the statistical results a higher value.

Therefore, in the cases not requiring tracheotomy, there were under the antitoxin treatment 1957 cases with 502 deaths = a mortality at the rate of 25.65 per cent.

These figures show, (1) in the tracheotomy cases, a large decrease in the rate of mortality (= 28.66 per cent.), viz., from 70.4 per cent. to 50.22 per cent., although here again the incidence of tracheotomy has increased (= 20.38 per cent.) from 8.57 per cent. to 10.31 per cent.; (2) in the non-tracheotomy cases, so small a decrease (= .61 per cent.) in the rate of mortality as to be practically negligible.

The above tables of the M.A.B. show clearly that under the antitoxin treatment the prognosis in the laryngeal forms of diphtheria, which are mechanically and otherwise dangerous to life, has greatly improved; but they also show that in cases where the elements of laryngeal obstruction play no part, the rate of mortality has practically remained unchanged, and therefore, from a comparison of laryngeal and pharyngeal, or tracheotomy and non-tracheotomy cases, as generally adopted, but little can be learned regarding the true value of the antitoxin. The mortality among the non-tracheotomy cases before the antitoxin days, is far too inconstant a factor to form a *fundamentum comparationis*; we must therefore, from this point of view, select the tracheotomy cases before and after the introduction of antitoxin, because the mortality among these cases before the antitoxin days has been less variable and consistently high. Applying this method of comparison, we are bound to acknowledge that the antitoxin has achieved something tangible, even in its infancy. From the statistics of the report of the American Pediatric Society we learn that of 1256 cases of laryngeal diphtheria, 672 were not tracheotomised, and of these 128 died = 19 per cent. The mortality amongst the intubated cases amounted to 25.9 per cent.; the mortality amongst the tracheotomies to 37.4 per cent.

That the incidence of tracheotomy has increased is partly due to chance, the disease during the antitoxin period having apparently more often assumed the laryngeal type than formerly, for the increased incidence existed not only in two general hospitals, which might be a mere coincidence, but also in those special hospitals where all varieties of the disease come under treatment; and at St. Bartholomew's it was almost as high in 1889—a year with a high mortality, as shown by table on p. 353, but it is partly due to the hopeful confidence which under the antitoxin treatment can now be placed in tracheotomy.

These figures of the M.A.B. and of our own Hospital do not agree with Dr. S. Martin's, according to which the mortality

in the mechanically and generally less dangerous forms, *i.e.*, the non-tracheotomy cases, has increased. They show that while in the forms complicated by laryngeal obstruction the mortality has materially decreased, in the forms without laryngeal obstruction, classified as non-tracheotomy cases, the mortality has also fallen, (1) in one case (S.B.H.), practically in a similar ratio; (2) in the other case (M.A.B.), so slightly as to be practically unchanged. There has, however, been no increase in the mortality in either case.

If asked to advance an explanation of the paradoxical results in pharyngeal diphtheria which Dr. S. Martin's figures show, we find that of the fatal so-called pharyngeal cases, "one had shown symptoms of palsy and dyspnoëic attacks," while "two were hæmorrhagic diphtheria;" and again later, in his remarks on the Duration of the Membrane, he states that "four" of the pharyngeal cases "also had laryngeal stridor, two of which had recession and croupy cough." We ask, can all those cases be included under the pharyngeal ones? In fact, it amounts to this, that although all tracheotomy cases are dangerous cases, it is not correct to imagine that all non-tracheotomy cases are mild cases, or necessarily milder than the former. We must avoid such a conclusion, especially in general hospitals, where, as a rule, only severe or dangerous cases are admitted, so that there we are dealing merely with degrees of severity or danger. This is also well shown by the notes on the fatal cases on pages 337 to 340, for of four cases in which tracheotomy was not performed, three had membranes in the larynx and trachea.

It has been shown that, as far as the class of cases admitted into general hospitals is concerned, the relative improvement under the antitoxin treatment has been most striking among the tracheotomy cases, but that in cases in which tracheotomy has not been performed the difference in mortality has been small compared with that existing under the old methods of treatment. This may seem remarkable, for hitherto, at all events, the tracheotomy cases have generally been considered to be the most severe forms of diphtheria, with the exception perhaps of the acute nasal and hæmorrhagic cases.

Now, we should naturally expect that milder forms of any disease, under any particular kind of treatment, would relatively and proportionately benefit more than the severer forms. But, on the view that laryngeal diphtheria is a more severe form of the disease than pharyngeal diphtheria, this assumption has received no practical support. The considerable reduction in the mortality in laryngeal, but the only slight reduction in

pharyngeal cases (except in a series given by Dr. S. Martin, with an apparently increased mortality in pharyngeal cases), is interesting as well as perplexing, and invites a more searching inquiry and closer statistical and mathematical reasoning.

Undoubtedly there is some very close and peculiar reaction between the membrane present and the antitoxin: all observers agree in saying that with the antitoxin the extension of the membrane is arrested, and the membrane broken up or loosened, and so either coughed up or made to vanish.

But bearing in mind the fact that the presence of a membrane does not of itself constitute the disease, albeit the clinical definition requires this *διφθερα*, an attempt may now be made to discover an explanation for the difference in results in the two forms of the disease irrespective of the presence of the membrane. If we consider the toxic nature of the disease, and disregard the element of risk introduced by laryngeal obstruction, it seems reasonable to ask whether the greater mortality in laryngeal diphtheria is not due rather to a *mechanical* cause—an obstructing membrane—than to the more serious *constitutional* effects *per se* of the toxins. In other words, apart from the presence of the membrane in the larynx or trachea, it may be believed that pharyngeal diphtheria often may be as severe a form as, if not severer than, laryngeal diphtheria.

That this is probably true for some cases the following lines will attempt to show, and if they do, then we shall be able to suggest at least one reason why the pharyngeal cases have benefited less under the antitoxin treatment, and how in the future an improved prognosis in these cases also may be attained.

In both forms the membrane present disappears under antitoxin, but the pharyngeal membrane does not occasion so much mechanical obstruction as does the laryngeal or tracheal, especially in the case of the small trachea of the young, in whom the worst forms of laryngeal diphtheria occur, so that many laryngeal cases no doubt die before the serum has been able to act on the obstruction. This leads to the inquiry whether, after the removal of the laryngeal obstruction, there is any reason to believe that severe laryngeal cases are more dangerous than severe pharyngeal ones, or whether, mechanical obstruction being absent, there might not be something peculiar to both which would tend to render them equally dangerous.

The following anatomical conditions and their effect upon the absorption of toxins must be considered. The pharynx is pro-

vided with a far larger blood and lymphatic supply than the larynx, by which greater absorption of the morbid products is obtained in the pharyngeal cases. Hence, in the absence of obstruction to respiration, a serious case of pharyngeal diphtheria such as finds admission into a general hospital is, on *à priori* grounds, more likely to be attended by severe toxic effects than the laryngeal form.

When, however, the disease (bacilli or membrane) has spread into the smaller bronchioles and alveoli of the lungs, a still more extensive path for the absorption of toxins (*viz.*, the pulmonary circulation) is established, and this accounts for the extreme danger to life of broncho-pneumonia as a diphtheritic complication. The pulmonary infection may, however, occur with either form, although of course it occurs more frequently when the larynx and trachea are involved.

In support of the view that toxic absorption takes place readily from the pharynx, may be mentioned those cases which are from time to time met with, in which the appearances had been uncertain clinically, and follicular tonsillitis had been diagnosed, but later the development of paralysis has pointed to the real nature of the disease. And Dr. Gee writes in Clifford Albutt's "System of Medicine," vol. i. p. 739: "Diphtheria, which is mainly or altogether laryngeal, is seldom followed by palsy; anything like extensive paralysis after diphtheritic croup must be most uncommon." Severe laryngeal cases are, however, rarely "unmixed;" there is generally an extension of the process into the pharynx, trachea, or lungs. The glands of the neck are more considerably swollen in severe pharyngeal forms than in laryngeal cases, and then diphtheria bacilli are almost always found in them. The danger in diphtheria, apart from mechanical obstruction, whether laryngeal or pharyngeal, in our opinion depends mainly on the escape of the diphtheria bacilli into the blood, lungs, or tissues. This may occur with either form, although the lungs are more jeopardised in laryngeal infection.

The mortality among the non-tracheotomised cases has always been lower than in the tracheotomy cases, both before and since the use of antitoxin, and consequently the decrease noticed under the new treatment in these tracheotomy cases is only at first sight much greater. But on closer consideration it will be found, that whereas at St. Bartholomew's Hospital for the years previous to the antitoxin treatment the mortality in tracheotomy cases was 76.75 per cent., and now (in our 108 cases) it is 36.76 per cent., the same figures for the non-tracheotomy cases for the same periods are 29.2 per cent. and

15 per cent., *i.e.*, the *percentage decrease* for these two classes of cases compared with the former rates of mortality is—

- | | |
|--------------------------------------|-----------------|
| (1.) Tracheotomy cases | 52.23 per cent. |
| (2.) Non-tracheotomy cases | 48.63 „ |

showing that on this system of classification the improvement in both forms is very nearly the same.

Dr. S. Martin's cases, on the other hand, if we allow his own figures to stand, show that, as compared with the four previous years, the mortality—

- | | |
|--|-----------------|
| (1.) In laryngeal (tracheotomy) cases has
<i>decreased</i> | 48.41 per cent. |
| (2.) In pharyngeal (non-tracheotomy) cases
has <i>increased</i> | 44.9 „ |

The M.A.B., on the other hand, show that the mortality—

- | | |
|---|-----------------|
| (1.) In tracheotomy cases has decreased | 28.66 per cent. |
| (2.) In non-tracheotomy cases has decreased
only | 00.61 „ |

The conclusion we must come to is that this method of classifying cases, *viz.*, into tracheotomy and non-tracheotomy cases, is somewhat misleading.

It is equally erroneous to consider a case as purely pharyngeal when tracheotomy was not required, since without laryngoscopic examination we cannot be certain that there is in all such cases no membrane in the larynx or trachea, and such examination is impossible in almost all cases of children under five.

This fallacious method of classifying cases of diphtheria has arisen from over-attention to the clinical evidence of the disease (the presence of the membrane), and under-estimation of the teaching of the pathology of the disease, the distribution of the bacillus, and the effects of its products, the toxins. In our opinion one of the greatest dangers is the extension of the diphtheria bacillus into the lungs.

The increased incidence of tracheotomy seems to show that the late epidemic has been of a dangerous type of disease. Those series of cases which show only a slightly decreased mortality in the non-tracheotomy cases probably show the same.

Dr. Welch says in his monograph that laboratory experiments show that “the serum arrests the spread of the local process and abates the symptoms of general toxæmia.” Most observers, it may fairly be assumed, consider now that this statement applies equally to the human being—certainly the first part of

it does; so that, when equal attention, at all events, is paid to the constitutional element of the disease as to the physical element in the disease, and the antitoxin is used accordingly, we may hope for a still greater recovery; for we have learnt by experience that (a.) the greatest benefit has occurred in those cases in which the treatment is begun early (before the third day); (b.) that the largest amounts of the antitoxin have been required in the severe cases whether tracheotomy was necessary or not, and in those in which, moreover, there has been broncho-pneumonia due to the bacillus diphtheriæ. If antitoxin is early and adequately administered in purely laryngeal diphtheria, whether tracheotomy is performed or not, the process remains local, there is no extension into the lungs, and the dangers of toxæmia are considerably lessened or even removed. The fatality of laryngeal cases, apart from the mechanical presence of the membrane, depends on the spread of the diphtheria bacilli, more especially into the lungs.

There is a consensus of opinion that the amount of antitoxin to be given shall be regulated by the severity and extent of the disease. Therefore, if the so-called pharyngeal forms are, as we believe, frequently *constitutionally* as severe as, and occasionally even severer than the so-called laryngeal ones, the slight improvement in the rate of mortality noted in the former, in spite of the antitoxin, is easy of explanation—it is due to insufficient or delayed antidotal treatment.

This lack of therapeutic energy, which arises in part from ignorance of pathological facts, would explain—

(1.) The disproportionately small improvement in pharyngeal cases.

(2.) The great fatality in cases complicated by broncho-pneumonia, which is always diphtheritic in nature.

(3.) The great fatality in all forms when the treatment is begun later than the third day.

(4.) The fatality of acute nasal diphtheria, which situation offers a very large area for the absorption of the toxic products of the disease, and favours pulmonary infection.

Undoubtedly at our Hospital the antitoxin has until recently generally been administered in too small quantities. A recognition of the teaching of pathology would admit of a much improved prognosis, if antitoxin were administered in much larger doses (*i.e.*, judging the doses by units and not by cubic centimetres) than has been done hitherto.

Clinical and pathological experience emphasises still more strongly than any other factor the necessity of early energetic

administration of the antitoxin without waiting for bacteriological confirmation of the clinical diagnosis or suspicion.

In connection with this last point it is well to bear in mind that a single negative bacteriological examination is of no value. Dr. S. Martin insists very strongly on this when he says that the presence of nerve degeneration is "the final and most accurate test of the diphtheria bacillus;"¹ and he has, moreover, drawn attention to the importance of the absence of the knee-jerk as the real test in the cases he published, and to which we have frequently referred²; but surely no one would advocate waiting until this has appeared before beginning the treatment.

We have not been able to investigate the relative severity of laryngeal and pharyngeal diphtheria more fully, and merely advance the views expressed above as worthy of some consideration. We should mention, however, that although in the experience of most observers post-diphtheritic paralysis most often follows pharyngeal diphtheria, this is at first sight contrary to our observations on the cases treated with antitoxin included in our series of 58, of which out of ten complicated by paralysis seven had previously been tracheotomised. This apparent contradiction is not merely due to a statistical error, our numbers being too small, but finds its explanation in the fact that a tracheotomised case is not necessarily purely laryngeal, the lungs and pharynx being frequently involved.

The incidence of hæmorrhagic diphtheria under the antitoxin treatment, as is well known, has been almost exactly the same as in former years, and, as heretofore, they have most frequently been pharyngeal or mixed cases: the antitoxin does not save them, though perhaps in a few cases death has been somewhat delayed.

It is significant that Dr. Gee writes (*op. cit.*, p. 748) of the laryngeal cases, that symptoms due to poisoning of the blood do not occur, and that death is due (p. 749) to one of two causes—either to laryngeal or pulmonary dyspnoea. This requires modification, for we know now that in fatal laryngeal cases the lungs are always full of diphtheria bacilli, so that the dyspnoea is due to pulmonary infection.

These last statements add much weight we think to the view that the apparently greater danger to life of the laryngeal form depends chiefly on the mechanical factor and on the greater liability to pulmonary infection—the presence of the membrane obstructing the tube and favouring broncho-pneumonia by aspiration—especially as it is in the young (under five years of

¹ Discussion at British Medical Association, 1895, reported in *British Medical Journal*, August 24, 1895, p. 513 (*q. v.*).

² *British Medical Journal*, January 25, 1896.

age) that the severest forms of laryngeal diphtheria are seen. Remove the membrane radically, which can only be done by early administration of antitoxin, generally with a tracheotomy in addition, and then it is questionable whether we are justified in regarding laryngeal cases as constitutionally more severe than grave pharyngeal ones, but it must also be remembered that if combined with antitoxin a tracheotomy saves life, it opens up fresh paths and opportunities for absorption of toxin.

Everything points to the extreme importance of proceeding no less energetically in the cases diagnosed as pharyngeal than in the laryngeal cases, and in a general hospital, where only severe cases are admitted, we would say often more energetically in the pharyngeal even than in the laryngeal cases, and without any delay whatsoever.

It is necessary that in all post-mortem examinations a careful bacteriological analysis be made of the spleen and heart's blood, for, as the records of Wright and Stokes and of our own Hospital show, in many cases there is a pyococcus septicæmia or extensive diphtheritic broncho-pneumonia. Death from pyococcus septicæmia cannot be prevented by a diphtheria antitoxin, but may occur even when the diphtheria has already been conquered. The two hæmorrhagic cases of Dr. S. Martin were probably septicæmic, which no amount of antitoxin could have cured.

From our figures we may arrive at the conclusion that antitoxin has been especially beneficial in tracheotomy cases, because, those being apparently more alarming, the antitoxin has been pushed more energetically, so that the process has remained local, and so long as it is purely laryngeal the chances of toxæmia are comparatively slight. The antitoxin does not prevent the necessity of tracheotomy, but this may be due to the fact that the laryngeal cases are only seen first when the disease has been in progress for some time. If this be the true explanation, then we may reasonably look towards antitoxin as a preventive of tracheotomy, provided the treatment can be begun sufficiently early and energetically in the disease.

This suggests the following inquiry into the incidence of tracheotomy and its results in cases treated with the antitoxin, according to the day of the disease on which the treatment is first begun. On this point we find, that out of a total number of 36 tracheotomies in the 58 cases (Series III.), there were 19 deaths = mortality of 52.77 per cent. Of these there were—

(a.) Admitted up to the third day of the disease, 15 cases with 6 deaths = mortality of 40.0 per cent.

(b.) Admitted after the third day of the disease, 21 cases with

13 deaths = mortality of 61.9 per cent. That is to say, for the 58 cases (see page 341):—

(A.) *Cases admitted up to the Third Day (25).*

No. of Cases.	Tracheotomies.	Percentage of Tracheotomies.	Deaths in Tracheotomies.	Mortality in Tracheotomies.
25	15	Per Cent. 60	6	Per Cent. 40.0

(B.) *Cases admitted after Third Day (33).*

No. of Cases.	Tracheotomies.	Percentage of Tracheotomies.	Deaths in Tracheotomies.	Mortality in Tracheotomies.
33	21	Per Cent. 63.63	13	Per Cent. 61.9

This shows that although the incidence of tracheotomy is only slightly less in cases admitted up to the third day, yet the mortality is largely decreased in these, the difference being from 61.9 per cent. to 40.0 per cent., or a death-rate of 35.37 per cent. better for those admitted up to the third day than those admitted after the third day, both being subjected to tracheotomy.

Cases in which Tracheotomy was not required.

(a.) *All ages*:—22 cases with 4 deaths, giving a mortality of 18.18 per cent.:—

Admitted up to the Third Day.			Admitted after the Third Day.		
No. of Cases.	Deaths.	Mortality.	No. of Cases.	Deaths.	Mortality.
10	0	Per Cent. 0	12	4	Per Cent. 33.3

(b.) *Under five years of age*:—14 cases with 2 deaths, giving a mortality of 14.28 per cent.:—

Admitted up to the Third Day.			Admitted after the Third Day.		
No. of Cases.	Deaths.	Mortality.	No. of Cases.	Deaths.	Mortality.
7	0	Per Cent. 0	7	2	Per Cent. 28.57

This table illustrates extremely well the good effect of antitoxin applied early, so as to exclude an extension of the process into the lungs and other parts.

We regret that their Report does not allow us to give a table on the same lines for the M.A.B. cases.

The date of the disease on which treatment is begun is as yet, therefore, perhaps no preventive of tracheotomy, *i.e.*, the antitoxin does not, in its present form and mode of administration, prevent tracheotomy, but when the treatment has been begun early in the disease, tracheotomy is much more successful, and less frequently followed by death. If performed at all, tracheotomy should be performed early, as a preventive measure, and should not be delayed till the symptoms are serious. It is only in this way that an extension of the infective process into the lungs can be avoided or counteracted.

The next point to inquire into is whether, other things being equal, the prognosis varies with the amount of antitoxin given. A table is appended of the volumetric dosage in the cases of Series III. (p. 341), for (*a*) all cases; (*b*) tracheotomy cases; (*c*) non-tracheotomy cases.

(A.) All Cases.

	Total Amount given for all Cases.	Total No. of Doses for all Cases.	Average Dose for each Injection.	Average No. of Doses per Case.	Average Total Amount per Case.
	c.c.		c.c.		c.c.
1. Total . . . 58	2226.0	313	7.11	5.39	38.37
2. Recoveries . 35	1143.5	161	7.10	4.6	32.67
3. Fatal cases . 23	1082.5	152	7.12	6.60	47.06

(B.) Tracheotomy Cases.

1. Total . . . 36	1738.5	251	6.92	6.97	48.29
2. Recoveries . 17	796.0	115	6.92	6.76	46.82
3. Fatal cases . 19	942.5	136	6.93	7.15	49.60

(C.) Non-Tracheotomy Cases.

1. Total . . . 22	487.5	62	7.86	2.81	22.15
2. Recoveries . 18	347.5	46	7.55	2.55	19.30
3. Fatal cases . 4	140.0	16	8.75	4.00	35.00

It must, however, be remembered that the volumetric measure is in no sense a measure of the value and strength of the antitoxin. The latter, so far as our experience reaches, can be judged only by the number of immunising units it contains. No antitoxin should be used unless its strength in units (Behring and Ehrlich or Roux) is given, and instead of recording the number of cubic centimetres injected, it is necessary to state the number of units. It is not always easy to obtain the latter number, and apparently we cannot always trust the figures printed upon the bottle, as was clearly shown by the Report of the Lancet Commission. We may, however, trust some of the antitoxins sent out from Germany, notably Behring's (Höchst) or Schering's (Aronson's); and therefore, until we are in possession of an accessible English carefully standardised antitoxin, we feel obliged, however reluctantly, to recommend that the article which has been made and tested in Germany should be used. According to Dr. S. Martin, less than 4000 units should never be injected.

Unfortunately, we are not in a position to calculate accurately the number of units which have been given in those cases treated at this Hospital which forms the basis of this inquiry. Dr. Martin advises to give all we want to give in one dose. This, we think, is too dogmatic. We should say, give *never* less than 1000 units, but go on giving them till you feel sure as to the patient's condition.

The above tables show that the most energetic volumetric administration was made in the more serious cases, both as regards the average number of doses per case and the average total amount per case.

In the less severe cases, both the average number of doses and the average total amount were much less.

The time of administering the first injection has in almost all cases been as soon as possible after admission of the patient, *i.e.*, within four hours after admission: in all but two cases, in which the first dose was given eight and twelve hours respectively after admission. Both of these were under two years of age, and died.

The largest single dose in any case has been 20 c.c., which amount was given in two cases, of which one recovered (total amount given being 43 c.c.), the other died (total amount given being 75 c.c.).

The larger amounts given in the first injection were in most cases in those which recovered, but this was by no means the rule, for cases have died in which larger doses were at first injected, but subsequently the administration has been either

discontinued, or it has been repeated in much smaller and less frequent doses.

The table in the Metropolitan Asylums Board Report shows the same facts with regard to the amount given for all cases: they have, however, no table of the dosage in tracheotomy and non-tracheotomy cases.

From their table (see Report, p. 27, Table LXVII.) we find—

	Average Dose for each Injection.	Average No. of Doses per Case.	Average Total Amount per Case.
	c.c.		c.c.
1. All cases	17.7	2.3	41.2
2. Recoveries	17.0	2.1	35.8
3. Fatal cases	19.1	2.8	55.1

It is evident that we cannot answer the question whether the prognosis varies with the amount of antitoxin given from these data, which merely show that the amount in c.c. of serum given varies with the severity of the case. But this direct variation affords some proof that the beneficial results obtained were due to the antitoxin.

We must, however, once more insist on the fact that it is absurd to measure the doses volumetrically. What we must know is the antitoxic or immunising value of the syringeful used. Large doses in earlier injections are better than an endless number of small injections. The largest immunising or antitoxic doses must be given in those cases in which the disease has existed some time, *e.g.* for more than three days, for in them the amount of poison to be overcome, other things being equal, must be far greater than in the earlier cases, and consequently a larger amount of antitoxin, which, however, may be contained in a few c.c., will be required to counteract the effect of the excessive amount of the poison, whatever be its nature. Whether it be produced, as Dr. S. Martin has declared, by a process of fermentation, or whether the poison is the direct product of the bacilli themselves, everything points to the extreme importance of adopting early energetic treatment with the antitoxin: on this point all must agree. It may even be necessary to inject the antitoxin into the circulation.

When, on post-mortem examination, the lungs are seen to be studded throughout with the patches of broncho-pneumonia, and when we find on bacteriological examination that the lungs are "full of diphtheria bacilli," we can realise what neglect of thorough, and efficiently thorough, administration

of antitoxin means, and we can understand how essential for success it is to begin early, and with sufficiently powerful doses, in order to counteract the effect of the millions of bacilli which must be present in these cases.

The tables given in other portions of this paper, showing the effect of the treatment according to the day of the disease when treatment is first begun, emphasise the importance of being in earnest about beginning the treatment early in the disease, when we may hope both to arrest the spread of the bacilli and to destroy the poison manufactured by them.

Having just now laid stress on the point that the doses must be measured by their antitoxic value, and not by cubic centimetres, we may proceed with an analysis of the volumetric table given above, and attempt to calculate the amount of immunising units administered. In this manner we may succeed in demonstrating that the prognosis depends on the proper use of antitoxin, *i.e.*, on the injecting a sufficient number of units. This we can only do by classifying our cases according to the preparation of antitoxin employed.

Rearranging the above statistics in this manner, we find that (I.) Klein's antitoxin was used in 35 cases; (II.) Aronson's antitoxin in 20 cases; and (III.) more than one kind of antitoxin was used in three cases.

I. KLEIN'S ANTITOXIN.

(A.) *All Cases.*

Mortality = 42.85 per cent.

	Total c.c. Given.	Total Number of Doses.	Average Amount per Dose.	Average Number of Doses per Case.	Average Total Amount per Case.
	c.c.		c.c.		c.c.
1. Total . . . 35	1305	196	6.65	5.6	37.28
2. Recoveries . 20	691	106	6.51	5.3	34.55
3. Deaths . . 15	614	90	6.82	7.06	40.93

(B.) *Tracheotomy Cases.*

Mortality = 50 per cent. Incidence = 68.57 per cent.

1. Total . . . 24	1109	168	6.60	7.0	46.20
2. Recoveries . 12	595	90	6.61	7.5	49.58
3. Deaths . . 12	514	78	6.58	6.58	42.83

(C.) *Non-Tracheotomy Cases.*

Mortality = 27.27 per cent.

	Total c.c. Given.	Total Number of Doses.	Average Amount per Dose.	Average Number of Doses per Case.	Average Total Amount per Case.
	c.c.		c.c.		c.c.
1. Total . . . 11	196	28	7	2.54	17.81
2. Recoveries . . 8	96	16	6	2.0	12.0
3. Deaths . . . 3	100	12	8.33	4.0	33.33

II. ARONSON'S ANTITOXIN.

(A.) *All Cases.*

Mortality = 40 per cent.

1. Total . . . 20	807.5	103	7.84	5.15	40.37
2. Recoveries . . 12	339.0	41	8.26	3.41	28.25
3. Deaths . . . 8	468.5	62	7.55	7.75	58.56

(B.) *Tracheotomy Cases.*

Mortality = 70 per cent. Incidence = 50 per cent.

1. Total . . . 10	533.5	71	7.51	7.1	53.35
2. Recoveries . . 3	105.0	13	8.07	4.33	35.0
3. Deaths . . . 7	428.5	58	7.38	8.28	61.21

(C.) *Non-Tracheotomy Cases.*

Mortality = 10 per cent.

1. Total . . . 10	274	32	8.56	3.2	27.4
2. Recoveries . . 9	234	28	8.35	3.11	26.0
3. Deaths . . . 1	40	4	10.0	4.0	40.0

III. *Cases in which more than One Preparation was Used.*

These were three in number—all recovered.

		No. of Injections.	Average per Dose.	Average No. of Doses per Case.	Average Total Amount per Case.
	c.c.		c.c.		c.c.
1.	{ Ruffer's . . 20 } { Klein's . . . 23 } = 43	5	8.6
2.	{ Klein's . . . 10 } { New York . . 7.5 } = 17.5	2	8.75
3.	{ Klein's . . . 20 } { Aronson's . . 33 } = 53	7	7.57
Total amount . . . 113.5		14	8.10	4.66	37.83

For age periods, according to date of disease, we obtain the following figures:—

I. KLEIN'S ANTITOXIN.

A. *All Ages.*—35 cases with 15 deaths.

Admitted up to Third Day of Disease.			Admitted after Third Day of Disease.		
Cases.	Deaths.	Mortality.	Cases.	Deaths.	Mortality.
19	5	Per Cent. 26.31	16	10	Per Cent. 62.5

B. *Under Five Years of Age.*—28 cases with 12 deaths.

Admitted up to Third Day of Disease.			Admitted after Third Day of Disease.		
Cases.	Deaths.	Mortality.	Cases.	Deaths.	Mortality.
16	5	Per Cent. 31 25	12	7	Per Cent. 58.33

II. ARONSON'S ANTITOXIN.

A. All Ages.—20 cases with 8 deaths.

Admitted up to Third Day of Disease.			Admitted after Third Day of Disease.		
Cases.	Deaths.	Mortality.	Cases.	Deaths.	Mortality.
5	1	Per Cent. 20	15	7	Per Cent. 46.66

B. Under Five Years of Age.—16 cases with 7 deaths.

Admitted up to Third Day of Disease.			Admitted after Third Day of Disease.		
Cases.	Deaths.	Mortality.	Cases.	Deaths.	Mortality.
5	1	Per Cent. 20	11	6	Per Cent. 54.54

If we compare these more detailed tables, we find that there is a somewhat smaller percentage of mortality amongst the cases treated with Aronson's antitoxin than amongst those treated with Klein's. It is perhaps not quite fair to compare them in this way, for whereas 35 cases were treated with Klein's antitoxin, only 20 were treated with Aronson's. Yet, if we consider it just to make a comparison, then we must ask for a reason to account for the appreciable difference in the percentage mortality. Our answer then would be that Aronson's serum was stronger than Klein's, *i.e.*, as regards its antitoxic value, disregarding the immunising value. While Aronson's antitoxin contained between 75–100 units, Klein's at the most contained but 20 units per 1 c.c. This in itself would suffice to explain the difference between the results obtained. But it must be evident that whatever antitoxin was used, there having been no proper standard, and the dosage by units not having been sufficiently understood, in few cases has the desirable number of units been injected. Thus, even making liberal allowances, the greatest average number of units which has been administered with Klein's serum is 1000, while with Aronson's serum it has been 6000, the average totals for all cases being with Klein's antitoxin 750 units, and with Aronson's 4000. We do not wish to criticise the method of preparing antitoxins and their relative value, nor enter into

any theoretical discussion as to the action of antitoxin, whether it is antitoxic, germicidal, or immunising, or whether it possesses a double or treble action ; but we must say that, if it is now recognised, after two to three years' experience, that at least 1000 units, or even 4000 units, should be given with the first injection, and that to ensure success in the class of cases admitted into general hospitals, several thousands of units are required, then we have used doses at St. Bartholomew's Hospital which were far too small, and may not that explain why our results have not quite come up to expectation ? We may repeat it, that the dose of antitoxin is not measured by the size of the syringe but by the number of units. This must be forcibly impressed on all minds. Larger doses of Aronson's serum or potent serum, prepared by the conjoint laboratories, are now being given at our Hospital, and it would be interesting to know how next year's result will read. In this connection we cannot do better than quote Dr. S. Martin,¹ with whom we entirely agree in all he says on this point:—

“There has undoubtedly been a saving of life in children under five years of age, and the better results in 1896 as compared with 1895 are again to be ascribed to the better serum used. The serum used in 1895 and the early part of 1896 was made at the British Institute of Preventive Medicine. During the last five months we have used serum kindly supplied from the laboratories of the Royal Colleges of Physicians and Surgeons by Dr. Sims Woodhead. The strength of this serum is about 4000 units in 5 c.c., and as this is a convenient amount to inject into a child, it is more serviceable than when 40 c.c. have to be given in order to administer 4000 normal units. In 1895 the strength of the serum used was not known, except that it was much less than that used this year. During 1896 only one case received as small a dose as 2000 units, four received doses of 3000 units, while the remaining cases received doses varying from 4000 to 16,000 units. We never now give a dose of less than 4000 units, and more frequently give 8000. It may be claimed for these larger doses that they have still further reduced the mortality from that of last year, and that we have had this year a minimum of cases of paralysis following the disease. In only three of the cases has there been evident paralysis, and one of these (a severe case of diphtheria) died in forty-four days from broncho-pneumonia.”

At the Hospitals under the Metropolitan Asylums Board also weak serum was used during 1895, viz., during the first eight months that supplied by the British Institute of Preven-

¹ *Lancet*, 1896, October 17.

tive Medicine (Ruffer's serum), and for the last four months that supplied by Dr. Sims Woodhead. It is stated in their Report that no difference was observed either in the antitoxic value or in the clinical behaviour of the two supplies. Since then Dr. Woodhead supplies serum of very much greater strength. Accepting matters, however, as they stood according to that Report, it is clear that the antitoxic equivalent of the doses used was far too low, for in 1895 the Preventive Institute serum did not contain more than 20-30 units, so that the average number of units given at these Hospitals, calculated from Table LXVIII. p. 27 of the Report, amounted to 670-2000 units per case. The Report argues that the best results may be obtained by giving a dose of 1000 units every twelve hours for the first twenty-four, thirty-six, or forty-eight hours, according to the gravity of the case, and, if necessary, a subsequent injection of half the amount daily for such time as the exudation may remain adherent. Dr. S. Martin, as we have seen, advises 3000 units as a dose. If we look at Table LXVII. on p. 27, we find that in no case did the average dose amount to 1000 units, and in most instances did the average amount per patient fall short of 1000 units. This explains to our mind that the results obtained there were no better than those obtained here; better, no doubt, than what had been achieved in former years, but not so good as what has been done in Germany, or by Dr. S. Martin, since he uses serum of high antitoxic value. It is well known that the Continental results in 1895 were better than ours: we now know the explanation. It is to be looked for primarily in the quality and method of administration of the serum. The prognosis undoubtedly depends on the antitoxic equivalent of the doses administered.¹ Let us realise this, and use the soundest measure of dosage, viz., the immunising unit, and we shall soon get results equal to those of other countries.

We may now pass to a brief consideration of the alleged ill effects which have been attributed to the antitoxin, and which might presumably affect the prognosis considerably. The most important of these are said to be the following, viz.:—

- (1.) Rashes.
- (2.) Joint affections.
- (3.) Inflammation about the seat of inoculation.

¹ Since the above was written larger doses of potent antitoxin have been used (measured by units), and we find that in 64 cases the mortality was 21.87 per cent.; of these there were 30 tracheotomies with a mortality of 36.6 per cent., and 34 non-tracheotomy cases with a mortality of 11.76 per cent. It will be of interest what next year's statistics will show, for already there is a considerable reduction.

- (4.) Abscesses.
- (5.) Albuminuria and nephritis.
- (6.) Collapse and sudden death.
- (7.) Pyrexia.

These may be taken in the above order :—

(1.) *Rashes*.—In the 58 cases (Series III.) a rash is stated to have occurred in 14. In seven of these cases the rash was an ordinary measles rash. In two there was both a measles rash and an antitoxin rash. In five there was an antitoxin rash only. So that measles complicating diphtheria is not an infrequent occurrence, being present in 15.51 per cent. of these cases.

The antitoxin rash occurring seven times gives a percentage incidence of 12.06 per cent. In no single case does it seem to have caused any worse trouble than a temporary rise of temperature, ranging from 100° F. to 103° F., and in two cases considerable itching. Its date of appearance was—

In 2 cases	7 days	} after the first injection.
„ 2 „	12 „	
„ 1 „	10 „	
„ 2 „	a few hours after the first injection.

In two cases the rash was described as erythematous; in two as urticarial; in one as petechial; in the remaining two, no note was made as to its nature.

The distribution has varied within wide limits; there seems to be no especially favoured area.

The temperature rose during the duration of the rash in four cases; in the other no alteration in the temperature occurred with the rash; no ill effects followed the rise of temperature.

The duration varied from a few hours in two cases, to four days in one case; in three cases the rash lasted two days, in the remaining case no note was made on the duration of the rash.

In no case was there a fatal result.

Albuminuria occurred in two of the cases.

We do not think that any importance is to be attached to the occurrence of a rash.

The incidence of an eruption in the M.A.B. hospitals was greater, viz., 45.9 per cent. (see Report, p. 26, Table LXVI.), but it does not appear from the Report that great importance can be attached to it.

(2.) *Joint affections*.—In the 58 cases collected by us (and in the 50 cases collected by Dr. Herringham) there was no affection of any joint. In the M.A.B. (see Table LXVI.) joints

were affected in 4.7 per cent. of the cases, but it is added that "they apparently leave no ill effects."

(3.) *Inflammation about the seat of inoculation.*—In one case only was tenderness complained of, but there was no actual lesion, nor was there any rise of temperature.

(4.) *Abscesses.*—In one case a small subcutaneous abscess occurred about the seat of inoculation. It was opened at once, and was entirely healed in four days; no ill effects followed. A bacteriological examination unfortunately was not made.

The M.A.B. gives an abscess incidence of 2.3 per cent., but attaches no immediate importance to it as an effect of the antitoxin (see M.A.B. Report, p. 26, Table LXVI. and below).

(5.) *Albuminuria and nephritis.*—In 29 of our 58 cases albumin is stated to have been present; of these, 13 were fatal cases. In 10 cases albumin was not found at any time; of these, none died. In 19 cases no note was made about albumin; of these, 10 were fatal cases.

Therefore, so far as our 58 cases are concerned, owing to the incomplete notes, it is impossible to make any trustworthy comparison, since the previous records are equally deficient.

The M.A.B. Report shows a considerable increase, viz., from 24.1 per cent. to 40.9 per cent. for albuminuria and a slight increase of nephritis, viz., 1.2 per cent. to 2 per cent. (see M.A.B. Report, pp. 21 and 23, Tables LI. and LVIII.).

We are not prepared to assert that there is any causal relation between albuminuria and antitoxin. It may be interesting, however, to recollect that some observers have maintained that the antitoxin produces cloudy swelling of the kidneys and albuminuria. On the other hand, this has been strenuously denied by others.

(6.) *Collapse and Sudden Death.*—No case of sudden death followed the injection of antitoxin. Collapse occurred in one case, a child aged 5 years, who was admitted moribund on the fourth day of the disease, and in whom signs of collapse followed each injection; in consequence three injections only were given, amounting in all to a total of 19 c.c. This case died two days after admission. In such a case the collapse may have been due to the syringe, independently of the antitoxin.

(7.) *Pyrexia* has occurred, generally in the shape of a slight and temporary rise of temperature after an injection, though this even has not followed every injection in the individual case. In no case did this temporary elevation of temperature seem to cause any detrimental effect.

The M.A.B. Report inclines towards the belief (M.A.B. Report, p. 26), that its occurrence somewhat retards convalescence.

Considered, therefore, as being actually due to the antitoxin, the ill effects following its use are more imaginary than real, and are not such as to cause any cessation in amount and frequency of the administration, except where collapse follows each injection.

We shall conclude by examining three other important features of diphtheria which are generally included as complications or sequelaë, viz., broncho-pneumonia, septicæmia, and paralysis, and their relation to the antitoxin. We shall begin with paralysis.

Paralysis—In the 58 cases at St. Bartholomew's Hospital with 23 deaths collected by us, paralysis occurred in 10 cases, giving an incidence of 17.24 per cent. These 10 cases include the three cited on pages 335, 336, which have been included among the recoveries from the acute attack for the reason given on page 336. Now these three cases all died, the paralysis being apparently the cause of death, in one, thirty-one days after admission; the cause of death in the other two was broncho-pneumonia complicating measles, which in each case was contracted in the hospital after the case had been considered by the physician under whose charge it was to have recovered from diphtheria in the acute form, the paralysis alone detaining the case in hospital. Unfortunately no post-mortem examination was allowed in any of these three cases, so that the absence of diphtheria bacilli in the tissues at the time of death was neither confirmed nor contradicted. In one of these, which died thirty-nine days after admission, for some reason or another, no bacteriological examination was ever made; in the other, which died fifty-nine days after admission, the only bacteriological examination made was that on the day of admission, which showed the presence of diphtheria bacilli. In the remaining one, which died from the results of the paralysis, the first signs of paralysis occurred twenty-four days after admission and death followed seven days later; and in this case no bacilli were found, nor was any membrane seen later than the eighth day after admission. Consequently in these three cases paralysis may be regarded as the direct cause of death in one only. Of the remaining seven cases, six recovered, while one died six days after admission from syncope. This case had signs of paralysis on admission on the sixth day of the disease, and death was due to diphtheritic paralysis: diphtheria bacilli were found in large numbers in the lungs post-mortem. Therefore out of the 10 cases in which paralysis occurred, it was the direct cause of death in two only, in one of which it occurred during the acute stage of diphtheria, and therefore may not itself have been the sole cause of death.

However, we may consider that diphtheritic paralysis directly caused death in two cases, *i.e.*, in 20 per cent. of the cases in which paralysis occurred. That is to say, after recovery from the acute stage of diphtheria, paralysis may be the cause of death in 2.85 per cent. of cases which have recovered from the acute attack.

The incidence of 17.24 per cent. of paralysis in these cases compares with an incidence of 23.2 per cent. at the hospitals of the M.A.B. (see Report, p. 21, Table LI.).

Owing to the difficulty of following up the progress of the cases after their discharge from the Hospital, we are unable to state in how many paralysis may have occurred after leaving St. Bartholomew's. In the Report of the M.A.B., we find a table giving the incidence in the non-antitoxin cases in 1895, as compared with the cases before antitoxin was used in 1894, which shows the incidence in the non-antitoxin cases of 1895 to have been 16.77 per cent. against 13.2 per cent. in 1894 (see p. 24, Table LIX.). Since these formed series of cases, neither of which were treated with antitoxin, they tend to support the view that the epidemic of 1895 was of a more severe type than the average of the preceding year (or years); and therefore that the incidence of paralysis is greater among severe cases.

The increased incidence of paralysis which the M.A.B. Report shows (Tables LI. and LII., p. 21) for the antitoxin cases compared with the pre-antitoxin year, *viz.*, from 10 per cent. to 23.2 per cent., may be due therefore to two causes, *viz.*—

(1.) A larger number of cases died previously which would, had they survived, probably have developed paralysis; for paralysis is a sequela rather than a complication; (2.) or owing to the greater survival among the more serious cases in a severer epidemic, a larger number have developed paralysis.

In the cases of Series III. in which paralysis occurred, the antitoxin was given in much greater amount than in any other cases, as is seen from the accompanying table, arranged similarly to those on page 365—

Cases.	Total Amount Given for all Cases.	Total No. of Doses for all Cases.	Average Dose for each Injection.	Average No. of Doses per Case.	Average Total Amount per Case.
10	538.5 c.c.	65	8.28 c.c.	6.5	53.85 c.c.

This shows that a much larger amount of antitoxin has been given in the cases in which paralysis followed than under any other circumstances in the St. Bartholomew's cases, both in the amount of each dose and in the total amount per case (*cf.* Tables of dosage, p. 365).

There is no similar table in the M.A.B. Report, but the above table shows merely that paralysis follows most often after the severe forms, as already stated, for both at St. Bartholomew's and at the hospitals of the M.A.B. the most copious administration of antitoxin has been found necessary in the severest cases, which is only what might be expected, and on which point we have dwelt at greater length above. The greater amount of antitoxin in these cases is certainly not the reason of the incidence of paralysis; on the contrary, we fully agree with Dr. Martin that the proper administration of antitoxin will undoubtedly diminish the incidence of paralysis.

Broncho-pneumonia.—In the 58 cases—

(*a.*) Signs of broncho-pneumonia were present during life in 22 cases, of which 14 died. Out of these 14 fatal cases an autopsy was performed in nine, and in all nine the diagnosis made during life was confirmed post-mortem.

(*b.*) The notes of the cases state that broncho-pneumonia was not present in 20 cases, of which five died. An autopsy was performed on these five cases, but no broncho-pneumonia was macroscopically found in them.

It must be remembered, however, that as far as our own observations go, the naked-eye diagnosis is of little value. We have often found typical acute broncho-pneumonia on microscopical examination, when at the autopsy the lungs were described as normal in appearance.

(*c.*) Of 16 cases, no note on the condition of the lungs could be found; among these 16 there were four deaths. An autopsy was made on one, but there was no macroscopic evidence of broncho-pneumonia discovered.

Broncho-pneumonia therefore occurred in at least 22 out of 58 cases of diphtheria, giving an incidence of at least 37.93 per cent., and this broncho-pneumonia we believe to have been caused by the diphtheria bacillus, because

(1.) In all the above 22 cases the first signs of broncho-pneumonia were first detected some time (1–5 days) later than the date on which the onset of diphtheria occurred. This reason, based on clinical evidence, is supported by

(2.) The bacteriological facts which Kanthack and Stephens have adduced on the nature of broncho-pneumonia complicating diphtheria (see *Jour. of Path. and Bact.*, July 1896).

Assuming, then, that broncho-pneumonia occurred in at least 14 out of 23 fatal cases, this means that diphtheritic broncho-pneumonia is associated with the fatal result, either as the immediate cause of death or in conjunction with some other cause, in at least 60.86 per cent. of the fatal cases. The view now held of the nature of this complication adds further weight to the probability of far greater improvement in the prognosis when the antitoxin treatment for diphtheria is begun early in the disease and a tracheotomy not feared or delayed.

The M.A.B. Report shows the incidence of broncho-pneumonia in diphtheria to have risen from 1.6 per cent. in 1894 to 3.6 per cent. in the antitoxin cases of 1895 (see Report, pp. 21 and 23, Tables LI. and LVIII.).

The only explanation we can offer of this is that it further supports the view that the 1895 epidemic was severer than its immediate predecessor.

It is important to remember that in almost every case of fatal diphtheria, whether pharyngeal or laryngeal diphtheria, bacilli are readily found in the lungs, and hence we believe that their escape into the lungs must be counteracted by energetic antitoxin treatment and early tracheotomy.

Septicæmia.—Septicæmia is not an uncommon cause of death in diphtheria, as shown by the researches of Wright and Stokes of Boston, and at our own Pathological Laboratory. It is astonishing how frequently pyogenetic cocci are found in fatal cases in the spleen and in the heart's blood. Hæmorrhagic diphtheria is probably always septic, and is generally, if not always fatal. The co-existence of septicæmia must be considered a grave sign, and portends a bad prognosis. It cannot be prevented or cured by the specific anti-diphtheritic serum, and we should welcome the further extension of the serum treatment, enabling us to grapple also with this complication. That can only be done by using a specific anti-streptococcus or anti-staphylococcus serum. This, it is said, is already being done in France. There can be no doubt that, if successful, this form of treatment will considerably decrease the mortality of diphtheria.

The causes of death.—We have analysed the causes of death in the 23 fatal cases out of the total of 58 cases at St. Bartholomew's Hospital. In these (1) a post-mortem examination was made in 15 cases; (2) no post-mortem examination was made in eight cases.

(1.) *The cases examined post-mortem* = (15).—Nine died of diphtheritic broncho-pneumonia and diphtheritic toxæmia. Three died of diphtheritic toxæmia without broncho-pneumonia. One

died of diphtheritic and secondary infective toxæmia. One died with purpura hæmorrhagica (septicæmia). One died of syncope due to paralysis and acute diphtheritic poisoning (bacilli being found in lungs and spleen post-mortem).

(2.) *The cases not examined post-mortem* = (8).—Five were considered to have died as the result of diphtheritic toxæmia associated with broncho-pneumonia. One died eight days after admission with sudden acute abdominal pain, with failure of pulse and respiration (the last injection having been two days previous to death). One died nine days after admission during tracheotomy for a severe attack of dyspnoea and coughing, during which the intubation tube was coughed out. One died two days after admission, of which we have been unable to find any notes of the clinical condition.

Four of the fatal cases were five years of age and upwards; in none of these was broncho-pneumonia diagnosed during life, and this was confirmed in three on which a post-mortem examination was performed.

Therefore broncho-pneumonia is especially fatal in cases under five years of age, for broncho-pneumonia was associated with 14 out of a total of 19 fatal cases under five years of age. *i.e.*, in 73.68 per cent. of these fatal cases.

From a study of the cases reported by the American Pediatric Society, we find that the commonest causes of death are (1.) sepsis and diphtheritic toxæmia, (2.) broncho-pneumonia, (3.) cardiac failure, (4.) laryngeal obstruction. 450 fatal cases have been especially enumerated: of these 58 were moribund on admission, 54 died of broncho-pneumonia, 53 of cardiac failure, 48 of laryngeal obstruction, and 105 of sepsis or toxæmia.

Summary.—1. The prognosis in cases of diphtheria treated by antitoxin is far better than previous to its introduction as a method of treatment, especially (1.) in the dangerous forms, *viz.* (a) those under five years of age, (b) those in which tracheotomy is required; (2.) in those in whom the treatment is begun early in the disease, and with sufficiently large injections.

2. The prognosis is markedly improved when the diagnosis is made early.

3. The use of the antitoxin does not appear to have lessened the necessity for tracheotomy.

4. The ill effects attending the use of antitoxin are very few, if any, and imaginary rather than real; and if real, they are not of such nature or severity as to be a contra-indication of its employment.

5. Broncho-pneumonia is of frequent occurrence, and is

diphtheritic in origin, and very frequently found in fatal cases (at least 60.86 per cent. in our cases).

6. The increased incidence of post-diphtheritic paralysis, if actual, is possibly due to the increased number of severe cases which have survived, on account of their having been treated with antitoxin, and is probably also due to the fact that too little antitoxin was used.

7. The method of administration should be energetic to begin with, the dose to be measured by units, and the frequency of injection to be regulated by the severity of the case.

8. Septicæmia implies a bad prognosis, and is a by no means infrequent cause of death. Special forms of serum should be administered to counteract its effects, and there can be little doubt that, when a suitable serum has been found, that with the prevention and cure of septicæmia the mortality of severe cases of diphtheria will be lessened to a still more remarkable degree.

In concluding, we desire to express our thanks to the Secretary of the Metropolitan Asylum Board for his courtesy in forwarding us a copy of the Special Report by the Medical Superintendents of the Hospitals of the Board upon the Treatment of Diphtheria by the Antitoxin during 1895. Further, we wish to point out the obvious lesson of this analytical inquiry. The best results are obtained on administering the antitoxin early and energetically in large doses, which must be measured by immunising units and not by cubic centimetres: 1000 units should be the minimum dose, and it should be repeated until all danger is passed. A larger number of units of course may be given: that must depend on the supply of serum. The injections must be repeated every 3, 4, or 6 hours, till the patient is safe or the physician feels at ease. Tracheotomy should not be delayed: if there is any dyspnoea it should be performed at once, and antitoxin vigorously administered. *Early tracheotomy, together with early and adequate antitoxic treatment, is the surest preventive of pulmonary infection.* Serious pharyngeal cases should be treated as energetically as serious laryngeal cases: they are just as dangerous, and pulmonary infection is common in them.

In gangrenous cases or in alarming cases antistreptococcus serum should be given as well as diphtheria antitoxin. Local treatment must not be neglected. Quickness of action, perseverance till both patient and physician feel at ease, common sense and good antitoxin are the surest means of succeeding.

ON THE ÆTIOLOGY OF CHOREA.

BY

H. MORLEY FLETCHER, M.D.

The ætiology of chorea has been so often discussed by many able writers, that some explanation is almost necessary of the reasons which led me to make further investigations into this difficult subject. During my period of office as Casualty Physician I collected full details of the many cases of chorea which were seen in the Out-patient Department, paying special attention to the date of onset, state of the heart, and history of rheumatic fever. These and other points were entered on printed tabular forms. Some of the cases were seen at the Hospital for Sick Children, Great Ormond Street, where I was acting as Clinical Assistant.

The 273 cases seen during the year 1894 may be considered to be cases of chorea which were not so severe as to require admission to a hospital.

It seemed that a profitable line of inquiry lay in ascertaining whether the ætiology of chorea in mild out-patient cases was the same as that in the severer forms of the disease such as are seen in the wards of a hospital. This is a point which, as far as I am aware, has not been previously discussed by the many writers on this subject.

The cases of chorea treated in the wards of St. Bartholomew's Hospital during the ten years 1885-94 inclusive, amount to 307, and I shall call this the *In-Patient Series*, in distinction to the *Out-Patient Series* of 273 cases mentioned above. I must take this opportunity of thanking the Physicians of the Hospital for permission to refer to the notes of the cases, and also to Mr. Hussey for assisting me in making the analysis.

In discussing the ætiology of chorea, it seems advisable to consider the main factors in their order without any subdivision into predisposing and exciting causes.

I. *Sex*.—The liability of the female sex to chorea is more

marked than in any other disease of ordinary frequency to which both sexes are liable.

In 273 out-patients there were 201 females and 72 males, or 2.8 females to 1 male.

In 327 in-patients there were 247 females and 80 males, a proportion of 3 females to 1 male. Trousseau,¹ Sturges,² and Hughes³ give practically the same figures, while Pye-Smith, Sée,⁴ and Watson give 2.5 females to 1 male.

Not only is the female sex more liable to chorea than the male in the proportion of about 3 to 1, but it is also associated with the more severe forms of the disease, the mortality being about 6 females to 1 male. To this point I shall refer later.

The greater liability of the female sex is probably due to their greater nervous susceptibility, which is accentuated by the important changes which occur at puberty.

Age.	In-Patients.		Out-Patients.		Total Number All Attacks.	Total Number First Attacks.
	All Attacks.	First Attacks.	All Attacks.	First Attacks.		
3	1	1	0	0	1	1
4	1	1	3	3	4	4
5	5	4	7	5	12	9
6	14	10	12	7	26	17
7	19	17	14	11	33	28
8	26	21	22	15	48	36
9	21	15	32	18	53	33
10	30	21	37	24	67	45
11	18	15	36	23	54	38
12	32	23	18	8	50	31
13	30	17	16	9	46	26
14	27	18	19	12	46	30
15	35	20	15	8	50	28
16	20	13	10	6	30	19
17	21	17	15	6	36	23
18	5	3	3	1	8	4
19	4	3	3	2	7	5
20	5	2	2	1	7	3
21	3	2	1	0	4	2
22	3	0	1	1	4	1
23 and } over }	7	1	7	1	14	2
Total	327	224	273	161	600	385

¹ Trousseau, Lectures on Medicine, Sydenham Society, vol. i. p. 392.

² Sturges on Chorea, p. 23.

³ Hughes, Guy's Hospital Reports, Second Series, vol. iv. p. 360, also Third Series, vol. i. p. 217.

⁴ Sée, De la Chorée et les Affections Nerveuses. Paris, 1851.

II. *Age.*—The earliest case in my series is one occurring at 3 years 4 months. Sturges mentions one at 2 years 11 months.

The preceding table shows the number of cases occurring for each year of life of the in-patients and out-patients, both series being arranged to show the number of first attacks, and also the number of all attacks, whether primary or recurrent.

An examination of this table shows that, taking the last two columns, which represent both out- and in-patients added together, the greatest liability is at 10 years both for primary and recurrent attacks.

Taking the out-patient series alone, we find that 50 per cent. of the first attacks occur during the years 8–11 inclusive. This may be considered as the period of greatest liability for first attacks among the less severe cases, such as attend an out-patient department.

This is not the case, however, with the more severe in-patient cases. In this, the age of the greatest liability for all attacks is at 15 years, as compared with 10 years for the out-patients, and the four years from 12–15 inclusive is the period of greatest liability, as compared with 8–11 for the out-patient series.

The first attacks in the in-patient series occur in considerable numbers at later ages than is the case in the other series.

These observations are in agreement with the well-known fact that the more severe cases of chorea occur at a later period of life than do the slighter cases, and this period coincides with the age of puberty.

The first attacks, especially the slighter ones, occur mostly during the early school-life of children.

Other authors give wider limits for the age of greatest frequency. Sturges finds the greatest liability between the sixth and fourteenth years;¹ Hughes, the tenth and fifteenth; Pye-Smith, the eighth and sixteenth.²

It would certainly appear that chorea, at or about fifteen years, is more serious than at other ages, and this is borne out by observations made by Watson, Ogle, and Hughes, that the majority of fatal cases occur in patients who are near or have just passed the age of puberty.

III. *Race.*—There is little doubt that chorea is a very rare disease amongst negroes and Indians, as has been pointed out by Osler,³ and I have not found a single case of chorea recorded in a coloured child in London. It is interesting to note in this

¹ Sturges, *Chorea*, 2nd edition.

² Pye-Smith, *Guy's Hospital Reports*, Third Series, vol. xix. p. 329.

³ W. Osler on *Chorea and Choreiform Affections*, 1894.

connection that the Army Medical Reports show that the black troops in Ceylon, Bermuda, West Africa, and the West Indies suffer at least as much from rheumatism as the white troops stationed in these regions, though it is difficult to ascertain from the returns the actual number of cases of rheumatic fever which are included under the heading rheumatism.

The rarity of chorea amongst the dark races may be due to their less highly educated, and therefore less susceptible, nervous systems.

IV. *Locality.*—I have been unable to collect statistics for this country bearing on this point, but there can be no doubt that chorea is far more prevalent in large cities than in rural districts.

Isambard Owen, in an interesting investigation on the distribution in the United Kingdom of rheumatic fever, rickets, chorea, &c.,¹ shows that chorea is by no means so widely distributed as rheumatic fever, which is very nearly universal throughout the British Islands. He shows, on the other hand, that the distribution of chorea closely resembles that of rickets, in that both accumulate in the great industrial centres, the rural districts being comparatively free. Speaking of London, he states that it is not so common in the south-western and south-eastern districts as in the rest of the metropolis. On this point I have no data.

The different conditions of life in children living in town and in the country are surely sufficient to account for the variations in the distribution of chorea—a disease in which mental effort, fright, and school pressure bear so important a part.

V. *Seasonal variation.*—The present inquiry was instituted largely with a view to ascertaining what relation, if any, could be established between the time of year and the date of onset of chorea; and further, to investigate what meteorological conditions might be associated with this.

Most writers agree that the disease is more common in the winter months. Hughes found the maximum number of cases in December, after which month the number falls, and is followed by a distinct rise in March.

The most elaborate inquiry into this seasonal relation is that by Morris J. Lewis.² In an analysis of 1383 cases he found that the maximum number of attacks occurred in March.

¹ Isambard Owen, Transactions of International Congress, Washington, vol. v. p. 157.

² Morris J. Lewis, Transactions of the Association of American Physicians, vol. vii. 1892.

After March the number of cases declines, reaching the minimum in November, and again begins to increase in December. He gives as an explanation of this that March is the month of most general sickness (in Philadelphia), and has some relation to "storm centres," mean barometric readings, and mean relative humidity. To these points I shall refer later.

My attention having been drawn to the large variation in the number of cases of chorea seen during the different seasons of the year, I considered it would be of interest to ascertain whether any such relations could be established from an inquiry on somewhat similar lines among cases treated at St. Bartholomew's Hospital. The same two series of cases were taken; the first a series of 273 out-patients seen during the year 1894, the second a series of 327 in-patients admitted to the Hospital during the ten years 1885-94 inclusive. Great care was taken to ascertain as accurately as possible the month of incidence of the chorea, all doubtful cases being excluded. Reliable data were obtained in 268 out-patients and 306 in-patients, the remainder being disregarded as doubtful. Charts I. and II. (p. 388) give the results showing the relative frequency of onset in the different months of the year. The thick line represents all attacks of chorea, whether primary or recurrent, the thin line below representing first attacks only.

An examination of both pairs of curves shows that the seasonal variations of first attacks is approximately the same as that for all attacks. It is probable, therefore, that the exciting cause or causes is the same for first as for subsequent attacks. There is, however, a distinct difference between the curves for the out-patient series and those for in-patients. In both the minimum number of cases occurred from May to September inclusive, but the months of greatest frequency do not coincide. Taking the out-patient curve, we find that December and January show a great increase in the number of cases, the latter month having the maximum for the year. In the in-patient curve there is also a marked rise in December and January, followed by a sharp fall in February; but then comes a great increase, reaching the maximum in March.

This increase in March corresponds to that found by Hughes in his analysis of cases treated in Guy's Hospital, the greatest frequency occurring in November, December, and March.

This seasonal variation of chorea is difficult to explain, and it will be convenient to discuss it later in its relation to other ætiological factors.

The explanation given by Morris of the maximum frequency occurring in March at Philadelphia, namely, that it is there the

month of greatest general sickness, does not hold for this country.

VI. *Relation to meteorological elements.*—Lewis investigated this relation with great care in his Philadelphian series. He

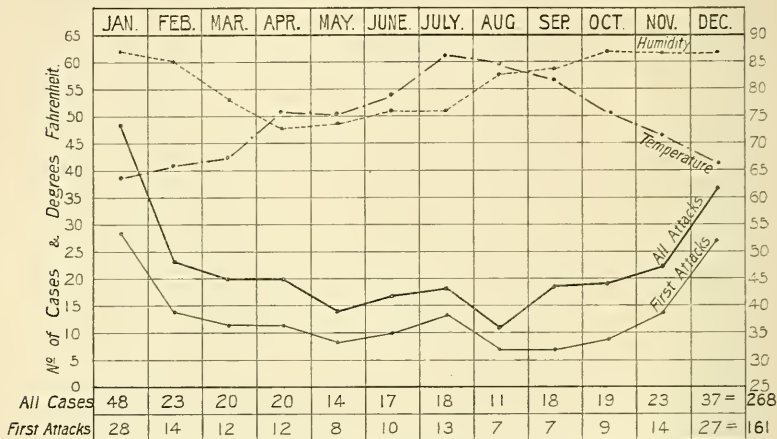


CHART I.—Showing 268 cases of chorea seen as out-patients during the year 1894, arranged in months according to the time of onset of the disease.¹

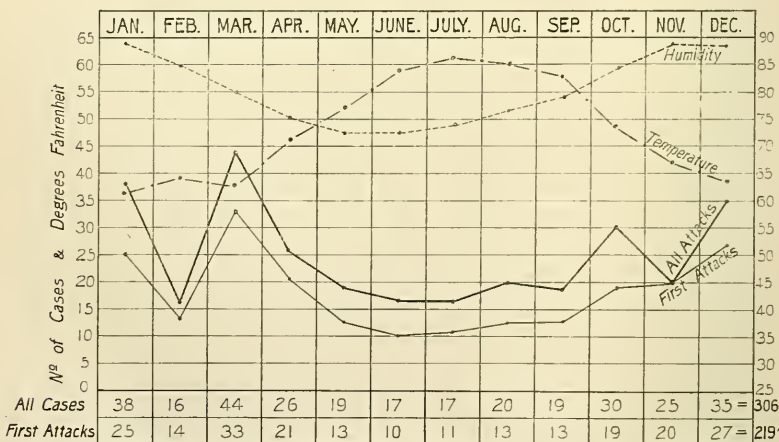


CHART II.—Showing 306 cases of chorea admitted to St. Bartholomew's Hospital during ten years, 1885-94, arranged in months according to the time of onset of the disease.¹

¹ The thick line represents all cases of chorea, the thin line first attacks alone. The dotted line represents the mean degree of humidity (saturation=100), and the broken line the temperature of the air. The figures on the right indicate the degrees of humidity, saturation=100.

found that curves of the mean barometer readings and mean relative humidity showed some relationship to the chorea curves. "Storm" curves, or curves of centres of low barometer, on the other hand, were found to coincide much more closely with the chorea curves.

He concludes that "weather" is the most important predisposing factor both in acute rheumatism and in chorea, though what element in weather is the most active in the causation of these diseases he admits is unknown.

I have investigated the following meteorological elements in connection with the two series of chorea cases I have given: (1) temperature of the air; (2) daily range of temperature; (3) humidity of the air; (4) rainfall; (5) barometric readings. The necessary data were obtained from the Registrar-General's Reports, from observations made at Greenwich by J. Glaisher, F.R.S.

1. *Temperature of the air.*—In the chorea charts I. and II. I have given the curves for the mean monthly temperature corresponding to the years considered, that for the in-patient series representing the average for the ten years 1885-94. The figures on the left of both charts represent degrees Fahrenheit, and also the number of cases of chorea.

There is a distinct inverse relationship between the temperature and chorea curves, as might be expected, for we have seen that chorea is most prevalent in the winter months and least prevalent in the warm summer months; but in Chart II. there is no fall of temperature at all proportional to the great rise in frequency which takes place in March.

2. *Daily range of temperature.*—It might be suggested that chorea, like pneumonia (Herringham¹), might be associated with wide variations of temperature occurring in the twenty-four hours. Herringham observed that lobar pneumonia is more frequent from the end of March to the end of June, and that this period presents the widest daily range of temperature in the year. In this country, however, the daily variation cannot be closely associated either with chorea or rheumatic fever, since both diseases are most prevalent in the winter months, when the daily range of temperature is far less than during the rest of the year.

3. *Humidity of the air.*—This is expressed in the Greenwich returns as the mean degree of humidity, saturation = 100. I have given curves on the chorea charts to show the monthly variations for the corresponding years, the figures on the right

¹ W. P. Herringham, Croupous Pneumonia in London during 1893, Brit. Med. Jour., May 12, 1894.

side of the charts giving the mean degree of humidity. A distinct resemblance to the chorea curves is apparent, but is of doubtful importance, since the degree of humidity varies inversely with the temperature.

4. *Rainfall.*—No result could be obtained of the least value by comparing the amount of rain with the frequency either of rheumatic fever or chorea, owing to the extreme irregularity of rainfall in this country.

5. *Barometric pressure.*—The same applies to this, and the results of this investigation were entirely negative.

VII. *Influence of previous attacks—Recurrence.*—In the out-patient series of 273 cases of chorea:—

	Per Cent.
In 161 cases it was the first attack,	= 59.
In 69 cases it was the second attack,	} = 41.
In 22 cases it was the third attack,	
In 21 cases it was the fourth, or more, attack,	

Of these 112 recurrent cases 24 were males and 88 females.
In the in-patient series of 327 cases:—

	Per Cent.
In 224 cases it was the first attack,	= 68.5.
In 61 cases it was the second attack,	} = 27.9.
In 16 cases it was the third attack,	
In 14 cases it was the fourth, or more, attack,	
In 12 cases doubtful.	

Of these 91 cases in which there was recurrence, 16 occurred in males and 75 in females.

On comparing these two tables, we find that recurrence occurred more often in the milder out-patient cases than in the more severe in-patient cases. As a general observation, it may be stated that recurrent attacks of chorea are less severe than primary attacks.

Taking both series together, in 600 cases recurrence took place in 203. Omitting 12 doubtful cases, this gives recurrence in 34.5 per cent. This may be compared with the 34 per cent. given by Sturges, 32.4 per cent. by Dickinson,¹ and the 31 per cent. by Mackenzie.

Sex plays a not unimportant part in the liability to recurrence, as is shown by the analysis of cases given above, from which it will be seen that females who have had chorea are more liable than males to subsequent attacks.

VIII. *Educational pressure.*—This is an important inciting cause of the disease. Its effect is most marked in boys and

¹ Dickinson, *Med. Chir. Trans.*, 1876, lix. p. 1.

girls below the age of puberty, the greatest number of cases occurring between eight and eleven years, boys and girls being affected in a much more equal proportion than usual. These children are generally particularly bright and intelligent. The chorea is usually not severe, but is very prone to recurrence.

School-board examinations, with the attendant extra preparation at home, are responsible for many of these cases. I have seen several children who had recurrent attacks of chorea, the recurrence in each case following immediately upon the annual school examination in consecutive years.

The movements are in some of the cases noticed to begin just before the examination, and are associated with the extra mental pressure which the child is undergoing. The majority of these cases, however, begin a few days, or as long as a fortnight, after the examination.

It is extremely difficult to obtain figures to illustrate the frequency of this important ætiological factor. Thanks to the courtesy of the officials of the London School Board, I have been able to gather some information with regard to these annual examinations. Until 1894, and including that year, annual compulsory examinations were held on a given date for each school. In the large area from which the cases dealt with in this paper were taken, there are many Board schools at which the dates of examination occur during different months of the year. August is the only month during which no examinations are held; and it is interesting to note on Chart I. that this is the month in which occur the fewest number of cases of chorea. With the exception of August, therefore, examinations may be said to be occurring during every month of the year. It is very probable that this accounts for a certain proportion out of the regular supply of chorea cases which occur during the year, and which is possibly distinct from the increased number in the winter months, which are more definitely associated with rheumatism.

The regulations for the annual examinations were altered in 1895, and they now partake more of the nature of an inspection.

The influence of "school" in chorea was a point to which Sturges attached considerable importance.

IX. *Fright.*—Forty-three in 273 out-patients, or 15.7 per cent., had a history of fright immediately preceding the attack of chorea. Great care must be taken in the investigation of this point, as it not unfrequently happens that a fright or sudden shock occurring to the child is put down as the cause of the chorea, although slight movements may have been present for some days before. There is little doubt, however, that fright,

mental worry, school, &c., undoubtedly do act as exciting causes in some cases of chorea in older children which are certainly what may be called rheumatic chorea. A large proportion of these fright cases, like those dealt with in the preceding section, are mild cases in young children, and are not attended with morbus cordis. It is also of importance to notice the fact that fright is the exciting cause in more cases of chorea in girls than in boys. Of the 43 cases recorded above, 34 were females and 9 males.

X. *Relation of chorea to rheumatic fever.*—A definite history of rheumatic fever was obtained in 88 cases of the series of 327 in-patients, or 26.9 per cent., a result which corresponds very closely with the 26 per cent. given by S. Mackenzie¹ in his Report of the Collective Investigation Committee. Of these 88 cases, 28 were males and 60 females, which gives, therefore, a history of rheumatic fever in 35 per cent. of the males, and 24.3 per cent. of the females in this series.

In the out-patient series rheumatic fever had occurred in 52 cases, or 19 per cent., and there was a history of vague joint-pains in 32 cases besides, or 11.7 per cent. Of the 52 cases with previous rheumatic fever, 16 were males and 36 were females, giving a percentage for males of 22.2 per cent., and for females of 17 per cent.

In both series it will be seen that a history of rheumatic fever is present more often in males than in females. There is also a very considerable difference between the out- and in-patient series as regards previous rheumatic fever, the percentages being 19 and 26.9 respectively. The difference is not so marked if we include the cases with vague joint-pains.

These figures show that the severer forms of chorea are more frequently associated with a definite history of rheumatic fever than are the less severe out-patient cases.

(I.) *Seasonal relation.*—Dr. C. P. Phillips has kindly furnished me with the accompanying chart (III.), showing the months of attack in 166 cases of rheumatic fever admitted as in-patients at St. Bartholomew's Hospital during the twelve years 1882–93 inclusive. The thick line represents *all* attacks, and the thin lower line the first attacks alone.

From these curves it will be seen that the maximum number of cases occurs in October.

Dr. Gabbit's² analysis of 2000 cases of rheumatic fever

¹ S. Mackenzie, Report of the Collective Investigation Committee. Brit. Med. Jour., February 26, 1887, p. 425.

² Gabbit, Seasons and Prevalence of Acute Rheumatism. Lancet, October 1883, p. 675.

admitted to the London Hospital from 1873 to 1881 shows the maximum to occur in November; but it must be borne in mind that he takes the date of admission, and not the date of incidence, as Phillips has done. We may fairly consider, therefore, that October and November are the months during which rheumatic fever is most prevalent in the East End of London. Statistics for various parts of Europe show that in the majority, rheumatic fever is most prevalent in the winter months, though, as in the table given by Garrod,¹ wide discrepancies are to be found in some cases—in Paris, for instance, Besnier's figures give the greatest frequency in the summer months.

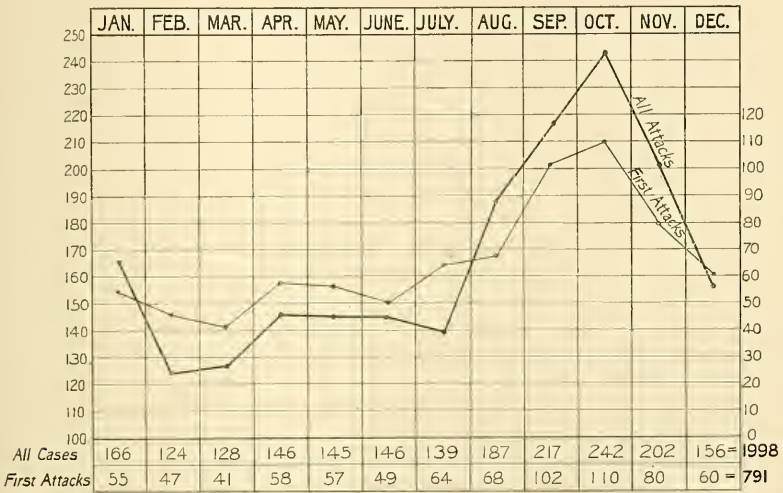


CHART III.—Showing the number of cases of rheumatic fever admitted to St. Bartholomew's Hospital during the twelve years 1882-93, arranged in months according to the time of onset of the disease.

If we now compare the rheumatic fever curves (Chart III.) with the chorea curves (Charts I. and II.), we find that the maximum number of cases of chorea occurs in December, January, and March, that is to say, in the months following the period of greatest prevalence of acute rheumatism. I consider that the frequency of chorea in December, January, and March is very largely dependent on the prevalence of rheumatic fever in November and December.

On the other hand, Morris found in his Philadelphian series that the curve for rheumatic fever, though it follows that for chorea with great regularity, attains its maximum in April, or

¹ A. E. Garrod. *A Treatise on Rheumatism*, p. 39.

one month later than does the chorea curve, so that he does not regard rheumatic fever as the predisposing factor. He considers rather that both diseases are influenced by some common cause, probably by the "weather." It is pure speculation, but one is drawn to the suggestion that possibly this common cause, which we may call "rheumatic poison," finds a more favourable soil, and therefore germinates more rapidly in the nervous system than in the fibro-serous tissues of the American people, who are often supposed to be pre-eminently highly strung and nervous.

It would be of interest to ascertain the months of greatest frequency of chorea in cities which have different months of greatest prevalence of rheumatic fever, but I have been unable to collect any statistics of value on this point.

(2.) *Frequency of heart affections.*—In 273 out-patients, heart murmurs were present in 61 = 22 per cent.; in 327 in-patients heart murmurs were present in 161 = 49.2 per cent. The great difference between these two results is what might have been expected from the difference in severity of the disease in the two series of cases. Taking both series together, in 600 patients heart affections were present in 222 cases = 37 per cent. Mackenzie in 439 cases found heart affections in 32 per cent. Hughes gives 50 per cent.

If we compare with this the frequency of cardiac affections in rheumatic fever, we find the figures do not differ so widely as one would have expected. Peacock¹ found heart disease present in 40 per cent. of first attacks of rheumatic fever, and 36.7 per cent. of the cases of recurrent attack. Church,² in an analysis of nearly 700 cases of rheumatic fever, found the heart affected in 56 per cent. of first attacks. West³ found 67 per cent. with cardiac disease in 1107 cases. Southey found cardiac complications present in 29.8 per cent. of a series of 3552 cases, a much lower percentage than is given by the majority of observers in this country.

(3.) *Mortality in chorea as compared with rheumatic fever.*—In the series of 327 cases of chorea admitted to this Hospital during ten years, seven deaths occurred = 2.1 per cent. Of these, six were females and one a male, giving a percentage of 2.4 death for females and 1.2 per cent. for males. The average age of the females who died was 13.7, and the male was 15 years of age. Mackenzie, in his analysis, puts the mortality at 2 per cent.

¹ Peacock, St. Thomas's Hospital Reports, 1873, p. 289.

² Church, St. Bartholomew's Hospital Reports, vol. xxiii. 1887, p. 269.

³ S. West, Practitioner, 1888, vol. xli.

During twenty-five years, 1871-95 inclusive, there have been 26 deaths from chorea, 22 females and 4 males, a proportion of 5.5 to 1; the average age of the females being 14.7, and of the males 12.3 per cent.

An examination of the post-mortem records of St. Bartholomew's Hospital shows that in by far the greater number of fatal cases there was well-marked and extensive endocarditis with pericarditis, and that in comparatively very few cases was the heart lesion limited to the beading of the edges of the mitral valve, which is generally supposed to be characteristic of chorea. This statement is supported by the table of fatal cases given by Dr. Sturges. Those cases which present this slight endocarditis are associated with very violent movements, not infrequently with pyrexia, and occasionally with pregnancy. As Sturges pointed out, death *from* chorea is very much rarer than death *with* chorea associated with extensive rheumatic heart disease. The mortality in rheumatic fever does not differ very widely from that in chorea. Southey,¹ in an analysis of a very large number of cases treated at this Hospital, placed it at 1.7 per cent.; Church at 1.307 per cent.; Pye-Smith gives 3.7 per cent.

It is interesting to note that whereas in chorea the female sex is far more liable to the disease and suffers more severely than the male, in rheumatic fever males suffer in somewhat larger numbers, though on this point statistics differ. The slightly greater liability of males is probably accounted for, as suggested by Dr. Fagge,² by the greater amount of exposure to which they are subjected, while, on the other hand, the sensitive nervous temperament of the female may render that sex more especially liable to chorea.

(4.) *Family history of rheumatic fever in chorea.*—In 273 out-patients there was a history of rheumatic fever (not rheumatism), either of the father or mother or of a brother or sister, in 70 cases = 25.6 per cent. Of these 70 cases, 52 were females and 18 males, which gives a percentage of 25.8 females in the total of 201 females dealt with, and of 25 per cent. males in the 72 males of this series. The figures for the in-patient series cannot be given with a sufficient degree of accuracy, as in some cases the family history is not stated, or in others is not sufficiently definite.

From the figures given above, therefore, it would appear that, given a strong family history of rheumatic fever, the sexes are almost equally liable to chorea.

¹ Southey, St. Bartholomew's Hospital Reports, vol. xiv. 1878, p. 1.

² Fagge, Principles and Practice of Medicine, 1888, vol. ii. p. 816.

The statistics which have been given in this section strongly support the view that chorea is a manifestation of rheumatic fever, in which the nervous system bears the brunt of the disease, and is analogous to erythema, tonsillitis, arthritis, and endocarditis. It is impossible to go further in the present state of our knowledge, seeing that we know neither the specific cause of rheumatic fever nor the way in which the cause produces its effect. A. E. Garrod¹ has suggested that chorea may bear a similar relation to rheumatic fever that paralysis does to diphtheria; but without entering into any discussion of this hypothesis, we are fully justified in entertaining the view that a very intimate relation exists between rheumatic fever and chorea.

Rheumatic fever may in some cases give rise to a persistent modification of the tissues and fluids of the body, during which an adequate exciting cause can produce either an attack of chorea or of rheumatic fever.

GENERAL CONCLUSIONS.

I. *Sex.*—The liability is in the proportion of three females to one male, the proportion being practically the same both in severe and in mild cases.

II. *Age.*—The greatest liability is at ten years of age both for first attacks and for all cases, taking mild and severe cases together. In mild cases (out-patients), 50 per cent. of the cases occur from eight to eleven years inclusive. Ten years is the age of greatest liability for first attacks in mild cases. In severe cases (in-patients), the age of greatest liability for all cases is fifteen years. This points to the great influence of puberty as an exciting factor.

III. *Race.*—Chorea is practically unknown among the coloured races. Rheumatic fever, on the other hand, is a common disease among them.

IV. *Locality.*—Chorea is prevalent in large cities, rare in rural districts. It is not so universally distributed as rheumatic fever.

V. *Seasonal variation.*—The maximum number of cases occurs in December and January for out-patients, with the minimum in August. For in-patients the maximum number of cases occurs in March, the numbers in December and January being fewer. The lowest numbers occur in June and July. This periodicity is the same both for primary attacks and for all

¹ A. E. Garrod, *Brit. Med. Jour.*, January 11, 1896, p. 71.

attacks, therefore the exciting factors are probably the same both for first and for recurrent attacks.

VI. *Meteorological elements.*—1. Temperature. The frequency of chorea varies inversely with the mean monthly temperature. 2. No relation can be found between the daily range of temperature and the frequency of chorea. 3. Humidity. The frequency of chorea varies directly with the mean degree of humidity of the air. With regard to (4) Rainfall and (5) Barometric pressure, no relation between chorea and these factors can be shown to exist.

VII. *Recurrence.*—In 600 cases there was recurrence in 34.5 per cent. Recurrence occurs more frequently in mild than in severe cases, and recurrent attacks are generally less severe than primary attacks. Sex liability in recurrent attacks is the same as for primary attacks.

VIII. *Educational pressure.*—This affects children below puberty in the majority of cases.

IX. *Fright.*—This was the exciting factor in 15.7 per cent. of the out-patient cases. Fright is more liable to cause chorea in girls than in boys.

X. *Relation of chorea to rheumatic fever.*—1. A past history of rheumatic fever is more common in in-patients than in out-patients, and in males than females. 2. Seasonal relation. There is a distinct relation between the monthly frequency of the two diseases, though this is less marked in the in-patient cases. 3. Heart affections are more than twice as frequent in the in-patient series as in the out-patient cases. The frequency of heart disease in chorea is not very much less than it is in rheumatic fever. 4. The mortality in chorea does not differ widely from that in rheumatic fever. The majority of fatal cases of chorea present post-mortem well-marked endocarditis, and generally pericarditis. The mortality in chorea is greater in females than in males. 5. A family history of rheumatic fever does not affect the sex liability to the disease.

XI. That chorea is a manifestation of rheumatic fever affecting the nervous system.

SOME CASES OF DEFORMITY.

BY

ARTHUR HEATH, M.B. (LOND.).

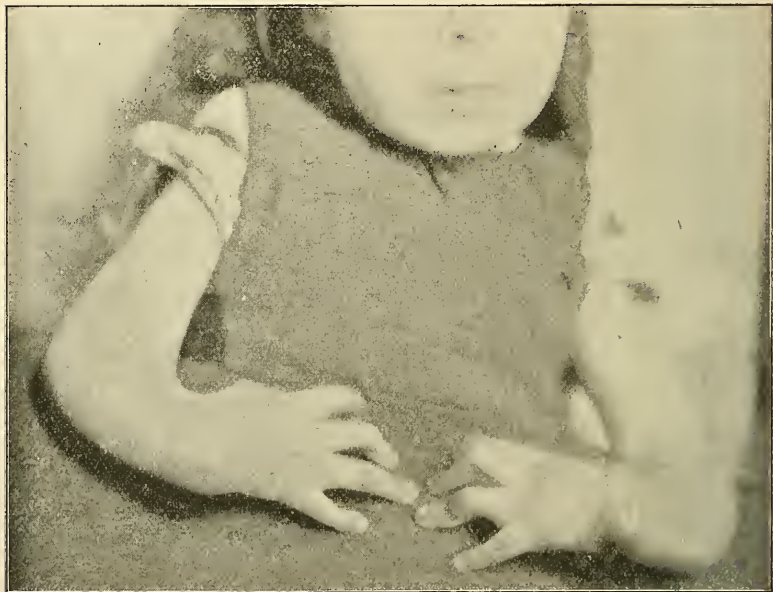
Congenital Absence of Radius as a One-Family Failing.

I have lately met with this troublesome omission in four members of the same family. The notes of two of the cases are as follows:—

R. C., a girl, aged 8, has no radius on either side; the ulna has not grown to its natural size, so that the length of the forearm is much reduced, the distance between the elbow and wrist joints being only four inches. The humerus is well developed. When the limb is at rest the hand lies in the prone position, and is drawn over to the radial side, so that instead of being in a line with the forearm it is at right angles to it. The end of the ulna at the wrist is prominent, the movement at the joint being quite free. Electrical reactions showed that the triceps and biceps were present and acted normally, the latter being apparently attached to the ulna. The supinator longus and flexor carpi radialis are present, and it is by these that the hand is held over to the side of the missing bone. Thus the muscles, curiously enough, are present on the side of the absent radius, while the flexor carpi ulnaris was not found, nor were the extensors of the wrist. The extensors of the fingers acted poorly, the flexors of the fingers strongly. The interossei and thenar and hypothenar muscles were present. The fingers are weak, and this seems to be partly due to the deficiency in power of the extensor muscles; the child, however, can make good use of the hands, and can even write by holding the pencil between the middle and ring fingers, and grasping it with the thumb below. She can raise the hands to the head, the chief difficulty being to reach objects which are to her side.

B. C., aged 2, and sister of the last case, presents a corresponding deformity—absence of radius on both sides, but she is

rather worse, for the fingers of the left hand are webbed, and the little finger wanting. The position in which the hands are held over to the radial side, and the deficient growth of the ulna, are exactly similar to the preceding case. The legs of this child are of chief interest, for each is rotated on the femur, so that the head of the fibula comes to be to the front in the middle line, and at first gives one the impression that it is a small patella, for the patella itself is absent on both sides. Owing to this rotation, the foot points inwards, and the child cannot be taught to walk, and has never been put on its legs at



all; the left leg is rotated a little more than half way round. (The photograph shows the position.) The fibulae are present throughout, and the malleoli stand out clearly, the foot not being in any way deformed. The muscles seem to be all present, but they have been so little used that they are in an ill-nourished condition, although the child is fat and well. The legs cannot be straightened at the knee-joint; this seems to be partly due to the head of the fibula coming into contact with the lower end of the femur, and partly to the contraction of the ham-string muscles from the legs having been constantly curled

up; for when an attempt is made to straighten the limb these tendons become very tense. The feet can be brought forward and held in the natural position by taking the limb and rotating it outwards at the hip-joint to the full extent. The legs have been put in plaster with the object of overcoming the flexion at

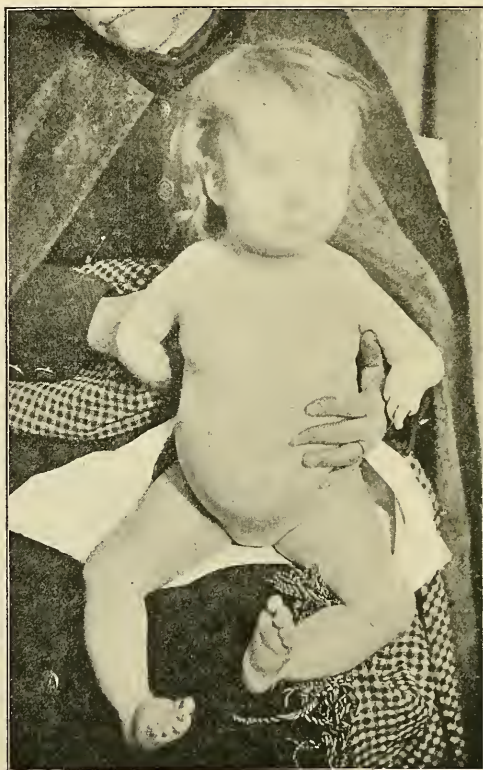


the knee, with a view to subsequent operation by osteoclasis to rotate the legs, so that the feet may be brought into such a position that walking will be possible.

Family history.—The mother of these children has had two families. Three children by the first husband were normal. Of six children by the second husband, four have had no radii.

Three of the four have been girls, but one was a boy. They were born in this order:—

Eldest, girl	.	.	No radii, dead.
Second, girl	.	.	(First case above, R. C.)
Third, boy	.	.	No radii, dead.
Fourth, boy	}	.	Normal.
Fifth, girl	}	.	
Sixth, girl	.	.	No radii (second case above).



Thus two normal children have been born in between. Since the first family escaped, it would appear that the father is the responsible parent. He rather indignantly denies the history of bony deformities of any kind in his family, nor is there any such history on the mother's side.

Osteitis Deformans Beginning in Early Life.

Miss F. is a young lady of 35 years of age, presenting extreme and very distressing deformity of all her limbs, so that she is rendered helpless; she cannot walk, but when she is alone can just manage to get about the room on her hands and feet. In the legs the tibiae are bowed forwards and very massive: the skin over them shines. The femora are also enlarged and curved, but at their lower ends are large prominences of bone. These quite prevent straightening of the knee-joints, and render walking impossible. The arms exactly correspond with the legs in the curving and enlargement of the bones, and also in the fact that the most marked increase of bone is at the lower end of the humerus, for besides the great increase in the size of the bone there are here superimposed bosses, some of which stand out almost like exostoses. At times the patient has a good deal of pain in these bones; they seem to actively inflame and become swollen. When the inflammation subsides the swellings diminish again, but with each attack the permanent condition is worse. The patient has not noticed any enlargement of her head, nor has she had any pain there, but the forehead is rather prominent, and there may have been considerable increase without its having been observed. When she was a child she was very liable to break her bones, and she had several fractures from slight accidents. The unaffected bones are small and delicate, and the patient is naturally of a frail build. She first noticed this permanent overgrowth when twenty years of age, and it has gradually increased. There seems to be no doubt that she is suffering from osteitis deformans, which is usually a disease of later life. The inflammation of the bones, though aggressive and lasting, is subject to paroxysmal increase in severity, particularly at the lower ends of the humeri and femora, and this is the time when they are particularly painful.

Arrest of Development of the Lower Jaw.

R. S., a boy, 18 years of age, shows suppression of growth of the inferior maxillary bone. The deformity is very obvious, for he has no chin at all; it is necessary to feel with the finger to find the border of the bone, as it does not in any part stand out from the soft parts, but is a mere ring of bone in the floor of the mouth. It appears to be quite complete, but simply dwarfed, of the size of a young child's jaw, and very thin. There has not been sufficient room for the teeth side by side,

and the lateral incisors have grown up behind the central ones, while the canines come forward next to these.

The jaw cannot be brought forward to permit of upper and lower incisors coming together; when the attempt is made, the lower are still an inch posterior; the lips, however, meet, and he speaks clearly; he is also able to eat with fair comfort, the chief complaints being inability to open the mouth properly, and the personal appearance. No adequate cause can be given for this deficiency in growth, and it would seem to be an hereditary tendency. The mother has had much difficulty with her teeth owing to the smallness of her jaws, but in her it is not so noticeable from her appearance; her teeth have had to be removed, and the dentist has fitted her with artificial ones of about half the ordinary size. The patient is the only son. Two girls do not show the defect.

Case of Probable Nerve Injury at Birth.

F. P. is now 3 years of age; when three months old it was noticed that the left arm was little used, and that the child never moved the shoulder. He was treated then for a doubtful fractured clavicle by bandaging the arm across the chest. Now he cannot raise this arm, but to get it up to the head assists it with his right hand. The deltoid is markedly wasted, and, to a less degree, the triceps also. The biceps has contracted, and will not admit of complete extension of the elbow; its tight tendon is felt to be the cause when the attempt is made. The left arm is shorter than the right, the measurement from the point of the shoulder to the flexure of the wrist shows 2 inches less on the left, of which $1\frac{1}{4}$ inches is accounted for by the arm and $\frac{3}{4}$ inch by the forearm. The whole left arm when it lies naturally is rotated inwards at the shoulder-joint, so that the palm of the hand is directed outwards, and though pronation and supination are completely performed, the child *apparently* does not thoroughly supinate owing to this rotation at the shoulder, which appears to be due to the action of the subscapularis unopposed by the external rotators. The question arises as to whether this partial disablement of the limb is sufficient to account for the deficient growth of the bones, or whether this, like the paralysis, is due to nerve injury at the time of birth (it was a breech presentation, attended by a midwife, and difficulty was experienced with the birth of the arms). It is possible that there was at the time injury to the epiphysis of the humerus.

Overgrowth Resembling Acromegaly.

A. D., a boy, aged 13, has hands and feet which, if he were older, would be diagnosed as acromegaly without doubt. The hands are no longer than natural, but overgrown in every other way, and the soft parts share as much as the bones in the enlargement; their shape is characteristically spade-like. The forefinger measures 3 inches round. The feet are enormous considering the age of the boy, and are quite symmetrical. The great toe measures $4\frac{1}{4}$ inches round and is adducted from the second toe; the second and third toes are webbed half way up. The boy is well developed about the chest, and no enlargement of other bones was detected. The circumference of the head is $22\frac{1}{4}$ inches. He suffers from cold hands, and says they are often blue in mild weather. His testicles are absent from the scrotum. He is myopic. His intelligence is quite up to the standard of his companions of the same age, and his skin is natural.

THE PATHOLOGY OF ALCOHOLIC PARALYSIS.

BY

HOWARD H. TOOTH, M.D.

Alcoholic paralysis or paraplegia, alcoholic polyneuritis, alcoholic pseudo-tabes, or peripheral neuro-tabes—these are a few of the synonyms of this disease. As the bibliography at the end of this article shows, the literature which has sprung up around this subject is considerable. The history of the disease has been told so often in all languages, that it would be needless repetition to rewrite it here, but the chronological arrangement of the appended bibliography will be sufficient guide to any reader who may wish to pursue such a study.* Good historical summaries will be found by Ettinger,³⁸ Payne,⁶⁴ Lafitte,⁸⁹ and H. Gudden,¹⁰⁸ and in fact most writers appear to have thought it necessary to discuss the bibliographical history of the disease more or less completely.

In the present article it is intended to keep to the pathological aspect of the question. As might be supposed, the earlier writers are either silent on the question, or vaguely assume the spinal origin of the disease.

Lancereaux⁸ in 1864 first definitely described a degenerated state of the peripheral nerves, and from that time writer after writer has noted similar lesions, as a rule, with integrity of the spinal centres and roots.

A large group of observers have adopted the view that the lesion is of a local inflammatory nature, caused by the circulation of the poison in the blood, a belief which is implied in the largely used term *neuritis*. Certain observations exist which lend colour to this view—Hadden,³⁴ Finlay,⁵³ Minkowski,⁶³ and some few others, who describe an actual increase of nuclei around the fibres, amounting to a sclerosis. But these are few compared to the great bulk of the recorded cases, in which the writers agree, tacitly or expressedly, that signs of active inflammation are generally absent, hence the term “degenerative

* The reference figures in the text refer to the bibliography.

neuritis" is very common. The intra-muscular nerve fibrils have scarcely received the attention one would have expected. Some, however (Thomson,⁸⁰ Gudden,¹⁰⁸ &c.), have described proliferation of nuclei in the perineurium along with degeneration of these fibres, and Siemerling⁷⁴ records an inflammatory condition of the muscle fibres (myositis).

In 1883 Erb²² made the important suggestion, on theoretical grounds, that the peripheral degenerative lesion might be secondary to "functional disturbance of the trophic centres of the cord." This view has been favoured by several writers, and among them may be mentioned Eisenlohr,* Babinski,† Brissaud,⁸² Goldscheider, and Moxter.‡

By far the larger number of writers, however much they may differ as to the origin of the lesion, agree that the cells of the anterior horns and posterior root ganglia are free from any degeneration to be discovered by the methods most commonly in use. Nevertheless, several cases are recorded in which the cells of the anterior horns were obviously affected. Sharkey,⁶⁵ Erlitzki,⁷¹ Schaffer,⁷³ Wilkin,⁷⁵ Reynolds,⁷⁰ Achard and Soupalt,⁹² Campbell,⁹⁵ Déjerine and Sottas.¹⁰⁷

Several writers also notice unusual pigmentation of cells, and some a vacuolation (Schultz⁴⁰).

It would appear that there remains yet much important work to be done in connection with the cells of the grey matter of the cord in these cases. By the use of certain basophile aniline stains most valuable information may be obtained concerning the nutrition of the cell, which can be obtained in no other way. One of the earliest degenerative changes in these cells is the disappearance of certain granules, mostly heaped about the nucleus, which have a special affinity for the basic dyes, such as methylene blue in the method of Nissl. A profound alteration, therefore, may be discovered by such means in a cell which would, under the ordinary methods, present quite natural appearances.

This has been demonstrated by Marinesco§ in a case of probable alcoholic paraplegia, but complicated by rheumatism. Again, in a case of atrophic paralysis of one leg,|| he found

* Eisenlohr: Ueber progressive atrophische Lähmungen, ihre Centralen oder periphere Natur. *Neurol. Centralbl.*, 1884, S. 145.

† Babinski: *Anatomie Pathologique des Névrites Périphériques*. *Gaz. Hebdom.*, 1890, p. 374.

‡ Goldscheider and Moxter: *Polyneuritis und Neuron-Erkrankung*. *Fortschr. der Med.*, Bd. 13, 1895.

§ Marinesco: *Les Polynévrites en rapport avec la Théorie des Neurones*. *Compt. Rend. Soc. de Biol.*, 1895, tom. ii. Ser. 10, p. 765.

|| Marinesco: *Sur un Nouveau Cas de Polynévrite avec Lésions de réaction à distance dans la Moelle épinière*. *Compt. Rend. Soc. de Biol.*, 1896, tom. iii. Ser. 10, p. 497.

by Nissl's method marked changes in the corresponding cells of the lumbar swelling, the general form unaltered, but the nucleus at the side of the cell of an ellipsoidal, not spherical contour, with disappearance of chromatophilic substance.

But this writer holds the view that the alteration of the cell is secondary to lesion of the fibre or neuron, and he shows by experiment* that such central changes follow section of a nerve trunk. This, however, seems to be only another proof of the oneness of cell and neuron, which has become more and more an article of belief. It has, of course, been long known that central cells waste and disappear after amputation of limbs, and this may be the earliest stage in the process.

It must be confessed, therefore, that the latest researches in this direction do not seem to solve the problem: the changes in the cells may be claimed by either party.

The more one considers the question the more reasonable does the theory become, that alcohol is a toxic material, resembling in some respects the metallic toxins, *e.g.*, lead, arsenic, &c.; or the bacterial toxins, *e.g.*, diphtheria, &c. Alcohol acts upon cell and neuron, and profoundly interferes with the nutritive power of the cell, so that it is no longer able to maintain its long neuron † (in the lower extremities it may be nearly 3 feet long). Hence follows paralysis, at first rapidly recoverable from on removal of the toxic cause, but later actual *degenerative* processes at the point farthest from the parent cell, which degenerative lesions gradually creep up the neuron towards the centre. Supposing all cells of the cord to be equally liable to nutritive defects, then the longest neurons will suffer first, and therefore those of the lower extremities, which is in accordance with the facts.

Whether the patient recovers from this stage depends upon his general recuperative powers, and the nerves, or rather centres, affected. The fatal cases are often those in which the vagus and respiratory centres suffer early; but in some cases the resisting power is so great that the muscles may be completely wasted, and then post-mortem may be found not only degeneration, but complete disappearance of fibres, which may possibly be replaced by sclerotic tissue with nuclear proliferation—an effect, not a cause.

For the sake of simplicity we have been considering only the motor side of the question. The pains, anæsthesia, hyper-

* Marinesco: Des Lésions Primitives et des Lésions Secondaires de la Cellule Nerveuse. Compt. Rend. Soc. de Biol., 1896, tom. iii. Ser. 10, p. 106.

† This idea was well expressed by Dr. Mott at the discussion on "Neuritis" at Carlisle last year.

æsthesia, and other sensory symptoms may be reasonably explained in the same way by supposing a similar toxic influence on the cells of the posterior root ganglia. And, in fact, the selective action of alcohol in some cases seems to be much more easily explained by action on the central cells than by any theory of inflammation of peripheral nerves. In some cases we find the motor paralysis predominating, in others the sensory, with a clinical picture indistinguishable at first sight from tabes (alcoholic pseudo-tabes).

The following cases present several points of interest in connection with the questions above discussed:—

CASE I.—*Chronic Alcoholism.*

The history of this case is short and incomplete, but significant enough.

Sarah R., aged 44, was admitted on May 14, 1894, to Hope Ward, under Dr. Gee. She was moribund on admission. From the medical man who had attended her it was ascertained that she had been a great drinker for four years. She had had delirium tremens three times in that period. For some months she drank a “bottle of whisky and one of brandy” every day. For three months she had suffered from muscular pains in all parts of the body, with wasting of muscles (? which) and morning sickness. The knee-jerks had been absent during that time.

For one week she had been unconscious, passing urine and fæces under her. No physical signs had been made out in the lungs, but the heart was suspected to be fatty. Bowels generally loose. Temperature always normal.

Family history.—She is the mother of one child, now aged 21. Her husband does not drink. Her father, one sister, and three brothers all “died of drink.”

On admission she was quite unconscious, with cold extremities and imperceptible pulse. In spite of ether and strychnine injections, the heart ceased to beat in about half an hour.

Post-mortem (Register No. xxi. 100) made on May 15, twenty-two hours after death.

Body fairly well nourished. Height $62\frac{1}{2}$ inches.

Brain, 35 oz.; apparently somewhat shrunken, with a considerable amount of cerebro-spinal fluid. Choroid plexuses very cystic.

Spinal cord.—Macroscopic appearances natural.

Lungs.—Right, $14\frac{1}{2}$ oz.; left, 11 oz. Right puckered at apex. Both somewhat emphysematous.

Heart.— $8\frac{1}{2}$ oz. No valvular disease. Milk spot on epicardium. Muscular substance soft, and of a patchy brown appearance. The blood smells strongly of alcohol.

Liver.— 81 oz.; large. Surface smooth on convexity, but on the lower aspect of the lobes rather roughened. Division into lobules not distinct.

Spleen.— 30 oz.; flabby and soft.

Kidneys.—Right, 6 oz.; left, 9 oz. Cysts on surface of left. Capsule retracts on incision. Surface granular on peeling capsule. Cortex yellow, swollen, patchy, and evidently fatty.

Microscopical examination.—*Spinal cord.*—Slices were taken from the cord at the levels of the following roots: Cervical, i. and vi.; dorsal, i. and vi.; and lumbar, i., and treated by Marchi's method. In spite of the rather long time (twenty-two hours) which had elapsed between the time of death and the post-mortem examination, the cord was well preserved, and there was little evidence of decomposition. The white matter appeared to be perfectly natural, and there was no evidence of degeneration, old or recent. There was also no degeneration of the myelinated fibres of the grey matter. Generally speaking, the cells of the anterior horns presented natural appearances—that is, their contour was perfect, their processes were sharp, and could be traced sometimes for a considerable distance. In some sections, however, the pericellular spaces were large, but this might be due to a shrinkage of the cells under the reagent. Here and there, also, may be seen a cell which is devoid of processes; this appearance, however, might possibly be explained by the plane of the section as regards that particular cell. All ganglion cells, whether in the anterior or posterior horns, or in the posterior vesicular column, are deeply loaded with pigment. This pigment, in some cells, is aggregated into a mass on one side of the cell, in others the pigment seems to occupy the whole cell; but, as a rule, by careful focussing, the nucleus and nucleolus can be seen in optical section. If the pigmentation then is a diseased condition, it has not led to a disorganisation of the anatomical features of the cell.

Peripheral nerves.—Pieces of the following were taken for examination: Left ulnar, median, and radial; right median; right phrenic; right and left vagus; left great sciatic; left small sciatic; left popliteal, and left posterior tibial. They were hardened in Müller's fluid, and longitudinal sections were made and stained in Weigert-Pal hæmatoxylin and alum carmine. By this method no degeneration or morbid change could be discovered in any of the above nerves, except in the posterior tibial. In all these nerves might be seen a

slight beading of the fibres here and there, but no more than might be accounted for by softening by decomposition and possible injury in removal.

In the posterior tibial there are, however, a few fibres broken up, with droplets of myeline taking their place in the sheath. This appearance is particularly well brought out by staining in Marchi's fluid, and then further staining in Weigert-Pal hæmatoxylin, a method recommended by Professor Schäfer (see Fig. 1). But in the study of degeneration the osmic acid method of Marchi has been of the greatest use. This reagent stains all free fat a deep black, but does not affect the myeline. One effect of degeneration is to cause, by a chemical decomposition, the liberation from the myeline of the fat, which then is acted upon by the osmic acid. Now, though the *form* of the fibre is altered as above described, it does not show any special affinity for osmic acid. This would suggest either that the liberated fat had been very rapidly absorbed by the blood-vessels, or that the appearances were not due to degeneration at all, but to rough handling and decomposition. There is no evidence whatever, in any of the preparations, of any inflammatory process. No increase of nuclei either of the primitive sheaths or of the connective tissue between the fibres.

Summary.—Even though the history is so meagre, there can be no doubt that the woman had been suffering from alcoholic paralysis for some months, as evidenced by the pains, muscular wasting, and loss of knee-jerks. Examination of the cord gives negative results, except for the pigmentation of the cells, to be discussed hereafter.

The nerves are generally natural, except for slight changes in the posterior tibial, which may possibly be of post-mortem origin. It is much to be regretted that the ultimate nerve fibrils were not examined.

CASE II.—*Chronic Alcoholism—Paraplegia—Pains in Limbs—Syncope Attacks—Fatty Cirrhosis of Liver.*

Emma H., aged 41, admitted on January 10, 1894, to Mary Ward, under Dr. Hensley.

History.—She is a spirit-drinker, quantity not ascertained. She has been ailing indefinitely for eighteen months. For six weeks she has been subject to vomiting, with pains in chest and back, apparently dyspeptic. For the same time she has been weak in her legs and easily tired; this has increased so that the gait became dragging, and now she is unable to stand even. This paralysis has been accompanied by pains more or

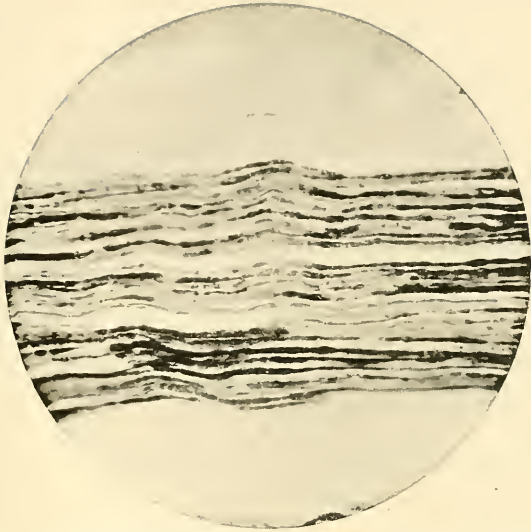


FIG. I.—A microphotograph of the posterior tibial nerve of Case I., stained by Marchi's and Pal's methods combined. A few of the fibres are swollen with broken-up myeline, but the segmentation and unequal staining may be due to post-mortem changes.

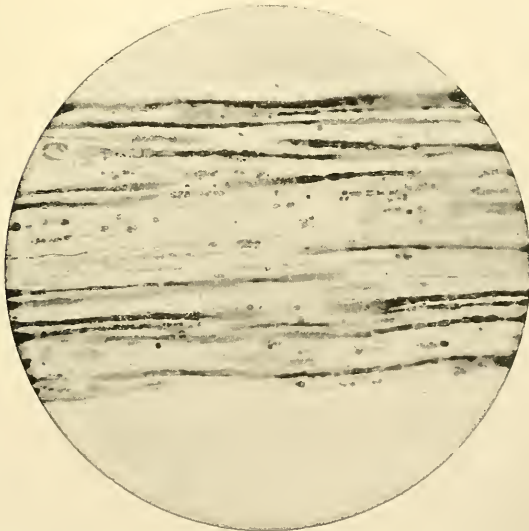


FIG. II.—From the anterior crural nerve of Case II., stained by the same methods as in Fig. I. A very extensive cleavage of the myeline and disorganisation of the fibres. The droplets of myeline do not stain black by the Marchi method alone, and therefore may be due to the liquefaction of decomposition.

less severe in the legs. She has taken to her bed for the last fourteen days.

Family history.—Father died of bronchitis, and had rheumatism. Mother died of gangrene (? of what), and suffered from asthma. Sister died of bronchitis. She is married, has had three healthy children and no miscarriages.

On admission.—She looks sallow and pinched, with stigmata on the cheeks. Expression dull and vacant. Tongue coated with a thin fur. Speech slow and hesitating. Memory poor. Generally listless; movements slow and tremulous.

Lungs.—Some rhonchus at left base.

Heart.—Natural.

Abdomen.—Tender all over, especially on the left side.

Lower extremities.—Cannot stand. Knee-jerks so feeble as to be noted almost absent. Legs very tender when handled.

Subsequent history.—January 12.—The pains in the legs are so severe as to keep her awake at night. The electrical reactions are normal to faradism and galvanism.

February 7.—Quite “off her head.” Arms, rigidity on extension, which causes pain.

February 17.—Temperature rose suddenly to 102.4° F.

February 21.—An attack of syncope. Resonance impaired. Right base looks dusky.

Died February 25 at 1.15 P.M.

Post-mortem examination.—(Register No. xxii. 53), made on February 26, twenty-three hours after death.

Body generally wasted. Height 60 inches.

Brain.—36 oz.; not apparently wasted.

Spinal cord.—Natural to naked eye.

Lungs.—Right, 20 oz.; left, 14 oz. Right lung shows in the upper lobe the mottled appearance of broncho-pneumonia.

Heart.—10 oz.; nothing abnormal.

Liver.—56 oz.; fatty and cirrhotic. Very tough in its consistence, but not granular on the surface.

Spleen.—8 oz.; rather large and firm.

Kidneys.—11 oz. the two. A large retention cyst in the right in one of the pyramids.

Uterus.—Very small.

Microscopical examination.—*Spinal cord.*—Slices were taken from the cord at various levels and treated by Marchi's method. The cells of the anterior horns are heavily pigmented. This pigment does not require osmic acid to show it; it is well seen, though of a more yellow colour, in sections stained by acid fuchsin.

In the cervical and lumbar enlargements some of the anterior

root bundles as they pass through the anterior root zone of the cord show a considerable breaking up of the myeline which takes the osmic acid stain very deeply, presenting rows of characteristic black masses. These masses can be seen streaming into the anterior horns, and in one or two sections can be also seen in the anterior commissure. So typical are they of degenerated fibres that one could scarcely have any doubt as to their pathological nature, were it not for the fact that they cannot be traced in most of the sections into the anterior root; this apparent degeneration stops sharply at the sheath of the cord. There is a similar but less marked appearance in the posterior root zone; but in the region of the cauda equina the change may be traced a short way into the posterior roots. It is probable therefore that this is a post-mortem change.

Peripheral nerves.—The left phrenic, vagus, ulnar, sciatic, anterior crural, and posterior tibial were examined in longitudinal sections and stained by the Pal hæmatoxylin and Marchi's methods, and also by a combination of the two. By the first-named method the fibre presented a peculiar mottled appearance, due apparently to an irregular cleavage of the myeline. By Marchi's method there was the same appearance, but no characteristic black staining suggesting the presence of free fat, but a uniform yellow tint. By combining the two processes, however, a very deep black stain is produced, and we then see that many of the fibres are profoundly affected, broken up into irregular black masses (see Fig. II.). Others are simply beaded in a multiform manner, and others apparently natural except for a transverse cleavage, which may be due to the reagents.

Summary.—There can be little doubt that this is a case of alcoholic paraplegia. Certain changes are found in the fibres of the anterior horns, and to a less degree of the posterior; but reasons are given for supposing them to be post-mortem changes. The peripheral nerves examined show very marked histological changes; but these alterations are of form rather than of chemical constitution, for by the Marchi method we find none of the black-stained masses which we should expect if the myeline had been broken up into a free fat, as is generally the case in degeneration. Either we must suppose that this fat has been absorbed, or that the changes in alcoholism differ very essentially from those of ordinary "Wallerian" degeneration, or that the alterations in form are post-mortem "artefacts," due to unconscious rough handling in removing the nerves, softened somewhat by decomposition.

CASE III.—*Chronic Alcoholism—Recent Paraplegia—Pains in Legs, with Patches of Anæsthesia—Sarcoma of Ovary, with Secondary Growths—Death Sudden.*

Ellen M., aged 43, admitted on April 11, 1895, to Faith Ward, under Dr. Church.

History.—Has been addicted to dram-drinking (rum) for two years. Seven weeks before admission she began to suffer from pain in the left side of the chest, which was aggravated by the movements of respiration. Then the pain seemed to appear in the left shoulder. For fourteen days she had had pain across the loins so severe as to prevent her from moving. There has been no cough, shivering, diarrhœa, nor vomiting.

She had rheumatic fever twenty-one years ago, and “pleurisy” five years ago.

On admission.—A large stout woman, of a neurotic temperament. Complexion reddish, face rather bloated. Pupils react to light. She is deaf in the left ear. Tongue red and dry. Skin “satiny.” She complains of pains in the back, and pain or tenderness in any part that may be touched.

Chest.—Well covered. Movements limited. Emphysematous. Slight rhoncal and sibilant sounds in both lungs.

Heart.—Apex beat not palpable. Cardiac dulness made out with difficulty, to extend upwards not beyond the fourth rib, and inwards to the left side sternal line. No murmur heard.

Abdomen.—Large, fat, and flabby.

Legs.—No œdema. Knee-jerks present on admission.

Urine.—No albumen.

April 16.—Urine found to contain a cloud of albumen.

April 18.—Legs are now paralysed. Tender on pressure over the region of the tibialis anticus. Several areas of anæsthesia to be made out on the legs. Left knee-jerk absent. Plantars present; no foot-drop.

April 20.—Paralysis of lower extremities more marked. (State of knee-jerks not noted.)

April 21.—At 2.30 A.M. she suddenly started up, saying she had a severe pain which she could not localise. She became blue in the face, pulseless, bathed in a cold sweat, pupils dilated. She revived for a few minutes after a strychnine injection, and the pulse was noted to be soft, small, and most irregular in volume and rhythm. She died in a few minutes after the first onset of the attack.

Post-mortem examination (Register No. xxii. 101) made on April 22, 33½ hours after death.

Body very large, strongly built, well nourished. Height, 69½ inches.

Brain.—48 oz. ; not shrunken. Cerebral arteries natural.

Spinal cord.—The body of the fifth dorsal vertebra is softened, infiltrated by a vascular growth protruding into the spinal canal, without, however, infiltrating the dura mater, nor pressing to any appreciable extent upon the cord. There are no naked eye signs of secondary degeneration. The posterior two-thirds of the left fifth rib is also infiltrated by a hard cartilaginous growth continuous with the above.

Lungs.—Right, 22 oz. ; left, 10 oz. Adhesions at both bases. Edema of right upper lobe. Both emphysematous.

Heart.—Weighs 12 oz. Milk spots on right ventricle. Considerable coating of epicardial fat. Muscle extremely flabby on both sides, of a brownish colour, and mottled somewhat. The blood is liquid, and faintly smelling of alcohol. In the pulmonary artery are many non-adherent worm-like clots.

Aorta.—Atheromatous.

Liver.—57 oz. ; large, soft, and greasy.

Spleen.—12 oz. ; fairly firm in consistence.

Kidneys.—11 oz. the two ; large, soft, and flabby (? due to post-mortem change). Cortex perhaps a little narrowed. Capsule tears away small pieces of the cortex on peeling.

Femoral glands on the right side infiltrated with cartilaginous growth.

Left ovary is practically replaced by a nodulated cartilaginous growth.

Microscopical examination—*Spinal cord.*—After hardening in Müller's fluid, slices of the cord were taken at the levels of the following roots : Cervical, iii. ; dorsal, i. ; dorsal, vi. ; and lumbar, ii. The histological elements of the cord are well preserved. There is no sign of any degeneration of the white matter or roots in any of the sections.

With the exception of a certain degree of pigmentation of the cells of the anterior and posterior horns, the grey matter may be said to be natural. This pigmentation is less marked than in the preceding cases.

Peripheral nerves.—Pieces of the left median, left radial, right and left vagus, right phrenic, anterior crural, sciatic, popliteal, and posterior tibial were hardened in Müller's fluid, then by Marchi's fluid, imbedded in celloidin, and cut by the ether microtome. The sections were then examined as Marchi preparations, and also further stained by Weigert-Pal hæmatoxylin, and counter-stained by Ehrlich's hæmatoxylin or alum carmine.

In none of these nerves is there to be found any appearance suggestive of degenerative disease. Here and there may be seen beaded fibres, but no more than would be accounted for by early decomposition changes, bearing in mind the long time which elapsed ($33\frac{1}{2}$ hours) between the death and the autopsy. Neither is there any evidence of inflammatory processes. The endo- and perineurium are quite natural.

Unfortunately the intra-muscular nerve-twigs were not examined. This is all the more to be regretted, because records of early cases are rare.

Remarks.—A disturbing element in the consideration of this case is the sarcoma of the ovary with the secondary growths, especially those of the left fifth rib and its corresponding dorsal vertebra. These, no doubt, may account for some of the pains from which she suffered, bearing in mind that she was also a neurotic subject. Supposing also that the secondary growth in the body of the fifth dorsal vertebra pressed upon the cord to any extent, the paraplegia might be explained, but not the loss of the knee-jerk on one side, nor the anæsthetic areas. That such a pressure really existed was rendered highly improbable by the post-mortem observation, and the microscopical examination of the spinal cord.

On the other hand, the woman was a dram-drinker, her pains were of the type so common in alcoholism. She became paraplegic while under observation, and though there were patches of anæsthesia about the legs, there was tenderness on pressure, two conditions usually co-existing in such cases. The disappearance of a knee-jerk is also extremely suggestive. The fatal syncope is a not unusual end of such cases, explained by a fatty, ill-nourished heart, and also probably by a paralysis of the vagus nerve endings.

The microscopical examination of the cord shows only a moderate degree of pigmentation of the ganglion cells. The nerve trunks show no sign of degeneration.

It may be suggested here that if the degeneration of the nerves in alcoholism be of an inflammatory nature, such an inflammation should precede, one would suppose, the degenerative process. But in this case, though sections were examined by several nuclear stains, no evidence of such inflammatory process could be found.

From consideration of these three cases one may make the following reflections:—

(1.) A person may present the symptoms of alcoholic paraplegia for a considerable period, and yet, post-mortem,

little or no degeneration of the fibres of the larger trunks be found.

(2.) The changes in the peripheral nerves are of a degenerative nature. No evidence can be found of any inflammatory process.

(3.) In a long nerve such as the sciatic, the degenerated fibres become more numerous as the periphery of the nerve is approached.

(4.) The behaviour of affected nerves under certain reagents seems to be of the greatest importance.

Weigert, and Weigert-Pal hæmatoxylin, and the latter combined with osmic acid, either by itself, or with Müller's fluid after Marchi's formula; or lastly, osmic acid, 1 per cent. solution alone, are all favourite methods for showing degeneration. But it must be remembered that by these methods the myeline stains black whether degenerated or not. But the Marchi method has a special value in that it will not stain normal myeline, but picks out at once free fat, which is one of the products of degeneration (? decomposition). The methods first mentioned show alterations in *form*, the last, alterations in *chemical constitution*.

The nerves in the preceding cases do not react to Marchi's fluid, a circumstance which suggests: (*a*) That this degeneration differs from similar degenerations in the central nervous system; or (*b*) that the free fat has been absorbed; or (*c*) that the appearances may be produced by rough handling in removal, and softening by decomposition. Delusive appearances may be easily produced, as every worker in neuro-pathology knows to his cost; and there must always be risks in dealing with material from bodies so long after death, as is often the case. Brissaud* sounds a note of warning in this connection which is not by any means superfluous.

(5.) The cells of the anterior horns are generally unaltered in form. But heavy pigmentation of these cells is a common feature. Granular pigment can be found in the cells of normal cords, in very small quantity and light in colour in children, and increasing in quantity and depth of colour as the age increases. It is possibly, therefore, a result of old age, and may be one of the many hints that premature old age may follow chronic alcoholism. But its presence in such quantities may have a still deeper significance in connection with the nutrition of the long neuron which is part of the cell. This pigmentation has been noted by several writers.

* Brissaud : Sur la prétendue dégénération nerveuse dans certaines névrites périphériques. *Compt. Rend. Soc. de Biol.*, 1890, p. 79.

BIBLIOGRAPHY.

The following works are arranged in chronological order. Where possible, the original articles have been read. The remarks appended do not pretend to be criticisms, but merely hints for the guidance of future readers. As many writers have included alcoholic paralysis under the larger head of neuritis, it is likely that many recorded cases may have been overlooked.

1. LETTSON. "History of Some Effects of Hard-Drinking." An Essay. 1789. Quoted by Buzzard ⁴, p. 70. A good description of alcoholic paraplegia.
2. JACKSON, JAMES. "On a Peculiar Malady Resulting from the Use of Ardent Spirits." *New England Journ. of Med. and Surg.*, vol. xi. 1822, 3rd Ser. vol. i. p. 351. Boston. Quoted by Dreschfeld, 33, p. 434. For those who cannot consult the original article, a French translation is given by Gilles de la Tourette, *Arch. de Neurol.*, xiv. 1887.
3. HUSS, MAGNUS. *Alcoholismus Chronicus*, 1852, p. 56.
4. MARCÉE. *Société des Hôpitaux*, tom. v. 1862, p. 241. In a discussion upon locomotor ataxia, mentions two alcoholic cases who exhibited the symptoms of ataxia, but who eventually recovered.
5. JACCOUD. "Des Paraplégies." Paris, 1864. Mentions alcohol as a rare cause of paraplegia.
6. TOPINARD. "De l'Ataxie Locomotrice." Paris, 1864, p. 40. Fully recognises an alcoholic ataxia.
7. LANCEREAUX. "Diet. *Encyclop. des Sci. Med.*," art. "Alcoolisme," 1864, tom. ii. p. 615. An exhaustive article on the general subject. Recognises alcoholic paraplegia, and a lesion of the peripheral nerves.
8. LANCEREAUX. "Études sur les Altérations produites par l'Abus des Boissons Alcooliques." *Gaz. Hebdom.*, 1865, p. 435. The second case is one of alcoholic paralysis with a post-mortem examination. Some cord changes are mentioned; in an addendum, p. 464, he notes degeneration of the nerves of the forearm.
9. LEUDET. "Étude Clinique de la Forme Hyperæsthésique de l'Alcoolisme Chronique, et de sa Relations avec les Maladies de la Moelle." *Arch. Gén. de Méd.*, 1867, tom. i. p. 5.
10. THOMPSON, REGINALD. "On Paralysis of the Extensors." *Med. Chir. Trans.*, 1868, p. 241.
11. HANDFIELD-JONES. "Notes on Epileptic Attacks, and of Paralysis as Results of Alcoholic Excess." *Practitioner*, 1871, vol. vii. p. 331.
12. LOCKHART-CLARKE. "Alcoholic Paresis and Paraplegia." *Lancet*, 1872, i. p. 427.
13. WILKS. "Alcoholic Paraplegia." *Lancet*, 1872, i. p. 320.
14. WESTPHAL. "Ueber eine bei Chronischen Alcoholisten beobachtete Form von Gehstörung." *Charité Annalen*, 1879, S. 395. A clinical paper drawing attention to the ataxic gait of such cases.

15. LEYDEN. "Ueber Poliomyelitis und Neuritis." *Zeitschr. für Klin. Med.*, 1880, Bd. i. S. 427.
16. LANCEREAUX. "De la Paralyse Alcoolique." *Gaz. Hebdom.*, 1881, p. 119. Describes degenerative lesions of the peripheral nerves in four cases.
17. MOELI. "Alcoholismus, Atrophische Lähmung der Extensoren am Oberschenkel." *Charité Annalen*, Jahrg. viii. 1881, S. 552.
18. FISCHER. "Ueber eine eigenthümliche Spinal-Erkrankung bei Trinkern." *Arch. f. Psych.*, 1882, S. 1.
19. MYRTLE. "Acute Ascending Paralysis. Chronic Alcoholism." *Brit. Med. Journ.*, 1882, ii. p. 312.
20. DÉJERINE. "Sur le Nervo-tabes périphérique." *Comptes Rendus de l'Acad. des Sci.*, 1883, tom. xcvii. p. 914.
21. DÉJERINE. "Étude sur le Nervo-tabes périphérique." *Arch. de Phys.*, 1884, 3rd Ser. tom. iii. p. 231. Two cases. Nerves found to be much degenerated. Root ganglion cells deeply pigmented.
22. ERB. "Bemerkungen über Gewisse Formen der Neurotischen Atrophie" (sog. Multiple Degenerative Neuritis). *Neurolog. Centralbl.*, 1883, S. 481. A plea for the central origin of the lesion.
23. GLYNN. "Cases of Alcoholic Paraplegia." *Liverpool Med. Chir. Journ.*, 1883, p. 374.
24. MÜLLER, F. C. "Ein Fall der Multipler Neuritis." *Arch. f. Psych.*, 1883, S. 669. Probably an alcoholic case. Degeneration of the small intramuscular nerves, less marked in the larger trunks.
25. PITRES ET VAILLARD. "Contribution à l'Étude des Nervites périphériques non-traumatiques." *Arch. de Neurol.*, 1883, p. 190.
26. STRÜMPPELL. "Zur Kenntniss der multiplen degenerativen Neuritis." *Arch. f. Psych.*, 1883, S. 339. Degeneration of peripheral nerves without any evidence of inflammation.
27. WILKS. "Diseases of Nervous System," 1883, p. 271.
28. BROADBENT. "On a Form of Alcoholic Spinal Paralysis." *Med. Chir. Trans.*, 1884, p. 133. Cord only examined, and found natural. For discussion see *Lancet*, 1884, i. p. 294.
29. CHARCOT. "Les Paralyxies Alcooliques." *Gaz. des Hôp.*, 1884, p. 785.
30. DRESCHFELD. "Alcoholic Paralysis." *Brain*, vol. vii. 1884, p. 200. Certain nerves of upper and lower extremities found degenerated.
31. KRÜCHE. "Die Pseudotabes der Alkoholiker." *Deutsch. Med. Zeitg.*, 1884, No. 72.
32. MOELI. "Statistisches und Klinisches über Alcoholismus." *Charité Annalen*, 1884, Jahrg. ix. S. 541. One case with post-mortem examination. Cord natural. Some peripheral nerve fibres degenerated.
33. DRESCHFELD. "Further Observations on Alcoholic Paralysis." *Brain*, vol. viii. 1885, p. 433.
34. HADDEN. "Two Fatal Cases of Alcoholic Paralysis." *Path. Trans.* 1885, p. 49. Examination of nerves of second case showed a sclerosed condition of the peripheral nerves.
35. HADDEN. "Cases Illustrating the Symptoms and Treatment of Chronic Alcoholism as it Affects the Nervous System." *Lancet*, 1885, ii. p. 610.

36. HUN. "Alcoholic Paralysis." *Amer. Journ. Med. Sci.*, 1885, April, p. 372. Cortical cells of second case granular. Cord natural. Nerves not examined.
37. LANCEREAUX. "De la Paralyse Alcoolique." *Gaz. Hebdom.*, 1885, p. 119. Four cases with marked degenerative changes in the nerves. None found in cord.
38. CÉTINGER. *Étude sur les Paralysies Alcooliques.* Paris, 1885. Figures characteristic degenerative lesion of nerves; notes absence of inflammation. Good bibliography to date.
39. REY. "Paralysies d'Origine Alcoolique." *Ann. Medico-Psycholog.*, 1885, p. 68.
40. SCHULTZ. "Beitrag zur Lehre der Multiplen Neuritis bei Potatoren." *Neur. Centralbl.*, 1885, S. 433. List of recorded cases of autopsies. Discusses value of vacuolation of ganglion cells of cord.
41. STRÜMPPELL. "Vortrag über die Nervenerkrankungen der Alkoholisten." *Berlin. Klin. Wochenschr.*, 1885, S. 519.
42. WEBBER. "Multiple Neuritis." *Arch. of Med. (Seguin)*, 1885, vol. xii. p. 23. Fourth case probably alcoholic. Extreme degeneration of nerves. Cord natural.
43. ZERNHARDT. "Ueber die Multiple Neuritis der Alkoholisten," &c. *Zeitschr. f. Klin. Med.*, Bd. xi. 1886, S. 363.
44. BUZZARD. "Paralysis from Peripheral Neuritis." London, 1886.
45. DUCKWORTH. "Three Cases of Multiple Peripheral (Alcoholic) Neuritis." *St. Bartholomew's Hosp. Repts.*, xxii. 1886, p. 253.
46. FRANCOTTE. "La Névrite Multiple." *Rev. de Méd.*, 1886, p. 377. Includes one alcoholic case.
47. GOMBAULT. "Sur les Lésions de la Névrite Alcoolique." *Comptes Rend. de l'Acad. des Sci.*, cii. 1886, p. 439. Takes strongly the view of the peripheral nature of the lesion.
48. OPPENHEIM. "Beiträge zur Pathologie der 'Multiple Neuritis' und Alkohol-Lähmung." *Zeitschr. f. Klin. Med.*, Bd. xi. 1886, S. 232.
49. ORMEROD. "Illustrations of Peripheral Nerve Disease." *St. Bartholomew's Hosp. Repts.*, xxii. 1886, p. 169. Three cases of alcoholic paralysis.
50. SAUNDBY. "A Case of Alcoholic Paraplegia." *Lancet*, 1886, ii. p. 241.
51. BIGGS. "Cord and Nerves in Case of Alcohol Paralysis." *Medical News*, March 10, 1887. Degenerative lesions of peripheral nerves. Cord and roots intact.
52. DÉJERNIE. "Contribution a l'Étude de la Névrite Alcoolique." *Arch. de Physiol.*, tom. x., 1887, p. 248. Degeneration of vagus found.
53. FINLAY. "Three Cases of Alcoholic Paralysis." *Med. Chir. Trans.*, 1887, p. 371. Found inflammatory lesions of nerves in two cases, with increase of nuclei in the perineurium.
54. KAST. "Primäre degenerative Neuritis." *Deutsch. Arch. f. Klin. Med.*, Bd. xl. 1887, S. 41. One case alcoholic, recovered.
55. KORSAKOW. "Ueber psychische Störungen bei Alcohollähmungen und multipler Neuritis." Abstract in *Neurol. Centralbl.*, 1887, S. 210.

56. OPPENHEIM UND SIEMERLING. "Beiträge zur Pathologie der Tabes Dorsalis, und der peripherischen Nervenerkrankung." Arch. f. Psych., Bd. xviii. 1887, S. 504. Cases 28 to 32 are alcoholic with degenerative lesion of peripheral nerves.
57. PITRES ET VAILLARD. "Des Névrites provoquées par les Injections d'Ether au Voisinage des Troncs Nerveux des Membres." Gaz. Méd. de Paris, 1887, p. 256.
58. WITKOWSKI. "Zur Klinik der Multiple Alkoholneuritis." Arch. f. Psych., Bd. xviii. 1887, S. 809.
59. BOISVERT. "Études clinique des Formes Atténuées de la Paralyse Alcoolique." Paris, 1888.
60. BRAMWELL. "A Case of Alcoholic Paralysis." Amer. Journ. Med. Sci., 1888, June, p. 575.
61. EICHHORST. "Neuritis fascians. Ein Beitrag zur Lehre von der Alkohol Neuritis." Virch. Arch., 1888, Bd. cxii. S. 237. See a criticism on this by Siemerling, Arch. f. Psych., Bd. xix. 1888, p. 824.
62. LEYDEN. "Die Entzündung der peripheren. Nerven. (Polyneuritis multiplex)." Berlin, 1888. Abstract in Brain, vol. xi. p. 257.
63. MINKOWSKI. "Beiträge zur Pathologie der Multiplen Neuritis." Mittheil. aus der Med. Klin. zu Königsberg, Leipzig, 1888. Found proliferation of nuclei in the nerves as well as atrophy of fibres.
64. PAYNE. "Address opening Discussion on Alcoholism." Path. Soc. Reported in Brit. Med. Journ., 1888, ii. p. 1275. Excellent resumé of subject.
65. SHARKEY. "Alcoholic Paralysis of the Phrenic, Pnœumogastric, and other Nerves." Path. Trans., 1888, p. 27. Cells of motor convolutions of brain, cervical and lumbar enlargement of cord, swollen and opaque. Inflammatory proliferation of nuclei in phrenic and vagus. Degeneration of popliteal.
66. SMART. "Case of Multiple Neuritis in a Woman, of Combined Syphilitic and Alcoholic Origin," &c. Edin. Med. Journ., July 1888.
67. SUCKLING. "Alcoholic Paralysis." Amer. Journ. Med. Sci., 1888, June, p. 567.
68. TAYLOR. "On Multiple Neuritis." Guy's Hosp. Repts., vol. xlv. 1888, p. 287. Case 1-8, alcoholic.
69. THOMSEN. "Zur Pathologie und pathologischen Anatomie der acuten completen (Alkoholischen) Augenmuskellähmung." Arch. f. Psych., 1888, Bd. xix. S. 185.
70. BUZZARD. "Case of Double Wrist Drop, apparently due to Multiple Neuritis of Alcoholic Origin," &c. Brain, vol. xi. 1889, p. 90.
71. ERLITZKI. "Ueber Paralysis Alcoholica." St. Petersburg, 1889. Abstract in Neurol. Centralbl., 1889, S. 210. Found degeneration of anterior horn cells of cervical and lumbar cord, also of anterior roots. Peripheral nerves not examined.
72. ROSS. "The Premonitory Symptoms of Alcoholic Paralysis." Lancet, 1889, i. p. 1125.
73. SCHAFFER. "Ein Fall von Alkohol Paralyse mit Central Befunde." Neurol. Centralbl., 1889, S. 156.

74. SIEMERLING. "Ein Fall von Alkoholneuritis," &c. *Charité Annalen*, 1889, S. 443. Marked degeneration of peripheral nerves, with myositis.
75. WILKIN. "Alcoholic Paralysis with Central Lesions." *Medical News*, Philadelphia, July 13, 1889.
76. BUZZARD. "Illustrations of some less-known Forms of Peripheral Neuritis." *Brit. Med. Journ.*, 1890, i. p. 1419.
77. FENWICK, J. C. "Case of Peripheral (Alcoholic) Neuritis." *Journ. Ment. Sci.*, 1890, p. 228.
78. LUNZ UND MAMUROWSKI. "Ein Fall acuter aufsteigender Alkohol-lähmung." *Neurol. Centralbl.*, 1890, S. 696. Cord natural. Extreme degeneration of nerves.
79. REYNOLDS. "Spinal Cord in Alcoholic Paralysis." *Path. Soc. of Manchester*, reported in *Brit. Med. Journ.*, 1890, i. p. 427. Ganglion cells of cord found swollen, vessels engorged, with great proliferation of cells in grey matter. State of roots and nerves not noted.
80. THOMSEN. "Zur Klinik und pathologischen Anatomie der Multiplen 'Alkoholneuritis.'" *Arch. f. Psych.*, 1890, S. 806. Cord natural. Intense degenerative lesion of nerves and intramuscular fibrils, also of muscle fibres with nucleus proliferation (myositis).
81. TILING. "Ueber die bei der Alkoholischen Neuritis multiplex beobachtete Geistesstörung." *Zeitschr. f. Psych.*, 1890, S. 232.
82. BRISSAUD. "De l'Influence des Centres Trophiques de la Moelle sur la Distribution topographique de certaines Névrites Toxiques." *Arch. de Neurol.*, 1891, vol. xxi. p. 261.
83. CARPENTIER. "Contribution à l'Étude des Paralysies Alcooliques." Thèse de Paris. Abstract in *Gaz. des Hôp.*, 1891, p. 297.
84. DUBRISAY. "Paralyse Alcoolique chez une jeune Femme dipsomane." *L'Union Méd.*, 1891, tom. lii. p. 719.
85. PAL. "Ueber Multiple Neuritis." *Wien. Klin. Wochenschr.*, 1891, S. 461.
86. BOEDEKER. "Zur Kenntniss der acuten alkoholischen Augenmuskellähmung." *Charité Annalen*, Jahrg. xvii. 1892, S. 790.
87. BRISTOWE. "Introduction to a Discussion on Peripheral Neuritis." *Brit. Med. Journ.*, 1892, ii. p. 1097.
88. EISENLOHR. "Beobachtungen über apoplektische Alkohol-lähmung." *Virch. Arch.*, 1892, Bd. cxxix. S. 140.
89. LAFITTE. "Les Paralysies Alcooliques." *Gaz. des Hôp.*, 1892, p. 1081.
90. PLANTH. "Ein Fall von Neuritis Multiplex Alcoholica." *Ann. der Städtisch. Allg. Krankenh. zu München*, 1890-92, S. 166. A copious historical survey.
91. RAKHMANINOFF. "Contribution à la Névrite Périphérique : un Cas de Gangren Symétrique, et deux Cas de Paralyse Alcoolique." *Rev. de Méd.*, 1892, p. 321.
92. ACHARD ET SOUPAULT. "Deux Cas de Paralyse Alcoolique." *Arch. de Méd. Experiment.*, 1893, p. 359. First case, degenerative changes of peripheral nerves. Second case, degeneration of cells of anterior horn, roots, and peripheral nerves.
93. ARNAUD. "Pronostic des Paralysies Alcooliques." *Gaz. des Hôp.*, 1893, p. 241.

94. BUZZARD. "Some Symptoms and Varieties of Multiple Neuritis." *Lancet*, 1893, ii. p. 1233.
95. CAMPBELL. A. W. "Ein Beitrag zur pathologischen Anatomie der sogenannten Polyneuritis alcoholica." *Prager Zeitschr. f. Heilk.*, 1893, S. 11. Four cases all showing more or less degeneration of cells in cord as well as of peripheral nerves.
96. HENNEBERG. "Beitrag zur Kenntniss der Polyneuritis alcoholica." *Inaug. Diss.*, 1893.
97. HERRENSTADT. "Beiträge zur Kenntniss der Neuritis Alcoholica." *Inaug. Diss.*, Berlin, 1893.
98. JACOBÆUS. "One Perifere Neuriter ved Chronisk Alcoholisme." *En Klinisk Studie*. Copenhagen, 1893.
99. RENNERT. "Beitrag zur Kenntniss der Multiplen Alkoholneuritis." *Deutsch. Arch. f. Klin. Med.*, 1893, Bd. 1. S. 213.
100. ROSS AND BURY. "Peripheral Neuritis." London, 1893, p. 121.
101. STAHN. "Ueber alcoholische Neuritis." *Inaug. Diss.*, Berlin, 1893.
102. THIROLOIX ET DUPASQUIER. "Marche et Curabilité des Paralysies alcooliques." *Gaz. Hebdom.*, 1893, No. 37.
103. TOOTH. "Degeneration of the Nerves in Alcoholism." *Path. Trans.*, 1894, p. 1.
104. BOEDEKER. "Zur Kenntniss der acuter alkoholischen Ophthalmoplegieen." *Arch. f. Psych.*, 1895, S. 810.
105. MAUDE. "Alcoholic Peripheral Neuritis in Advanced Age." *Brain*, 1895, p. 315.
106. NOTHNAGEL. "Ein Fall von Polyneuritis alcoholica." *Wien. Allg. Med. Zeitg.*, 10, 1895.
107. DÉJERINE ET SOTTAS. "Sur un Cas de Polynévrite Motrice à Marche lente. Paralyse Spinale, Antérieure Subaigue. *Compt. Rend. Soc. de Biol.*, 1896, tom. iii. Ser. 10, p. 193. An alcoholic and syphilitic subject. Anterior horn cells degenerated, also atrophy of anterior roots and degeneration of peripheral nerves.
108. GUDDEN, H. "Beiträge zur Kenntniss der multiplen Alkoholneuritis." *Arch. f. Psych.*, 1896, Bd. xxviii. S. 643.
109. HERTER. "Report of a Case of Acute Alcoholic Intoxication in a Young Child followed by . . . Multiple Neuritis." *N. Y. Med. Journ.*, vol. lxiv. 1896, p. 608.
110. NEIL. "A Case of Alcoholic Neuritis with Prominent Nervous Symptoms." *Brit. Med. Journ.*, 1896, i. p. 906.

ACUTE ATROPHY OF THE LIVER.

BY

T. LAUDER BRUNTON, M.D., F.R.S.,

AND

F. W. TUNNICLIFFE, M.D.

Acute atrophy of the liver is a very rare disease. How rare it is may be inferred from the fact that in the statistics of this hospital we have only been able to find seven cases as having occurred in twenty-five years. Over 150,000 patients are treated either in the wards or casualty department of this hospital every year. If we reckon on this estimate the number treated in twenty-five years, it will come out at about 3,750,000, or in other words, we shall find that one case of acute atrophy of the liver occurs in about every 500,000 cases that apply to this hospital for treatment. Thierfelder,* in his article upon this subject in "Ziemssen's Handbuch," was only able to collect from all sources 143 cases of primary acute atrophy of the liver in which the diagnosis had been verified by a post-mortem examination. Even when he admitted into his statistics those cases unverified by post-mortem examination, the number only amounted to 200. Henoeh † has only observed three cases of acute atrophy of the liver in children, and only one of these was verified post-mortem.

This disease is naturally very interesting on account of its rarity. It is interesting also on account of its medico-legal relations, because it simulates acute poisoning by phosphorus so closely that it is difficult, or impossible, to distinguish a case of acute yellow atrophy from one of phosphorus poisoning unless by the history showing that phosphorus has been administered, by finding phosphorus in the vomit, or by

* Handbuch der speciellen Pathologie u. Therapie, Band viii. p. 215. 1873.

† Henoeh: Vorlesungen über Kinderkrankheiten. Berlin, 1831, p. 501.

noticing that the fumes which proceed from the patient's mouth or his breath are luminous in the dark. But acute yellow atrophy of the liver has another interest, which is perhaps still greater. It is especially interesting at this period of the development of medical science, because just now pathology in its onward progress is passing from the consideration of the relationship between microbes and disease to that of the relationship between the products of microbial life and disease. It is beginning to look upon the symptoms of disease as caused not so much by microbes themselves, but rather by the poisons formed by them. We have, then, in acute yellow atrophy of the liver a sort of connecting link between poisoning by an inorganic substance, viz., phosphorus, and a disease or diseases, such as yellow fever, for example, which are most certainly due to microbes.

There is another disease due to microbes, viz., cholera, which resembles acute yellow atrophy, not in its symptoms, but in the fact that it, too, closely simulates poisoning by an inorganic substance—arsenic. The late Sir Robert Christison used to say that the symptoms of poisoning by arsenic were so very much like those of cholera, that he felt quite certain that cases of sporadic cholera occurring just about the beginning of an epidemic had been taken for cases of arsenical poisoning, and, *vice versa*, that cases of true arsenical poisoning had probably been passed over entirely unnoticed during the height of a cholera epidemic. It is curious to note that the three different forms of cholera may be imitated by arsenical poisoning. In the first form there are symptoms of well marked gastro-enteritis, violent vomiting, copious watery purging, followed by collapse and death; in the second, there is collapse without the previous irritation of the gastro-intestinal canal, the effect of the poison being exerted apparently chiefly upon the organs of circulation; in the third form, the poison exerts its influence chiefly upon the nervous system, and then there may be convulsions, delirium, and death.

Under the term primary acute atrophy of the liver are included those cases of icterus gravis, or jaundice with profound nervous symptoms, fever, and death, with or without the presence of leucin or tyrosin and a corresponding diminution of urea in the urine, in which no cause could be found, or rather was found post-mortem, for the acute atrophy of the liver which was present. Those cases in which a similar symptom-complex occurred during life, and a practically identical pathological condition was found after death, were, if phosphorus or antimony, for instance, were found in the

stomach, called phosphorus or antimony poisoning, the essential difference between the two classes of cases being the finding of an irritant poison in the one case, and the non-finding of it in the other. Another class of acute yellow atrophy seems accompanied by the presence of micrococci in the biliary canaliculi* and liver cell detritus.† A third class of case is associated with the presence of micrococci in the hepatic blood vessels. The micrococci were seen in the portal canals filling the arteries, and in the peripheric part of the lobule between the liver cells, filling up apparently the capillaries between them.‡ Bouiet et Boy Tossier,§ in a case of acute yellow atrophy, thirty-six hours before death obtained from the blood of the patient a micrococcus, which was also present in the liver and renal epithelium. The result of their inoculations is, however, not given. Before leaving this part of the subject, we think it will be well to mention three other conditions all due to causes widely different in one sense, but not so different in another, which closely resemble acute yellow atrophy of the liver. These three are (1) poisoning by phosphorus, (2) poisoning by lupins (so-called lupinosis), (3) poisoning by toluylenediamine.

Perhaps it would be best to recapitulate shortly here the symptoms of phosphorus poisoning. Phosphorus poisoning is also very rarely met with in this hospital. In twenty-five years we have only been able to collect seventeen cases, one of which was fatal. The rest all recovered.

It is not, however, so rare elsewhere, being very much commoner abroad in Germany, Austria, and France than in England. The deaths from phosphorus in England and Wales occurring in the ten years ending 1892, from all causes—*i.e.*, accident, negligence, and suicide—amounted to 147.|| It seems to be especially common in Vienna, where a favourite method of committing suicide consists in scraping the heads off two or three hundred lucifer matches, mixing with water, and swallowing the mixture.

The symptoms in phosphorus poisoning are as follows:—First of all there are the ordinary indications of gastro-enteritis, and more especially of gastritis, as shown by pain in the stomach and vomiting. These primary symptoms pass off after a greater or less length of time, according to the treatment

* Klebs' Tagebl. des naturf. Vers. zu Leipzig, 1872.

† Eppinger, Prager Vierteljahrsch. f. prakt. Heilkunde, 1875.

‡ A case of acute atrophy of the liver. Tomkins and Dreschfeld, Lancet, 1884, i. p. 606.

§ Recherches sur le microbe de l'ictère grave. Rev. de Med., vi. p. 334. 1886.

|| Poisons: their Effects and Detection. Wynter Elyth, Lond. 1895, p. 215.

adopted. There is then an interval during which the patient appears to be almost well, but after a day or two new symptoms make their appearance, the patient becomes jaundiced, there is tenderness of the abdomen, and then nervous symptoms rapidly appear, delirium followed by convulsions, weak intermittent breathing, and death. There is only one symptom, practically, in which phosphorus poisoning and acute yellow atrophy of the liver have been said to differ, and this is in the interval which elapses between the primary and secondary symptoms in the case of phosphorus poisoning. This interval has been stated to be absent in cases of acute yellow atrophy of the liver. It will be noticed, however, that in the case of F. G., which we shall discuss fully later on, an interval did appear to take place between the primary and secondary symptoms, but this interval, although carefully marked and noted in the case before us, might have very easily been missed if one had not been on the look-out for it.

Another condition which has a close resemblance both to acute yellow atrophy of the liver and phosphorus poisoning is the disease known as lupinosis. This disease does not occur in man, but is responsible annually for the deaths of hundreds, one might say thousands, of sheep in Germany. It is contracted by the sheep as a result of eating certain kinds of lupins (*Leguminosæ*). The poison is contained in the husks of the seeds, and is soluble in dilute alkalies, and to some extent in water. Its actual chemical nature is unknown. It has received various names—Ictrogen,* Lupinotoxin,† &c.‡ The results of the ingestion of the poison are vomiting, pain, and tenderness at the epigastrium and over the liver; later, jaundice, with hæmorrhages under the skin into the muscles and serous membranes, twitching, paralysis, delirium, coma, and death. On post-mortem examination the liver is found to be in a condition of acute yellow atrophy. The average course of the disease seems to be about a week. Two or three days generally intervene between the ingestion of the poison and the appearance of jaundice, then a rapid onset of the nervous symptoms occurs, which is soon followed by death. According to Liebscher,§ the poison is of the nature of a toxin, and is produced by the agency of certain fungi.

* Liebscher: Untersuchungen über die Lupinosen Krankheit der Schafe. Bericht aus d. physiol. Lab. des landwirth. Instituts zu Halle. Herausgegeben v. J. Kühn. ii. Heft., p. 53, 1880.

† Arnold u. Schneidemühl, Vierter Beitrag Lupinose. Lüneberg, 1883.

‡ *Vide* also Roloff: Über die Lupinose. Archiv. für Thierheilkunde, Band ix., 1883.

§ *Loc. cit.*

The third condition, which is of special interest with regard to acute yellow atrophy of the liver, is toluylenediamine poisoning. This substance is a methylated aromatic diamine, of strongly basic properties. The action of this substance was discovered by Professor Schmiedeberg, and has been worked at by Stadelmann,* Afanassiew,† Hunter,‡ and others. The injection of this substance into the circulation, or into the stomach, causes a very profound jaundice. This substance exerts a destructive action on the red blood corpuscles, and renders the bile thick and viscid, so that it cannot flow through the bile ducts, which, at least in their smaller or capillary ramifications, become blocked by inspissated bile. If the drug is pushed to a fatal issue, the nervous system becomes affected, and the animal dies in a comatose condition.

To recapitulate then—Acute atrophy of the liver is interesting from a medico-legal aspect on account of the great similarity between it and phosphorus, arsenic, and antimony poisoning. It is also interesting in its relation to those diseases, such as yellow liver and cholera, which are due to specific micro-organisms. And lastly, it is interesting when compared with cases of poisoning due to other more complex organic substances, such, for instance, as toluylenediamine and the toxins in the condition known as lupinosis. Many of the products of the putrefaction of meat belong to the class of diamines; they are mostly substituted diamines, such as toluylenediamine. Some of these substances are non-poisonous, as, for instance, putrescine, cadaverine, &c.; some are poisonous, as collidine and muscarin, and the degree of their toxicity seems to be to some extent dependent upon the nature and extent of the substitution. Just now, when attention is being turned from bacteria themselves to their products as causes of disease, this subject is a particularly interesting one, for it seems as if the two cases which we are going to shortly describe form a connecting link between simple mineral poisoning on the one hand, and specific infective disease on the other.

One case occurred in a child, F. G., a boy, aged $3\frac{1}{2}$ years. He was apparently healthy, and had been apparently more than usually healthy all his life. On November 9th, he felt sick after breakfast, he vomited after dinner, and from that time he continued to vomit everything that he took for two days, but no pain whatever was complained of, and the child went about as if comparatively little was the matter with him. After two

* Stadelmann : *Arch. für exp. Path. v. Pharm.*, Band xiv. p. 241. 1881.

† Afanassiew : *Arch. f. d. ges. Physiologie*, xxx. pp. 385-436. 1883.

‡ Hunter : *Journal of Path.*, iii., 1896, p. 259.

days, on the Wednesday, the seat of the symptoms shifted. During the first two days the stomach was partly affected, but now the stomach appeared to get better, vomiting entirely ceased, and the milk which was taken was not only completely retained, but caused no pain. The liver, however, appeared on this day to become affected, because jaundice set in, and there was a certain amount of tenderness and pain complained of by the child in the abdomen, chiefly in the epigastric and hypogastric regions. But still the child upon this day was apparently very fairly well; he was able to go about as usual, and with the exception of the slight pain in his stomach and the slight jaundice, he was really to all appearances well. The vomiting had completely ceased, and the pain was not such as to attract much attention. On the Thursday, however, there was a marked change. After an apparent lull in the symptoms for twenty-four hours, the child began to suffer much more severely. He shrieked with pain, and was put to bed. The bowels had been constipated for two days; but after an aperient powder which was given to him the bowels were opened towards evening, and the motions were then of a dark-greenish offensive character. From this time forward the child became unconscious, and passed all his motions under him. Subsequently, and on until death, the nervous symptoms took the upper hand; the child became unconscious, delirious, and did not know his parents, and towards the end of the time, shortly after his admission to hospital, he had a convulsion—on the evening of Friday. Towards the midnight of Friday the breathing, which had before been somewhat stertorous, began to get feeble and intermittent, the coma deepened, and the child died. It will be seen that there is almost no difference between the symptoms presented by this case and the symptoms of acute poisoning by phosphorus. The diagnosis was therefore uncertain, the only way of making sure being to ascertain whether phosphorus or antimony was present in the contents of the stomach. This was done, and they were found to be absent; but, on the other hand, we succeeded in finding a body, or a mixture of bodies, which belonged to the class of substances known as diamines, but the specific reactions of which did not correspond to any known diamine. The chemical tests, however, left little doubt that the substance was of the nature of a substituted, in all probability methylated, diamine. The changes which were found post-mortem were chiefly those of irritation of the stomach and intestines, a soft fatty condition of the liver, and a tendency to ecchymosis throughout the body generally. We ought to have mentioned that indications

of these conditions were present before death; that on admission the sound over the abdomen on percussion was tympanitic throughout, and yet on palpation the edge of the liver could be discovered coming down about two-thirds of the way to the umbilicus. The liver therefore was apparently enlarged, and yet it was soft and flabby, so that it fell back towards the spinal column and the intestines came in front of it. There was towards the end of life vomiting, first of all of a matter looking like beef-tea, and consisting of altered blood, and later on of pure blood. There was further, as in toluylenediamine poisoning, an alteration in the blood, which was shown by the presence of little extravasated points, little ecchymoses upon the neck and upon the body. At the post-mortem examination ecchymoses were found on the pericardium. The liver was thin, and of a somewhat deep ochre colour internally on section, on the outside it presented a curious purple or lilac reflection. In this case the urine did not contain leucin or tyrosin. Bile pigment was present in large quantities, and a cloud of albumen.

There is a condition of the liver called red atrophy, regarding which comparatively little is known. It is, if possible, a still rarer condition than yellow atrophy, and so we have thought it worth while in this connection to relate a case which was admitted into the Hospital a good many years ago. The patient, J. D. S., aged 43, a schoolmaster, was admitted March 7, 1889, and he died March 12, 1889. The history was that he had had a pain in the back, somewhat like a stiff neck, for about three weeks, but to this he attached little or no importance. On February 28th he had been dining with the other masters of the school in the middle of the day, and he had some kind of meat which they all noticed to have a somewhat disagreeable taste. He was, however, the only one who suffered from partaking of it. Between four and five on the same afternoon he had a kind of fit, like a fainting fit, but did not quite lose consciousness. He was sick once, and he slept fairly well that night, and had no symptom of any important kind, and more especially there was no trace of paralysis. Next day he did not feel very well, and stayed indoors. The same was the case the day after, when he sent for a doctor. On the third day he again fell down in a fainting fit with violent pains in the head, and was then sick every quarter of an hour for twelve hours. His vision was not impaired; there was no diplopia; he had pins and needles all over the body; the voice was noticed to be husky, and he went to bed. On March 4th he had difficulty in swallowing liquids, although he could get down jellies. He

further noticed that his vision was bad. On March 7th the condition was rather better; he lost control of the movements of the right hand from March 3rd, and he could not walk on March 7th. He had no delusions, no dyspnoea, no difficulty in passing water, no girdle pains, no shooting pains in the legs. When admitted on March 7th, his face was flushed, his manner was nervous, he felt rather shaky, his voice was nasal, his face moved slightly more on the left than on the right side; he had no headache, no pain anywhere. The pupils were both contracted, the right markedly so, it being half the size of the left. The pupils reacted well to light and accommodation; there was no nystagmus or strabismus. An attempt to swallow milk was unsuccessful and brought on hawking and cough. The milk did not pass up through the patient's nose, although he said that it would have done so had he not spit it out. The movements of the right hand and arm were disorderly and restless; there was no impairment of tactile and painful sensation; the liver dulness was normal, the spleen was not felt. We may pass over the remainder of the symptoms just now, only mentioning that the paralysis appeared to pass away and the patient's brain became affected, so that he was delirious, restless, and noisy, and had to be removed to the Casualty Ward, where he remained for less than a day. Shortly after being transferred there he vomited several times, the vomited matter being black. He could not sleep, but was constantly groaning; the pulse became almost imperceptible; the extremities were cold, and he died. The result of the post-mortem can be put very shortly. There was really no cause of death to be found. All that could be found in the body was some hæmorrhage into the cord, and a liver which was soft, small, and red. On pressing into it, it felt to one very much like the consistence of a typhoid spleen. It was very much softer than usual, almost diffluent, and Dr. Norman Moore, who was then pathologist, said that the only diagnosis that could be come to was, that the patient had died of red atrophy of the liver. It is, however, to be noted here that the patient had no well-marked jaundice, and the symptoms which he presented were those of poisoning, although they did not correspond with those of any known poison. The contraction of the pupils, one would have said, pointed to something like physostigmine, but of course the other symptoms that he presented were not like those of physostigmine poisoning, and the curious fleeting character of the paralysis was very remarkable, because at one time there was marked ptosis in the left eye, and a short time afterwards this had apparently disappeared. The dysphagia, due

doubtless to paresis of the muscles composing the pharynx and œsophagus, as well as the other paralysis, was almost certainly of peripheral origin, and further of peripheral nervous origin, *i.e.*, due to paralysis of the motor nerve-endings in the affected muscular structures. This action of the poison in these two cases is especially interesting from the fact that the products of many bacteria, when injected into animals, exert this paralysing action, sometimes acting like curara on the motor nerve-endings of striped muscle, at other times like atropine, affecting chiefly the motor nerve-endings in unstriped muscle. The dysphagia, cardiac irregularity, and the other paralysees following diphtheria, are phenomena of this class.

Taking these two cases together, then, we find that in neither of them was there any injury to the vital organs which was apparently sufficient to produce death. Both of them seemed to have died from the effect of a poison. We could not trace in either the one or the other any account of the administration of a poison either of an organic or inorganic nature, and we are practically forced to the belief that death in both was due to poisons formed within the bodies of the patients themselves by microbes, the nature of the microbes not yet being known. Probably if all the organs had been analysed with sufficient care one might have been able to separate from them some definite ptomaine or chemically allied body, but the amount of the diamine which we found in the stomach contents of the first case was not sufficient to give conclusive evidence as to its exact nature, although there was no doubt that such a body, or a mixture of such bodies, was present. It would be beyond our purpose here to enter exactly into the chemical reaction upon which this conclusion is based. It is, however, of interest to mention that the substance obtained from the stomach contents of the first case was poisonous, and when injected under the skin of a small guinea-pig produced a rise of temperature, twitchings of the limbs, followed by paresis and general lethargy. A second dose produced identical symptoms, with the exception of the rise of temperature. In both instances the guinea-pig recovered. An examination of its urine showed the presence of peculiar crystals, which, however, we were unable to identify as certainly tyrosin, although they had many resemblances to tyrosin crystals. In the case of yellow fever, poisons have been separated from the contents of the stomach and intestines, and these are supposed to have the power of producing the disease in animals, or at least symptoms of the disease. Gautier,¹ in his work on toxins, gives a reference to a

¹ Gautier, *Les Toxines*, Paris, 1896.

French work which we have not been able to consult in the original.

The symptoms in the two cases we have mentioned are those of gastro-intestinal disturbance, followed in the one case by jaundice, showing implication of the liver; in the other not followed by jaundice, but in both succeeded by symptoms of grave disease of the cerebro-spinal system. In the child who was admitted a very short time ago, the cerebro-spinal centres seemed to be involved; and in the case of S., the man who was admitted several years ago, the peripheral motor nerves seem to have been also affected. It is difficult, upon any other assumption, to explain the fleeting paralyses.

Now, there is one class of poisons that produce symptoms perhaps more nearly resembling those of acute yellow atrophy than anything else; we allude to some of the viperine venoms, *i.e.*, venoms from various species of snake of the viperine as distinguished from the colubrine class. These substances have the power of producing dissolution of the blood; they cause, when introduced into the stomach, very active vomiting indeed. They produce paralysis, weakness of the respiration, and consequent death, and not unfrequently death is preceded by twitchings, or even convulsions—a phenomenon which we have noticed as occurring in the case of the child with acute yellow atrophy, and which also occurred in the guinea-pig poisoned with the diamine which we obtained from the stomach contents of this child. Perhaps you may be able to understand the occurrence of the different symptoms in acute yellow atrophy more easily by following them downwards from their cause to the appearances presented, than by following them upwards to their cause. Let us take it for granted that the poison, whatever it may be, tends to destroy the blood corpuscles, and therefore to produce certain symptoms. By destroying the blood corpuscles it will tend to cause jaundice, and jaundice of a peculiar kind; because one of the noteworthy points in this case was that there was no obstruction whatever to the gall ducts. The bile could flow perfectly freely through all the ducts, and there was no obstruction by means of mucus or anything else. The mucus was not increased, and this is a point of considerable importance, for in many such cases the occurrence of jaundice has been explained on the supposition that the amount of mucus in the gall ducts was increased, and had offered an obstruction to the flow of bile out into the duodenum. This, however, did not occur here. The only way in which we can explain the occurrence of jaundice is by supposing that the bile had stayed in the capillaries of the liver, and had been absorbed

back into the system. If there be no obstruction in the larger ducts, obstruction can still occur in the biliary capillaries in one of two ways: either the liver cells may swell very much, and thus obstruct the flow of bile through the capillaries so much that the tension in them being much increased reabsorption of bile may take place and jaundice occur. A similar absorption may take place if the bile contains such an undue proportion of solids that it is too thick and viscid to flow through the biliary capillaries. It will consequently stick in them and block them, more especially if at the same time the liver cells are somewhat swollen; thus the tension of the bile in the capillaries is increased and reabsorption takes place. This is probably the mechanism of the occurrence of jaundice in acute yellow atrophy.

Another interesting phenomenon occurring in these cases are the ecchymoses. They probably depend partly upon a dissolution of the blood, and partly upon some affection of the blood-vessels. The venom of the rattlesnake, when put upon the mesentery of a frog, will cause ecchymosis to occur to a tremendous extent. If you put a minute drop of the poison upon the mesentery of a frog spread under the microscope, things seem to be all right at first, but suddenly there is a flow of blood over the whole field, so much so that it is exceedingly difficult to make out the exact mechanism by which the blood finds its way out from the vessel into the tissue around it; so that the ecchymoses have a twofold origin, destruction of the blood corpuscles and partial degeneration of the vessels.

With regard to the diagnosis of acute yellow atrophy, there is not very much to be said. At first it is impossible to recognise the difference between acute yellow atrophy and ordinary slight catarrhal jaundice. It can only be done when the symptoms characteristic of yellow atrophy, viz., the delirium, the altered respiration, the convulsions, and the coma, come on. The one diagnostic symptom in the course of the disease between it and poisoning by phosphorus, viz., the intermission which is said to occur in phosphorus poisoning and not to occur in yellow atrophy, does not seem to hold. In the case of F. G., as we have said, there seemed to be a distinct intermission, so that many people who believe in the statement that there is an intermission only in cases of phosphorus poisoning, and not in cases of acute yellow atrophy, would be inclined to class the case we had here as one of phosphorus poisoning. But against such a diagnosis is the fact that there was absolutely no history of phosphorus having been taken by the child, no history of any peculiar smell in the breath, no peculiar smell or

appearance in the vomited matter, and an entire failure to find any evidence whatever of phosphorus in the contents of the stomach. This being so, we feel justified in diagnosing it as a case of acute yellow atrophy.

Treatment is practically nil. So far as is known, almost all the cases of acute yellow atrophy have died. It is said that some have recovered, and it has been stated that alleviation at least of the symptoms has been produced by free purgation. One can see that this would be to a certain extent a rational method of treatment, because if the symptoms depend upon the presence of a toxin in the intestine, clearing the intestine out, more especially by large doses of saline purgatives, will help to clear away the poison which has given rise to the symptoms. Thus one might hope, to a certain extent, for alleviation, if not complete recovery. Of course there is always this doubt in those cases that have recovered, viz., whether they were really cases of acute yellow atrophy or not. They may have been cases of phosphorus poisoning, because in the cases of undoubted phosphorus poisoning collected from the Hospital Reports during the last twenty-five years, seventeen in all, sixteen cases recovered and were discharged, while only one died. So that in cases of phosphorus poisoning, where quickly treated, the prognosis is good, and as the pathological conditions produced by phosphorus are so nearly allied to those which occur in acute yellow atrophy of the liver, one might hope that recovery would take place in the latter as well as the former condition. The difficulty, of course, is that before one thinks of applying the treatment for acute yellow atrophy the case is so far advanced that it has become almost hopeless. The proper treatment would be to give saline purgatives in large doses just after the jaundice had begun, and before the graver symptoms, *i.e.*, the nervous symptoms, had come on. But, on the other hand, one does not know that it is going to be a case of acute yellow atrophy of the liver until those symptoms do appear, and therefore, the only rule to lay down is that perhaps it would be the best treatment in a case of jaundice, whenever there are indications of the approach of nervous symptoms, to purge freely, employing especially the salines, which would clear the intestine thoroughly out.

We have thought it advisable to append the clinical notes of the two cases, with the kind permission of Dr. Walker, under whose care J. D. S. was.

CASE I.—J. D. S., æt. 43, schoolmaster. Admitted March 7, 1889; died March 12, 1889.

History.—Pain in the back like a stiff neck for three weeks.

February 28.—About one o'clock had for lunch some meat, which was noticed by himself and others to have a disagreeable taste. Between 5 and 6 P.M. had a kind of fit like a fainting fit. Did not quite lose consciousness. Was sick once. Was not paralysed after the fit. Slept fairly well that night.

March 1.—Stayed indoors.

March 2.—Consulted Dr. C. R. Walker of Leytonstone.

March 3.—Got up. Fell down in a fainting fit, with violent pains in the head. Was then sick every quarter of an hour for eighteen hours. Vision not impaired. No diplopia. Pins and needles all over body; not since. Voice noticed to be husky. Took to bed.

March 4.—Had difficulty in swallowing liquids. Can get jellies down with care. Vision bad. Was seen by Dr. Lauder Brunton in consultation with Dr. C. R. Walker. No objective signs of disease could be found.

March 6.—Dr. Walker noted paralysis of right pharynx and palate, with impairment of sensations of the same; right facial paresis with slight anaesthesia, also some paresis of right extremities and side. At times there is nystagmus (this was noticed on Sunday after the "fit"). He is being fed by peptonised beef suppositories.

March 7.—Admitted into St. Bartholomew's Hospital under the care of Dr. Lauder Brunton, and the following notes were made by the House-Physician, Dr. H. D. Rolleston:—

Felt rather better. Has lost control over movements of right hand since March 3. Found he could not walk. No difficulty in passing his water. No dyspnoea. Bowels open last on March 3. No delusions. No girdle pain. No shooting pains in the legs.

Is repeatedly exposed to cold, but not on one occasion more than another. No history of a blow on the spine. Has been working in a room without a fire. Denies morbus veneris. Usually a healthy man. Typhoid fever five years ago. Rather a nervous man. Never had a fit. Never had discharge from the ears. Mother paralysed for four years. Father found in bed dead, æt. 54 years. One of four. One sister "funny." No history of insanity.

Present condition.—Face flushed. Manner rather nervous. Feels "shaking." Voice nasal. Face moves slightly more to the left than to the right. No headache. No pain anywhere. Pupils both contracted, the right markedly; right pupil is half

the size of the left; pupils react to light and accommodation. No nystagmus. No strabismus.

Ophthalmoscopically.—Right disc swollen. Margin indistinct. Vessels congested. Left fundus normal.

Tongue protruded without tremor and fairly straight. Furred on dorsum. Edges red. Palate moves. Back of larynx covered with mucus.

An attempt to swallow milk was unsuccessful, and brought on hawking and cough. The milk did not on this occasion come through patient's nose. He says it would if he did not spit it out. Unable to stand. Tendency to fall towards the right. Grasp of hands equal. No paralysis of any limb. Movements of right hand and arm disorderly and restless. No impairment of tactile or painful sensation. No formication. Knee-jerks present. No ankle-clonus. No cremasteric or abdominal reflex.

Abdomen.—Liver dulness normal. Spleen not felt. No dulness above the pubes. Passed urine naturally. Urine 10.23. acid. Trace of albumen.

No bed-sore. No pain on percussing any part of the spine.

Thorax.—Movement unimpaired.

Heart: Dulness normal. Impulse diffuse. Apex in fourth space half an inch inside the nipple-line. Sounds clear.

Lungs: Percussion good. Sounds clear.

March 8.—Slept comfortably last night. Nystagmus. Manner strange.

March 9.—Tongue furred on dorsum. Did not sleep well. Swallowed some jelly this morning. Said he would go out if not fed with solids. He was seen by Dr. Church this afternoon. Dr. Rolleston writes: "Dr. Church did not feel quite easy about him. Whatever else the patient might have the matter with him, there was some insanity about him. His manner had not struck me till this afternoon, when he certainly got very excited when made to walk."

March 10.—Swallowing all night. Sleepy. Drowsy. No pain. Tongue foul. Voice nasal. Pupils equal, somewhat dilated.

March 11.—Noisy night. No sleep after sulphonal grs. xx. Poor pulse. Has had some occipital headache. Strange, but easily managed. Tongue edges red. Dorsum furred. Takes fairly. Continually groaning, and behaving in a quite idiotic fashion. 3.15 P.M., transferred to private room.

Patient has been very restless ever since he came down from the ward to private room, talking a good deal. Took about half an ounce of brandy and milk by mouth, and has been very

sick on and off all the evening. Black-looking vomit. Had an enema composed of an egg, brandy, and milk at a quarter to six (4 oz.); also a Slinger's suppository at half-past seven. Temperature, 96°. Bowels not open. Has not passed water.

10.15 P.M.—Patient had an enema of 4 oz. beef essence, 1 oz. wine. Vomited a small quantity of black vomit. Very restless.

12.30 A.M.—Had enema, 1 oz. brandy, 1 oz. essence, 15 grs. chloral. Breathing seemed easier.

2 A.M.—Died. Just before dying vomited, bringing up small quantity of black vomit.

Post-mortem (March 13).—No cause for death found by naked-eye examination.

External appearances.—Well made, muscular, well nourished. Rigor mortis gone from arms; present in legs. All internal organs somewhat soft and flabby, suggesting an early stage of decomposition.

Head.—Brain: Some general softness, probably commencing decomposition. No naked-eye lesion.

Spinal cord.—A little blood in arachnoid on its posterior aspect, forming a leash of distended veins. Cord, membranes, and spinal column normal to naked eye.

Chest.—Œsophagus: Some longitudinal streaks of pigmentation at lower edge of œsophagus. Vagi and sympathetic of neck seem perfectly normal. Lungs: Some emphysema and congestion. Small white spots on pleural surface of upper lobes. Heart: Rather larger than normal. Slight patch of atheroma on mitral valves; otherwise no disease.

Abdomen.—Peritoneum: Much fat in mesentery and omentum. Stomach: Mucous membrane covered with thick darkly stained mucus. Intestines: Normal, lumpy, somewhat pale stools in the bowel. Coats themselves were normal. Liver: Bile in bladder very black. Some slight adhesion of liver to diaphragm. Liver small, weighing 42 oz. Capsule smooth, not wrinkled. Section rather flabby in consistence (? decomposition). Lobular structure visible. Other organs normal, except kidneys, which were soft.

CASE II.—F. G., aged 3½ years. Admitted November 13; died 1 A.M. November 14, 1896.

History.—Child been very healthy all his life. After breakfast, November 9th, retched; vomited his dinner same day.

November 10.—Better.

November 11.—Vomited everything he took, but complained

of no pain. Child complained of pain in epi- and hypogastric region.

November 12.—Morning, child ran about; 2 P.M. he screamed with pain, was put to bed, and rapidly became unconscious. Bowels had been constipated for two days, but after an aperient powder he passed a green offensive motion on evening of 12th; from this time everything was passed under him.

November 13.—Condition upon admission.—Deeply jaundiced, petechiæ over body and neck. Face slightly flushed. Chest moved freely, no cough; *alæ nasi* did not dilate; lungs normal. Respirations 34, quiet, but occasionally with a sigh. Cheyne Stokes breathing absent.

Circulatory system.—Pulse 134, otherwise normal.

Nervous system.—Pupils dilated, veins of retina swollen and full. Outer side of left disc looked blurred. Nystagmus. Arms and legs rigid.

Abdomen retracted, resonant, tender to manipulation. Edge of liver felt two-thirds of distance between umbilicus and ensiform cartilage.

Considerable dysphagia.

Temperature on admission, 98.4°; 7 P.M., 99.4°; 9 P.M., 100.00°; 11 P.M., 102.8°. Soon after admission convulsion occurred, followed by vomiting, first of altered milk, then of blood. Respiration became stertorous and irregular. Condition deepened into coma, and death took place about 1 A.M., November 14.

Urine post-mortem.—No blood, no sugar; trace of albumen; no leucin or tyrosin. Bile pigments.

Post-mortem notes.—November 14.—3 ft. 2 in. high, well nourished, markedly jaundiced. Petechial eruption on trunk and neck, and to some extent on limbs. Bruise on right frontal eminence. Rigor mortis not present.

Brain.—44 oz.; bones, membranes, and sinuses normal.

Eyes.—No evidence of disease of discs or choroid. Ears, no otitis media.

Chest.—Œsophagus, larynx, trachea, natural. Lungs, R., 6 oz.; L., 5 oz.; collapsed in places, crepitant all over.

Heart.—Natural weight, 3 oz. Sub-endothelial hæmorrhage in left ventricle. Pericardial fluid bile stained.

Stomach.—Bile stained; contained 3 oz. sweet-smelling blood-stained mucus. Mucous membrane softened and shaggy. Cardiac end of stomach apparently healthy, and sharply defined from rest of mucous membrane.

Intestines natural, except for a number of petechial spots on peritoneal surface.

Liver.—Weight 22 oz., pale, with ill-defined lobules on section. Portal vein natural. Liver edge firm and sharp. Areas of better defined lobules present, with pinkish spots. Gall bladder and ducts natural and free from obstruction.

Spleen.—4 oz.; natural.

Lymphatic glands enlarged. *Kidneys, ureters, and bladder* natural.

[We owe the notes of this case to the House-Physician, Dr. Hedges.]

PROCEEDINGS
OF
THE ABERNETHIAN SOCIETY
FOR THE SESSION 1895-96.

OFFICERS.

<i>Presidents</i> . . .	Dr. H. B. MEAKIN and Mr. J. K. MURPHY.
<i>Vice-Presidents</i> . . .	Mr. S. GILLIES and Mr. F. A. SMITH.
<i>Secretaries</i> . . .	Mr. R. H. BREMRIDGE and Mr. W. R. STOWE.
<i>Additional Committee-men</i>	Mr. A. L. ORMEROD and Mr. J. A. WILLET.

June 20, 1895.

The Midsummer Meeting was held, Mr. Murphy, President, in the chair.

Dr. Thorne-Thorne delivered his address on 'Some Difficulties in Connection with the Isolation of Infectious Cases.'

A vote of thanks to Dr. Thorne-Thorne was unanimously carried.

October 10.

The Introductory Address was delivered by Dr. Church, Mr. Meakin, President, in the chair.

A vote of thanks to Dr. Church for his interesting address was proposed by the President, and seconded by Mr. D'Arcy Power.

October 17.

First clinical evening, Mr. Meakin in the chair.

Mr. Phillips showed a case of Kaposi's disease in a girl *æt.* 10.

Mr. Murphy showed a case of supposed thrombosis of the superior vena cava, a specimen of ruptured tubal gestation, four photographs of an early human ovum only ten days old, and microscopical specimens of the liver from a case of congenital ascites.

Mr. Meakin showed two cases where he had successfully treated chronic ulcers by a plastic operation.

Mr. Christopherson showed a case of mercurial tremors.

Mr. Bremridge exhibited a series of microscopical specimens kindly lent by Dr. Kanthack.

October 24.

First ordinary meeting, Mr. Murphy in the chair.

Mr. S. D. Rowland read a paper on 'Foreign Methods of Sero-Therapy in Diphtheria.' A long discussion followed.

October 31.

Second ordinary meeting, Mr. Meakin in the chair.

Mr. W. H. Maidlow read a paper entitled 'Biblical Medicine,' which was followed by some discussion.

November 7.

Second clinical evening, Mr. Murphy in the chair.

The President showed cases of spastic paraplegia, disseminated sclerosis, and syringo-myelia, and a case of 'cor bovinum' in a boy.

Mr. Phillips showed a case of epithelioma involving Stenson's duct, and a series of microscopical specimens.

Mr. Collins showed a case with enormously dilated veins on the surface of the abdomen, in which the question of thrombosis of the inferior vena cava was raised.

Mr. Belben showed a case of spastic paraplegia in a girl of ten, with mental symptoms.

November 14.

Third ordinary meeting, Mr. Murphy in the chair.

Mr. W. Langdon Brown read a paper on 'The Mechanism of Phagocytosis.' A short discussion followed.

November 21.

Fourth ordinary meeting, Mr. Meakin in the chair.

Mr. Hussey read a paper on 'The Blood Parasites of Malaria,' which was followed by a long discussion.

November 28.

Fifth ordinary meeting, Mr. Murphy in the chair.

Mr. Paterson read a paper on 'Some Points in the Diagnosis of Typhoid Fever.' A discussion followed.

December 5.

A meeting held in the Anatomical Theatre, Mr. Meakin in the chair.

Dr. Kanthack showed a photograph of a drawing of actinomycosis made by Mr. Thomas Smith thirty years ago.

Messrs. Roughton and Cosens read a paper on 'Photo-micrography,' illustrated by a practical demonstration.

December 12.

Sixth ordinary meeting, Mr. Murphy in the chair.

Dr. Gow read a paper on 'Some Forms of Tubal Disease.' A discussion followed.

January 16, 1896.

The Mid-Sessional Address was delivered by Mr. Henry Power, Mr. Murphy, President, in the chair. The subject was 'Medicine and Music,' and was illustrated by violin solos by Mr. Haydon.

A vote of thanks concluded the meeting.

January 23.

Seventh ordinary meeting, Mr. Meakin in the chair.

Dr. W. J. Collins read a paper on 'The Pathology of Cataract.' A discussion followed.

January 30.

Eighth ordinary meeting, Mr. Murphy in the chair.

Mr. Furnivall read a paper on 'Training.' A discussion followed.

February 6.

Third clinical evening, Mr. Meakin in the chair.

Mr. Murphy showed cases of thrombosis of the inferior vena cava, and pseudo-hypertrophic paralysis. He also showed (for Dr. Garrod) cases of congenital spastic paraplegia and diplegia, facial hemi-atrophy, and congenital pulmonary stenosis.

A series of microscopical specimens, lent by Dr. Kanthack, were exhibited.

February 13.

Ninth ordinary meeting, Mr. Meakin in the chair.

Dr. Claye Shaw read a paper on 'The Sexual Branch of the Connate Instincts.' There was a large attendance, and a short discussion followed the paper.

February 20.

Tenth ordinary meeting, Mr. Murphy in the chair.

Mr. L. C. P. Phillips showed cases of malformation of the external ear, and lymph-angiectasis in a girl.

Mr. Phillips then read a paper on 'Head Injuries.' A discussion followed.

February 27.

Eleventh ordinary meeting, Mr. Murphy in the chair.

Mr. Morland read a paper entitled 'An Aid to Human Anatomy: a Suggestion.' An animated discussion followed.

March 5.

Twelfth ordinary meeting, Mr. Meakin in the chair.

Mr. J. W. W. Stephens read a paper on 'Marriage of Kin.' A discussion followed.

March 12.

Thirteenth ordinary meeting, Mr. Murphy in the chair.

Mr. H. B. Meakin read a paper on 'The Shape of the Hard Palate as an Indication of Neurotic Inheritance.' Two cases were shown in which an abnormally high palate was associated with mental deficiency.

March 19.

Fourteenth ordinary meeting, Mr. Murphy in the chair.

A paper entitled 'Medicine in the Seventeenth Century,' written by Mr. C. C. Ingram Turnbull, was read by the Secretary, Mr. Turnbull being unavoidably absent.

March 26.

Annual General Meeting.

The report of the outgoing Committee was read and adopted.

The balance-sheet was read and passed.

The names of office-bearers elected for the ensuing year were then read :—

Presidents—Mr. J. W. Stephens and Mr. W. R. Stowe.

Vice-Presidents—Mr. R. H. Bremridge and Mr. J. Hussey.

Treasurer—Mr. Alfred Willett, F.R.C.S.

Secretaries—Mr. W. Langdon Brown and Mr. A. L. Ormerod.

Additional Committce-men—Mr. A. Compton and Mr. J. S. Williamson.

DESCRIPTIVE LIST

OF

SPECIMENS REVISED, REJECTED, AND
ADDED TO THE MUSEUM

DURING THE YEAR 1896.

Secondly, It has been found necessary to examine a number of specimens to which no proper or no definite diagnosis was appended, or which appeared to be wrongly described. This revision is of extreme importance, and ought to be continually practised. This year we have thus revised fifteen specimens, and in some instances corrected serious mistakes. The more important corrections have been tabulated in the following Table:—

No. of Specimen.	Diagnosis in Catalogue.	Diagnosis on Re-examination.
XX. 2188 <i>b</i>	Intussusception of sigmoid flexure into rectum	} Columnar-celled carcinoma.
XXI. 2194 <i>b</i>		
XXVIII. 2390	Tubercle of liver	Actinomycosis.
XLIII. 2962	Cancerous growth in kidney	Columnar-celled carcinoma.
XVIII. 2022 <i>a</i>	Adenoma of uterus	Alveolar carcinoma.
	Angio-sarcoma of small intestine	} Columnar-celled carcinoma (in parts colloid).
XVIII. 1987	} Contracted dysenteric ulcer of colon	} Columnar-celled carcinoma (in parts colloid).
XLIV. 3030	Polypoid growths of vagina	Mixed-celled sarcoma.

These revised specimens have been described under the heading of "Corrigenda."

Thirdly, An attempt has been made to supply a microscopic section with every specimen added, wherever, even though the diagnosis could hardly have been in doubt, this seems to be an advantage, or rather a necessity. A new departure has been made in establishing a fresh section of the catalogue, viz., the "Histological Records of Museum Specimens." Each microscopic section has been numbered exactly like the specimen itself, the Roman number indicating the Series, the other number the Specimen, so that there can be no difficulty in identifying it. It remains to revise the catalogue of the microscopic specimens in use, and to transfer all those specimens which are records of Museum Specimens to this section, and at the same time to prepare a special cabinet of typical histological specimens for the use of students. The Histological Records and the Students' Cabinet, however, should be kept apart, since they serve entirely different purposes.

Fourthly, It may be mentioned that a few specimens have been added which have been prepared with formalin and spirit and put up in glycerine, according to a method described in the *British Medical Journal*, 1896, vol. i. Epitome No. 468. It appears that this method preserves the colour of the tissues extremely well.

Fifthly, An Alphabetical Index has been prepared of the drawings and photographs in the Museum. This appeared to be necessary, since through repeated additions the same diseases or lesions have been classified under different headings, so that it is difficult to select all the drawings or photographs illustrating a given morbid lesion. Similar alphabetical indices must gradually be prepared of the different sections, and next year I hope to treat the Casts similarly.

The following is a table of the specimens added, re-numbered, or re-mounted, of casts, photographs, drawings, and microscopic specimens added :—

Old specimens re-mounted	156
Old specimens re-numbered	68
New specimens added	150
Casts added	22
Photographs added	107
Drawings added	29
Microscopic sections added	45

Before passing on to the description of the specimens added, I must allude to the great assistance which the Photographic Society, and especially Mr. T. J. Horder, has given me ; but still more necessary it is to mention the most useful help I have derived from Mr. T. Strangeways Pigg and the Assistant E. H. Shaw, who have cut almost all the sections required for the Museum, of which a fraction only will be found described below. From a pathological point of view the histological description of a specimen is a matter of necessity, and not of choice, and without their assistance this would have been very incomplete.

The following is the list of the donors of specimens whose names appear in this year's catalogue :—

- | | | |
|------------------------|-------------------------|---------------------------|
| C. E. Baker, Esq. | C. O'B. Harding, Esq. | C. B. Lockwood, Esq. |
| J. B. Burnett, Esq. | A. Harper, Esq. | S. Moberly, Esq. |
| H. T. Butlin, Esq. | W. H. Jessop, Esq. | M. G. Pearson, Esq. |
| C. B. Dale, Esq. | Dr. A. A. Kanthack. | D'A. Power, Esq. |
| H. S. Elworthy, Esq. | T. Kirsopp, Esq. | H. Power, Esq. |
| Dr. W. S. A. Griffith. | P. Lambert, Esq. | T. Strangeways Pigg, Esq. |
| J. H. Griffiths, Esq. | Dr. G. Lindsay Johnson. | B. Vernon, Esq. |
| Dr. A. Hamilton. | J. Langton, Esq. | Dr. S. West. |

I.

CORRIGENDA.

4. Two Photographs of "Partial Hypertrophy" of the foot. The second toe of the left foot is the part most affected.

The patient was a young Hindoo, aged 18. For details of the case see *British Medical Journal*, vol. ii. (1891), p. 188, where it was wrongly described as acromegaly.

Presented by A. A. Kanthack, M.D.

MELANOTIC SARCOMA.

- 1886f. Portion of Mesentery with multiple deposits, discoloured by an abundance of pigment, which determines their melanotic character. Microscopically the nodules are true melanotic sarcomas. There is no record whether these growths were primary, or what is more probable, secondary deposits. 2293 (1892).

A microscopic specimen has been preserved in the *Histological Records*, xvi. 1886f.

1987. Contraction, with extreme narrowing of the intestinal canal, of a portion of Colon, consequent, it was thought, upon the healing and cicatrisation of a dysenteric ulcer; but on microscopic examination (in 1896) this proved to be a columnar-celled carcinoma, in some parts colloid.

For a microscopic specimen see *Histological Records*, xviii. 1987.

From the collection of J. R. Farre, Esq., M.D.

- 2022a. Portion of Small Intestine obstructed by a new growth. From the jejunum to the ileo-cæcal valve the small intestine was thickened and dilated. Above the valve there was some pigmentation; the orifice of the valve was narrower than natural. On section of the walls of the intestine, there was found to be some increase of connective tissue, but no new growth. From the ileo-cæcal valve to the descending colon the intestine was dilated, and at the commencement of the descending colon it was obstructed by a large ragged new growth which extended round the mucous membrane. Microscopic examination showed that the growth was a columnar-celled carcinoma, colloid in parts. (1884.)

For a microscopic specimen see *Histological Records*, xviii. 2022a.

- 2087a. The Sac of a Femoral Hernia from the wall of which a cystic out-growth has been developed. This cyst did not communicate with the

peritoneal cavity, the hole behind, through which a green glass rod has been passed, having been made accidentally.

The specimen was removed in the course of an operation for the radical cure of the hernia. (1894.)

See *Female Surgical Register*, vol. iii. (1894), No. 194.

2188b. Intussusception of the Sigmoid Flexure into the Rectum in an adult.

The specimen consists of part of the descending colon and sigmoid flexure, which have been invaginated into the rectum; a piece of the latter has been removed, showing the lower orifice of the invaginated intestine, into which a piece of black catheter has been passed; the coats of the bowel in the neighbourhood of the intussusception are thickened, and there is a coating of coagulated mucus between the contiguous layers of mucous membrane: there is also some distension of the colon above the constriction.

The presenting part of the intussusception is thick, irregular, hard and nodular on section; it shows marked infiltration of the muscular and other coats by some form of new growth.

Microscopically the lesion proved to be a columnar-celled carcinoma.

The patient was a woman about 40 years of age, who died in the Surrey County Lunatic Asylum. She suffered from attacks of obstruction, sometimes accompanied with faecal vomiting, for the last two years of her life. She ultimately died from exhaustion following gangrene of the foot and leg. Post-mortem it was discovered that she also had two partially calcified uterine fibroids which pressed slightly on the rectum.

A section will be found in the *Histological Records*, xx. 2188b.

Presented by A. M. Jackson, M.D.

2217a. A Section of a Liver affected with diffuse sarcoma. The gland was uniformly enlarged, and was nearly white. Its surface was smooth. It weighed forty ounces. There were no isolated growths.

F., æt. 5 years, in perfect health until six months before death. Tubercle in both lungs. Microscopic examination showed the liver to be infiltrated with small round cells. The glandular destruction was not so great towards the centre as towards the surface, and there was a considerable amount of fibrous tissue in the central part. This tumour had been described as a lympho-sarcoma in the *Addenda* (1894), but on re-examining it in 1895, no reason could be found why the growth should be lympho-sarcoma rather than round-celled sarcoma.

A microscopical specimen will be found in the *Histological Records*, xxi. 2217a.

See *Transactions of the Pathological Society*, vol. xxxvi. (1885), p. 236.

ACTINOMYCOSIS.

2239c. A Piece of Liver which had been in this Museum for fourteen years, and had been described in the catalogue (see *Addenda*, 1894, No. 2194b, p. 157) as tubercular disease, while it evidently is, what it microscopically proved to be, viz. actinomycosis. The honey-combed appearance is very characteristic. 2194b (1894).

From a lad who died with amyloid degeneration of the abdominal viscera. He had previously suffered from symptoms of tubercular disease of the genito-urinary organs. There was no evidence of syphilis. Cicatricial contractions of

the upper and lower ends of the right ureter from old ulcerations were found, and hydronephrosis of the corresponding kidney, but there was no tubercular ulceration in progress.

A microscopical specimen has been preserved in the *Histological Records*, xxi. 2239c.

Presented by F. S. Eve, Esq.

Re-examined in 1894 by A. A. Kanthack, M.D.

2254. Portion of a Duodenum, with a small carcinomatous growth, surrounding and closing the orifice of the common bile duct. The tumour appears to grow from the mucous membrane. The adjacent parts are healthy. Microscopically it is a typical columnar-celled carcinoma (see *Histological Records*, xxii. 2254). xix. 5.

The patient, a woman 27 years old, was intensely jaundiced for three months before death. For the last nine days of her life she had copious hæmorrhage from the gums, nose, and intestines, and in the last two days discharged from the latter scarcely anything but blood. She died comatose. The case is recorded by Dr. Ormerod, in the *Lancet*, 1846.

2265. A Gall-Bladder exhibiting a soft cancerous growth sprouting from its lining membrane. Except at the seat of this growth it appears healthy. The growth is a soft alveolar carcinoma (see *Histological Records*, xxii. 2265).

From the same man as No. 2222 in the preceding Series.

- 2390a. A Cancerous Growth in the Right Kidney. The new tissue has infiltrated the whole of the organ, which was enlarged to the size of a fist. The tubular substance of the kidney has almost entirely disappeared, except for traces of the calyces. In the pelvis there was a ragged calculus about half an inch in length. The lining membrane of the ureter, which is considerably deflected in its course, is villous in appearance, being covered by innumerable papillomatous fringes. This villous appearance extends into the calyces. Both the pelvis and the calyces have been greatly altered in shape and size by the growth. Microscopically the latter was found to be a columnar-celled carcinoma, with an extensive but delicate fibrous stroma.

From a woman æt. 48 years. There were cancerous deposits in the liver and lungs.

Microscopical specimens have been preserved in the *Histological Records*, xxviii. 2390a.

See *Medical Post-Mortem Register*, vol. xi. p. 341.

CARCINOMA OF BODY OF UTERUS.

- 2962a. The Mucous Membrane of the Body of the Uterus is seen to be so greatly thickened as to form small masses which project into the uterine cavity. The cavity of the uterus was filled with thick tenacious mucus similar to that which is secreted by the cervical glands. The cervix is normal. Microscopically the growth is an alveolar carcinoma.

The Fallopian tubes presented only senile changes. The ovaries were small, hard, and white; a pedunculated cyst, as large as a marble, was in connection with the left ovary. From a woman æt. 63, who for fifteen or twenty years before her

death had suffered from myxœdema with atrophy of the thyroid gland. There was an effusion into the chief serous cavities of the body. In the *Addenda* (1894) this specimen was described as a simple adenoma.

A section has been preserved in the *Histological Records*, xliii. 2962a.

Presented by James Berry, Esq.

3030. A Large Cluster of Polypoid Growths removed from the nymphæ and walls of the vagina of a child. The largest growth is of oval shape, and nearly three inches in its chief diameter; the others are various in size and shape, some spheroidal, others pyriform, and the smallest not more than one or two lines in length. They are grouped without order; the largest is attached to the upper wall of the vagina and to the nymphæ, and at its upper part is traversed by the urethra; the others were attached to different parts of the vagina. In minute structure they appeared to be mixed-celled sarcomas, exhibiting also numerous large cells almost like giant-cells and extensive myxomatous degeneration. The largest was soft, elastic, opaque, white; the others, more like gelatinous polypi of the nose, were nearly pellucid, succulent, pale-yellowish, purple-pink, and white in various shades. xxxii. 66.

For a microscopic specimen see *Histological Records*, xliv. 3030.

3034. The Labia Pudendi affected with squamous-celled epithelioma. They are both enlarged and indurated. In the left labium, which is the most diseased, the cancer forms an elevated, circumscribed, and superficially ulcerated swelling.

They were removed from a middle-aged woman.

For a microscopic specimen see *Histological Records*, xliv. 3034.

SERIES LVII. 317.—SUBLINGUAL CYST (? DERMOID).

Drawing of the Face and Neck of a boy æt. 12 years, with a large Sublingual Cyst, which formerly had been described in this catalogue as a large ranula. Such it evidently is not; it may be a dermoid cyst, developed in the "mesobranchial area of His" (*v.* paper by Kostanecki and Mielecki in *Virchow's Archives*, vol. cxxi. p. 69). (T. Godart.)

II.

ADDENDA.

SERIES I.

DISEASES OF BONE.

EXOSTOSIS.

380a. A Typical Exostosis, which sprang from the lower end of the femur on its inner aspect, near the epiphysial line, and was removed by operation from a boy æt. 11 years. The outer shell of hyaline cartilage, enclosing soft, spongy, and in part as yet imperfect bone, is very characteristic, as was also the age of the patient and the site of the growth, which had been first noticed three years before the operation. There were no other exostoses.

See *Male Surgical Register*, vol. iv. (1895), No. 1828.

SPONTANEOUS FRACTURE.

507a. Upper portion of the Left Femur, removed after death from a woman æt. 30 years, who had suffered from scirrhus in the left breast. The breast and axillary glands had been extirpated $5\frac{1}{2}$ months before death; about three weeks later she complained of pain in the left lumbar and sacral regions, extending down the back of the thigh. A month later, slipping slightly, she broke her left femur clean across near the junction of the upper and middle thirds. The fracture was treated in the ordinary manner. The liver on admission was felt to be enlarged and nodulated, and both iliac crests were softened and the clavicles atrophied.

As seen from the specimen, there is practically no union. The post-mortem examination showed that there were secondary nodules in the liver and in the iliac crests, and the preparation further reveals a large nodule at the site of the fracture. Curiously enough, this had not been recognised at the autopsy. Microscopic examination proved the presence of scirrhus in this situation, which also had not been recognised in the original published account, in which the case was wrongly described as "one in which cancerous dissemination and osteomalachic processes progressed simultaneously."

A microscopic section has been preserved in the *Histological Records*, i. 507a. See also *Transactions of the Pathological Society*, March 3, 1896.

Presented by S. Moberly, Esq.

SERIES II.

DISEASES OF JOINTS.

TUBERCULAR KNEE-JOINT.

569c₁. A Knee-Joint removed by operation from a man æt. 52 years, which has been laid open. It is affected by tubercular disease; the synovial membrane is "pulpy," the cartilage has disappeared in places, and the bone has been in part destroyed by caries. All these changes are well shown. The joint when opened also contained pus.

See *Male Surgical Register*, vol. v. (1893), No. 3280.

SERIES VI.

DISEASES AND INJURIES OF MUSCLES,
TENDONS, AND BURSAE.

MELON-SEED BODIES FROM A BURSA.

1216e. A large collection of so-called Melon-seed Bodies removed from a large intermuscular bursa in the thigh of a man æt. 30 years.

See *Male Surgical Register*, vol. i. (1896), No. 3612*.

SERIES VII.

DISEASES AND INJURIES OF THE PERICARDIUM
AND HEART.

MEDIASTINAL ACTINOMYCOSIS.

1238l. A Section through the Heart and neighbouring Mediastinal Structures, obtained from a young man æt. 19 years. The heart is embedded in a curious honeycombed tissue, which microscopically was shown to be actinomycotic in nature. The pleura was extensively infiltrated with actinomycosis.

The patient had been treated for empyema, but the nature of the lesion, although seriously suspected, had not been detected after repeated histological examinations.

See *Surgical Post-Mortem Register* (1895), p. 167; and *Male Medical Register*, vol. iii. Part 2 (1895), No. 135.

CARDIAC ANEURYSM.

1260a. Section through the Left Ventricle of a man æt. 72 years, showing an aneurysm on its outer wall. The cardiac wall, just above and below the aneurysm, shows marked interstitial fibrosis (so-called interstitial

myocarditis). The ventricular wall generally is hypertrophied, and apparently shows slight interstitial changes in parts other than those mentioned before. The epicardial fat is also generally increased. Fibroid changes can also be made out under the endocardium, especially over the papillary muscle and some of the chordæ tendineæ.

The history of the case is indefinite, the notable feature having been repeated attacks of dyspnœa. At the post-mortem examination there was found atheroma of the aorta, œdema of the lungs, large kidneys and a congested spleen.

Presented by C. O'B. Harding, Esq.

1264a. The specimen shows a fairly large Aneurysm in the wall of the left ventricle at the apex of the heart. No references or notes were available; the valves of the heart were normal, but on examining the heart wall, there is certain evidence of pericarditis and of fibroid changes in the myocardium at and near the apex, and this latter condition satisfactorily explains the origin of the aneurysm.

1264b. Section through the Ventricular Septum of a Heart obtained from a man æt. 43 years, who was brought in dead. There is a large aneurysm, produced by a considerable dilatation of the right sinus of Valsalva, which extends downwards into the septum. The left coronary artery is plainly seen, but no trace of the right coronary artery could be found. There is also atheroma of the aorta and the marked fibrosis around the aneurysm is well shown.

See *Medical Post-Mortem Register*, vol. xxiii. (1896), p. 208.

INFECTIVE ENDOCARDITIS.

1302b. A Mitral Valve covered extensively on the auricular surfaces of both flaps with thick fibrinous vegetations. The latter spread also widely into the auricle. The endocarditis had appeared in the course of croupous pneumonia.

The history of the case could not be found in the Ward Notes.

See *Medical Post-Mortem Register*, vol. xx. (1893), p. 127.

DILATED HEART.

1327a. This specimen was obtained from a woman æt. 38 years. There is marked mitral stenosis, shortening and thickening of the valvular flaps and of the chordæ tendineæ, and thickening of the muscoli papillares. At the time of the examination the muscular substance was generally flabby, soft, and of a brownish tinge, and showed the tabby-cat striation (fatty degeneration) to a marked degree in both ventricles, though more in the left than in the right. The left auricle is enormously distended, and at the time of the autopsy could hold a good-sized fist. The coronary sinus is also considerably dilated. There was also considerable tricuspid stenosis and incompetence, with dilatation of the right ventricle and auricle, and the aortic valves were thickened and stunted. Amongst other points this specimen illustrates the mechanical effect of a dilated auricle on the aorta.

See *Medical Post-Mortem Register*, vol. xxii. (1895), p. 74; and *Female Medical Register*, vol. i. Part I (1895), No. 53.

SERIES VIII.

DISEASES AND INJURIES OF ARTERIES.

ATHEROMA OF AORTA.

1429a. This preparation shows the Arch of the Aorta and portion of the Pulmonary Artery of a man *æt.* 61 years. The aorta is markedly atheromatous, and there is a communication between the aorta and the pulmonary artery, at the site of the depression which corresponds to the ductus arteriosus, which no doubt is due to a perforation of an "atheromatous ulcer." The man died soon after coming to the Surgery.

See *Medical Post-Mortem Register*, vol. xxii. (1895), p. 326.

DISSECTING ANEURYSM OF THE AORTA.

1464a. Arch of the Aorta removed from the body of a man (*insane*) *æt.* 51 years. He was suddenly seized with a severe paroxysm of pain, and died twelve hours later.

The outer coat has been laid open, and reveals an almost complete transverse rupture through the inner and middle coats of the aorta one inch above the semilunar valves. The outer coat was separated by blood from the underlying middle coat along the convexity of the arch, and had ruptured into the pericardium by a vertical slit three-quarter inch long on the posterior aspect of the first part of the arch, the centre of which corresponded to the transverse rupture of the other coats. The blood had passed a little way up between the middle and outer coats of the innominate as well as the left common carotid artery, and down along the aorta as far as the left external iliac artery (see the next specimen).

The large vessels and those at the base of the brain were atheromatous; 10 oz. of blood-clot were found in the pericardium.

Presented by H. S. Elworthy, Esq., F.R.C.S.

1464b. Longitudinal section through the Descending Thoracic and Abdominal Aorta, Common Iliacs and their Bifurcations, showing an extensive effusion of blood between the middle and outer coats. This extends as far as the left external iliac artery. The aorta is atheromatous. Further details will be found under Specimen No. 1464a. The other half of this specimen is preserved in the Museum of the Royal College of Surgeons.

See *British Medical Journal*, vol. i. (1896), p. 398.

Presented by H. S. Elworthy, Esq., F.R.C.S.

SERIES X.

DISEASES AND INJURIES OF THE LARYNX
AND TRACHEA.

SEPTIC INFLAMMATION.

1615a. Posterior view of a Larynx, removed from a woman *æt.* 42 years, who died of so-called Angina Ludovici. The œdematous swelling of the mucosa over the epiglottis, of the aryteno-epiglottidean folds, and over the arytenoid and cricoid cartilages is well shown. The mucous membrane of the pharynx was also swollen. The swelling is so considerable that the entrance to the larynx is greatly narrowed.

The lesion was evidently septic in nature, and was followed by a miscarriage (7½ months). She was admitted into the Surgery suffering from dyspnoea and general debility and difficulty in swallowing; there was œdema of both cords. Before she could be moved from the Surgery to the Ward she suddenly aborted; she died next morning. Bacteriologically pneumococci were found locally in the neck and larynx, and also in the spleen. The œdematous infiltration extended beyond the larynx into the œsophagus and tissues of the neck generally, which were described as brawny, and also contained pus. Further, there was purulent pleurisy and pericarditis. The uterus contained a small mass of clot (? septic).

See *Medical Post-Mortem Register*, vol. xxii. (1895), p. 106; and *Martha Ward Book*, No. 1224; and for the Pathology of this subject see a paper by Dr. F. Semon in the *Transactions of the Medico-Chirurgical Society*, vol. lxxviii. p. 181.

TYPHOID ULCERATION.

1641a₁. The Larynx removed from the body of a girl *æt.* 17 years, who died between the third and fourth week of typhoid fever. The tip of the epiglottis is eroded and superficially ulcerated. The intestines showed well-marked typhoid ulcers, most of them still with adherent sloughs.

See *Medical Post-Mortem Register*, vol. xx. (1893), p. 376; and *Faith Ward Book* (1893), No. 193; also *St. Bartholomew's Hospital Reports*, vol. xxxi. p. 113.

1641a₂. Larynx of a man *æt.* 20 years, who died in the fifth week of typhoid fever. The tip of the epiglottis is ulcerated in its whole extent, and the cartilage exposed and bare in a necrotic condition. The intestines showed fresh typhoid ulcers.

See *Medical Post-Mortem Register*, vol. xxii. (1895), p. 264; and *Mule Medical Register*, vol. iv. Part 2 (1895), No. 193; also *St. Bartholomew's Hospital Reports*, vol. xxxi. p. 113.

1641b₁. Larynx removed from the body of a man *æt.* 28 years, who died in the sixth week of typhoid fever from general peritonitis (not perforative in nature). The specimen shows a deep ulcer just under the left processus vocalis; a probe passed into it downwards and forwards grated against the bare cartilage (or bone) of the cricoid ring. At the autopsy, pus could be squeezed out from below through the slit-like

opening of the ulcer. The intestines showed healing and cicatrising ulcers, except on the ileo-cæcal valve, where fresh ulcers still existed.

See also Specimen No. 2006b; and *Medical Post-Mortem Register*, vol. xxii. (1895), p. 276; also *St. Bartholomew's Hospital Reports*, vol. xxxi. p. 113.

DIFFUSE PAPILOMA.

1645b. Looking into the aditus laryngis from above, it will be seen that it is completely blocked by densely papillomatous growths, which occupy the whole of the larynx as seen from below, where parts of the cricoid cartilage and posterior wall of the trachea have been removed. On the anterior wall of the trachea is an irregular tracheotomy opening. A few small warty growths were found on the anterior wall of the trachea, one of which has been left, the others having been removed for histological purposes.

The larynx was removed from an infant æt. 2 years, who had suffered from difficulty of breathing and hoarseness for about six months. On one occasion intubation had to be performed, and subsequently the child could not get on without the tube, and it was through loss of the tube, which was coughed up, that the child died.

Microscopically the growths both in the larynx and in the trachea were ordinary papillomata, covered in either situation with squamous epithelium.

A microscopical section is preserved in the *Histological Records*, x. 1645b.

See *Surgical Post-Mortem Register* (1895), p. 240; *Male Surgical Register*, vol. iv. (1895), No. 1400; and *Male Medical Register*, vol. ii. Part 2 (1895), No. 239; also *Transactions of the Laryngological Society*, vol. iii. 1895-96.

1645c. The Larynx of a child æt. 4 years. The mucosa is profusely studded with delicate papillomatous growths, roughly arranged on either side in two rows, one along and below the true vocal cord, and the other above the false cord. Similar growths are seen on the posterior wall and also some distance below the cords. Tracheotomy had been performed on account of dyspnœa. Delicate papillomatous growths appear through the tracheal opening, which during life resembled granulations, and had been described as such.

The child died of asphyxia a day before it was to have been operated upon; the tube had been forced out accidentally, and, in spite of dilatation and artificial respiration, the child soon died. During life the child had suffered from occasional attacks of severe dyspnœa, probably caused by obstruction of the tube by the growths found at the upper part of the trachea.

Microscopically the endolaryngeal growths were proved to be ordinary squamous-celled papillomata, while the growths around the tracheal opening show a lining of stratified columnar epithelium.

A microscopical specimen has been preserved in the *Histological Records*, x. 1645c.

See *Surgical Post-Mortem Register* (1895), p. 179; *Female Surgical Register*, vol. iv. (1895), No. 2544*; and *Transactions of the Laryngological Society*, vol. iii. 1895-96.

CARCINOMA.

1656c. Sagittal section through the Tongue, Larynx, Trachea, Pharynx, and Œsophagus removed from a man *æt.* 49 years. The whole larynx is entirely filled up by a growth (microscopically a squamous-celled carcinoma). Below, at the upper end of the trachea, a channel is seen; this represents the track of the tracheotomy tube which had been inserted four months before death. At that time the laryngoscope revealed an endolaryngeal growth on the right vocal cord, involving also the ventricular band. A month later thyrotomy was performed, and a sessile growth removed from the right half of the larynx. Subsequently the growth sprouted rapidly along the track of the tube, fungating outside over the skin. This is well shown in the specimen, where a large cauliflower-like growth is found under the chin, continuous with the laryngeal growth. Posteriorly the growth had also fungated through the pharyngeal mucosa over the posterior wall of the larynx, as may be seen in the specimen.

See *Surgical Post-Mortem Register* (1893), p. 45; *Male Surgical Register*, vol. iii. (1893), No. 2871*; and *Proceedings of the Laryngological Society*, vol. iii. 1895.

1656e. Epithelioma of the Larynx of a man *æt.* 43 years, who died with phthisis pulmonalis. A section has been made into the growth of the right vocal cord to show its infiltrating nature. This specimen illustrates the co-existence of carcinoma and tuberculosis.

A microscopic specimen has been preserved in the *Histological Records*, x. 1656e. See *Medical Post-Mortem Register*, vol. xxiii. (1896), p. 133; and *Male Medical Register*, vol. ii. (1896), *sub* James Smith.

1657a. The pharynx has been opened from behind and the larynx from the front. From behind a large piece of the necrosed cricoid cartilage is seen lying in an irregular mass of swollen and breaking-down tissue, which microscopically proved to be a squamous-celled carcinoma (epithelioma). There is much destruction of tissue in the right sinus pyriformis, while the left sinus is encroached upon by a mass of growth. From the front, it will be seen that the larynx is greatly narrowed, distorted, and obstructed by the growth, which is infiltrating the posterior wall, the lateral folds, and the ventricular bands.

The specimen was obtained after death from a woman who had presented no symptoms until towards the end, when there was much dyspnoea.

A microscopic section is preserved in the *Histological Records*, x. 1657a.

Presented by T. Kirsopp, M.B.

STENOSIS AFTER TRACHEOTOMY.

1659d. Specimen of the Larynx and Trachea of an infant *æt.* 2 years, upon whom tracheotomy had been performed five weeks before death on account of diphtheria. The skin wound had completely scarred; the larynx is normal, and the hole of the tracheal opening, which had involved the upper two or three rings, is closed by fibrous tissue, which, on contracting, had caused marked narrowing of the upper part of the trachea, so that before the posterior wall of the trachea

and larynx was divided, the diameter of the lumen here was said to have measured $\frac{1}{3}$ in., if not less.

See *Medical Post-Mortem Register*, vol. xx. (1893), p. 379; and *Radcliffe Ward Book* (1893), No. 76.

FOREIGN BODY IN LARYNX.

1660a₁. A sagittal section through the Tongue, Larynx, and Pharynx of a man who was admitted in a dying condition. The aditus laryngis is completely obstructed by a piece of meat, which is firmly locked between the epiglottis and the posterior and outer walls of the larynx. At the post-mortem examination it was found that the epiglottis was pushed forward against the base of the tongue.

See *Medical Post-Mortem Register*, vol. xx. (1894), p. 261; and *Transactions of the Laryngological Society*, vol. iii. (1895-96).

TUBERCULAR GLANDS.

1664l. A complete dissection of the Larynx, Trachea, and its Bifurcation, with the adjoining structures, from a case of tuberculosis. The lymphatic glands around the aorta and the bifurcation, as well as the cervical glands, are all enlarged, yet not fused, but more or less discrete. One of the tracheal glands behind has been laid open to show the caseation. It is interesting to note the encroachment upon the arch of the aorta and the large vessels, which must have caused some narrowing and some resistance to the circulation. The left bronchus is also somewhat pressed upon and slightly constricted. Amongst other things, the specimen also shows a small ulcer at the right processus vocalis. At the post-mortem examination the lungs were found to be tubercular, as also the pericardium and peritoneum, the omentum especially being much thickened and converted into a thick triangular mass. The spleen and abdominal glands were also tubercular.

See *Medical Post-Mortem Register*, vol. xxii. (1895), p. 337; and *Male Medical Register*, vol. i. Part 2 (1895), No. 292.

1664m. The Lungs, Larynx, and Trachea, with the adjoining structures, removed from a child *æt.* 5 months. The lungs are tubercular and studded with caseous nodules (caseous broncho-pneumonia); the bronchial glands are considerably enlarged, and in part caseous (tubercular) and adherent to each other. They surround and press upon the arch of the aorta and the large vessels, the aorta especially having been pressed upon. Behind there is an abscess cavity below the bifurcation of the trachea, which communicates with a ragged cavity in the left lung. The latter is also in direct communication with the left bronchus, the wall of which has in part been destroyed by ulceration. The mucous membrane of the trachea near the bifurcation, and of the right bronchus, is in a condition of superficial ulceration (erosion). There were also tubercular ulcers in the intestines and tubercular deposits in the spleen. The abdominal lymphatic glands were generally caseous.

See *Medical Post-Mortem Register*, vol. xxii. (1895), p. 81; and *Male Medical Register*, vol. iii. Part 2 (1895), No. 201.

SERIES XI.

DISEASES AND INJURIES OF THE PLEURA,
BRONCHIAL TUBES, AND LUNGS.**GANGRENE OF THE LUNG.**

1704c₁. Section through the Right Lung of a girl *æt.* 4 years. The whole upper lobe and a part of the lower lobe are completely gangrenous. The lower lobe shows distinct, though not extensive, bronchiectatic changes, and also at the lower and inner angle destructive or ulcerative broncho-pneumonic conditions. It is probable, therefore, that the gangrene was secondary to an acute broncho-pneumonia. There was also pleurisy (as well shown in the specimen), and the pleural cavity at the time of the post-mortem examination contained a small quantity of foul-smelling brownish dirty fluid. The left lung was natural.

See *Medical Post-Mortem Register*, vol. xx. (1893), p. 294; and *Mary Ward Book* (1893), sub Elizabeth Rowland.

HÆMORRHAGIC INFARCTION.

1708a. Section through the Lower Lobe of the Left Lung of a man *æt.* 31 years, who died with mitral and tricuspid stenosis and incompetence. The specimen shows the characteristic appearances of a hæmorrhagic infarction in the lung; the pleural surface is slightly bulged, the infarction red and hæmorrhagic, with consolidation around; the branch of the pulmonary artery running into this lobe is plugged.

The engorged intestine of this case has been preserved in Series xviii. No. 1956b.

See *Medical Post-Mortem Register*, vol. xxii. (1895), p. 80; and *Male Medical Register*, vol. ii. Part 2 (1895), No. 70.

PYÆMIC LUNG.

1711c. The two halves of a Lung obtained from a young man *æt.* 18 years, who died from pyæmia and septicæmia after otitis media and suppurative thrombosis of the lateral sinus. The sections of the lung show under the pleura several pyæmic foci (infarcts) in various stages of development. At the upper part there is a comparatively fresh infarct, marked (1) in the specimen; then follow (2) one a little more advanced; (3) one breaking down; and (4) a true pyæmic abscess. The pleura itself shows evident signs of inflammatory changes.

The parts about the ear have been preserved in Series xxxiv. No. 2676e.

See *Male Surgical Register*, vol. ii. (1895), No. 807; and *Surgical Post-Mortem Register* (1895), p. 74.

CASEOUS PNEUMONIA.

1718c₁. A coronal Section through a whole Lung, which was completely solidified on account of chronic tubercular changes. It is an instance of so-called "caseous pneumonia." There is also a cavity under the pleura

which had ruptured through the serous coat and caused a pneumothorax. Further, the bronchi are ectatic, and ulcerative processes are evident in several places in the shape of smaller cavities, and the general caseation is beautifully shown. The pleura was adherent, as can be seen from its roughened and fibrinous appearance.

See *Medical Post-Mortem Register*, vol. xxii. (1895), p. 275; and *Female Medical Register*, vol. ii. Part 2 (1895), No. 160.

MILIARY TUBERCULOSIS.

1719a. Section through a Lobe of a Lung from a child *æt.* 10 months, showing typical military grey tubercles. The post-mortem examination further revealed tubercular meningitis and tubercles in the liver and spleen and suppurative otitis media. (Fixed with formalin and preserved in glycerine.)

See *Medical Post-Mortem Register*, vol. xxiii. (1896), p. 177.

TUBERCULAR LUNG.

1724d. A Solid Lung, possibly tubercular in nature, obtained from a child *æt.* 11 months, who was admitted with diphtheria and died twenty days later. There was also adhesive pleurisy with serous effusion. The lung is fleshy and solid, and shows scattered purulent or caseating foci. (Fixed with formalin and preserved in glycerine.)

See *Medical Post-Mortem Register*, vol. xxiii. (1896), p. 193.

ACTINOMYCOSIS OF LUNG.

1727e. Section through the Right Lung of a man *æt.* 31 years. The pleura is much thickened, fibrous, and fibrinous. The section of the lung is studded with numerous small tubercle-like masses, which now, however, mostly appear in the form of small cavities, because their contents have fallen out. When fresh, it was almost, if not quite, impossible to distinguish them from military tubercles. Some of them were translucent, others yellow, suggesting caseating tubercles, with this difference, that most of them on section appeared to be hollow or dimpled, and some of them had a sinuous outline. The numerous sinuous hollows are well seen in this hardened specimen, and also their relation to the bronchi and bronchioles. Microscopically the lesion was an actinomycotic broncho-pneumonia.

Both lungs were affected; the right lung was tightly adherent to the diaphragm, and between it and the diaphragm a mass of yellow cheesy matter was found, suggesting a local empyema. This "empyema" communicated with the liver, which was also actinomycotic, and has been preserved in Series xxi. No. 2239d. Actinomycotic foci were found further in the right suprarenal capsule, the right kidney, and the spleen, the suprarenal capsule and kidney being infected by extension of the disease from the liver. Actinomycotic (pyæmic) abscesses existed in the right elbow-joint, and in the middle of the calf of the left leg near the inner side, and also over and in the left deltoid muscle.

See *Transactions of the Pathological Society*, vol. xlv. (1894), p. 233; *Medical Post-Mortem Register*, vol. xx. (1893), p. 344; and *Mark Ward Book* (1893), No. 240.

SARCOMA OF LUNG.

1728d. Section through the Right Lung and part of the anterior Chest Wall. The skin is bulged forward by a growth which has destroyed the ribs to some extent, and has passed backwards into the lung, infiltrating it. Microscopically the tumour is a large-celled sarcoma, presenting a decidedly alveolar arrangement; bone was absent. It is possible that the growth began in the pleura, but its starting-point may also have been the chest wall. During life the case had presented some difficulty in the diagnosis, aneurysm having at one time been suspected.

Presented by J. Howell Griffiths, M.B.

SERIES XII.

DISEASES OF THE MOUTH, TONGUE, PALATE,
AND FAUCES.**ANGIO-FIBROMA.**

1770c. Two Large Polypoid Masses removed by operation from the nasopharynx of a boy *æt.* 14 years. To obtain access to the growth the right superior maxilla had to be removed. The two polypi appeared to be attached to the periosteum of the sphenoid bone. The growths had been diagnosed on clinical grounds as sarcomatous. There had been very frequent epistaxis and pain and swelling in the face; the right nostril was completely obstructed. After removal the growths were shown to be angio-fibromata. A microscopic specimen has been preserved in the *Histological Records*, xii. 1770c. The vascularity of the growth is well seen on examining the surface of the section. The superior maxilla of this case has been preserved (see No. 1774l).

See *Male Surgical Register*, vol. ii. (1894), No. 3634*; and *Lancet*, vol. ii. (1895), p. 913.

1770d. A Large Polypoid Mass, weighing 4 ounces, removed by operation from the nasopharynx of a young man *æt.* 19 years. As in the previous case, to obtain access to the growth the left superior maxilla had to be removed. The tumour was attached to the base of the skull. Microscopically the growth proved to be a vascular fibroma (angio-fibroma). The vascularity of the same is well shown on section in the lower part of the specimen, the surface of the section being riddled with little holes, the openings of the vessels.

The superior maxilla of this case has been preserved in this Series (No. 1774m), and microscopic specimens in the *Histological Records*, xii. 1770d.

See *Male Surgical Register*, vol. iv. (1896), No. 575. See also *Lancet*, vol. ii. (1895), p. 913.

1770e. The Two Halves of a large Nasopharyngeal Polypus, removed from a young man *æt.* 19 years by operation after removal of the upper jaw and splitting of the palate. The polypus consists of soft cellular fibrous tissue,

and was excessively vascular. The enormous number of blood-vessels is well shown, the patent openings of the vessels being very evident. The tumour after removal measured $2\frac{1}{2}$ by $1\frac{1}{2}$ inches, had a well-defined capsule, and was elastic to the touch. On section, numerous vessels and sinuses were seen, one large enough to admit the tip of the little finger.

History.—The patient had suffered from nasal obstruction, but had never had epistaxis.

See *Male Surgical Register*, vol. iv. (1894), No. 1893; also *Lancet*, vol. ii. (1895), p. 913.

EMPHYEMA OF ANTRUM.

1774b. Right Upper Maxilla with Antrum Highmori, which had been the seat of an empyema. The antrum is enlarged, posteriorly a polypoid mass is found, and the antral lining generally is thickened. The anterior alveolar plate is partially necrosed and destroyed, and the first molar tooth is missing, having been removed to supply an outlet for the pus. A blue glass rod has been passed into the antrum to show the communication between the antrum and the fang cavity.

History.—The patient, a boy æt. 11 years, had a severe attack of toothache, followed by swelling of the right cheek, discharge from the right ear, and severe constitutional disturbances. There was also a purulent discharge from the right nostril and from the right posterior naris. The antrum was opened from the alveolus, but the symptoms persisted, rigors became frequent, the inflammation spread over the face and down the neck, and the boy died within two months of the onset of the toothache. The post-mortem examination revealed meningitis, suppurative thrombosis of the cavernous sinuses, gangrenous cellulitis of the left orbit, necrosis of the right lower maxilla, and abscesses in the lungs.

See *Male Surgical Register*, vol. v. (1894), No. 2790; and *Surgical Post-Mortem Register* (1894), p. 157.

ATROPHY OF SUPERIOR MAXILLA.

1774l. The Right Superior Maxilla, removed from a boy æt. 14 years, for the purpose of gaining access to two large nasopharyngeal growths, which were successfully removed. The growths have been preserved in this Series (No. 1770c.)

This specimen illustrates the effects of continually increasing pressure on bone and soft tissues.

The turbinated bones have almost disappeared, the antrum of Highmore is considerably reduced in size; the alveolar process has suffered, so that the teeth are most irregular and crowded.

See *Male Surgical Register*, vol. ii. (1894), No. 3634*.

1774m. A Left Upper Maxilla, removed from a young man æt. 19 years, for the purpose of gaining access to a very large nasopharyngeal tumour, which was successfully removed, and has been preserved in this Series (No. 1770d). The specimen demonstrates the effects of pressure, which led to absorption of bone and considerable flattening of the antrum of Highmore. A small polypus is found in the antrum, springing from its nasal wall.

See *Male Surgical Register*, vol. iv. (1896), No. 575.

CYST OF TONGUE.

1779a₁. Sagittal Section through the Tongue and Larynx of a child æt. 1 year, who died of diphtheria with a post-pharyngeal abscess. There is a large cyst at the basis linguæ, and below this a number of other small cysts. The large cyst was filled with transparent jelly-like material, and communicated with the buccal cavity through a fairly large oval opening, situated behind the foramen cæcum. There was no evidence of a lingual or thyreo-glossal duct.

See *Medical Post-Mortem Register*, vol. xx. (1893), p. 151.

LACERATION.

1795a. A Tongue mutilated and lacerated by gunpowder exploded in the mouth with intent of committing suicide, by filling some gas-piping with powder and setting a light to it, while the tube was held in the mouth. The injury ended fatally shortly afterwards.

See *Male Surgical Register*, vol. iv. (1894), No. 2618; and *Surgical Post-Mortem Register* (1894), p. 146.

TUMOUR OF PALATE.

1800d. A Tumour removed from the hard palate of a woman æt. 40 years, where it had been growing for ten years. Microscopically it proved to be one of those curious adenomatous growths containing concentric bodies described by Mr. Stephen Paget in the *Transactions of the Pathological Society*, vol. xxxviii. p. 348.

A microscopic section of this specimen has been preserved in the *Histological Records*, xii. 1800d.

See *Female Surgical Register*, vol. i. (1896), No. 2825*.

PAPILLOMA OF TONSIL.

1807d. A papillomatous Hypertrophied Tonsil removed by operation from a woman.

A microscopic section has been preserved in the *Histological Records*, xii. 1807d.

SERIES XV.

DISEASES AND INJURIES OF PHARYNX AND
ŒSOPHAGUS.**ŒSOPHAGEAL POUCH.**

1833b. A Pressure Pouch removed by operation from the back of the œsophagus at its junction with the pharynx. The patient was a man 73 years of age, in whom symptoms had existed for about four years. The early symptoms were difficulty in swallowing, and the return of tiny fragments of food many hours after they had been taken: the fragments of food were but little altered, and could be readily recognised. The

later symptoms, in addition to those mentioned, were difficulty of introducing an instrument into the stomach, and the appearance of an ill-defined, soft swelling in the posterior triangle of the neck on the left side during the taking of food. The patient made a good recovery from the operation.

A microscopic section has been preserved in the *Histo'ogical Records*, xii. 1833b.; it shows the presence of striped muscular fibres in the wall of the pouch.

Presented by H. T. Butlin, Esq., F.R.C.S.

SERIES XVI.

DISEASES OF THE PERITONEUM, OMENTUM, AND MESENTERY.

TUBERCULAR OMENTUM.

1883b. A typical specimen of the Large Omentum, extensively and generally infiltrated with tubercle.

HYDATID CYST.

1893a. Section through a large sterile Hydatid Cyst growing near the peritoneum in connection with the sigmoid flexure opposite its mesenteric attachment. Cysts were also found in the liver and in the neighbourhood of the kidney, and have been preserved in Series xxi. No. 2230b., and Series xxviii. No. 2393i. respectively.

See *Surgical Post-Mortem Register* (1895), p. 87; and *Male Surgical Register*, vol. iii. (1895), No. 1015.

SERIES XVIII.

DISEASES AND INJURIES OF THE INTESTINES.

ENGORGEMENT OF INTESTINE.

1956b. A Piece of Jejunum, obtained from a man æt. 31 years, who died with mitral and tricuspid stenosis and incompetence, and had extensive infarction of the left lung. The mucous membrane of the bowel is dark, and considerably engorged with blood and thickened. The liver was also engorged (nutmeg liver.)

A specimen of the pulmonary infarct has been preserved in Series xi. No. 1708a.

See *Medical Post-Mortem Register*, vol. xxii. (1895), p. 80; and *Male Medical Register*, vol. ii. Part 2 (1895), No. 70.

INTESTINAL ULCERATION.

1963b. Portion of Ileum of an infant aged 7 weeks, showing numerous small round and oval ulcers, about the size of lentils, clearly punched

out, with sharp and slightly thickened edges. At the post-mortem examination it was found that the ileum contained numerous ulcers for $4\frac{1}{2}$ feet above the cæcum. There was collapse of the lungs and recent pleurisy; on the pleuræ covering both lungs, a few very small white spots, closely (?) resembling tubercle, but not caseous, were found. During life the only symptoms were vomiting and diarrhœa. All other organs were natural.

See *Medical Post-Mortem Register*, vol. xxi. (1894), p. 203.

ULCERATIVE COLITIS.

1987d. Piece of Large Intestine, removed from a woman æt. 27 years, showing the lesions characteristic of ulcerative colitis.

There was a history of a previous attack of colitis two years before death. The fatal attack had lasted thirty-one days, and it appeared that two brothers of the patient died of the same complaint at about the same age.

Presented by J. B. Burnett, M.B.

1987e. A portion of the Rectum from a woman æt. 36 years, who died of general ulcerative colitis. The mucous membrane shows very extensive ulceration, the surface being considerably undermined and destroyed in parts. In two places the ulceration has led to curious pouching. The cæcum and colon also presented an extraordinary degree of ulceration. The case has been described by Dr. H. Tooth in the *Transactions of the Pathological Society*, vol. xlv. (1894), p. 66.

See *Medical Post-Mortem Register*, vol. xx. (1893), p. 318; and *Mary Ward Book* (1893), No. 166.

SWINE FEVER.

1987f. Two portions of Large Intestine from a pig which died of swine fever. The upper piece is a portion of cæcum, the lower piece a portion of the colon. Both show a number of ulcers characteristic of the lesion.

See also *Transactions of Pathological Society*, vol. xlvi. (1895), p. 334.

Presented by T. Strangeways Pigg, Esq.

ENTERIC FEVER.

2006b. Portion of Ileum from a man æt. 28 years, who died of enteric fever in the seventh week. The serous surface is covered with thick "organised" lymph. There was general peritonitis, the coils of the small intestine being matted together by fresh adhesions. The ulcers were in a condition of healing and cicatrisation: some such ulcers may be seen on the mucous surface of the gut. No perforation was found at the post-mortem examination.

The larynx of this case, showing perichondritis and necrosis, has been preserved in Series x. No. 1641b₁.

Reference to this case in the *Medical Register* could not be found. See *Medical Post-Mortem Register*, vol. xxii. (1895), p. 276.

TUBERCULAR DISEASE.

2012c. A longitudinal section through a loop of Small Intestine and the Cæcum, removed by operation from a man æt. 34 years, who had suffered for some months with symptoms of chronic obstruction. The ends of the gut remaining behind were joined by means of a Murphy's button, and the patient did extremely well after the operation. The wall of the gut below the cæcum (as the specimen hangs) is greatly thickened for a distance of several inches, so that there is a considerable stricture. This thickening also extends into the ileocæcal valve and beyond it into the cæcum. On examining the loop further away from the cæcum, several annular strictures will be observed, where the ridges of the valvulæ conniventes and the intestinal wall are especially thickened; but also between these annular strictures the wall is considerably infiltrated and thickened. It will further be seen that there are a number of ulcers corresponding to the sites of the annular strictures, best seen on the free border of the gut, opposite to the mesenteric attachment. On turning the specimen round, it will be seen that under the serous coat, corresponding to the sites of the annular strictures, there are small collections of nodules; but smaller nodules will also be found scattered about in other situations, especially below and in the part just below the cæcum where the infiltration is most exaggerated and the stricture most pronounced. The lymphatic glands are enlarged.

A small portion of the loop has been hung up separately to show the appearance of the annular constrictions before the bowel was opened. The lesion is a tubercular one, as proved by microscopical examination.

The patient made an uninterruptedly good recovery, the button coming away in ten days. The patient was alive and well ten months afterwards.

Presented by J. Langton, Esq., F.R.C.S.

2012d. A portion of Jejunum showing typical tubercular ulceration. The ulcers run in the transverse axis of the gut, their bases are irregular and tuberculated, and the serous surface of the gut is covered with numerous miliary tubercles, corresponding mostly to the course of the ulcers.

Note the distribution of the tubercles along the blood-vessels on the serous surface, especially at the lower part of the specimen.

A portion of the ileum is preserved; see next specimen.

See *Medical Post-Mortem Register*, vol. xxiii. (1896), p. 112.

2012e. A portion of the Ileum from the same case as the preceding specimen, showing typical tubercular ulceration. Most of the ulcers are of considerable size, and arranged in the transverse axis of the gut, but besides there are a number of small follicular ulcers. The serous surface of the gut is studded with small miliary tubercles, which are most numerous in situations corresponding to the ulcers on the mucous surface. The distribution of the tubercles along the blood-vessels is well shown.

POLYPI.

2019e. Portion of Jejunum of a girl æt. 13 years, who died of septi-cæmia.

The valvulæ conniventes are profusely studded with small, short, stumpy polypoid excrescences, which have thick short stalks, and are velvety in appearance from the presence of villi on their surface. The polypi are not entirely restricted to the ridges of the valvulæ, but a few may also be seen between the ridges. There were similar excrescences in the duodenum and ileum, and a few larger polypi in the sigmoid flexure.

Microscopically these polypi are seen to consist of all the various elements of the mucosa, and they are all of them beset with villi; dilated lymphatics, as described in a similar case by Drs. Alchin and Hebb (see *Transactions of Pathological Society*, vol. xlvi. (1895), p. 220), are absent.

Two microscopic specimens have been preserved in the *Histological Records*, xviii. 2019e.

A portion of the sigmoid flexure has been preserved in this Series (No. 2019f.).

See *Medical Post-Mortem Register*, vol. xxii. (1895), p. 107; and *Female Medical Register*, vol. i. Part 1 (1895), No. 79.

2019f. Portion of Sigmoid Flexure from the same case as the preceding specimen, showing a small pedunculated polypus. The mucosa of the small intestine was studded with numerous small polypi.

2019g. Portion of Small Intestine from a man who died at the Radcliffe Infirmary, Oxford. From its mucous membrane there spring a number of comparatively large polypi, which microscopically are identical with those more generally observed in the large intestine and rectum. There were a large number of such large polypi distributed over the stomach and the whole length of the small intestine.

For a full description of the case and specimen, see *Transactions of the Pathological Society*, vol. xlvii. (1896), p. ; and *British Medical Journal*, vol. ii. (1895), p. 973.

Presented by Dr. W. Collier (Oxford).

MYXO-SARCOMA.

2027i. A portion of the Cæcum and the Ascending Colon opened by a longitudinal section, removed after death from a boy æt. 17 years, on whom a right lumbar colotomy had been performed on account of acute intestinal obstruction. Seven months afterwards the artificial anus was closed, with the result that the wound did not heal and fæcal matter was discharged through it, and 4½ months later the boy died. The bowel is extensively affected and altered by a "colloid myxomatous" growth, more especially pronounced at the upper part (as the specimen hangs) near the cæcum. This growth also extends into and infiltrates the outer coats of the bowel.

The peritoneal cavity at the post-mortem examination contained much

deeply bile-stained fluid, and the peritoneum all over was studded with nodules of new growth, colloid in appearance. The main mass of the new growth occupied about the middle of the ascending colon, and it was here that the colotomy opening was. The growth had spread up as far as the hepatic flexure and infiltrated the wall of the gall-bladder. The cæcum also is considerably involved. Microscopically it proved to be a myxo-sarcoma, in parts recalling the descriptions given by some writers of the badly defined group of *Cylindroma*.

See *Male Surgical Register*, vol. iii. (1895), No. 734; and *Surgical Post-Mortem Register* (1895), p. 224.

MURPHY'S BUTTON.

2040q₁. Longitudinal section through a piece of gut, the free ends of which had been joined by means of a Murphy's button after an enterectomy. The section has been made so as to demonstrate the mechanism of the button. The specimen was obtained from a woman æt. 40 years, who died soon after the operation, performed on account of a strangulated femoral hernia. For the history of the case see No. 2106a.

See *Female Surgical Register*, vol. v. (1896), No. 931.

SERIES XIX.

DISEASES OF THE RECTUM AND ANUS.

MELANOTIC SARCOMA.

2073d. A Melanotic Sarcoma removed by operation from the Rectum of a woman æt. 39 years. Eighteen months after the operation there were no recurrences or metastatic deposits.

A microscopic specimen has been preserved in the *Histological Records*, xix. 2073d. See *Female Surgical Register*, vol. ii. (1895), No. 1048.

SERIES XX.

HERNIÆ, OR PROTRUSIONS AND OTHER DIS- PLACEMENTS OF THE INTESTINAL CANAL AND OMENTUM.

HERNIAL SAC.

2081b. Portion of the Wall of a Hernial Sac, obtained from a man æt. 46 years, which contains in its substance a thick fibro-cartilaginous plate.

See *Surgical Post-Mortem Register* (1895), p. 253.

RADICAL CURE OF HERNIA.

2090a. A specimen to show the changes which occur after the operation of radical cure for hernia. A male child, *æt.* 3 years, was operated upon for congenital inguinal hydrocele and hernia. The sac was opened, twisted, and sewn to the abdominal wall. Tubercles were found in the wall of the sac removed (see No. 2124a). The child died seven months later of acute miliary tuberculosis (peritoneum and meninges). It will be seen that at the seat of operation the peritoneum shows practically nothing abnormal. Glass rods have been passed into the external iliac artery and vein.

See *Male Surgical Register*, vol. v. (1895), No. 1523 and 2053; and *Surgical Post-Mortem Register* (1895), p. 303; and *Transactions of the Pathological Society*, vol. xlvii. (1896), p. .

2090b. A specimen to show the changes which occur in the peritoneum after the operation of radical cure for hernia. The operation had been done 2½ years ago in a woman *æt.* 48 years, who had an inguinal hernia. She died of a perforating ulcer of the duodenum. On examining the right inguinal region after death, the inguinal canal was found to be represented by a thickened band of fibrous tissue; no external or internal ring could be seen; the peritoneum over the site of the internal ring showed puckering and scarring. There was no trace of a sac or of adhesions. A red glass rod has been fixed on the abdominal surface to indicate the line of incision.

See *Female Surgical Register*, vol. ii. (1895), No. 2260; and *Surgical Post-Mortem Register* (1895), p. 257; also *Transactions of the Pathological Society*, vol. xlvii. (1896), p. .

STRANGULATED BOWEL.

2106a. A piece of Small Intestine removed by operation from a woman *æt.* 40 years, who was admitted with a strangulated femoral hernia. Taxis having proved unsuccessful, the abdomen was opened, the stricture divided, the gangrenous gut resected and the ends joined by Murphy's button. The woman died next day.

The resected loop of bowel is greatly discoloured, being dark, and contrasting strongly with the upper ends of the loop. The lines of constriction at the boundary zones between the dark and light portions are well shown. The dark portion had been strangulated and is distended, the distension being caused by fluid and gas.

The portion of gut with the Murphy's button *in situ* has been preserved in this Series (No. 2040q1).

See *Female Surgical Register*, vol. v. (1896), No. 931.

TUBERCLE IN A HERNIAL SAC.

2124a. A Hernial Sac (inguinal hernia) removed by operation from a boy *æt.* 3 years, which shows numerous typical miliary tubercles. The results of the radical operation are shown in Specimen No. 2090a. The boy died of tubercular meningitis seven months after the operation, and

there was also tubercular peritonitis, which undoubtedly existed before the operation for radical cure.

See *Male Surgical Register*, vol. v. (1895), Nos. 1523 and 2053; and *Surgical Post-Mortem Register* (1895), p. 303.

UMBILICAL HERNIA.

2156a. Sac of an Umbilical Hernia of a new-born child, with a portion of the cord attached to it. The gut had protruded into the umbilical cord, which was thinned out and expanded, and formed the sac of the hernia. The umbilical vessels and the remains of the urachus could be seen in the sac wall. The hernia was reduced and a light binder applied. A few hours later sloughing of the sac was noticed, and therefore it was cut away and the abdominal opening closed, the infant dying two days later.

See *Female Surgical Register*, vol. iii. (1895), No. 2839; and *Surgical Post-Mortem Register* (1895), p. 299.

2156c. Vertical Section through an Umbilical Hernia obtained after death from an obese man *æt.* 55 years, who died with pneumonia and pericarditis. The hernial contents consist of omental tissue firmly incorporated with the wall of the sac. The extreme obesity of the abdominal wall is well seen.

See *Medical Post-Mortem Register*, vol. xxii. (1895), p. 268.

TRAUMATIC HERNIA.

2158a. This specimen was obtained after death from a man *æt.* 28 years, who had received a punctured wound in the abdomen, having been gored by a cow. The scar of this wound is still recognisable, and was situated midway between the umbilicus and the symphysis pubis. The wound had healed by first intention, but four days after the injury a hernia appeared at its site, which was promptly reduced and a pad applied. Eight days later, however, symptoms of obstruction appeared, and the man died next day, without any operation having been undertaken.

The specimen shows that the reduction had not been complete, the gut (small intestine) being nipped at the opening through the sheath of the rectus muscle and the peritoneum, produced by the injury. The proximal end of the gut is enlarged as compared with the distal end, into which a blue glass rod has been passed. A knuckle of gut is seen in front of the sheath of the rectus abdominis, lying between the aponeurosis and the skin in the subcutaneous tissue.

At the post-mortem examination no evidence of general peritonitis was obtained, but it was said that 4 inches below the point shown in the specimen the intestine was doubled upon itself and bound down by adhesions to the front of the spine, and the constriction at this point appeared to be tighter than that at the neck of the hernia.

Presented by C. B. Dale, Esq.

VOLVULUS.

2177c. Section through a small portion of the Small Intestine to which a subserous tumour is attached, removed by operation from a man *æt.* 48 years, who suddenly developed symptoms of acute obstruction. The growth, on microscopical examination, was found to be a fibro-cellular tumour, and is situated under the peritoneum without encroaching on the lumen of the gut. The stout pedicle of the tumour had become twisted, and thus strangulated: hence the marked engorgement; and the gut in its turn had become involved in this twist, so that an intestinal strangulation resulted. The site of the strangulation is well shown; for there considerable thinning and ulceration have taken place. The portion of the gut was removed and the ends joined by a Murphy's button. The patient died thirty hours after the operation with septic peritonitis.

A microscopical specimen has been preserved in the *Histological Records*, xx. 2177c.

Presented by J. Langton, Esq., F.R.C.S.

SERIES XXI.

DISEASES AND INJURIES OF THE LIVER.

CIRRHOSIS.

2193c. The Liver of a woman *æt.* 43 years, showing the appearances characteristic of perihepatic cirrhosis. The section through the right lobe shows the thickening of Glisson's capsule, and the lobular cirrhotic condition of the liver. The anterior margin is greatly thinned and curled upwards, and the liver is considerably deformed on account of the contraction of the newly formed fibrous tissue, so that the organ has become lobular, some of the small lobules being almost completely severed from the general mass of the liver. The wall of the gall-bladder is considerably thickened.

Presented by A. Harper, Esq.

2198a. A typical Atrophic Cirrhotic Liver, obtained from a woman *æt.* 30 years, who died of profuse hæmatemesis. At the post-mortem examination the stomach was found to be full of blood, its mucous membrane discoloured in patches, with minute hæmorrhages in many places; there were no ulcers. The spleen (see Series xxv. No. 2296a) was considerably enlarged, and its capsule greatly thickened. There was no clear history, but only a suspicion of spirit-drinking, and the woman worked well until thirty-six hours before her death.

Presented by C. E. Baker, Esq., F.R.C.S.

SARCOMA.

2215c. The Liver of a boy *æt.* 10 years, enormously enlarged and studded with large nodules, presenting all the appearances character-

istic of secondary malignant deposits. A drawing, a cast, and a microscopical specimen have been preserved (see Series lvii. No. 502a, Series lvi. 138a₇, and *Histological Records*, xxi. 2215c). Microscopically the growth was a sarcoma; it was secondary to a growth of the kidney.

See *Medical Post-Mortem Register*, vol. xxii. (1896), p. 45.

HYDATID CYST.

2230b. Portion of the left lobe of a Liver with a Hydatid Cyst, removed from the body of a man *æt.* 68 years, who died after herniotomy for strangulated umbilical hernia. The cyst contains numerous daughter-cysts and caseous matter. The cysts are dead and their walls greatly thickened.

Other cysts were found, one beneath the peritoneum near the sigmoid flexure, and another in connection with the right kidney, and have been preserved in Series xvi. No. 1893a, and Series xxviii. No. 2393i respectively.

See *Surgical Post-Mortem Register* (1895), p. 87; and *Male Surgical Register*, vol. iii. (1895), No. 1015.

2233a. Section through a portion of a Liver with a large Cyst, probably hydatid in nature, the walls of which are calcified. It was removed from the body of a man aged about 60 years, who died with pleurisy and pericarditis, but no history pointing to hydatids or liver trouble was obtainable. The liver was normal in size and texture, and from the lower anterior margin of the right lobe, just to the right of the gall-bladder, a tumour projected, in size equal to a cricket-ball, of stony consistence and calcified. It was densely adherent to the duodenum and colon, and partially to the right kidney. On sawing through this tumour, it was found to contain bile-stained fluid and a few jelly-like and putty-like masses, from which all trace of structure had disappeared.

Obtained in exchange from the Royal Free Hospital.

2233b. Section of a Liver removed from the body of a man *æt.* 42, who died of pyæmia (perinephritic and retropleural abscesses and multiple hepatic abscesses). In the Spigelian lobe there was a cyst enclosed by a calcareous wall containing degenerated and bile-stained hydatids. Two abscesses will be seen on the free surfaces of the section.

See *Medical Post-Mortem Register*, vol. xxii. (1895), p. 250; and *Male Medical Register*, vol. ii. Part 2 (1895), No. 207.

ACTINOMYCOSIS.

2239d. Section through the Liver of a man *æt.* 31 years, who died of extensive actinomycosis, pyæmic in its distribution. The appearances are typical, and resemble those of specimens Nos. 2239a, b, c. The liver is firmly adherent to the diaphragm, which is involved by the process, and has been perforated, a communication having thus been established with the right pleural cavity, which contained an "empyema." The lung of this case has been preserved in Series xi. No. 1727e, where also a fuller account and references will be found.

SERIES XXII.

DISEASES AND INJURIES OF THE GALL-
BLADDER AND BILIARY DUCTS.

2264d. A portion of Liver with half the Gall-Bladder attached to it. It was removed from a man *æt.* 47 years, on whom cholelithotomy had been performed and cholecyst-enterotomy had been attempted. The gall-bladder is slightly enlarged; its fundus, which had been opened during the operation, was surrounded by granulation tissue. The wall of the bladder is considerably thickened, ragged and shaggy, and microscopically was shown to be in a cancerous condition (columnar-celled carcinoma). The growth extended into the cystic duct and the tissues around the same, and had blocked the main hepatic duct. The liver was jaundiced and the lumbar glands contained secondary deposits, but the liver was free.

The patient had been admitted on account of a tumour on the right side of the abdomen with pains in the epigastrium; jaundice appeared shortly before admission. A few days before death the gall-bladder was opened and twenty-five calculi removed; one was felt in the common bile duct, but could not be got at; a biliary fistula was made and a tube inserted. A few hours before death an attempt was made to join the bladder to the duodenum by means of a Murphy's button, but the patient became collapsed; a stone, of the size of a hazel-nut, was removed from the common duct, but it was impossible to join the gall-bladder to the intestine.

A microscopic section of a lumbar gland has been preserved in the *Histological Records*, xxiii. 2264d.

See *Male Medical Register*, vol. i. Part 1 (1895), No. 96; and also *Male Surgical Register*, vol. i. (1895), No. 598; and *Surgical Post-Mortem Register* (1895), p. 69.

2264e. Section through the Liver and Gall-Bladder of a man *æt.* 53 years. The gall-bladder is greatly diminished in size and contains a calculus; its walls are thickened and infiltrated by a malignant growth, which spreads into the adjoining liver tissue in all directions for about $1\frac{1}{2}$ to $2\frac{1}{2}$ inches. At the time of the post-mortem examination the shrivelled gall-bladder contained a milky thin mucoid fluid. The bile ducts are enormously distended and contained a thin yellow fluid. The liver was deeply jaundiced; the mesenteric glands were infiltrated with growth.

Microscopical Report by Dr. W. P. Herringham:—The edge of the primary growth showed fibrous tissue running in between the liver cells, and here and there a glandular alveolus lined with cubical epithelium. The lymphatic glands showed fibrosis with similar alveoli. No columnar cells seen. The pancreas was free from growth.

See *Male Medical Register*, vol. i. Part 1 (1895), No. 37; and *Medical Post-Mortem Register*, vol. xxii. (1895), p. 50.

SERIES XXV.

DISEASES AND INJURIES OF THE SPLEEN.

SPLENIC INFARCTS.

2295e. A Spleen showing typical "septic" infarcts. It was removed from the body of a man *æt.* 33 years, who died of infective endocarditis, with gangrene of both feet, embolism of both axillary arteries and the right common iliac artery; there were also infarcts in the kidneys and several small infarcts in the intestines.

See *Medical Post-Mortem Register*, vol. xxii. (1895), p. 359; and *Male Medical Register*, vol. ii. Part 2 (1895), No. 304.

ABSCESS.

2295l. Section through a Spleen which was occupied by an abscess of considerable size, so that but little has remained of the spleen substance. The remnants can still be seen in the wall of the abscess as darker blood-stained areas. The wall of the abscess is shaggy and shreddy. At the post-mortem examination the spleen was described as being in size equal to two fists; it was adherent both to the diaphragm and the omentum (the adherent diaphragm is shown in the specimen); it weighed 50 ounces before it was cut into. The abscess was described as a cyst. The cystic contents were a reddish turbid fluid like chocolate. At the upper and inner part, and also below, fragments of normal spleen tissue are visible, and between these and the cyst there is some yellowish firm tissue resembling that seen in infarcts.

The spleen was removed from a man *æt.* 49 years, who died with pneumonia. The right lung was consolidated in its upper lobe, and it contained an abscess cavity filled with reddish turbid fluid, "not particularly foul in odour." It is unfortunate that no bacteriological examination was made of the fluid in the pulmonary abscess and the splenic cyst. There can, however, be but little doubt that the latter was an abscess, probably of pneumococcus origin, *i.e.*, a secondary pneumonic process.

See *Medical Post-Mortem Register*, vol. xix. (1892), p. 77; and *John Ward Book* (1892), *sub* John Newman.

ENLARGEMENT.

2296a. A Large Spleen obtained from a woman *æt.* 30 years, who died of profuse hæmatemesis. At the post-mortem examination her liver was found to be greatly cirrhotic and diminished in size (see Series xxi. No. 2198a). The capsule of the spleen is considerably thickened, and in some places shows, though imperfectly, those curious depressions which are so common in well-marked perihepatitis.

Presented by C. E. Baker, Esq., F.R.C.S.

SERIES XXVI.

DISEASES OF THE THYROID GLAND.

2310g. A Tumour of the Thyroid Gland, laid open after it had been hardened in spirit. It had been enucleated from the right lobe of the thyroid gland of a man *æt.* 20 years, having been diagnosed as a cyst. Microscopically it proved to be a cystic adenoma.

See *Male Surgical Register*, vol. v. (1896), No. 586.

2314o. Section of an Adenoma of the Thyroid Gland, removed by operation from a woman *æt.* 47 years. (Fixed in formalin and preserved in glycerine.)

See *Female Surgical Register*, vol. ii. (1896), No. 1599.

SERIES XXVIII.

DISEASES AND INJURIES OF THE KIDNEYS, THEIR
PELVES, AND THE URETERS.**LARGE WHITE KIDNEY.**

2332a. Sections through a Large White Kidney, obtained from a boy *æt.* 15 years, who died with symptoms of uræmia. It has been fixed with formalin and preserved in glycerine, and has retained its colour fairly well.

See *Medical Post-Mortem Register*, vol. xxiii. (1896), p. 154.

CONTRACTING WHITE KIDNEY.

2334a. A Kidney obtained after death from a woman *æt.* 20 years. At the time of the post-mortem examination it was pale on section, the cortex compressed and thinned; the capsule, though rather thick, separated fairly easily, leaving a typically granular surface. Unfortunately the appearances on section are lost; still the specimen is important, because the woman had had scarlet fever, which left a chronic nephritis behind, which eventually resulted in a white granular kidney.

See *Medical Post-Mortem Register*, vol. xix. (1892), p. 104.

GRANULAR KIDNEY.

2335f. Heart and Kidneys of a girl *æt.* 11 years. The *kidneys* show the characteristic appearances of interstitial nephritis. The appearances at the time of the autopsy were described as follows:—(a) Right kidney on section shows a much attenuated cortex; its structure is much blurred, the capsule thick, and on separation it left a typically granular surface; there were no cysts; the general colour was rather dark: (b) the left kidney was more flabby to the touch than the right kidney; the colour was dark; in other respects it was like the right.

The heart shows considerable hypertrophy of the left ventricle, and there is also well-marked fibrinous pericarditis. A well-defined patch of atheroma is seen on the ventricular surface of the anterior flap of the mitral valve, and there are also a few patches above the aortic cusps, and atheromatous changes were also detected in the abdominal aorta.

The previous history of the child could not be found in the Ward Notes.

See *Medical Post-Mortem Register*, vol. xx. (1893), p. 133.

TUBERCULAR DISEASE.

2341f. Section through a Right Kidney removed by operation from a man æt. 25 years. It shows the lesions characteristic of tubercular pyonephrosis.

See *Male Surgical Register*, vol. iii. (1895), No. 172.

2341g. Section through a Tubercular Kidney obtained from a boy æt. 10 years, who died with hip-disease.

(The specimen has been fixed with formalin and preserved in glycerine.)

See *Surgical Post-Mortem Register* (1896), p. 158.

SARCOMA OF KIDNEY.

2391b. Section through a Left Kidney one half of which is occupied by a growth which microscopically proved to be a round-celled sarcoma. The lower half of the kidney is free from growth. The kidney was removed by operation from a woman æt. 27 years, and weighed 6 lbs. She recovered from the operation, but was readmitted two months later with recurrence and paraplegia.

See *Female Surgical Register*, vol. ii. (1895), Nos. 2042 and 2663.

HYDATID CYST.

2393i. Section through a large Hydatid Cyst in connection with the right kidney. In the same body two other cysts were found, one in the liver (see Series xxi. No. 2230b), and the other behind the peritoneum near the sigmoid flexure (Series xvi. No. 1893a).

See *Surgical Post-Mortem Register* (1895), p. 87; and *Male Surgical Register*, vol. iii. (1895), No. 1015.

SERIES XXIX.

DISEASES AND INJURIES OF THE URINARY BLADDER.

2405b. An everted Bladder removed post-mortem from a girl æt. 16 years, who died with purpura hæmorrhagica. There are numerous hæmorrhages under the mucosa, which after preservation have become almost entirely decolourised.

See *Female Medical Register*, vol. iii. Part 2 (1895), No. 134; and *Medical Post-Mortem Register*, vol. xxii. (1895), p. 177.

MYXOMATOUS POLYPI OF BLADDER.

2417a₁. A Bladder with Ureters and Kidneys obtained from a child *æt.* 2 years and 2 months. The bladder has been everted, and is almost completely occupied by curious polypoid growths, partly sessile, partly stalked, which, when the specimen was fresh, were very delicate and transparent, undoubtedly myxomatous. Bristles have been passed into the openings of the ureters. The latter are thickened and much dilated, and the kidneys hydronephrotic, with a few scattered suppurating nodules, which, in this preserved condition, however, are hardly recognisable. Microscopically the polypi proved to be myxomata, possibly myxosarcomata.

A microscopic specimen has been preserved in the *Histological Records*, xxix. 2417a₁.

The child had been well until five months before death, when he complained of suprapubic pain, and since then, although pain abated, he had always suffered from frequent micturition, and eventually from inability to pass urine without much straining and pain. There were no symptoms of kidney trouble.

Presented by Percy Lambert, Esq.

VILLOUS TUMOUR.

2417e. A beautiful and well-preserved Villous Tumour of the Bladder removed from a man *æt.* 58 years by suprapubic cystotomy.

See *Male Surgical Register*, vol. iii. (1894), No. 2869; and *Surgical Post-Mortem Register* (1894), p. 211.

 SERIES XXX.

DISEASES AND INJURIES OF THE BRAIN AND ITS MEMBRANES.
MICROCEPHALIC BRAIN.

2483b. Specimen of a Microcephalic Brain obtained from the body of a girl *æt.* 4 years. The brain weighed 18 ounces and is smaller than natural. The posterior lobes are especially small and shrunken, causing the cerebellum to appear more prominent. On the left side there is a zone of sclerosis, extending from the frontal lobe (orbital aspect) along the hemisphere to the posterior lobe. In the frontal region it is limited to the lower frontal convolution and involves the ascending frontal convolution largely. The posterior Rolandic region is almost entirely sclerosed, and the convolutions are remarkably shrunken, very hard, and they were yellow in colour before hardening. The shrinking is not so marked in the convolutions anterior to the Rolandic fissure, but the convolutions have a granular, roughened surface. On the left side there seems to be more substance about the angular gyrus than on the right side, but the gyri all around it are shrunken.

The description above given applies almost exactly to the right hemisphere, but there is a fairly complete sclerosis of the superior temporo-sphenoidal convolution and some of the posterior third of the middle temporo-sphenoidal convolution. A longitudinal section was made through the sclerosed area on the left side. It was seen that the cortex was thinned to $\frac{1}{16}$ th inch in places, its outline being very irregular; the white matter looked natural. The posterior horn of the lateral ventricle was opened and was evidently dilated. The lenticular nucleus was natural. The cord was apparently natural, but has not yet been examined microscopically.

See *Medical Post-Mortem Register*, vol. xxii. (1895), p. 29; and *Female Medical Register*, vol. iv. Part 1 (1895), No. 35.

SERIES XXXIII.

DISEASES AND INJURIES OF THE EYE AND ITS APPENDAGES.

ORBITAL SARCOMA.

2570b. A Tumour removed from the Orbit of a boy *æt.* 6 years. It had caused swelling of the right upper lid and displacement and proptosis of the right eye; it was of hard consistence and occupied the region of the lachrymal gland. Recurrences took place both in and around the orbit, and the præ-auricular glands also became involved; gradually the disease spread to the neighbouring bones, and the boy died about eight to nine months after the lesion had been detected. Microscopically it is a mixed-celled sarcoma, myxomatous in parts.

A histological specimen has been preserved in the *Histological Records*, xxxiii. 2570b.

Presented by W. H. Jessop, Esq., F.R.C.S.

ORBITAL TUBERCULOSIS.

25701. Section through a mass removed from the Orbit (from the region of the lachrymal gland, which had been destroyed by the growth) of a young man *æt.* 18 years. The growth when fresh was distinctly caseous, and microscopically also undoubtedly tubercular, although Koch's bacillus was not discovered in stained sections. Six months previous to the operation a small lump appeared in the right upper lid, which gradually increased to the size of a walnut, displacing the eye downwards. There were signs of tuberculosis of the lungs, and also enlarged glands and scars in the neck.

See *Ophthalmic Ward Book* (1896), No. 3102.

PERFORATING ULCER.

2579. The Anterior Half of an Eye removed from a man *æt.* 73 years, showing a perforating ulcer of the Cornea with considerable destruction of the Iris. No history of an injury could be obtained; the patient is described as an old, withered, unhealthy man.

See *Ophthalmic Ward Notes* (1896), No. 3575*.

CORNEAL OPACITY.

2579a. The Anterior Half of an Eye showing considerable thickening and opacity of the cornea, consequent upon keratitis and corneal ulceration following removal of an epithelioma of the orbit. The specimen shows distinctly the encroachment of the conjunctival and sclerotic vessels upon the opaque cornea.

See *Female Surgical Register*, vol. v. (1895), No. 659.

HYPOPYON.

2579b. Section of an Eye excised from a girl *æt.* 13 years, showing a Hypopyon. Five needling operations had been performed for anterior polar cataract; hypopyon resulted, and pus appeared in the vitreous humour; and the eye, being blind and painful, was removed.

See *Ophthalmic Ward Book* (1895), No. 1558.

CORNEAL ULCERS.

2579c. Two Halves of an Eye showing elongation of the globe (myopia) and adhesions of the iris. The adherent part of the iris is perforated by a small circular ulcer, which makes it apparent that there had been a perforating corneal ulcer. The lens is flattened and the choroid thinned.

Presented by W. H. Jessop, Esq., F.R.C.S.

HYPHÆMA.

2579h. The Anterior Half of an Eye removed from a woman *æt.* 44 years on account of plastic iritis and intraocular hæmorrhage. The blood in the anterior chamber and the discoloration of the iris are easily recognised.

See *Ophthalmic Ward Notes* (1896), No. 307.

XEROPHTHALMOS.

2588b. Two Portions of an Eye removed from a man *æt.* 44 years on account of the condition called Xerophthalmos resulting from a scald with boiling fat. The xerosed cornea is well seen, as well as the atrophied lens; the sclerotic is thinned and translucent.

See *Ophthalmic Ward Book* (1895), No. 1390.

FOREIGN BODY.

2592a. Anterior Half of an Eye enucleated from a man *æt.* 39 years, showing the presence of a piece of iron, which had penetrated through the cornea and become impacted, causing great damage to the eye. The neighbourhood of the corneal wound is discoloured by rust. The injury had occurred eighteen years before. For drawings of this case see the *Ophthalmic Ward Notes*.

See *Ophthalmic Ward Book* (1893), No. 1940.

INJURY.

2593c. Section through an Eye of a woman *æt.* 37 years, which had been destroyed through a severe injury, causing a rupture of the sclerotic. The cornea is swollen and suffused with blood (chemosis); the anterior chamber is distended by a large clot, and the hæmorrhage has extended backwards behind the iris; the retina is partly detached. The lesion of the sclerotic on the left or inner side is well shown.

See *Ophthalmic Ward Book* (1896), No. 75.

INTRAOCULAR HÆMORRHAGE.

2602. Section through an Eye removed by operation from a man *æt.* 40 years. The whole globe is completely filled up with solid matter, probably resulting from intraocular hæmorrhage and inflammation. The lens is opaque, the anterior chamber obliterated, and behind the lens is some white denser substance. Three months before the operation the patient had been struck with the handle of a chisel.

See *Ophthalmic Ward Book* (1896), No. 405.

NORMAL EYE.

2602a. Anterior Portion of a Normal Eye removed from a boy whose orbit had to be cleared out on account of sarcoma of the lachrymal gland. The latter has been preserved in this Series, No. 2570b.

Presented by W. H. Jessop, Esq., F.R.C.S.

IRIDECTOMY.

2605c. Portion of an Eye of a woman *æt.* 73 years, to demonstrate the result of an iridectomy, performed on a glaucomatous eye with cataract and central choroiditis, which had been blind and painful.

See *Ophthalmic Ward Book* (1895), No. 1791.

MELANOTIC SARCOMA OF IRIS.

2606a. Section through an Eye removed by operation, showing a small black growth in the iris. This microscopically proved to be a melanotic sarcoma. The iris is pushed forward by the growth, which has sprung from the posterior pigmented layer (the uvea), and the anterior chamber is almost completely obliterated. (Hardened in Müller's fluid.)

Presented by Henry Power, Esq., F.R.C.S.

GLAUCOMA.

2608c. Section through a Glaucomatous Eye excised from a woman *æt.* 54 years. The cupping and the course of the retinal artery across the disc are well shown, and also the macula lutea on the outer side. An iridectomy had been performed three years before, as may be seen on looking at the front of the eye; the lens was absent and the anterior chamber obliterated.

See *Ophthalmic Ward Book* (1896), No. 146.

PANOPHTHALMITIS.

2613e. Antero-posterior Section through the anterior half of the same Eye from which the next specimen was prepared. It shows old iritic changes, plastic occlusion of the pupil, adhesion of the lens, and exudation of lymph (so-called) on the anterior surface of the lens. At one side there is a white pyramidal mass where the detached retina is attached to the sclerotic. This mass microscopically showed bony or osteoid structure. A foreign body was not found in this eye on cutting it open. See also next specimen, No. 2613f.

There was a history of an injury to this eye sixteen years before removal—a piece of metal entering the eye; this was removed; vision gradually failed; five weeks before admission painful inflammation set in.

See *Ophthalmic Ward Book* (1895), No. 3618.

2613f. Coronal Section through an Eye of a man *æt.* 30 years, removed on account of total blindness and painful inflammation. The eye had been completely blind and the lens transmitted no light. The iris was adherent to the lens at its pupillary margin and bulging (*iris bombé*) and the pupil blocked by the opaque lens. The specimen shows detachment of the retina produced by a peculiar, semi-solid exudation between the retina and the choroid coat.

Another portion of the same eye is preserved. See the previous specimen, No. 2613e.

See *Ophthalmic Ward Book* (1895), No. 3618.

TUBERCLE OF THE CHOROID.

2621b. The Posterior Half of an Eye which has been everted. The retina has been removed so that the choroid coat is shown, and to one side of the disc a typical tubercle is seen. The specimen was obtained from an infant *æt.* 7 months, who died with general tuberculosis.

See *Medical Post-Mortem Register*, vol. xxiii. (1896), p. 52.

TUBERCULAR DISEASE.

2621c. Section through an Eye removed from a girl *æt.* 16 years. It is occupied by a growth, thought during life to be a pseudoglioma, which on microscopical examination, however, turned out to be tubercular.

There was a strong tubercular history. The eye was enucleated on March 13, 1896, and early in April she had undoubted signs and symptoms of meningitis, of

which she died. After death the examination revealed tubercular meningitis and multiple tubercles in the lungs.

A microscopic specimen has been preserved in the *Histological Records*, xxxiii. 26216.

See *Ophthalmic Ward Notes* (1896), No. 507.

DISLOCATION OF THE LENS.

2640. The Anterior Half of an Eye embedded in gelatine, showing dislocation of the lens forward into the anterior chamber. It was removed from a woman *æt.* 46 years, who eighteen years before had been injured by a "tip-cat."

See *Ophthalmic Ward Notes* (1896), No. 1445.

LENSES.

2641c. Five Lenses, of which (1) is normal, and has become opaque through hardening in formalin solution; (2), (3), (4), (5) illustrate various forms of cataract.

INJURY.

2642b. Sections through an Eye removed from a man *æt.* 41 years, who had been struck in the face by a branch. The eye became painful and inflamed, and the lens cataractous (traumatic cataract). The sections show marked iritis, the lens is replaced by a grey mass and the cornea hazy. A thorn was found in the vitreous humour.

See *Ophthalmic Ward Book* (1896), No. 225.

NORMAL FUNDUS.

2652a. Coronal Section through a normal Eye showing the disc and the retinal vessels, which have kept their natural colour. To the outer side of the disc is the yellow spot, easily recognised by its colour, and situated on a peculiar triradiate fold.

Presented by W. H. Jessop, Esq., F.R.C.S.

ALBINISM AND OPAQUE NERVE FIBRES.

2652b. The Posterior Halves of Two Eyes, removed (*a*) one from a dark rabbit, (*b*) the other from an albino. In (*a*) the choroidal pigment is black, and the opaque nerve fibres are very evident. In (*b*) the choroidal pigment is absent, but on holding the eye up to the light, the band of opaque nerve fibres is well seen.

Presented by A. A. Kanthack, M.D.

OPTIC NEURITIS.

2652c. The Posterior Half of an Eye obtained from a man *æt.* 21 years, who died with a cerebral tumour. The fundus shows the appearances characteristic of optic neuritis, viz., marked cloudiness around the disc, extending as far as the macula lutea. In this cloudy area the vessels are greatly obscured and almost invisible. The whiteness of the macula is very striking.

See *Medical Post-Mortem Register*, vol. xxiii. (1896), p. 65; and *Male Medical Register*, vol. v. (1896), sub George Bryant.

SERIES XXXIV.

DISEASES OF THE EAR.

OTITIS MEDIA.

2676d. The Left Temporal Bone and Jugular Vein of a child *æt.* 4 years, who died from the sequelæ of an acute suppurative otitis media. The bone has been opened up so as to expose the various parts more clearly. (1) The meatus, the middle ear with the attic, are filled with pus, and the structures in the tympanic cavity disorganised; the suppuration has extended into the labyrinth. (2) From the attic the process has passed into the mastoid cells; this is indicated by the bristle. The mastoid antrum had been laid open during life by an operation. (3) The jugular vein down to its end, the innominate vein and the superior vena cava show the characteristic appearances of phlebitis, the vessel-wall being roughened and covered by fibrinous clots over the whole of the circumference. (4) On turning the specimen round, the thrombosis of the lateral sinus becomes evident: a red glass rod has been passed from this sinus into the jugular vein, in order to show the continuity of the process. (5) The superior petrosal sinus is also thrombosed. It goes without saying that there was septicæmia with pyæmia in this case.

See *Male Surgical Register*, vol. iii. (1893), No. 1058; and *Surgical Post-Mortem Register* (1893), p. 81.

2676e. Portion of the Skull, including the right temporal bone with the lateral sinus and internal jugular vein, laid open in such a way as to show the various conditions which have resulted from a suppurative otitis media. Looking at the specimen from the front, the following points will be noticed: (1) the destructive changes which have taken place in the middle ear, the membrana tympani and some of the ossicles having disappeared; (2) the attic is enormously enlarged, and still contains some pus, as best seen on the left side; (3) the jugular vein is filled and blocked by a dark breaking down clot, and is in a condition of phlebitis down to its termination; (4) to the left the opening of a chiselling operation is visible. On turning the specimen round, there will be seen (5), on the left, the lateral sinus opening into the jugular vein, its wall roughened from the suppurative phlebitis which had existed during life; (6) further away, and especially on the right, the clot has been left in the sinus, and is well shown on section and through a slit made into the dura mater; (8) the mastoid cells are partly opened up by the chiselling operation, and still contain pus, as is best seen on the left; (9) a slit has been made into the superior petrosal sinus, and it also is thrombosed.

There was at the time of the post-mortem examination no clot at the lower end of the jugular vein, so that the putrid contents of the lateral sinus and the jugular vein passed directly into the right innominate

veins, and thus into the heart, leading inevitably to septicæmia, and in this case also to pyæmia, as shown by the specimen of the lung preserved in Series xi. No. 1711c.

See *Male Surgical Register*, vol. ii. (1895), No. 807; and *Surgical Post-Mortem Register* (1895), pp. 73 and 74.

SERIES XXXVI.

DISEASES OF THE TESTICLE.

SARCOMA OF TESTIS.

2797m. Section through the Right Testis, removed after death from a man æt. 24 years, which is occupied by a cystic malignant growth, which microscopically proved to be a cystic sarcoma. There were secondary deposits in the lumbar, supraclavicular and cervical, mediastinal, and bronchial glands, in the lungs, in the cavity of the right auricle, and in the liver, which was studded with them. The growth had also invaded the inferior vena cava and the right ureter from the infected lumbar glands, causing an obstruction both in the vena cava and in the right ureter. The man died with symptoms of suppression of urine.

Specimens of the intravenous and intracardiac growths will be found under the two following numbers.

See *Surgical Post-Mortem Register* (1894), p. 88; and *Male Surgical Register*, vol. i. (1894), No. 1359.

2797n. This specimen was obtained from the same man as the preceding specimen. The preparation shows a portion of the aorta abdominalis surrounded as far as and beyond its bifurcation by the infected lymphatic glands which on section are riddled with numerous small cysts, and microscopically present the same appearances as the primary testicular growth, viz., cysto-sarcoma. The inferior vena cava is shown on the left, and has been laid open. It will be seen that its lumen is considerably encroached upon by the infected glands, and further, that it is in part obstructed by a mass extending into it from the glandular growth. This mass, which at first sight resembles a clot, microscopically proved to be sarcomatous, and there can be no doubt that the curious growths which were found in the right cavities of the heart and at the cardiac end of the inferior vena cava owe their origin to this mass, which had invaded the abdominal vena cava. The growth has extended from the vena cava into the right renal vein, and thence into the right ureter, which it completely blocked.

2797o. This curiously branched mass was found in the right auricle and in the inferior vena cava of the man from whom the two preceding specimens were obtained. It was fixed to the free border of the tricuspid

valve, and extended thence partly into the right ventricle and partly into the inferior vena cava. The curiously shaped lateral branches contain small cysts, filled by a small amount of clear fluid. Microscopically the growth is a cystic sarcoma, almost identical in its structure with the primary testicular growth.

SERIES XL.

DISEASES AND INJURIES OF THE URETHRA
AND PENIS.

2890c. Complete slough of the Skin of the Penis, which separated in one piece while the patient, a man aged 40 years, was in the bath. He had been admitted on account of acute swelling of the penis and testes, for which no adequate cause could be found. The penis was swollen to about three times its normal size. Ulceration and sloughing set in, for which the patient was submerged in the bath.

See *Male Surgical Register*, vol. i. (1895), No. 2473.

SERIES XLII.

DISEASES OF THE UTERINE APPENDAGES.

HYDROSALPINX.

2936. Two Tubes removed by operation from a woman æt. 30 years. Both specimens illustrate the conditions of hydrosalpinx. The lower specimen is of special interest, because there the hydrosalpinx communicates by a small round opening with what appears to be an ovarian cyst.

There was no definite history: "the woman was ailing for twelve years, and probably there was gonorrhœal infection at that time."

Presented by C. B. Lockwood, Esq., F.R.C.S.

SERIES XLIII.

DISEASES OF THE UTERUS.

CARCINOMA OF CERVIX.

3006c. Sagittal Section through a Uterus removed by vaginal hysterectomy from a woman æt. 33 years, the cervix of which is cancerous (microscopically squamous-celled carcinoma). Externally there is but little more to be seen than a superficial ulcer; this, however, was malignant,

and on section found to communicate with a small ragged cavity in the substance of the cervix. The body of the uterus was free from malignant disease.

A microscopical specimen has been preserved in the *Histological Records*, xliii. 3006c.

See *Martha Ward Book* (1895), No. 1478.

3006e. Sagittal Section through a Uterus removed by vaginal hysterectomy, the cervix of which is greatly enlarged in its vaginal portion, and microscopically was found to be an alveolar carcinoma. The body of the uterus is free from carcinoma.

A microscopical specimen has been preserved in the *Histological Records*, xliii. 3006e.

Presented by W. S. A. Griffith, M.D.

SERIES XLIV.

DISEASES OF THE VAGINA AND EXTERNAL ORGANS OF GENERATION IN THE FEMALE.

SARCOMA OF VAGINAL WALL.

3030a. This specimen was obtained from the body of a girl æt. 2 years and 4 months. It shows a large tumour, of firm consistence, between the rectum and the bladder. This, microscopically, was a typical mixed-celled sarcoma. On turning the specimen round, it will be seen that the tumour extends and bulges into the vagina, on the anterior and posterior walls of which numerous polypoid growths are found, growing partly from the vaginal walls and partly from the surface of the tumour. These polypoid growths are myxomatous in nature, but hardly sarcomatous, although it is quite possible that they may have begun as true sarcomatous deposits which subsequently became myxomatous.

A vaginal discharge had been observed for fourteen months, and for the last five months the girl had suffered from severe bearing-down pains. During the last week of her life she had retention of urine, and she died with uræmic symptoms. During life the vagina, which easily admitted two fingers, was seen to be studded with œdematous polypoid growths, which bled when handled roughly. They were covered with a slight purulent discharge. A dense mass of new growth was attached to the right side and posterior wall of the vagina. The tumour was somewhat cylindrical in shape and seemed to infiltrate the recto-vaginal septum, for it was not circumscribed. Post-mortem the bladder was distended, its wall thick and muscular, the urethra elongated, being 2 inches in length.

Microscopical specimens (*a*) of the main tumour and (*b*) of the polypoid growths have been preserved in the *Histological Records*, xlv. 3030a.

See *Transactions of the Pathological Society*, vol. xlvii. (1896), p. .

Presented by D'Arcy Power, Esq., F.R.C.S.

SERIES L.

GENERAL PATHOLOGY.

3233d. The Right Foot of a girl æt. 16 years, removed by Syme's amputation. There was a perforating ulcer (neuropathic ulceration) involving the outer toes, which have necrosed away by a process of so-called "liquefactive necrosis." After healing, a similar ulcer appeared at the stump, and a second amputation had to be performed. The nerves were examined after this second operation and showed degenerative changes (neuritis).

The girl had measles when three years old, followed by "inflammation of the right foot," leading to decay of the fourth and fifth metatarsal bones. In 1891 the fourth metatarsal bone was removed, together with some dead bone in the stump of the fifth toe. In 1893 she was treated in Stanley Ward for "talipes equino-varus" and a sinus in the right foot. The latter had been discharging since, and on admission in 1894 the right foot was still in the same deformed position; the fourth and fifth toes were absent, and there were a perforating ulcer on the sole and two sinuses. The knee-jerk was absent on the right side, normal on the left; there was anæsthesia over the dorsum of the right foot; the right leg was one inch shorter than the left; there was no reaction of degeneration in the muscles of the right leg or foot. Three days after the operation a slough appeared at the stump, which came away four weeks later, leaving an unhealed wound. She was re-admitted a few months later with an ulcer on the anterior surface of the stump, and an amputation was performed just below the knee-joint. The wound did not heal by first intention.

The stump after the first operation has been preserved in this Series. See No. 3233e.

See *Female Surgical Register*, vol. iv. (1895), Nos. 1871* and 186.

PERFORATING ULCER OF STUMP.

3233e. The special interest of this specimen lies in the history of the case, which will be found in the description of the previous specimen. The stump shows a round ulcer on its lower and anterior aspect, about 1 inch in diameter and $\frac{3}{4}$ inch in depth, without any granulations on its surface or edges.

For history and references see the previous specimen.

CONGENITAL LIPOMA.

3248a. A curious Pendulous Lipoma, which grew from the lower part of the back of a child aged 2 years, and had existed since birth. Two photographs and a coloured drawing of this case will be found in Series lvii. Nos. 1328a, b, and c.

See *Male Surgical Register*, vol. i. (1894), No. 2931.

RODENT ULCER.

3324a₁. Specimen of a Rodent Ulcer removed by operation from the face of a man æt. 70 years. (Hardened in formalin and preserved in glycerine.)

A microscopical specimen has been preserved in the *Histological Records*, l. 3324a₁.

See *Male Surgical Register*, vol. v. (1896), No. 1987.

EPITHELIOMATOUS WART.

3325g. A Malignant Wart (squamous-celled carcinoma), which appeared in the neighbourhood of the chief growth, affecting the cheek and lip. The infiltrating nature of the disease can be made out fairly distinctly on examining the cut surface of the specimen.

See *Male Surgical Register*, vol. ii. (1894), No. 2434.

EPITHELIOMA AND GOUT.

3327b. Portion of the Pinna of the Left Ear, removed by operation from a man *æt.* 64 years. The pinna is occupied by an ovoid growth, the left half of which is warty and ulcerated, and typically epitheliomatous, while the right half is smoother, and when fresh had all the appearances of a typical gouty tophus.

In July 1894 a white spot was noticed on the left pinna which the patient scratched off; a scab formed, which he again picked off. This process was repeated several times, and gradually a sore appeared, which increased in size and was accompanied by severe darting pain in the ear. He also had had frequently pains and swelling of the left foot and elsewhere in the joints.

In October 1894 the growth had assumed the size of an acorn, and was destroyed at another Hospital, but grew again, so that by December it reached the size of the specimen in the bottle. There were also tophi in other situations, viz., on the right pinna and on the left below the growth.

Microscopically the mixed nature of the growth was verified, so that this is an instance of a cancerous neoplasm appearing on a gouty soil, and apparently actually on an injured tophus.

See *Male Surgical Register*, vol. ii. (1894), No. 3689*.

EPITHELIOMATOUS CYST.

3327c. A Larynx and Trachea, with the Arch of the Aorta and the large vessels of the root of the neck. On the right side of the larynx there is a large cystic growth, which during life was occupied by a serous, blood-stained fluid. The wall of the cyst is studded with tuberos masses which, on microscopical examination, proved to be typical squamous-celled carcinoma. No sign of any primary disease was discovered anywhere, although carefully looked for; nor were any secondary deposits found in any of the viscera or glands. The cyst measured three inches vertically, extending from the level of the hyoid bone to the clavicle, and measured nearly as much transversely, lying beneath the sterno-mastoid muscle. The prævertebral muscles, the sterno-mastoid, the brachial and cervical plexuses are described as being infiltrated by the growth, the internal jugular vein as completely destroyed, and the carotid artery, vertebræ, clavicle, larynx, trachea, &c., as unaffected, neither adherent nor infiltrated. Outside the cyst, and close to it, were a few small nodules resembling growths, but apparently glands. The glands on the left side of the neck and those of the thorax were free from disease. In the absence of any primary growth on the skin or mucous membranes, it was thought that

the case was one of primary epitheliomatous cyst of the neck developing from similar epithelium of fœtal origin in the branchial clefts.

A microscopic specimen has been preserved in the *Histological Records*, l. 3327c.

See *Male Surgical Register*, vol. iv. (1894), No. 2478; *Surgical Post-Mortem Register* (1894), p. 148; and *Transactions of the Pathological Society*, vol. xlv. (1895), p. 166, and also, for a similar case, *ibid.* p. 169.

CUTANEOUS NÆVUS.

3347b. A Cutaneous Nævus removed by operation from the skin of the leg of a boy aged 4 years.

See *Male Surgical Register*, vol. iii. (1895), No. 1851.

SEBACEOUS CYST.

3364d. Sections through a small Sebaceous Cyst removed by operation, showing the thin capsule in its lower half. It was obtained from a man æt. 35 years, who suffered repeatedly from sebaceous cysts. Thus in 1887 some appearing below the right ear had been removed; in 1888 a crop of them appeared on the face and forehead, and in 1894 he had others on the neck. These cysts varied in size from that of a pea to that of a walnut, and from thirteen to sixteen were removed.

See *Male Surgical Register*, vol. ii. (1895), No. 3471*.

3364e. A Sebaceous Cyst removed after death from the chest-wall of a woman æt. 64 years. It has been divided into two halves along its long axis, and one half emptied of its contents. The circumscribed character and its superficial situation are well shown; it is surrounded by a distinct capsule, and is situated in the skin proper superficial to the panniculus adiposus. The right half further shows the little black speck on the epidermis generally described with these cysts.

POST-MORTEM EMPHYSEMA.

3382f. A Slice of Brain showing post-mortem emphysema in a typical form, which gives it the appearance of "Gruyère cheese."

From the Dissecting-Room.

3382g. A Slice of Liver from a case of pyæmia with meningitis and liver abscesses, which shows post-mortem emphysema remarkably well. The liver is riddled with small cavities, which at the post-mortem examination were found to contain gas, so that the tissues crackled on pressure.

Other portions of this liver were allowed to incubate at 38° C., which caused the air spaces to enlarge considerably, as is seen in the next specimen.

See *Medical Post-Mortem Register*, vol. xxii. (1895), p. 270.

3382h. A Slice of Liver from the previous case after incubation. This has caused considerable enlargement of the air-spaces, and also an increase in their number.

3382i. A Slice of Spleen showing post-mortem emphysema. The organ had been obtained from a case of typhoid fever, and had been incubated for twenty-four hours at 38° C. The spleen pulp is riddled with numerous small cavities.

3382j. Liver of a Rabbit in which the post-mortem emphysema had been artificially produced in the following manner. From the above liver and spleen pure cultures of a bacillus indistinguishable from the *Bacterium coli commune* had been obtained. 1 cc. of a broth culture was injected into the ear vein of the rabbit, and the animal killed by a stroke on the neck 20-30 minutes later, and then kept in a warm room over-night, with the result that the liver, spleen, and brain became highly emphysematous, and gas was also found in many of the vessels. From the liver the same bacillus was easily recovered.

Presented by A. A. Kanthack, M.D.

SERIES LVI

CASTS AND MODELS.

ACROMEGALY.

1b₁. Cast of the Left Hand from a case of Acromegaly in a man æt. 28 years. A photograph has been preserved in Series lvii. No. 3b.

ACHONDROPLASIA.

1m. Two Casts of the Hands of a girl æt. 13 years, supposed to suffer from Achondroplasia. Five photographs of this girl and her sisters will be found in Series lvii. No. 4m, where also a short account of the case is recorded. Casts of the legs of this girl will be found under the next number.

See *Orthopædic Department* (1896), No. 345.

1n. Casts of the Legs of a girl æt. 13 years, said to have Achondroplasia. Photographs of this girl and her sisters will be found in Series lvii. No. 4m, where also a short account of the case is recorded. For casts of the hands see the previous specimen.

See *Orthopædic Department* (1896), No. 345.

CONGENITAL DEFORMITY OF HANDS.

66g. Casts of the Hands of a man æt. 29 years possessing a curious congenital deformity, both hands being in a condition of extreme adduction. An operation was performed with some benefit therefrom. A photograph of these hands has been preserved in Series lvii. No. 1327d.

See *Male Surgical Register*, vol. ii. (1896), No. 1258.

66h. The Hands of a child æt. 3½ years, the fingers of which are in a position of over-extension. The feet of this child were also deformed, the right in a position of Valgus and the left in a position of Varus. Casts of them have been preserved in this Series, No. 87h. These deformities were of congenital origin.

CONGENITAL TALIPES.

85n. Casts of the Right and Left Feet of a boy æt. 11 years, showing Congenital Talipes Equino-Varus.

See *Male Surgical Register*, vol. i. (1895), No. 3028.

87f. Casts of the Right and Left Feet of a boy æt. 16 years, both in the position of Talipes Varus, which was of congenital origin.

See *Male Surgical Register*, vol. i. (1896), No. 1719.

87g. Cast of a Congenital Talipes Valgus in a child æt. 1 month.

See *Orthopædic Department* (1896), No. 225.

87h. Casts of the Feet of a child æt. 3½ years, showing Talipes Valgus on the right side and Talipes Varus on the left side. This deformity was of congenital origin. A photograph of the feet has been preserved in Series lvii. No. 1342l. Casts of the hands of this case have been preserved in this Series, No. 66h.

See *Orthopædic Department* (1896), No. 269.

DEFORMED TEETH.

106a. Cast of the Teeth and Gums of a girl æt. 17 years, who had congenital proptosis and a curious deformity of the face. A photograph and a drawing of this case will be found in Series lvii. No. 1331b.

CIRRHOSIS OF THE LIVER.

138. Cast of a Liver to demonstrate the external appearances characteristic of the ordinary form of cirrhosis (atrophic, alcoholic, or lobular cirrhosis).

SARCOMA OF LIVER.

138a₇. Cast of the Liver of a child æt. 10 years, showing numerous secondary sarcomatous deposits, which present the typical umbilicated appearance. The primary growth was in the kidney.

Microscopically the primary and secondary growths were shown to be sarcomatous.

A drawing of the liver will be found in Series lvii. No. 502a.

GOÏTRE.

138p. Goitre in a woman æt. 47 years.

See *Female Surgical Register*, vol. ii. (1896), No. 705.

ULNAR PARALYSIS.

144k. Cast of the Right Hand of a boy æt. 16 years suffering from ulnar paralysis, the result of an injury. Two photographs have been preserved in Series lvii. No. 733a, where also a short account of the history is found.

See *Male Surgical Register*, vol. v. (1896), No. 1286.

TERATOLOGICAL CATALOGUE.

SERIES I.—ABNORMAL CONDITIONS OF THE
AXIS.

CLASS V.—ARREST OF DEVELOPMENT.

SUB-CLASS V.—DEFECTIVE CLOSURE OF THE AXIAL CANAL OF
THE CEREBRO-SPINAL SYSTEM.**MENINGOCELE.**

3472b. Section through the Head and Neck of a female child 7 weeks old, demonstrating the relations of a typical meningocele. The latter is situated posteriorly at the back of the head, the basi-occipital being absent, so that the foramen magnum is enormously enlarged. The fluid (arachnoid) has passed through this aperture under the skin, and in that manner produced the bulging sac. The latter is in part separated from the cranial cavity by a portion of the occipital bone, and from the spinal canal by the laminæ and spines and their aponeuroses. Two incomplete septa run through the meningocele on its outer and upper walls. Looking into the cranial cavity, it will be seen that the cerebellum has been pushed up against the lower part of the tentorium, and has atrophied or has been insufficiently developed in consequence of pressure, so that it has, to a great extent at least, moved out of relation to the tentorium. The pons and medulla have also suffered considerably from the undue pressure. The meningocele has been in direct communication with the ventricles through the foramen of Majendie and the iter. A blue glass rod has been passed through this communication. The lateral ventricle is obviously distended. The pia mater covering of the meningocele is well shown both in the intra- and extra-cranial parts of the cyst.

[The letters M, P, and C have been affixed to the Medulla, Pons, and Cerebellum respectively.]

The child's head was very hydrocephalic; there was no marked proptosis, nor cleft palate or spina bifida; clinically convulsions were absent. The cyst was tapped on three occasions, and about 13 ounces of clear fluid removed without any benefit.

See *Female Surgical Register*, vol. iii. (1894), No. 2669*; and *Surgical Post-Mortem Register* (1895), p. 21.

SERIES IV.—ABNORMAL CONDITIONS OF THE VASCULAR SYSTEM.

CLASS V.—ARREST OF DEVELOPMENT.

3600a. Heart of a child showing a large round hole in the auricular septum, which is behind and independent of the foramen ovale, through which a green glass rod has been passed. Otherwise the heart is normal.

See *Medical Post-Mortem Register*, vol. xxii. (1895), p. 351.

ANATOMICAL AND PHYSIOLOGICAL CATALOGUE.

SERIES XIV.

THE INTESTINAL CANAL.

LONG PEYER'S PATCHES.

723a. Two specimens (see front and back) of Intestinal Mucous Membrane (Ileum), showing curiously elongated Peyer's patches, obtained from individuals free from intestinal lesions, one having died of bronchitis and the other from the effect of carbolic acid.

See *Medical Post-Mortem Register*, vol. xxii. (1895), pp. 253 and 278.

SERIES XXXII.

EARLY OVUM.

1196c. A specimen of an Early Ovum expelled from the uterus. It is more or less completely surrounded by chorionic villi, and on holding it up to the light the embryo can be seen floating in the amniotic cavity. The embryo was said to be about four weeks old.

See *Elizabeth Ward Notes* (1896), *sub* Sarah Parker.

PREGNANCY.

1208a. Sagittal Section through a Pregnant Uterus containing a complete foetus with its membranes, obtained from a woman *æt.* 30 years, who died after an enterectomy. Observe the shape and length of the cervical canal and the mucous plug at the external os. The more important parts have been lettered thus: *p* = Placenta; *v* = Decidua vera; *r* = Decidua reflexa; *c* = Cervix.

The cavity enclosed between the decidua vera and the decidua reflexa still exists, so that the pregnancy cannot have advanced beyond the fourth month; and the amnion can also be made out.

See *Surgical Post-Mortem Register* (1894), p. 53.

SERIES LVII.

DRAWINGS AND PHOTOGRAPHS OF DISEASED OR INJURED PARTS.

ACROMEGALY.

3a. Two Photographs of the man "Westphalen," described by Virchow as a typical case of Acromegaly. This man exhibited himself in London at the Royal Music Hall in 1890 under the name of Goliath, and was shown in that year at the Pathological Society, London.

A cast of this man's hand has been preserved in Series lvi. No. 1b.

For a description of the case, see *Illustrated Medical News* and *Berliner Klinische Wochenschrift*, 1889.

Presented by A. A. Kanthack, M.D.

3b. Photograph of the Hands of a man who presented the appearances characteristic of Acromegaly. The fingers are thick and fleshy ("tatzenartig"), and much increased in measurements, as a comparison with the photographs of two normal hands shows.

A cast of the left hand of this man has been preserved in Series lvi. No. 1b₁.

ACHONDROPLASIA.

4m. A Series of five Photographs showing the appearances associated with the condition known as Achondroplasia. The ages of the three girls are 13, 11, and 7 years respectively. They are the youngest of a family of six, their three elder brothers being grown up and full size. Both father and mother were healthy, and the girls are said to have been normal in appearance at birth and up to the fifth year.

Casts of the hands, legs, and feet will be found in Series lvi. Nos. 1m, n.

OSTEITIS DEFORMANS.

16b. Two Photographs from a case of Osteitis Deformans. (See also No. 1371 in this Series.)

See *Female Surgical Register*, vol. v. (1895), No. 2199.

16c. A Series of eight Photographs of the case of Osteitis Deformans, originally described by Sir James Paget in the *Transactions of the Medico-Chirurgical Society*, vol. lx. They require no further explanation. The central photograph shows to the left the cap worn by the patient before the disease appeared, to the right his hat when his condition had been fully developed.

Various bones of this man are preserved in Series i. No. 73.

Presented by A. Hamilton, M.D., of Windermere.

24b. Two Photographs of a girl *æt.* 14 years, to show the results of an osteotomy for Genu Valgum.

See *Female Surgical Register*, vol. ii. (1895), No. 2364*.

LEPROSY.

- 31m. A Photograph of the Bones of two Lepers' Hands, showing the characteristic atrophy of the phalanges in leprosy. (From Molde in Norway.)

Presented by G. Lindsay Johnson, M.D.

CONGENITAL PARALYSIS.

- 143d. Two Photographs taken of a boy affected with Congenital Paralysis of the left arm.

See *Male Surgical Register*, vol. iv. (1896), No. 219.

MORBUS CORDIS.

- 181a. Photograph of the Face and Chest of a boy suffering from morbus cordis.

See *Male Medical Register*, vol. v. (1896), *sub* Dennis Malone.

ORBITAL ANEURYSM.

- 191b. Photograph of a man *æt.* 35 years suffering from cirroid aneurysm of the orbit.

Twelve years ago the left common carotid artery had been tied; this was followed by temporary relief; six years afterwards galvano-puncture had been performed four times; gradually, however, the original condition returned, and the right carotid artery was tied, but without any benefit.

See *Male Surgical Register*, vol. i. (1895), No. 637.

- 193a. A Photograph of the same case (193), taken in 1895.

See *Male Surgical Register*, vol. iii. (1895), No. 425.

VARICOSE VEINS.

- 203a. Two Photographs of Varicose Veins on the anterior abdominal wall of a man *æt.* 35 years.

See *Male Medical Register*, vol. v. (1895), No. 32.

OBSTRUCTION OF SUPERIOR VENA CAVA.

- 204a. Photograph of a man *æt.* 44 years. There is considerable *œdema* of the face and upper half of the body, due to obstruction of the superior vena cava and the innominate veins, caused by an aneurysm of the arch of the aorta. After death it was seen that the superior vena cava lay practically in the wall of the aneurysm, and was thus obstructed.

See *Medical Post-Mortem Register*, vol. xxii. (1895), p. 306; and *Male Medical Register*, vol. ii. Part 2 (1895), No. 276.

PLASTIC OPERATION OF NOSE.

- 285a. Three Photographs of the Face of a man which had been shattered by a gun explosion. (*a*) Shows the face in its mutilated condition; (*b*) and (*c*) show the results of several plastic operations.

See *Male Surgical Register*, vol. iv. (1896), No. 2918*.

NIGRITIES LINGUÆ.

304a. Nigrities Linguæ in a man æt. 68 years. (L. Mark.)

See *Male Surgical Register*, vol. iii. (1896), No. 1657.

PERITONEAL FOLDS.

361-1. A curious Fold of the Peritoneum, probably of congenital origin, discovered during the autopsy of a man æt. 59 years, who died from strangulation of the intestine. (L. Mark.)

C=Cæcum. D=Duodenum. I=Ileum. L=Liver. M=Cut edge of mesentery. P=Pancreas. T.c.=Transverse colon. V=Vertebral column. 1=Right border of the fold, arising from one side of the vertebral column and from the posterior surface of the ascending colon. 2=Left border of the fold, arising from the left side of the vertebral column. 3=The upper portion of the fold, surrounding the duodenum and arising from the pancreas. 4=An additional fold with crescentic margin, arising from the junction of the ascending with the transverse colon and inserted into the main portion of the membrane. 5=The lower portion arising from the pelvis and the sacro-iliac synchondrosis, perforated near its free edge by a canal through which the ileum passes. The ileum is much narrowed for the last 3 inches of its course.

See *Surgical Post-Mortem Register* (1896), p. 109; and *Male Surgical Register*, vol. iii. (1896), No. 1477.

FUNICULAR HERNIA.

459. Right Funicular Hernia in a man æt. 22 years (photograph).

See *Male Surgical Register*, vol. iii. (1895), No. 885.

VENTRAL HERNIA.

467a. Drawing of a Ventral Hernia in a woman æt. 36 years. (L. Mark.)

See *Female Surgical Register*, vol. iv. (1896), No. 170.

SARCOMA OF LIVER.

502a. Drawing of a Sarcoma of the Liver (secondary to a kidney-growth) in a child æt. 10 years. (L. Mark.)

The liver and a cast of it have been kept in Series xxi. No. 2215c, and in Series lvi. No. 138a₇ respectively.

A microscopic specimen has been preserved in the *Histological Records*, lvii. No. 2215c.

BRONCHOCELE.

533b. Two Photographs of the Face and Neck of a woman æt. 35 years who had a bilateral goitre. There was much dyspnœa, and the trachea was displaced to the left.

See *Female Surgical Register*, vol. iv. (1896), No. 1675.

574a. Two Photographs of a woman æt. 21 years, who in 1890 had been operated upon for a goitre, the right lobe of the thyroid gland having been removed. Subsequently the left lobe had become enlarged, and she developed slight symptoms of myxœdema or "cachexia thyreopriva."

See *Female Surgical Register*, vol. iii. (1894), No. 2078.

MYXŒDEMA.

585d. Photograph of a woman suffering from myxœdema.

CONGENITAL BAND.

649a. Drawing of an Everted Bladder affected with cystitis, and showing a congenital band stretching downwards from the opening of the right ureter. Obtained from the body of a woman æt. 34 years, who died with tubercular disease of the kidney and bladder. (L. Mark.)

See *Female Surgical Register*, vol. iv. (1895), No. 2588; and *Surgical Post-Mortem Register* (1895), p. 283.

SYMPATHETIC PARALYSIS.

724b. A Photograph showing the appearances in the face following pressure upon the cervical sympathetic nerve.

From a man æt. 56 years, admitted with carcinoma of the œsophagus.

See *Male Surgical Register*, vol. v. (1895), No. 921.

PARALYSIS OF SERRATUS MAGNUS.

726a. Two Photographs showing the characteristic position of the scapula due to paralysis of the serratus magnus.

726b. Photograph showing paralysis of the left serratus magnus.

See *Male Medical Register*, vol. ii. (1896), *sub* William Carver.

DIPHTHERITIC PARALYSIS (?).

726c. Two Photographs of a child æt. 4 years, thought to be suffering from diphtheritic paralysis of the muscles of the lips and tongue. The palate was natural, but there was partial loss of power in the arms, and to a lesser extent in the legs; the knee-jerks were temporarily absent. Later rigidity of the arms and hands appeared, rigid flexion of the wrist and finger-joints, and also aphasia and paralysis of the tongue. The history of diphtheria was doubtful.

See *Male Medical Register*, vol. ii. (1896), *sub* Christopher Bland.

PARALYSIS OF ULNAR NERVE.

733a. Two Photographs of the Right Hand and Forearm of a boy æt. 16 years suffering from paralysis of the ulnar nerve, due to an injury caused by falling out of a window on to a skylight, the nerve being divided in two places, viz., at the middle of the forearm and just above the wrist. The divided ends were sutured on the same afternoon, and union by "first intention" was obtained. There was no return of sensation or of power, and the hand remained in the position typical of ulnar paralysis. He was re-admitted twice since, and two further operations performed with slight benefit. The injury occurred on August 18, 1895; the photograph was taken May 15, 1896.

See *Male Surgical Register*, vol. v. (1895), No. 3391, and vol. v. (1896), No. 1286. A cast of the hand is preserved in Series lvi. No. 144k.

ULCERATION OF CORNEA.

736a. A Photograph of a young woman's Face showing marked exophthalmos, accompanied by extensive and severe ulceration of the cornea.

DERMATITIS HERPETIFORMIS.

773c. Photograph of the Hands in a case of Dermatitis Herpetiformis occurring in a woman.

See *St. Bartholomew's Hospital Reports* for 1893, p. 313 (Dr. Samuel West).
Presented by Dr. S. West.

773d. Two Photographs from a case of Dermatitis Herpetiformis occurring in a man.

See *St. Bartholomew's Hospital Reports* for 1893, p. 313 (Dr. Samuel West).

PITYRIASIS RUBRA PILARIS.

773e. Two Photographs (*a*) of Abdominal Wall and (*b*) of Hands and Feet of a man suffering from Pityriasis rubra pilaris (Devergie).

See *British Journal of Dermatology*, No. 83, vol. vii.

CHIRO-POMPHOLYX.

814a. Three Photographs of the Hands of a man suffering from Chiro-pompholyx.

See *Skin Department*, 1894, sub J. Goodall.

KAPOSI'S DISEASE.

833a. A Drawing and a Photograph of a case of so-called Kaposi's disease (Xeroderma pigmentosa).

A microscopic specimen of one of the nodules has been preserved in the *Histological Records*, lvii. 833a.

See *Female Surgical Register*, vol. iii. (1895), No. 2200; and *Female Medical Register*, vol. ii. Part 2 (1895), sub Kathleen Taylor.

Cf. *British Journal of Dermatology* for 1895 (Dr. S. West); and *St. Bartholomew's Hospital Reports* for 1895.

ATROPHY OF NAILS.

842a. Photograph showing severe atrophic changes in the nails after influenza.

MOLLUSCUM CONTAGIOSUM.

861a. A Drawing and a Photograph of a case of confluent Molluscum Contagiosum on the head of a child 7 months old.

See *Female Surgical Register*, vol. iii. (1896), No. 1343.

ELEPHANTIASIS.

864a. A Photograph of a case of "Elephantiasis." From a woman *æt.* 57 years, suffering from Carcinoma Mammæ, who had not been out of England.

See *Female Surgical Register*, vol. iii. (1895), No. 376.

864b. "Elephantiasis" of the Leg in a boy *æt.* 16 years. (Photograph.)

See *Male Surgical Register*, vol. v. (1895), No. 2741.

PIGMENTED MOLE.

882a. Photograph of the Face of a woman *æt.* 26 years, showing a large pigmented mole.

See *Female Surgical Register*, vol. v. (1895), No. 612.

SKIN DISEASES OF UNCERTAIN NATURE.

924a. Photograph of the Back of a man *æt.* 57 years, presenting a curiously scarred appearance. The nature of the affection remained uncertain, and was diagnosed as either Phthiriasis, or Erythema Iris, or Syphilide.

See *Male Surgical Register*, vol. v. (1895), No. 2102.

BURNS.

1064b. Severe scarring and contraction of the skin of the neck and face after a burn. (Photograph.)

See *Male Surgical Register*, vol. iv. (1895), No. 2949.

1064c. Severe scarring and contraction of the skin of the neck and face after an extensive burn. (Photograph.)

See *Male Surgical Register*, vol. i. (1895), No. 3142.

ULCER ON SCALP.

1077a. A curious Ulcer which appeared on the scalp of an infant *æt.* 2 years, and was treated with nitric acid (its nature remained uncertain). (L. Mark.)

See *Female Surgical Register*, vol. iii. (1896), No. 537.

GANGRENE OF THE SCROTUM AND PENIS.

1081a. Drawing of Gangrene of the Scrotum and Penis in an infant 14 days old (cellulitis or erysipelas neonatorum), which caused death of the child from septicæmia. The streptococcus pyogenes was found in the skin, the spleen, and the blood. (L. Mark.)

See *Male Surgical Register*, vol. iv. (1895), No. 3645; and *Surgical Post-Mortem Register* (1895), p. 289.

GANGRENOUS CELLULITIS.

1082a. Photograph of the Left Leg of a man *æt.* 75 years, in a condition of gangrenous cellulitis. The leg was amputated through the thigh, but the man died soon afterwards of shock. All the arteries were found to be rigid and calcareous at the post-mortem examination; there was also infective endocarditis.

See *Male Surgical Register*, vol. iii. (1894), No. 1300; and *Surgical Post-Mortem Register* (1894), p. 80.

GANGRENE.

- 1095a. Drawing of Gangrene of the Right Leg of a man æt. 34 years, who was admitted with gangrene of both legs, the right being in a more advanced condition than the left. The former was amputated, while the latter recovered. (L. Mark.)

See *Male Surgical Register*, vol. iv. (1896), No. 13.

FUNGATING ULCER (TUBERCULAR).

- 1105b. Drawing of the Leg of a woman æt. 50 years, showing large fungating masses which have sprung up from pre-existing ulcers.

Microscopic examination showed these masses to be tubercular, and a microscopic specimen has been kept in the *Histological Records*, lvii.

1105b. (L. Mark.)

See *Female Surgical Register*, vol. v. (1896), No. 2914*.

TUBERCULOSIS.

- 1105c. A Photograph exhibiting extensive tubercular disease of the Tarsus in a man æt. 60 years.

See *Male Surgical Register*, vol. ii. (1896), No. 789.

LUPUS.

- 1110a. Chronic Tubercular Ulceration around the left angle of the Mouth (lupus) of a boy æt. 18 years. (L. Mark.)

See *Male Surgical Register*, vol. i. (1895), No. 3105.

ERUPTION ON NOSE.

- 1112a. A Lupus-like Eruption, of uncertain nature, on the Nose of a woman æt. 53 years. (L. Mark.)

See *Female Surgical Register*, vol. i. (1896), No. 1403.

VARIOLA.

- 1116a. Photograph of a man suffering from smallpox, who had been in a Salvation Army Shelter for three nights, and was eventually picked up in the streets by a policeman.

Presented by M. G. Pearson, Esq.

SYPHILITIC SORES.

- 1117a. Primary Syphilitic Sores on both Nipples of the Breast of a woman æt. 31 years. Her child was syphilitic, and a drawing of her pudenda will be found under No. 1132. (L. Mark.)

MUCOUS TUBERCLES.

1132. Drawing of the Pudenda and Perinæum of an infant æt. 16 months affected with congenital syphilis (mucous tubercles and ulceration). This infant is the child of the woman whose breasts are depicted in Drawing No. 1117a. The mother had primary syphilitic sores on both nipples. (L. Mark.)

TERTIARY SYPHILIS.

1150a. Two Photographs of the Head of a man æt. 39 years who had contracted syphilis twelve years previously.

See *Male Surgical Register*, vol. iv. (1894), No. 740.

1152a. Two Photographs of the Face of a woman showing the typical "syphilitic nose."

See *Male Surgical Register*, vol. iv. (1894), No. 2107.

HÆMOPHILIA.

1179. Drawing showing a curious Hæmorrhagic appearance of the Skin of the Chest and Abdomen (subcutaneous hæmorrhage) associated with hæmophilia. From a man æt. 48 years. (L. Mark.)

See *Male Surgical Register*, vol. iv. (1896), No. 785.

LIPOMA.

1193c. Photograph of the Face and Neck of a woman æt. 50 years, with a lipoma in the neck.

See *Female Surgical Register*, vol. iii. (1896), No. 433.

SARCOMA.

1233a. Photograph of a man æt. 49 years with an enormous Sarcoma growing from the left scapular region.

See *Male Surgical Register*, vol. v. (1895), No. 3469*.

1233b. A Sarcoma developed upon a Chronic Ulcer of the Leg. (Photograph.)

See *Male Surgical Register*, vol. iii. (1896), No. 3093.

TUMOURS OF UNCERTAIN NATURE.

1242a. Photograph and a coloured Drawing of a curious reddish (copper-coloured) growth on the back of a man æt. 45 years, of nine years' duration, of uncertain nature, diagnosed microscopically by some as fibro-sarcoma, by others as merely inflammatory. It was removed by operation, and six months later there had been no recurrence.

A microscopic specimen has been preserved in the *Histological Records*, lvii. 1242a.

See *Male Surgical Register*, vol. iii. (1896), No. 1075.

EPITHELIOMA.

1271a. Two Photographs of a woman æt. 42 years, showing a large Tumour in the neck, secondary to a malignant growth (epithelioma) on the posterior aspect of the cricoid cartilage which had caused no symptoms.

See *Female Surgical Register*, vol. v. (1896), No. 1097.

1277b. Photograph of the Left Leg of a woman æt. 66 years with an epitheliomatous growth developed upon an old ulcer.

See *Female Surgical Register*, vol. v. (1896), No. 615.

1278a. Photograph of the Back of the Head of an old woman occupied by an epitheliomatous growth.

See *Female Surgical Register*, vol. iv. (1895), No. 1897 and 2594*.

1280. Two drawings of an Epithelioma over the Scapula in a man æt. 45 years. (L. Mark.)

See *Male Surgical Register*, vol. ii. (1895), No. 3065.

CYSTIC HYGROMA.

1301a. Cystic Hygroma in the left groin of a child æt. 4 years. (Photograph.)

See *Male Surgical Register*, vol. iii. (1895), No. 678.

SEBACEOUS CYST.

1305. Photograph of a Tumour, thought to be a fibroma, growing from the navel of a boy æt. 12 years. It contained atheromatous matter, and was therefore probably a sebaceous cyst.

On microscopical examination the cyst was seen to be lined with squamous epithelium, and in one part numerous giant cells were found. Evidence of any dermoid structures could not be obtained.

A microscopic section has been preserved in the *Histological Records*, lvii. 1305.

See *Male Surgical Register*, vol. ii. (1896), No. 1852.

DERMOID CYST.

1305a. Drawing of a Dermoid Cyst about the angle of the lower jaw in a girl æt. 15 years. (L. Mark.)

See *Female Surgical Register*, vol. v. (1896), No. 600.

ECTOPIA VESICÆ.

1312c. Drawing of an Ectopia Vesicæ in a man æt. 23 years, after operation. (L. Mark.)

See *Male Surgical Register*, vol. iii. (1896), No. 2254*.

HYOSPADIAS.

1315l. Three Photographs of the External Genitalia of a man which resembled the pudenda to such an extent that for some years the individual had been brought up as a girl.

No. 1 is a photograph of the man showing the appearance of the parts in the erect posture.

No. 2 represents the appearance of the parts themselves, showing the rudimentary penis, the urethral opening, and the divided scrotum.

No. 3 represents the parts after an operation had been performed in

order to restore the meatus urinarius. (A black catheter is placed in the urethra.)

It is evident that this is not a case of hermaphroditism, but simply one of an extensive and complete hypospadias.

See *Male Surgical Register*, vol. i. (1895), No. 2632, and vol. i. (1896), No. 463a.

PTEROMALLEUS.

1327c. Photograph illustrating the condition described as Pteromalleus.

See *Female Surgical Register*, vol. iii. (1895), No. 1561.

CONGENITAL DEFORMITY OF HANDS.

1327d. Two Photographs exhibiting a curious Congenital Deformity of the Hands in a man *æt.* 29 years.

Casts of the hands will be found in Series lvi. No. 66g.

See *Male Surgical Register*, vol. ii. (1896), No. 1258.

CLEFT PALATE.

1327e. Photograph of a girl *æt.* 6 years, exhibiting a cleft of the soft palate.

See *Female Surgical Register*, vol. iv. (1896), No. 421.

1331b Photograph and Drawing of a girl *æt.* 17 years with marked proptosis and other curious deformities of the face, of congenital origin. Casts of the teeth and gums have been preserved in Series lvi. No. 106a.

See *Ophthalmic Ward Book* (1895), No. 1896.

CONGENITAL FISTULA.

1335l. Congenital Fistula over the Sacrum in a girl *æt.* 15 years. (L. Mark.)

See *Female Surgical Register*, vol. v. (1896), No. 1025.

CONGENITAL DEFORMITY OF EAR.

1340a. Two Photographs showing congenital deficiency of the external meatus of the ear.

See *Male Surgical Register*, vol. i. (1896), No. 3401*.

DIPYGUS PARASITICUS.

1342a. Three Photographs of a female child *æt.* 6 years with a supernumerary leg growing from the left buttock (a dipygus parasiticus). This case forms no exception to the law that the completeness of the parasite varies directly with its proximity to the head of its host; growing from the pelvis, the parasite is represented merely by a lower extremity.

See *Female Surgical Register*, vol. v. (1896), No. 1506.

CLUB-FOOT.

- 1342l. Photograph of a child æt. $3\frac{1}{2}$ years with Talipes Valgus on the right side and Talipes Varus on the left side.

Casts of the hands and feet will be found in Series lvi. Nos. 66h and 87h.
See *Orthopædic Department* (1896), No. 269.

POTT'S FRACTURE.

- 1342m. A badly united or badly set Pott's Fracture, leaving a marked deformity behind (pseudo-varus). (Photograph.)

See *Orthopædic Department* (1896), sub Fanny Green.

LYMPHATIC OBSTRUCTION.

- 1350a. Two Photographs of the face of a man æt. 45 years, showing partial swelling of the right side of the face, especially around the right eye, said to have been due to blocking of the right lymphatic duct.

See *Male Surgical Register*, vol. v. (1895), No. 1738.

UNCERTAIN TUMOUR.

- 1351a. Photograph of a woman æt. 42 years, showing a tumour at the root of the neck on the left side, which was cystic in nature and probably malignant. The woman refused an operation, and the diagnosis could therefore not be confirmed.

See *Female Surgical Register*, vol. i. (1896), No. 190.

HYSTERIA.

1358. Two Photographs of an emaciated woman, æt. 17 years, suffering from "Hysteria," who refused food on account of anorexia (anorexia nervosa).

See *Female Medical Register*, vol. iii. (1896), No. 55.

OPERATIVE SURGERY.

- 1360a. A set of Seven Drawings and a Photograph illustrating the methods of arranging the skin flaps in radical operations for the removal of cancerous breasts, so as to successfully cover in the denuded area.

See a paper read by H. T. Butlin, Esq., F.R.C.S. (*Transactions of the Royal Medico-Chirurgical Society*, vol. lxxix. (1896); and *Female Surgical Register*, vol. v. (1894), No. 833.

Presented by H. T. Butlin, Esq., F.R.C.S.

FACIAL DIAGNOSIS.

IDIOCY.

1370. Two Photographs of an Idiot.

Presented by B. Vernon, Esq., F.R.C.S.

OSTEITIS DEFORMANS.

1371. Photograph of the head of a woman suffering from Osteitis Deformans. For further photographs of the same woman see this Series, No. 16b, where also a reference to her case will be found.

EXOPHTHALMIC GOÏTRE.

1372. Photograph to show the Typical Facial Appearance of a woman suffering from Exophthalmic Goitre.

ADENOID VEGETATIONS.

1373. Photograph of a young woman exhibiting the Typical Facial Appearance accompanying Adenoid Vegetations.

III.

HISTOLOGICAL RECORDS OF MUSEUM
SPECIMENS.*

- I. 507a. Microscopic Section of Specimen No. 507a in Series i., from a femur which had fractured spontaneously. The patient, a woman *æt.* 30 years, had carcinoma of the breast. The specimen shows that there was a cancerous growth at the seat of fracture (an adenoid or glandular cancer, with well-marked fibrous stroma). (Prepared by E. L. Lloyd.)

See Series i. No. 507a.

- VIII. 1464b. Microscopic Specimens illustrating the appearance of one of the iliac arteries in the case of the dissecting aneurysm preserved in Series viii. No. 1464b. The intima is thickened and lifted from the tunica media by coagulum. (Prepared by E. H. Shaw.)

See Series viii. No. 1464b.

- X. 1645b. Microscopic Specimen, stained with hæmatoxylin, of a papillomatous growth in the trachea, from a case of diffuse papilloma of the larynx. These tracheal growths are lined by squamous epithelium. (Prepared by E. H. Shaw.)

See Series x. No. 1645b; and *Male Surgical Register*, vol. iv. (1895). No. 1400.

- X. 1645c. Microscopic Specimens, stained by hæmatoxylin, of the papillomatous growths around the tracheotomy opening in a case of diffuse papilloma of the larynx. The growths are lined by an irregular columnar epithelium. (Prepared by E. H. Shaw.)

See Series x. No. 1645c.

- X. 1657a. Microscopic Section of Specimen No. 1657a in Series x. It is a typical squamous-celled carcinoma (epithelioma). (Prepared by E. H. Shaw.)

See Series x. No. 1657a.

- XII. 1770d. Microscopic Section from Specimen No. 1770d in Series xii., a large nasopharyngeal growth. It is a very vascular fibroma, an angio-fibroma, presenting no malignant, *i.e.*, sarcomatous, appearances. (Prepared by E. H. Shaw.)

See Series xii. No. 1770d.

* The Roman figure refers to the Series, the ordinary figure to the number of the Specimen.

- XII. **1800d.** Microscopic Section of Specimen No. 1800d. in Series xii. It agrees in its character more or less with the growths described by Stephen Paget, Esq., in the *Transactions of the Pathological Society, London*, vol. xxiii. p. 348, of which examples will be found in Series lv. No. 220. (Prepared by E. H. Shaw.)
See Series xii. No. 1800d.
- XII. **1807d.** Microscopic Section of Specimen No. 1807d. in Series xii. It is an ordinary papillomatous hypertrophy of the tonsil, lined by squamous epithelium. (Prepared by E. H. Shaw.)
See Series xii. No. 1807d.
- XV. **1833b.** Section through the wall of the Oesophageal Pouch, preserved in Series xv. No. 1833b. It is lined by squamous epithelium, and contains striped muscle. (Prepared by E. H. Shaw.)
See Series xv. No. 1833b.
- XVI. **1886f.** A Microscopic Section from Specimen No. 1886f in Series xvi., multiple melanotic deposits in the mesentery. The latter are typical melanotic sarcomatous growths. (Prepared by E. H. Shaw.)
See Series xvi. No. 1886f.
- XVIII. **1987a.** Microscopic Section from Specimen 1987a in Series xviii., which was said to be a cicatrised dysenteric ulcer, which had led to an intestinal stricture. The specimen is a typical columnar-celled carcinoma, colloid in parts. (Prepared by T. Strangeways Pigg.)
See Series xviii. No. 1987a.
- XVIII. **2022a.** Microscopic Section of Specimen 2022a in Series xviii., an intestinal new growth, formerly described as an angio-sarcoma. It is a columnar-celled carcinoma, colloid in parts. (Prepared by T. Strangeways Pigg.)
See Series xviii. No. 2022a.
- XVIII. **2019e.** Microscopic Sections of Specimen No. 2019e preserved in Series xviii. The section has been made in the long axis of the bowel through some of the polypoid excrescences. The latter are due to a hypertrophy of the mucosa, and not due to dilatation of the lymphatics. They possess all the normal elements of the mucosa, villi, follicles, &c. (Prepared by E. H. Shaw.)
See Series xviii. No. 2019e.
- XIX. **2073d.** Two Microscopic Slides from a case of Melanotic Sarcoma of the Rectum, preserved in Series xix. No. 2073d. They confirm the diagnosis, demonstrating the typical appearances of a melanotic sarcoma, in this case consisting chiefly of spindle cells, with pigmented cells, but no alveolation. Specimen (i.) is taken right through the growth; specimen (ii.) through a little submucous melanotic nodule near the anus. (Prepared by T. Strangeways Pigg.)
See Series xix. No. 2073d.

xx. 2177c. Sections from the Specimen 2177c in Series No. xx., a subserous tumour growing from the free margin of the intestine, which had caused strangulation of the bowel. It is a fibrous (fibro-cellular) tumour, containing much blood. (Prepared by E. H. Shaw.)

See Series xx. No. 2177c.

xx. 2188b. Microscopic Section of Specimen 2188b in Series xx. The specimen had been described in former catalogues as one of chronic intussusception, but the microscopic specimen shows undoubtedly that the cause of the trouble was a carcinoma (a columnar-celled carcinoma). (Prepared by E. H. Shaw.)

See Series xx. No. 2188b.

xxi. 2215c. Microscopic Section of Specimen No. 2215c in Series xxi., of which also a cast and a drawing have been preserved in Series lvi. No. 1387a₁, and Series lvii. No. 502a respectively. It is a sarcoma with curious tubular structures. The growth in the liver was secondary to a growth in the kidney, which presented the same appearance, but was rather more tubular. (Prepared by T. Strangeways Pigg.)

See Series xxi. No. 2215c; and Series lvi. No. 138a₇; and also Series lvii. No. 502a.

xxi. 2217a. Microscopic Section of Specimen No. 2217a in Series xxi., in a former catalogue described as diffuse lympho-sarcoma of the liver. The liver is diffusely infiltrated with small round cells, but there is no reason why the growth should be a lympho-sarcoma rather than a round-celled sarcoma. (Prepared by E. L. Lloyd.)

See Series xxi. No. 2217a.

xxi. 2239c. Microscopic Section of Specimen 2239c in Series xxi. It has been stained by Weigert's method, and shows the presence of the actinomyces fungus. (Prepared by A. A. Kanthack.)

See Series xxi. No. 2239c.

xxii. 2254. Microscopic Section of Specimen 2254 in Series xxii. The growth which surrounded and closed the orifice of the common bile duct is a typical columnar-celled carcinoma. (Prepared by E. H. Shaw.)

See Series xxii. No. 2254.

xxii. 2264c. Microscopic Section of a portion of a Lumbar Gland from a case of carcinoma of the gall-bladder, which has been preserved in Series xxii. No. 2264c. It is a columnar-celled carcinoma. (From the Surgical Registrar.)

See Series xxii. No. 2264c.

xxii. 2265. Microscopic Section of Specimen 2265 in Series xxii., a cancerous growth in the gall-bladder. It is a soft alveolar carcinoma. (Prepared by E. H. Shaw.)

See Series xxii. No. 2265.

xxiv. 2294. Microscopic Section of Specimen 2294 in Series xxiv., formerly described as a melanotic tumour. It is a typical melanotic sarcoma. (Prepared by E. H. Shaw.)

See Series xxiv. No. 2294

xxviii. 2390a. Microscopic Sections ((i.) kidney, (ii.) ureter) from Specimen No. 2390a in Series xxviii., a cancerous growth in the kidney and ureter. Microscopically the growth in the kidney is a columnar-celled carcinoma with an extensive, delicate, fibrous stroma. The cells occupying the alveoli are irregular in size and shape and mostly cubical or short columnar, but in some parts rather tall columnar cells are seen. Some of the alveoli are completely filled up by these pleomorphic cells; others are merely lined by a single layer of either tall or short columnar cells. In the ureter the growth was villous, but it is the same form of carcinoma, which here, however, has assumed a villous character, a recognised type of carcinoma of the ureter. It is highly probable, therefore, that the growth in the kidney arose from the epithelium of the pelvis, calyces, and ureter. (*Cf.* Paper by Dr. Voelker, *Transactions of the Pathological Society*, vol. xlvi. p. 133.) (Prepared by E. H. Shaw.)

See Series xxviii. No. 2390a.

xxix. 2417a₁. Microscopic Section through one of the myxomatous polypi of Specimen No. 2417a₁ in Series xxix. In one part it is a pure myxoma, but under the surface the growth is very cellular and not unlike a mixed-celled sarcoma. In other parts, again, it is distinctly fibrous. The surface epithelium is much proliferated, and approaches the columnar type. The spaces near the surface, lined by proliferated epithelium, are no doubt crypts and follicles in section, and not glands. The growth, therefore, is possibly a fibro-myxo-sarcoma. (Specimen prepared by E. H. Shaw.)

See Series xxix. No. 2417a₁.

xxxiii. 2570a. Microscopic Sections of Specimen 2570a in Series xxxiii., an Orbital Sarcoma occurring in a boy. The growth is a typical mixed-celled sarcoma, more or less myxomatous in nature. (Prepared by F. F. Wesbrook.)

See Series xxxiii. No. 2570a.

xxxiii. 2613e. Microscopic Section from the Eye preserved in Series xxxiii. No. 2613e. The section was taken from the ciliary region, and shows the presence of bone, or rather osteoid tissue, in the inflammatory and fibrous material which has appeared there in consequence of the lesion. (Prepared by E. H. Shaw.)

See Series xxxiii. No. 2613e.

xl. 2962a. Microscopic Section of Specimen No. 2962a in Series xli., taken from the thickened mucous membrane of the uterus. This specimen was formerly described as an adenoma; it is, however, a typical alveolar carcinoma. (Prepared by E. H. Shaw.)

See Series xli. No. 2962a.

XLIII. 3006c. Microscopic Section from a growth in the Cervix Uteri in a woman *æt.* 33 years. The growth has been preserved in Series xliii. No. 3006c. It is a typical squamous-celled epithelioma. (Prepared by T. Strangeways Pigg.)

See Series xliii. No. 3006c.

XLIII. 3006e. Microscopic Sections from a malignant growth in the Cervix Uteri. The growth has been preserved in Series xliii. No. 3006e. It has the histological characters of an adeno-carcinoma, there being numerous alveoli completely filled by polygonal or cubical cells; but on examining the sections carefully it will be seen that the origin of the growth was from columnar epithelium, for in some of the alveoli columnar cells will be found. These have proliferated to such an extent as to fill the alveoli, and through pressure they have assumed cubical or polygonal shapes. (Prepared by T. Strangeways Pigg.)

See Series xliii. No. 3006e.

XLIV. 3030. Microscopic Section from Specimen 3030 in Series xliv., Polypoid Growths removed from the nymphæ and walls of the vagina of a child. The growths are mixed-celled sarcomas, with numerous large cells, resembling giant cells. There is also much mucous degeneration. The growths are hardly true myxo-sarcomas, but rather mixed-celled sarcomas, undergoing myxomatous degeneration. (Prepared by E. H. Shaw.)

See Series xliv. No. 3030.

XLIV. 3030a. Microscopic Sections (three slides) from Specimen 3030a in Series xliv., a vaginal growth in a child *æt.* $2\frac{1}{3}$ years. The growth in its firmer parts is a typical mixed-celled sarcoma; in its softer parts it is myxomatous in nature. (Prepared by T. Strangeways Pigg.)

See Series xliv. No. 3030a.

XLIV. 3034. Microscopic Section of a growth in the left labium, preserved in Series xliv. No. 3034. The growth is a typical squamous-celled carcinoma (epithelioma). (Prepared by E. H. Shaw.)

See Series xliv. No. 3034.

L. 3327c. Sections through the wall of the epitheliomatous cyst preserved in Series l. No. 3327c. They show the presence of an undoubtedly epitheliomatous growth. (Prepared by E. Willett, Esq.)

See Series l. No. 3327c.

L. 3324a₁. Microscopic Section from the Specimen No. 3324a₁ in Series l. It is a typical rodent ulcer. (Prepared by T. Strangeways Pigg.)

See Series l. No. 3324a₁.

LVII. 833a. Microscopic Specimen of a Nodule removed from the skin of a girl suffering from Kaposi's disease (Xerodermia pigmentosa). (Prepared by E. H. Shaw.)

For a drawing and a photograph of this case see Series lvii. No. 833a.

LVII. 1105b. Microscopic Section from the Fungating Ulcer of the leg depicted in Series lvii. No. 1105b. The fungating masses are undoubtedly tubercular. (Prepared by E. H. Shaw.)

See Series lvii. No. 1105b.

LVII. 1242a. Section of the Tumour on the back of a man *æt.* 48 years, depicted in Series lvii. No. 1242a. It is stained by carmine, and microscopically appears to be a sarcoma, but the clinical opinion and appearances were not in favour of this view. (Prepared by A. A. Kanthack.)

See drawing and photograph in Series lvii. No. 1242a.

LVII. 1305. Microscopic Section through the wall of an Umbilical Cyst, of which a photograph will be found in Series lvii. No. 1305. It had been described as a fibroma, but the section shows no evidence of any fibroid or dermoid structures. The cyst is lined by squamous epithelium, and in one part numerous giant-cells will be found. (Prepared by T. Strangeways Pigg.)

See Series lvii. No. 1305.

IV
ALPHABETICAL INDEX

OF THE
DRAWINGS AND PHOTOGRAPHS OF DISEASED
OR INJURED PARTS

(SERIES LVII.)

IN THE
MUSEUM OF ST. BARTHOLOMEW'S HOSPITAL

1896

- ABORTION, 1012
Abscess, brain, 689, 692-694
— breast, 1055
— cardiac, 161
— cerebral, 689, 692-694
— lachrymal, 740
— liver, 484
— psoas, 135
Acarus, 912
Achondroplasia, 4m
Acne, 815, 816, 856, 857
— varioliformis, 815, 816
— vulgaris, 857
Acromegaly, 3-4
Actinomycosis, 1106-1106b
Adenocarcinoma, *see* Carcinoma
Adenoid vegetations, 1373
Addison's disease, 593-599b, 601, 1352
Addison's keloid, *see* Keloid
Alcoholic neuritis, 725
Ammonia, *see* Poisoning
Amyloid degeneration, liver, 495
— degeneration, spleen, 523
Anæmia, brain, 680; *see* Leucocythæmia
Aneurysm, 185-193, 195-199, 204a
— anastomotic, 191, 1200
— aortic, 185-187, 204a
— axillary, 190
— cirroid, 191-191b, 1200
Aneurysm, embolic, 195-199
— false, 197, 198
— femoral, 188
— multiple, 195
— orbital, 191a, b
— pulmonary artery, 281
— varicose, 192-193a
Aneurysmal varix, 192
Angioma, arterial, 191, 1200
— dura mater, 702
Ankle, tubercular disease, 1105c
Ankylosis, *see* Joints
Anorexia nervosa, 1358
Anteflexion of uterus, 991
Ante-mortem clots, 156-160
Anus, 450, 451, 453-455
— condylomata, 453, 454
— imperforate, 450, 451
— papilloma, 455
— syphilis, 453, 454
— warts, 455
Aorta, aneurysm, 185-187, 204a
— atheroma, 182, 184
— valves, 172-175
Aphasia, 699a-c
Apoplexy, lung, 257-260
— brain, *see* Brain
Arachnoid, *see* Meninges
Arsenic, *see* Poisoning

- Arteries, 182-201
 — obstruction of, 194-200
 — plugging of, 194-200.
 — rupture of, 201.
 Arthrectomy, *see* Joints and Operations
 Arthritis, *see* Joints
 Articular cartilage, *see* Joints
 Ascites, 367, 368
 Ataxia, locomotor, 1072
 Atelectasis, 1332
 Atheroma, 182-184
 — aorta, 182-184
 — pulmonary artery, 183
 Atrophy—
 — acute yellow, 482
 — bone, 1
 — brain, 652
 — heart, 154
 — nails, *see* Nails
 — progressive muscular, 136-138
 — thyroid gland, 542
 — tongue, 296, 297
 — yellow, 482
 Auricles, supernumerary, 1338-1339, 1341
 Axilla, cancer, 1258
 Axillary artery, aneurysm, 190
- BANDL'S ring, 1004
 Bichromate of potash, *see* Poisoning
 Bile ducts, 483, 503, 508
 Bladder, 641, 643-650, 1312-1313a
 — band, 649, 649a
 — ecchymoses, 643
 — ectopia, 1312-1313a
 — enteric, 643
 — fistula, recto-vesical, 647
 — hæmorrhages, 643, 644
 — inter-uretal fold, 649
 — papilloma, 650
 — pouch, 649
 — tubercle, 629, 645, 649
 — tumours, multiple, 641
 Blood, leukæmia, 1349
 Bones, absorption. 23
 — achondroplasia, 4m
 — acromegaly, 3-4
 — atrophy, 1
 — caries, 1105c
 — cephalhydrocele (traumatic), 58
 — chondroma, 34, 35, 37, 38
 — chromium (enlargement), 2
 — — syphilis, 31, 57; *see* also
 Skull
 — enchondroma, 34, 35, 37, 38
 — fracture, 103-112, 124-130, 1342m
 — fracture, compound, 22; *see* under
 Fracture
 — jaw, *see* under Jaw
 — leprosy, 31m
- Bones, malignant growths, 39, 56, 59,
 123
 — maxilla, *see* under Jaw
 — necrosis, 11-14, 18, 20-22, 28
 — nodes, 30, 57
 — nodes, Parrot's, 57
 — osteitis deformans, 1517, 1371
 — osteoma, 32, 33
 — osteomyelitis, 5-8
 — osteotomy, 24a-b
 — Parrot's nodes, 57
 — periostitis, 18, 19
 — rickets, 24, 25
 — rickets, scurvy, 26, 27
 — sarcoma, 42, 44-49, 52-55, 59
 — sclerosis, 9, 10, 122
 — scurvy rickets, 26, 27
 — syphilis, 28, 30, 31
 — syphilis, congenital, 31, 57
 — syphilis, necrosis, 28, 31
 — ulceration (articular ends), 29
 Brain, abscess, 689, 692-694
 — anæmia, 680
 — aphasia, 692a-c
 — apoplexy, 657-671
 — atrophy, 652
 — cerebellum, 697, 708
 — circulation, 672-677
 — congestion, 653
 — corpus striatum, 652, 669
 — crura, 662, 663, 696
 — encephalitis, 690
 — encephalocele, 713
 — fracture of skull, abscess, 689, 693
 — glioma, 703
 — hæmorrhage, 657-671
 — hernia, 710
 — hydrocephalus, 654
 — optic thalamus, 652, 658, 705
 — pineal gland, 709
 — pons, 662-665, 697, 698
 — sarcoma, 708, 712
 — sclerosis, 700, 701
 — sloughing, 691
 — softening, 670, 671
 — tuberculosis, 696-698
 — tumours, 696-698, 704-708
 — ventricle, fourth, 662, 664
 Breast, 1025-1059
 — abscess, 1055
 — atrophic scirrhus, 1039
 — carcinoma, 1034-1048, 1052-1054,
 1057, 1058
 — chondrosarcoma, 1032a-c
 — colloid cancer, 1048
 — cysts, 1028-1031, 1043
 — duct cancer, 1050
 — duct papilloma, 1049
 — eczema of nipple, 1051-1058

- Breast, epithelioma of nipple, 1034
 — hypertrophy, 1025
 — lupus, 1027
 — male breast, 1044, 1049, 1059
 — male breast, duct papilloma, 1049
 — male breast inflammation, 1059
 — male breast scirrhus, 1044
 — mammary abscess, 1055
 — nipple, 1034, 1051-1058
 — nipple, eczema, 830, 1051-1058
 — nipple, epithelioma, 1034; *see also* under Nipple for further references
 — operation, 1360a
 — Paget's disease, 830, 1051-1058
 — proliferous cysts, 1030
 — sarcoma, 1032-1033
 — scirrhus, 1035-1037, 1039-1044, 1046a-b, 1052-1054, 1057, 1058
 — scirrhus, atrophic, 1039
 — scirrhus, fungating, 1040
 — scirrhus, male breast, 1044
 — serocystic disease, 1028, 1029
 Broad ligaments, 967
 Bromide rash, *see* Rashes
 Bronchial glands, malignant growth, 270, 271
 Bronchiectasis, 238-240
 Bronchitis, capillary, 237
 Bronchocele, *see* Goitre
 Bubo, 1129-1131b
 Bullæ, 769, 802a, 803, 804c-d, 806, 807, 813
 Bullet-wounds, 1345
 Burns, 1062-1064c
 Burnett's fluid, *see* Poisoning
 Bursa patellæ, 148; *see also* Intermuscular cysts
- CACHEXIA strumipriva, 574a, 575-578; *see also* Myxœdema
 Calculus, biliary, 1353
 — renal, 631, 632, 639
 — vesical, 1307, 1308
 Cancrum oris, 293, 294, 1078a
 Carbonic acid, *see* under Poisoning
 Carbonate of potash, *see* under Poisoning
 Carcinoma, adeno-, *see* Glandular
 — axilla, 1258
 — bile ducts, 503
 — breast, *see* under Breast
 — chimney-sweep's, 940-943
 — colloid, 1261-1262
 — gall-bladder, 511
 — glandular (general pathology), 1258-1263
 — heart, 165
 — kidney, 639, 640
 — larynx, 230
 — liver, 499-503, 511
 — meninges, 711
 — œsophagus, 358-359
 — penis, 959, 960
 — peritoneum, 366
 — pharynx, 357
 — rectum, 456, 457
 — serotum, 940-944
 — stomach, 398, 399, 1263
 — thyroid, 1259
 — tongue, *see* Tongue
 — uterus, 995, 1001
 — wax refiner's, 1276, 1277
See also under various organs
 Caries, 1106
 Carotid artery, ligature, 691
 Caseous glands, 515
 Cavities in lung, 267-268a
 Cellulitis, 1051, 1066, 1067, 1075, 1081a, 1082a, 1083a
 Cephalhydrocele, 58
 Cerebellum, 697, 708, 925
 Cerebral arteries, 184, 200
 Cervix uteri, 976, 977, 981, 1002, 1003; *see also* Uterus
 Charcot's disease, 86-94, 96
 Cheek, epithelioma, 1265a, 1268, 1270, 1271
 — sarcoma, 1214-1215, 1221-1222
 — syphilis, 1122
 Chimney-sweep's cancer, *see* under Carcinoma
 Chin, syphilis, 1119
 Chiropompholyx, 814a
 Chloral rash, *see* Rashes
 Chloroma, 1244
 Cholera, 408-410
 Chondroma, 34, 35, 37, 38
 Chondrophytes, 76
 Chyle, 1354
 Cirrhosis, liver, 394, 480, 481, 483, 485
 — lung, 242
 — stomach, 394
 Cleft palate, 1329-1329b
 Clitoris, 1007
 Clots in heart, 156-160
 Clubbed fingers, 179
 Club-foot, 1342l
 Coccygeal cyst, 1304, 1328a-b, 1336
 Colitis, 427
 Colloid carcinoma, *see* under Carcinoma
 Condylomata, 453, 454
 Congenital, *see* Malformation, Paralysis, and Syphilis
 Conjunctiva, *see* Eye
 Contraction ring, 1004

- Cord, *see* Larynx, Spinal Cord, and Umbilicus
 Cornea, *see* Eye
 Corpus luteum, 1020-1022
 — striatum, *see* Brain
 Cranium, 2, 31, 58
 — cephalhydrocele, 58
 — enlargement, 2
 — fracture, *see* Fracture
 — sarcoma, 580
 — syphilis, 31, 57
 Cretin, 538-540, 570-573, 1177, 1178
 — after thyroid feeding, 540
 — foetal, 1325a
 Croup, 220-224
 Crura, *see* Brain
 Crush, 1346-1347
 Cutaneous horn, 823a
 Cut-throat, 234, 1357
 Curvature, spinal, 131
 Cyanosis, 177, 178, 181
 Cysts around joints, *see* Joints
 — breast, 1028-1031, 1043
 — coccygeal, 1304, 1336
 — dermoid, 1305, 1305a
 — diaphragm, 1277
 — hydatid, 166
 — hygroma, 1301-1302
 — intermuscular, *see* Joints
 — lingual, 318
 — neck, 1299, 1300, 1303
 — ovarian, 961
 — parovarian, 967
 — peritoneal, 365
 — placenta, 1013
 — proliferous (breast), 1030
 — renal, 633-635
 — retinal, 755
 — sebaceous, 1305
 — sublingual, 317, 318a
 — thigh, 1298
 — thyroid gland, *see* under Goitre
 — tubo-ovarian, 963
- DEGENERATION, caseous, 515
 — cystic, of kidney, 635
 — fatty, of muscles, 134
 — fatty, of heart, 155
 — fatty, of liver, 478-480, 485
 — pulpy, 73-75
 Dermatitis herpetiformis, 773a-d
 Dermoid cyst, 1305, 1305a
 Diagnosis, facial, adenoid vegetations, 1373
 — facial, exophthalmic goitre, 1372
 — facial, idiocy, 1370
 — facial, osteitis deformans, 1371
 Diaphragm, 134, 1297
 Diphtheria, stomach, 402, 402a
 Diphtheria, tongue, 302, 303
 — tonsils and larynx, 216, 220-226, 726c
 Dipygus parasiticus, 1342a
 Dislocations, 114-120
 — congenital (hip), 119
 — hip, 117-119
 — idiopathic, 120
 — lens, 752
 — shoulder, 114-116
 Drug rashes, 915-924; *see* Rashes
 Duct cancer, 1050
 — papilloma, 1049
 Duodenum, hæmorrhage, 406a
 Dura mater, *see* Meninges
 Dysentery, 412-426
- EAR, 756, 757, 1328d, 1338-1341
 Ecchymoses, bladder, 643
 — heart, 162, 163, 180
 — face, 1347
 — pericardial, 180
 Echinococcus, 505
 Ectopia vesicæ, 1312-1313a
 Ectopic gestation, 1016-1019
 Ectropion, *see* Eye
 Eczema, 775b-c, 780-782, 830
 — chronic, 830
 — circinatum, 782
 — epidemic, 780a
 — nipple, 830, 1051-1058
 — pustular, 775b-c, 780
 Elbow, synovial cyst, 87
 Electrolysis (uterus), 1005
 Elephantiasis, 862, 864-864b, 936-938
 Emboli, 195, 196
 Embolic aneurysms, 195-199
 Embolism of lung, 255, 256
 Emphysema, 241
 — interstitial, 241
 Empyema, 236
 Encephalitis, *see* Brain
 Encephalocele, *see* Brain
 Enchondroma, 34, 35, 37, 38, 1187-1189
 Endocarditis, 155, 167-169, 175
 — infective, *see* Ulcerative endocarditis
 — rheumatic, 167, 172
 — ulcerative, 169-171, 174, 175, 1082a, 1206
 — *See* also Valvular diseases
 Enteric fever, 430-441, 1000
 Enteritis, 406
 Epiglottis, 217
 Epiphysitis, 64
 Epispadias, 1315
 Epithelioma (general pathology), 1265-1279

- Epithelioma, back, 1280
 — cheek, 1265a, 1268, 1270, 1271
 — eyelid, 1279
 — hand, 1272, 1274
 — head, 1278a
 — larynx, 230, 1271a
 — leg, 1277a
 — lip, 287, 1265-1267
 — melanotic, 1281, 1282
 — neck, 1271a
 — nipple, 1034
 — nose, 1273
 — penis, 959, 960
 — temple, 1278
 — tongue, *see* under Tongue
 — ulcer, 1277b
 — wax refiner, 1276, 1277
See also Carcinoma
- Erosions, hæmorrhagic (stomach), 352, 354
 Eruptions (skin), 762, 773-775, 865, 897, 905, 906
 — parasitic, 908-914; *see also* Rashes
 Erysipelas, 1066, 1067, 1075, 1081a
 Erythema, 759-761b, 763-772
 — circinatum, 760-764
 — circumscriptum, 763
 — iris, 924a
 — marginatum, 759
 — multifforme, 767-769
 — nodosum, 761-761b
 — serpens, 765, 766
 — tuberculatum, 772
 — vesiculatum, 770, 771
 Exanthematous rashes, *see* Rashes
 Excision of joints, *see* Joints
 Exophthalmic goitre, *see* Goitre
 Exophthalmos, 763a; *see also* Goitre and Proptosis
 Extrauterine gestation, 1016-1019
 Eye, abscess, lachrymal, 740
 — congenital malformations, 1331, 1331a, 1337
 — conjunctiva, 736, 739
 — cornea, 736a, 737, 742
 — ectropion, 753
 — foreign body, 754
 — glioma, 1211
 — intra-ocular tumour, 744
 — iris, 749, 749a
 — lachrymal gland, 740, 741
 — lens, 752
 — lid, 741, 753, 1124, 1124a, 1212, 1279
 — melanotic sarcoma, 745-748, 1251-1253, 1255
 — pupil, 738
 — retina, 755
 — sarcoma, 744, 751
- FACIAL diagnosis, 1370-1373
 — paralysis, 724
 Fallopian tubes, 963, 970
 False passages (urethra), 957
 Fatty degeneration, muscles, 134
 — heart, 155
 — liver, 478-480, 485
 Favus, 908-911
 Feet, *see* under Malformation
 Femur, *see* Fracture
 Fever rashes, 887-900; *see* Rashes
 Fibrinous polyp (uterus), 990
 Fibrocellular tumours, 1180-1185
 Fibroid, *see* Fibroma
 Fibroma (general pathology), 1180-1185
 — ovary, 964
 — recurrent, 1223-1230, 1240
 — uterus, 983-989
 — vagina, 982
 Fibrosis of lung, 242
 Fibula, *see* Fracture
 Finger, hammer, 146
 Fissure, cervix uteri, 976, 977, 1002, 1003
 Fistula, branchial, 1338
 — recto-vesical, 647
 — sacral, 1335l
 — urinary, 956
 Fracture, 103-112, 124-130, 1342m, &c.
 — compound, 22
 — depressed (skull), 104
 — femur, 103, 107-109
 — fibrous union of, 107-109
 — fibula, 112
 — humerus, 110
 — leg, 112
 — olecranon, 111
 — Pott's, 1342m
 — skull, 104, 689, 693
 — spine, 124-130, 717-719
 — spontaneous, 103
 — tibia, 105, 106, 112, 1342m
 — ununited, 110
 Frost-bite, 1087, 1088
 Fungating ulcer, 1105b
 Fungus disease, Indian, 1106b
- GALL bladder, 509-511
 — stone, 1353
 Gangrene, 1078-1103
 — cancrum oris, 293, 294, 1078a
 — cellulitis, 1081a, 1082a, 1083a
 — dry, 1086a-e, 1088, 1093, 1094
 — frost-bite, 1087, 1088
 — hospital, 1083a-f
 — idiopathic, 1090, 1091
 — lung, 246-248
 — moist, 1095
 — necrosis, 1078

- Gangrene, noma, 1081; *see* Vagina
 — phagedæna, 1078-1080, 1082, 1081a;
 see Phagedæna
 — Raynaud's, 1096-1103
 — slough, 1084
 — symmetrical, 1096-1103
 — traumatic, 1089
 Gastrostomy, 400, 401
 Genu valgum, 24a-b
 Glanders, 214, 903-904, 907
 Glioma, 703, 1211
 Glossitis, 298
 Goitre, 532-537, 543-569, 573-575, 579-
 584, 601
 — cancerous, 537, 582-584
 — cystic, 534, 543-557, 560-564, 579,
 580
 — exophthalmic, 535-536, 569, 600
 — parenchymatous, 558, 559, 565-568,
 573
 See also Myxœdema and Cachexia
 strumipriva
 Gout, 95, 935, 1158-1161a
 Grafting, skin, 1074, 1075
 — sponge, 1076
 Granular kidney, *see* under Kidney
 Graves' disease, *see* Goitre
 Grease, 901-902
 Greasy warts, 886
 Gumma, liver, 495
 — muscle, 147
 — skin, 1134
 — testicle, 928
 — tongue, 344, Pl. ix. 2
 Gums, hypertrophy, 288, 289
- HÆMOPHILIA**, 82, 83, 1179
 — joints, 82, 83
 — skin, 1179
 Hæmorrhage, subcutaneous, 1065; *see*
 also under various organs, Heart, &c.
 Hammer-finger, 146
 Hand, congenital deformity, *see* Mal-
 formation
 — enchondroma, *see* under Enchon-
 droma
 — epithelioma, 1272, 1274
 Hare-lip, 1329, 1329a, 1330
 Heart, abscesses, 161
 — aortic disease, 172-175
 — atrophy, 154
 — carcinoma, 165
 — congenital malformation, 177-179
 — dilatation, 176
 — disease, 181a
 — ecchymoses, 162, 163, 880
 — fatty, 155
 — hæmorrhages, 162, 163, 180
- Heart, hydatid, 166
 — mitral disease, 181, 183
 — purpuric hæmorrhages, 162, 163,
 180
 — thrombus, 156-160
 — tricuspid disease, 181
 See also Pericardium, Endocarditis, &c.
 Heberden's nodes, 875-878
 Hemiatrophy, tongue, 296, 297
 Hemiplegia, tongue, 296
 Hepatic duct, 508
 Hereditary syphilis, *see* Syphilis
 Hermaphrodite, 1315e
 Hernia, 460-471
 — cerebri, 710
 — diaphragmatic, 470, 471
 — encysted, 463
 — femoral, 463a
 — fossa intersigmoidea, 460
 — funicular, 459, 463
 — gall-bladder, 509
 — inguinal, 464
 — interstitial, 468, 469
 — obturator, 461, 462
 — ovary, 464
 — strangulated, 460, 509
 — umbilical, 465, 466
 — ventral, 467, 467a
 Herpes, 795-800, 855
 — frontalis, 797
 — symmetrical, 799-800
 — zoster, 796, 798, 855
 Hip, ankylosis, 63a-b, 66
 — Charcot's, 96
 — dislocations, 117-119
 — disease, 63a-b
 — pyæmia, 61, 62
 — rheumatic disease, 101
 — rheumatism, acute, 65
 — tuberculosis, 63a-b
 Horn, cutaneous, 823a
 — sebaceous (penis), 959a-b
 Hospital gangrene, 1083a-f
 Humerus, *see* Fracture
 Hutchinson's teeth, 347-349
 Hydatids, heart, 166
 — liver, 504
 — spleen, 530
 Hydrocephalus, 654
 Hydrochloric acid, *see* Poisoning
 Hydrosalpinx, 970
 Hygroma, 1301-1302
 Hymen, imperforate, 972, 974
 Hypospadias, 1315l
 Hypertrophy, breast, 1025
 — cervix uteri, 981, 1002
 — gums, 288, 289
 — lip, 290
 — muscles, 1350

- Hypertrophy, tongue, 295
 Hysteria, 1358
- ICHTHYOSIS, skin, 819-822a; *see also*
 Tongue
- Idiocy, 1370
 Idiopathic gangrene, 1090, 1091
 Imperforate anus, 450, 451
 ——— hymen, 972, 974
 Indian fungus disease, 1106b
 Infantile paralysis, 143, 144, 722, 723
 Infarcts, lung, 249, 250a
 ——— kidney, 623, 624
 ——— pyæmia, 250a
 ——— spleen, 526, 531
 Intermuscular cysts, *see* Cysts of joints
 under Joints
- Intestines, 406-448
 ——— catarrh, 406
 ——— cholera, 408-410
 ——— duodenum, 406a
 ——— dysentery, 412-426
 ——— enteric fever, 430-442
 ——— enteritis, 406
 ——— faecal staining, 447
 ——— fibromata, 448
 ——— hæmorrhage, 406a, 424a
 ——— leucocythæmia, 411
 ——— lymphatic glands, 408-410
 ——— obstruction, 1353
 ——— solitary glands, 408-410
 ——— tuberculosis, 443-446
 ——— typhoid fever, 430-442
 ——— ulcerative colitis, 427
 ——— ulcers, 407, 412-429, 443-446, 452
 ——— ulcers, colitis, 427
 ——— ulcers, distension, 428
 ——— ulcers, dysenteric, 412-426
 ——— ulcers, follicular, 407
 ——— ulcers, syphilis, 452
 ——— ulcers, tubercular, 443-446
 ——— ulcers, typhoid, *see* above
 ——— ulcers, vermiform appendix, 429
 Intussusception, 475-477
 Involution of uterus, 1004
 Iodide rash, *see* Rashes
 Iris, *see* Eye
 Itch, parasites, 912
- JAW, lower, 59, 1219, 1220, 1305a
 ——— sarcoma, 1219, 1220
 ——— necrosis, 1078
 ——— upper, antrum, 1216
 ——— upper, sarcoma, 1213, 1216, 1218
- Joints, 61-101
 ——— ankle, tubercular disease, 1105c
 ——— ankylosis, 63a, 63b, 66
- Joints, arthrectomy, 78, 78a
 ——— arthritis, acute, 69-72, 86
 ——— arthritis, rheumatoid, 67, 88, 97, 101
 ——— arthritis, tubercular, 73-75, 1105c
 ——— articular cartilages, 77
 ——— artificial, 66
 ——— Charcot's disease, 88-94, 96
 ——— chondrophytes, 76
 ——— cysts, 79-81, 84-87, 98-101
 ——— dislocations, 114-120
 ——— elbow, cyst, 87
 ——— epiphysitis, 64
 ——— excision, 78, 78a
 ——— gout, 95
 ——— hæmophilia, 82, 83
 ——— hip, 61-62, 63a-b, 65, 66, 96, 101,
 117-119
 ——— injuries, 114-120
 ——— intermuscular cysts, *see* Cysts of
 joints
 ——— knee, 68-79, 81-83, 89-94, 97-100
 ——— pulpy degeneration, 73-75
 ——— pyæmia, 61, 62, 70
 ——— rheumatism (acute), 65
 ——— rheumatoid arthritis, 67, 88, 97, 101
 ——— shoulder, 86, 114-116
 ——— synovial cysts, *see* Cysts
 ——— synovitis, 61, 62, 65, 68, 69
 ——— tuberculosis, 73-75, 1105c
 ——— ulceration (articular cartilages), 77
- KAPOSI'S disease, 833a
 Keloid, Addison, 824-826; *see also* Mor-
 phœa and Scleroderma
 ——— Alibert, 848-858
 ——— true, 848-858
- Kidneys, 603-642, 507a
 ——— calculus, 631, 632, 639
 ——— carcinoma, 639, 640
 ——— cystic degeneration, 635
 ——— cysts, 633-635
 ——— granular, 612-615
 ——— granular, large, 612, 613
 ——— granular, small, 614, 615 (?)
 ——— hæmorrhage, 636-638
 ——— infarct, 623-624
 ——— mottled, 611
 ——— pyelitis, 621
 ——— pyonephrosis, *see* Suppurative ne-
 phritis
 ——— scarlatinal, 608-610
 ——— tubercle, 625-630
 ——— tumours, 641
 ——— white, large, 617; *see also* Chronic
 parenchymatous nephritis
 ——— white, small, 607
See also Nephritis
- Knee, 68-79, 81-83, 89-94, 97-100

- LABIA, 1080
 Lachrymal gland, *see* Eye
 Laryngitis, 211-213, 219
 Larynx, 211-213, 216-225, 227-234
 — epithelioma, 230, 1271a
 — inflammation (acute), 211-213, 219
 — œdema, 211, 217, 218
 — papilloma, 229
 — syphilis, 227, 228
 — tumour, 231
 — ulceration, 213
 See also Croup and Diphtheria
 Lateral curvature, 131
 Lead poisoning, 1163-1165
 Leg, epithelioma, 1277a
 — fracture, 112
 Lens, *see* Eye
 Leonine face, 1350
 Leprosy, 31a, 1114 1114a
 Leucocythæmia, 160, 411, 1349
 Leukæmia, *see* Leucocythæmia
 Leukoma, *see* Tongue under Leukoplakia
 Leukoplakia, *see* Tongue
 Lichen, 784-789
 — pilaris, 788a
 — ruber, 786, 786a, 789
 — planus, 209, 784, 786a
 Lid, *see* Eye
 Linæ transversæ, 775a
 Lipoma, 1190, 1193c, 1197
 — diffuse, 1192, 1193a-b, 1197
 Lips, 287-290, &c.
 — epithelioma, 287, 1265-1267
 — hypertrophy, 290
 — syphilis (primary), 1120-1121
 — tubercular ulcer, 1104, 1105
 Liver, 478-507a
 — abscesses, 484
 — accessory lobule, 506
 — acute yellow atrophy, 482
 — amyloid, 495
 — carcinoma, 499-503, 511
 — cardiac, 486-487
 — cirrhosis, 480, 481, 483, 485
 — echinococcus, 505
 — fatty, 478-480, 485
 — fatty cirrhosis, 485
 — gumma, 495
 — hydatid, 504
 — hypertrophic cirrhosis, 481
 — melanotic sarcoma, 497, 498
 — nævus, 496
 — nutmeg, 486
 — pylephlebitis, 488-492
 — rickets, 493, 494
 — rupture, 507, 507a
 — sarcoma, 502a
 — surface markings, 493, 494
 Locomotor ataxia, 1072
 Lung apoplexy, 257-260
 — atelectasis, 1332
 — bronchiectasis, 238-240
 — capillary bronchitis, 237
 — cavity, 267-268a
 — cirrhosis, 242
 — embolism, 255, 256
 — emphysema, 241
 — fibrosis, 242
 — gangrene, 246-248
 — growths, cartilaginous, 269
 — growths, sarcoma, 271, 273, 274
 — infarcts, 249, 250a
 — new growths, 269, 271-274
 — pneumonia, 243-245
 — pyæmia, 250-254
 — pyæmic infarcts, 250a
 — spanæmic, 261
 — syphilis, 275
 — tubercle, 262-268a
 Lupus erythematosus, 835a-b, 1113
 — exedens and non-exedens, 835, 836
 — lymphaticus, 520, 521
 — papillary, 820, 821
 — vulgaris, 739, 1027, 1108-1112a
 Lymphadenoma, 516
 Lymphangiectasis, 519
 Lymphangioma, 319-321a
 Lymphangitis, 1066
 Lymphatic glands, 515-517
 — glands, caseous, 515
 — glands, sarcoma, 517
 — vessels, 518, 520, 521
 — vessels, obstruction of, 1350a
 Lymphosarcoma, 1245-1247, &c.
 — mediastinal, 152-153, 206, 270, 271

 MADURA disease, 1106b
 Male breast, 1044, 1049, 1059
 Malformations (congenital), 177-179,
 1311-1342
 — bladder, 1312-1313a
 — brain, 1331, 1331a, 1334
 — breasts, 1318-1322a
 — ears, 1328, 1338-1341
 — eye, 1331, 1331a, 1337
 — face, 1331b
 — feet, 1311
 — fistula, 1335l
 — hands, 1311, 1326-1328, 1330
 — heart, 177-179
 — limbs, 1342
 — lip, 1329-1330
 — palate, 1327e, 1329
 — parasite, 1342a
 — penis, 1315, 1315l
 — peritoneum, 361l
 — pteromalleus, 1327c

- Malformations (congenital), spine, 1335, 1335a
 — viscera, 1317
 Malignant growths, bone, 39, 42, 44-49, 52-56, 59, 123
 — mediastinum, 152, 153
 See also Carcinoma, Sarcoma, and special organs.
 Mammary abscess, 1055
 Mater, dura and pia, *see* Meninges
 Maxilla, lower, *see* Jaw
 — upper, *see* Jaw
 Measles, 178, 1081
 Median nerve, 729-733
 Mediastinal growth, 152, 153, 206, 270, 271, 1245, 1246
 Melanosis, 1250
 Melanotic epithelioma, 1281, 1282
 — sarcoma, *see* Sarcoma
 — wart, 1254
 Meninges, angioma, 702
 — carcinoma, 711
 — hæmorrhage, dura mater, 655, 714
 — hæmorrhage, arachnoid, 655a, 656
 — spinal, 714, 715
 Meningitis, 682-688, 695, 715
 — tubercular, 695
 Meningocele (traumatic), 713a, 1334
 Menstruation, 974, 975
 Mercurial poisoning, 1166
 Microcephalus, 1331, 1331c
 Mineral acids, *see* Poisoning
 Mitral disease, 155, 158, 167-169, 173, 181, 183, 257
 — disease, pulmonary apoplexy, 257
 Moist gangrene, 1095
 Mole, hairy, 881-886
 — pigmented, 882a
 Molluscum contagiosum, 860-861a
 — fibrosum, 1186, 1186a
 — simplex, 859
 Morbus cordis, *see* Heart
 Morphœa, 824-830, 832, 832a; *see* also Keloid and Scleroderma
 Muscle, 134-147
 — fatty, 134
 — gumma, 147
 — hypertrophy, 1350
 — myositis ossificans, 139, 140
 — rupture of, 144a
 — sarcoma, 1236, 1237, 1239
 — serratus magnus, 726a-b
 Mycetoma, 1106b
 Myelitis, *see* Spinal cord
 Myositis ossificans, 139, 140
 Myxœdema, 542, 574a, 576-578, 585-585d, 999, 1167-1176
 — male, 1167-1169; *see* also Cachexia strumipriva and Thyroid gland
 NÆVUS, 208-210, 1200-1207
 — congenital, 1200a, 1201, 1202, 1205
 — degenerated, 1283
 — face, 209-210
 — liver, 496
 — multiple, 1207
 — tongue, 323a
 Nails, absence, 843
 — atrophy, 842, 842a
 — onychia, 914
 Nasopharynx, 353-356
 Neck, cysts, 1299, 1300, 1303
 Necrosis (bone), 11-14, 18, 20-22, 28
 — gangrene, 1078
 Nephritis, acute, 603
 — chronic parenchymatous, 617-619
 — interstitial, 604, 606, 607, 614
 — scarlatinal, 608-610
 — suppurative, 605, 616, 620, 622
 — tubercular, 625-629, 630
 Nerves, 724-735
 — facial, 724
 — injury, 729-734
 — median, 729-733
 — neuritis, 725-728
 — radial, 734
 — sympathetic, 724a-b
 — tibial, 735
 — tumour, 735
 — ulnar, 733a
 Neuritis, 725-728
 — alcoholic, 725
 — chronic, 727-728
 — peripheral, 726
 Nigrities linguæ, 304a
 Nipple, eczema, 830, 1051-1058
 — epithelioma, 1304
 — Paget's disease, *see* Eczema
 — supernumerary, 1318-1322a
 — syphilis, primary, 1117, 1118
 — syphilis, tertiary, 1135
 Nitrate of silver rash, 924
 Nitric acid, *see* Poisons
 Nitro-hydrochloric acid, *see* Poisons
 Nodes, 30, 57
 — Parrot's, 57
 Noma, 1081
 Nose, 285-286
 — epithelioma, 1273
 — morbid anatomy, 286
 — normal anatomy, 286
 — rhinoplasty, 285, 285a
 — syphilis, 1152, 1152a, 1154, 1155
 Nutmeg liver, 486
 OBSTRUCTION, intestinal, 1353; *see* under Intestines
 — lymphatic, *see* under Lymphatics

- Obstruction, veins, *see* under Vena cava and Veins
- Œdema, epiglottis, 217
— larynx, 211, 217, 218
- Œsophagus, 352, 356a, 358-359, 360, 361, 374, 375
— cancer, 358-359
— congestion, 352
— poisoning, 360, 361, 374, 375
— pouches, 356a
- Olecranon, fracture, 111
- Onychia mycosis, 914
- Onychogryphosis, 1311
- Operations, arthrectomy, 78, 78a
— breasts, 1360a
— excision of joints, 78, 78a
— gastrostomy, 400, 401
— hare-lip, 1329a
— osteotomy, 24a-b
— ovariectomy, 1343
— plastic, 285, 285a, 1312c, 1315l, 1360, 1360a; *see* also Grafting
— rhinoplasty, 285
— Syme's, 1071
— Tagliacotian, 285
— thyroid gland, 533a, 543, 544, 546, 547, 574, 575; *see* also Grafting
- Optic thalamus, *see* Brain
- Orbital aneurysm, 191a-b
— sarcoma, 1217
- Ossifying sarcoma, 1241
- Osteitis deformans, 15-17, 1371
- Osteoma, 32, 33
- Osteomyelitis, 5-8
- Osteosarcoma of lung, 273, 274, 1241
- Osteotomy, *see* Operations
- Otitis media, 756
- Ovariectomy, *see* Operations
- Ovary, 961-964
— cyst, 961
— fibroma, 964
— tubo-ovarian cyst, 963
- Ovum, 1012, 1013, 1016-1022
- Oxalic acid, *see* Poisoning
- PAGET'S disease, 830, 1051-1058; *see* also Nipple
- Palate, 340, 342
— cleft, 1327e, 1329
- Pancreas, 512
- Papilloma, anus, 455
— bladder, 650
— skin, 1209
— tongue, 322
— vocal cord, 229
- Paralysis, congenital, 143d
— diphtheritic (?), 726c
— facial, 724
— infantile, 143, 144, 722-723
- Paralysis, median nerve, 729-733
— pseudo-hypertrophic, 145
— radial nerve, 734
— sefratus magnus, 726a-b
— sympathetic nerve, 724a-b
— ulnar, 733a
- Parametritis, 993, 994
- Paraplegia, spastic, 720, 721
- Parasites (skin), 908-914
— acarus, 912
— favus, 908-911
— itch, 912
— onychia mycosis, 914
— ringworm, 913
— scabies, 912
— tinea circinata, 914
— tinea favosa, 908-911
— tinea tonsurans, 913
— trichophyton tonsurans, 913
— congenital malformation, 1342, 1342a
- Parenchymatous nephritis, *see* Nephritis and Kidney
- Parotid tumours, 1188, 1189
- Parovarian cyst, 967
- Parrot's nodes, 57
- Patella, bursa, 148
- Pearl ash, *see* Poisoning
- Pemphigus, 769, 801-814; *see* also Bullæ
— hæmorrhagicus, 805
— pruriginosus, 814
- Penis, 958-960, 1081a, 1315, 1315l
- Perchloride of mercury, *see* Poisoning
- Perforating ulcer, 1072-1072b
- Pericardial ecchymoses, 180
— hæmorrhages, 180
— new growth, 152, 153
— tuberculosis, 164
- Pericarditis, 149-151
- Perimetritis, 994a-997, 1000
- Periostitis, 18-19
- Periosteal sarcoma, 1234
- Peripheral neuritis, 726
- Peritoneum, 361l, 362-368
- Peritonitis, 362-364
- Phagedæna, 1077a, 1078-1080, 1082
— of labia, 1080
— of scalp, 1077a
- Pharynx, adenoids, 1373
— cancer, 357
— stenosis, 343
— tumour of, 231
- Phlegmonous erysipelas, 1075
- Phthiasis, 924a
- Pigmentation of skin, 1352; *see* also Addison's disease
- Pigmented mole, 882a
- Pineal gland, *see* Brain
- Pityriasis rubra pilaris, 773e

- Placenta, 1013
 Plastic operation, 285, 285a, 1312c, 1315l, 1360, 1360a; *see also* Operations
 Pleurisy, 235, 236
 Plugging of arteries, 184, 200
 Pneumonia, 243-245
 Poisoning, ammonia (stomach), 386, 387
 — arsenic (uterus), 998
 — bichromate of potash (stomach), 393
 — Burnett's disinfecting fluid (stomach), 391
 — carbolic (stomach), 390; (tongue), 292
 — carbonate of potash (stomach), 392
 — hydrochloric acid (stomach), 384
 — lead, 1163-1165
 — mercury, 1166
 — nitric acid (stomach), 379, 382, 385; (urine), 642
 — nitro-hydrochloric acid (stomach), 382a
 — oxalic acid (stomach), 374-377, 403
 — pearl ash (stomach), 392
 — perchloride of mercury (stomach), 388, 389
 — prussic acid (stomach), 378
 — sublimate (stomach), 388, 389
 — sulphuric acid (stomach), 383, 385
 Polymastia, 1318-1322
 Polypi, ear, 757
 — fibrinous, 990
 — stomach, 373, 397
 — uterus, 978-980, 990
 Pons, *see* Brain
 Post-mortem changes, 1355, 1356
 Pott's fracture, 1342m
 Pouch, cesophageal, 356a
 — vesical, 649
 Primary syphilis, *see* Syphilis
 Progressive muscular atrophy, 136-138
 Prolapse of vagina, 1008
 Proliferous cyst, 1030
 Proptosis, 1331, 1331a; *see also* Exophthalmos
 Prostate, 1238
 Prurigo (Hebra), 783, 784
 Prussic acid, *see* Poisoning
 Pseudo-hypertrophic paralysis, 145
 Psoas abscess, 135
 Psoriasis, skin, 790-794
 — tongue, 344, Pl. vi. 3
 Pteromalleus, 1327c
 Pulmonary artery, 176, 183, 276-279, 281
 Pulp disease, 73-75
 Pupil, *see* Eye
 Purpura hæmorrhagica, 62, 162, 163, 180, 680, 866, 867, 870-874
 Purpura hæmorrhagica, rheumatica, 868
 Pyæmia, infarct, 250a
 — joints, 61, 62, 70
 — kidney, 171
 — liver, 488-492
 — lung, 250-254
 — vein, 202
 Pyelitis, 621
 Pylephlebitis, 488-492
 Pyonephrosis, *see* Suppurative nephritis
 RADIAL nerve, 734
 Radius, absence of, 1330
 Rashes, bromide, 916, 923, 923a
 — chloral, 919, 920
 — drugs, 915-924
 — fevers, 887-900
 — iodide, 915, 917-918, 921, 922
 — silver, 924
 — syphilis, 1120-1121, 1128, 1144; *see also* Rupia and Syphilis, &c.
 — typhoid, 889-900
 — uncertain, 901-902
 — vaccination, 893-895
 — varicella gangrenosa, 887-892, 896
 — variola, *see* under Variola
 Raynaud's disease, 1096-1103
 Recto-vaginal fistula, 451
 Rectum, 450-452, 456, 457
 — cancer, 456, 457
 — distension, 450, 451
 — stricture, 456, 457
 — syphilis, 452
 — ulcer, 452
 Recurrent fibroid, 1223-1230, 1240
 Renal, *see* Calculus and Kidney
 Retina, *see* Eye
 Retroversion of uterus, 992
 Rheumatism, acute, joints, 65
 — endocarditis, 167, 172
 — nodes, *see* under Nodes
 — nodules, 875-878
 — purpura, 868
 Rheumatoid arthritis, 67, 88, 97, 101
 Rhinoplasty, 285, 285a
 Rickets, 4m, 24, 25, 493, 494; *see also* Achondroplasia
 — scurvy, 26, 27
 Ring, Bandl's, 1004
 — contraction, 1004
 Ringworm, 913; *see also* Tinea and Trichophyton
 Rodent ulcer, 1285-1296
 Rudimentary limbs, 1342
 Rupia, 1137-1138, 1149
 Rupture, artery, 201
 — intestines, *see* Herain
 — muscles, 144a

- SACRO-COCYGEAL tumour, 1304, 1328a, 1328b, 1336
- Sarcoma, antrum, 1216
- back, 1242a
- brain, 708, 712
- bone, 42, 44-49, 52-55, 59
- breast, 1032-1033
- cheek, 1214-1215, 1221-1222
- chondro, testis, 929-929b
- eye, 744-748, 751, 1211, 1251-1253, 1255
- eyelid, 1212
- glioma, 1211
- jaw, lower, 1219, 1220
- jaw, upper, 1213, 1216, 1218
- liver, 502a
- lung, 271, 273, 274
- lymphatic glands, 517
- lymphosarcoma, 1245-1247
- maxilla, upper, 1213, 1216, 1218; *see* under Jaw
- melanotic, abdomen, 1249
- melanotic, eye, 745-748, 1251-1253, 1255
- melanotic, liver, 497, 498
- melanotic, mouth, 1253a
- melanotic, wart, 1254
- muscles, 1236, 1237, 1239
- orbit, 1217
- ossifying, 1241
- osteo-, 1241
- periosteal, 1234
- prostate, 1238
- recurrent fibroid, 1223-1230, 1240
- scapula, 1233a
- spindle-celled, 1223-1227
- thigh, 1235
- ulcer, 1233, 1233b
- ulcerating, 1233
- uterus, 1231
- withering, 1232, 1232a
- See also special organs*
- Scabies, 912
- Scalp, 1077a
- Scarlatina, 608-610
- Scars, 924a, 1009, 1063-1064c
- vagina, 1009
- syphilitic, 1150
- tongue, *see* under Tongue
- Scirrhous, *see* under Breast
- Sclerema, 833
- Scleroderma, 824-833; *see also* Morphœa and Sclerema
- Sclerosis, bone, 9, 10, 122
- brain, 700, 701
- Scorbutus, 879, 880; *see also* Scurvy
- Scrofuloderma, 837
- Scrotum, 935-944
- carcinoma, 940-944
- Scrotum, chimney-sweep's cancer, 940-943
- elephantiasis, 936-938
- erysipelas, 1081a
- gangrene, 1081a
- gout, 935
- topi, 935
- ulceration, 939
- Scurvy, 879, 880
- rickets, 26, 27
- Sebaceous cyst, 1305
- horn (penis), 959a-b
- Serocystic disease, breast, 1028, 1029
- Serratus magnus, paralysis, 726a-b
- Shoulder, 86
- dislocation, 114-116
- Skin, 759-924
- grafting, 1074, 1075
- pigmentation, 1352; *see also* Addison's disease
- Skull, *see* Cranium
- Slough, 1084
- Softening, brain, 670, 671
- Sore, *see* Syphilis
- Spanæmic lung, 261
- Spastic paraplegia, 720, 721
- Spina bifida, 1335, 1335a
- Spinal cord, 714-723
- crush, 719
- fracture, 717-719
- infantile paralysis, 722, 723
- meninges, 714, 715
- myelitis, 716-718
- paraplegia, 720, 721
- softening, 716-718
- Spine, 122-131
- curvature, 131
- fracture, 124-130
- lateral curvature, 131
- malignant disease, 122
- sclerosis, 122
- Spleen, 523-531
- amyloid, 523
- capillary phlebitis, 525
- enlarged Malpighian bodies, 524
- hydatid, 530
- infarcts, 526, 531
- tubercle, 527-529
- Sponge grafting, 1076
- Stomach, 369-403
- cancer, 398, 399, 1263
- congestion, 371, 372
- digestion, 369
- dilatation, 390
- diphtheria, 402, 402a
- engorgement (cirrhosis), 394
- erosions, 352, 394
- gastrostomy, 400, 401
- hæmorrhagic erosions, 352, 394
- poisons, *see* Poisoning

- Stomach, polypi, 373, 397
 — thickening (of mucosa), 395, 396
 Stomatitis, 291
 Stricture, urethra, 952-957
 Stump, 1060
 Sublimate, *see* Poisoning
 Sublingual cyst, 317, 318a
 Suicide, 1344, 1357
 Sulphuric acid, *see* Poisoning
 Suprarenal bodies, 586-601
 — Addison's disease, 593-596a, 597-599b, 601
 — bronzing of skin, 593-596a, 597b, 601
 — hæmorrhage, 588
 — hypertrophy, 586, 587
 — tubercle, 589-592
 Syme's operation, 1071
 Symmetrical gangrene, 1096-1103
 Sympathetic nerve, 724a-b
 Syndesmitis ossificans, 140
 Synovial cysts, *see* Joints
 Synovitis, *see* Joints
 Syphilides, *see* under Syphilis
 Syphilis (general pathology), 1112, 1117-1155
 — anus, 453, 454
 — arm, 1125, 1126
 — bones, 28, 30, 31
 — bubo, 1129-1131b
 — cheek (primary), 1122
 — chin (primary), 1119
 — condylomata, 453, 454
 — congenital, 31, 57, 306, 1132, 1154, 1155
 — eyelids, 1124, 1124a
 — groin, 1126a, 1133
 — gumma, *see* Gumma
 — hand (primary), 1123
 — hereditary, 1112
 — intestines, 452
 — larynx, 227, 228
 — lip (primary), 1120-1121
 — lung, 275
 — mucous tubercles, 1132
 — nose, 1152, 1152a, 1154, 1155
 — nipple (primary sore), 1117-1118, 1135
 — penis (primary sore), 958, 1117-1126a
 — rash (secondary), 1120, 1121, 1128
 — rectum, 452
 — rupia, 1137-1138, 1149
 — scars, 1150
 — sore (primary), 1117, 1126a
 — syphilides, 924a, 1136, 1139-1142, 1146-1148, 1151
 — teeth, 347-349
 — tertiary, 1133-1153
 — tongue, 1126; *see* also Tongue
 Syphilis (general pathology), tubercles, 1132
 — ulcers, 1133, 1143-1145a, 1150-1151, 1153
 — vaccinated, 1125
 — warts, 1133
 TABES dorsalis, 1072
 Taches bleuâtres, 898a
 Tagliacotian operation, 285, 285a
 Talipes, 1342l
 Teeth, 345-349
 — congenital syphilis, 347-349
 Temple, epithelioma, 1278
 — tumours, 1350, 1351
 Tertiary syphilis, *see* Syphilis
 Testicle, 925-933
 — displacement, 933
 — gumma, 928
 — malignant disease, 929-932
 — sarcoma, 929-929b
 — tubercle, 925-927
 Thigh, cyst of, 1298; *see* also Sarcoma
 Thrombus, heart, 156-160
 — pulmonary artery, 276-279
 — septic, 202
 — veins, 202
 Thumb, bifid, 1330
 Thyroid gland, 532-585c
 — anatomy of, 541
 — atrophy of, 542
 — bronchocele, *see* Goitre
 — cachexia strumipriva, 575-578
 — cancer, 537, 582-584, 1259
 — cretin, 538-540, 570-573
 — cystic adenoma, *see* Cystic goitre
 — cystic goitre, 534, 543-557, 560-564, 579-580
 — exophthalmic goitre, 535-536, 569, 601
 — feeding with, 540
 — goitre, 532-537, 543-569, 573-575, 579-584, 601; *see* above and below
 — myxœdema, 542, 574a, 576-578, 585, 585c, 1167-1176; *see* Myxœdema
 — operations, 533a, 543, 544, 546, 547, 574-575
 — parenchymatous goitre, 558, 559, 565-568, 573
 — treatment, 1174-1176
 Tibia, fracture, *see* Fracture
 Tibial nerve (posterior), 735
 Tinea, 908-911, 913, 914; *see* Parasites
 Tongue, 295-339, 344, &c.
 — acute inflammation, 298
 — adenoma, 325a
 — carcinoma, 344, Pl. vi. 1, vii. 1-3, xv. 2-4, xvi. 1-4, 1269, 1270; *see* also under Epithelioma of tongue

- Tongue cysts, 317-318a
 — diphtheritic, 302, 304
 — dyspeptic ulcer, 301
 — epithelioma, 331, 332, 335-339; *see*
 also under Carcinoma of tongue
 — excoriation, 344, Pl. vi. 4, vii. 4
 — fibroma, 324, 325
 — fissures, 326
 — glossitis, 298
 — gumma, 344, Pl. ix. 2
 — hemiatrophy, 296, 297
 — hemiplegia, 296
 — hypertrophy, 295
 — ichthyosis, 327-329, 344, Pl. vii. 2,
 xvi. 4
 — leukoplakia, 330-332, 344, Pl. vi. 1,
 4, vii. 1, 2, xv. 2, 3
 — lingual cyst, 318
 — lymphangioma, 319-321a
 — nævus, 323a
 — nigrities, 304a
 — papilloma, 322
 — plaques, 344, Pl. i. 2
 — psoriasis, 344, Pl. vi. 3, ix. 2
 — scars, 344, Pl. i. 3, ii. 2-4, x. 4
 — sublingual cyst, 317, 318a
 — syphilis, 306-316, 344, Pl. v. 1, viii.
 1-4, 1126
 — tubercular ulcer, 305, 305a, 344,
 Pl. iv. 3, ix. 4
 — ulcer, 299-301, 305, 305a, 307, 309,
 310, 314, 344, Pl. iii. 12, iv. 3, v. 2-4,
 ix. 4
 — ulcer, dyspeptic, 301
 — ulcer, syphilitic, 307, 309, 310-314,
 344, Pl. v. 2-4
 — ulcer, tubercular, 305, 305a, 344, Pl.
 iv. 3, ix. 4
 Tonsil, diphtheria, 216, 226
 — gangrenous, 215, 216
 Tophi, 935, 1158-1161a
 — scrotum, 935
 Torticollis, 141, 142
 Trachea, croup and diphtheria. 222, 223,
 225
 — glanders, 214
 — tracheotomy, 232, 233
 — ulceration, 232
 Tracheotomy, 232, 233
 Traumatic gangrene, 1089
 Transplantation, 1348; *see* also Grafting
 Transposition of viscera, 1317
 Trichophyton, *see* Parasites
 Tricuspid disease, 181
 Tubes, Fallopian, 963, 970
 Tubercles, mucous, 1132
 Tuberculosis (general pathology), 1104c
 — ankle, 1105c
 — bladder, 629, 645, 649
 Tuberculosis (general pathology), brain,
 696-698, 925
 — general, 164
 — intestines, 443-446
 — joints, 73-75, 1105c
 — kidney, 625-630
 — lip, 1105, 1105a
 — lung, 262-268a
 — meninges, 695
 — miliary, lung, 262
 — pancreas, 512
 — pericardium, 164
 — spleen, 527-529
 — suprarenal bodies, 589-592
 — tarsus, 1105c
 — testicle, 925-927
 — tongue, 305, 305a
 — ulcers, 1104-1105a
 — ulcers, fungating, 1105b
See also under Lupus, and the various
 organs
 Tubo-ovarian cyst, 963
 Tumours of uncertain nature, 1242-1243,
 1305a, 1351a
 Typhoid fever, 430-432
 — rash, 898-900
 — stool, 442
 ULCERATION (general pathology), 1066,
 1068-1077, 1104-1105a
 — articular cartilages, 77
 — bones, 29
 — cervix uteri, 976, 977, 1002, 1003
 — cornea, 736a
 — larynx, 213
 — scrotum, 939
 — stump, 1060
 — tongue, *see* Tongue
 — trachea, 232
 — tubercular, 1104-1105a
 — uterus (cervix), 976, 977, 1002, 1003
See also Ulcers
 Ulcerative colitis, 427
 Ulcerative endocarditis, *see* Endocarditis
 Ulcers, cancerous, 1277b
 — fungating, 1105b
 — gall-bladder, 510
 — gouty, 1158, 1159
 — intestine, 407, 412-429, 443-446,
 452; *see* under Intestine
 — perforating, 1072-1072b
 — rectum, 452
 — rodent, 1285-1296
 — scalp, 1077a
 — skin, 844-847, 860, 866
 — skin, chronic, 844-846
 — skin, circular, 846
 — skin, strumous, 847
 — sarcoma, 1233, 1233b

- Ulcers, syphilis, 1133, 1143-1145a, 1150-1151, 1153
 See Carcinoma, Syphilis, and Tuberculosis
- Ulnar nerve, 733a
- Umbilicus, 1305
- Uncertain skin diseases, 942a, 1112a
 — tumours, 1242a, 1351a
- Urethra, 952-957
 — dilatation, 955
 — false passages, 957
 — fistulae, 956
 — stricture, 952-957
- Urine, 642
- Urticaria pigmentosa, 776-777a
 — hæmorrhagica, 778-779
- Uterus, 972-1005
 — antelexion, 991
 — arsenic poison, 998
 — Bandl's ring, 1004
 — carcinoma, 995, 1001
 — cervix, 976, 977, 981, 1002, 1003
 — contraction ring, 1004
 — electrolysis, 1005
 — enteric fever, 1000
 — fibrinous polyp, 990
 — fibroid, 983, 989, 1005
 — hymen, imperforate, 972, 974
 — involution, 1004
 — myxœdema, 999
 — parametritis, 993, 994
 — perimetritis, 994a-997, 1000
 — polyp, 978-980, 990
 — retroversion, 992
 — sarcoma, 1231
 — ulceration, cervix, 976, 977, 1002, 1003
- VACCINATION, 893-895, 1125
- Vagina, 972, 974, 982, 1007-1009, 1080, 1081
- Valvular disease, aortic valves, 172-175
 — mitral valves, 158, 181, 183
 — pulmonary valves, 176
 — tricuspid valves, 181
- Varicella, 887-892, 896
- Varicose aneurysm, 192-193b
 — veins, 203-207
- Variola, 1116-1116a
- Varix, 192
- Vegetations, mitral, 155, 167-169, 173
- Veins, 202-210
 — clot, 202
 — thrombus, 202
 — varicose, 203-207
- Vena cava, inferior, 204
 — inferior, obstruction, 204-204a
 — superior, 203, 206
 — superior, obstruction, 203, 206
- Ventricles, brain, *see* Brain
- Vermiform appendix, 429
- Vertebrae, *see* Spine
- Vitiligo, 834
- Vitiligoidea, 838-841; *see also* Xanthelasma
- Vocal cord, papilloma of, 229; *see also* Larynx
- Volvulus, 472-474
- WART, clitoris, 1007
 — rectum, 455
 — skin, 817, 820, 821, 823, 886, 1209
 — skin, greasy, 886
 — syphilis (tertiary), 1133
- Wax refiner's carcinoma, 1276, 1277
- White kidney, *see* Kidney and Nephritis
- Wry neck, 141, 142
- XANTHELASMA, skin, 838-841
 — tongue, 304
- Xanthoma skin, 838-841
- Xeroderma pigmentosa, 833a

BOOKS PRESENTED TO THE LIBRARY.

1896.

Presented by

Injuries and Diseases of the Genital Urinary Organs, by Henry Morris, F.R.C.S.	}	The AUTHOR.
Report of the Indian Drugs Commission, 1893-94	}	The UNDER-SECRETARY OF STATE FOR INDIA.
Aseptic Surgery, by C. B. Lockwood, F.R.C.S.	}	The AUTHOR.
The Schott Methods of the Treatment of Chronic Diseases of the Heart, by Dr. Bezley Thorne (2 copies)	}	The AUTHOR.
The Harverian Oration for 1896, by Dr. William Selby Church	}	The AUTHOR.
Uric Acid in Causation of Disease, 3rd edition, by Alexander Haig, M.D.	}	The AUTHOR.
The Medical Annual for 1896	}	The PUBLISHERS.
Macallaster's Zoology and Morphology of Vertebrate Animals	}	
Eeker on the Convulsions of the Human Brain	}	
M'Cook's Agricultural Ant of Texas	}	
Barker's Manual of Surgical Operations	}	EDGAR WILLETT, Esq., F.R.C.S.
Howship's Discrimination and Appearances of Surgical Disease	}	
Ferguson's Lectures on the Progress of Anatomy and Surgery	}	
Transactions of International Medical Congress (4 vols.)	}	
Surgical Diseases of the Ovaries and Fallopian Tubes, including Tubal Preg- nancy, by J. Bland Sutton, F.R.C.S.	}	The PUBLISHERS, MESSRS. CASSELL & Co.
Diseases of the Nose and Throat, by F. de Havilland Hall, M.D.	}	The AUTHOR.

Presented by

Coroners' Act, Notes on, 1896, by E. L. Hussey, Esq.	}	The AUTHOR.
Elementary Anatomy and Surgery for Nurses, by W. M'Adam Eccles, F.R.C.S.		
Surgery of the Chest, 1896, by Stephen Paget, F.R.C.S.	}	The AUTHOR.
Micro-Organisms and Disease, by E. Klein, M.D. (1896)		
The Girdle of Truth, by the Rev. E. J. Brewster	}	The AUTHOR.
Consoling Thoughts, by M. Augusta Brewster		
The Sequels of Disease, with Observations on Prognosis in Disease, by Sir Dyce Duckworth, M.D.	}	The AUTHOR.

SUMMARY OF SCHOLARSHIPS AND PRIZES

OBTAINABLE BY STUDENTS AT ST. BARTHOLOMEW'S HOSPITAL.

AT ENTRANCE :—

Senior Entrance Scholarship in Physics and Chemistry	£75 0 0
Senior Entrance Scholarship in Biology and Physiology	75 0 0
Junior Entrance Scholarship in Science	150 0 0
Preliminary Scientific Exhibition	50 0 0
Jeaffreson Exhibition	25 0 0
Shuter Scholarship	50 0 0

AT END OF FIRST YEAR :—

Junior Scholarship in Anatomy and Biology (First)	30 0 0
Junior Scholarship in Anatomy and Biology (Second)	20 0 0
Junior Scholarship in Chemistry and Physics and Histology (First)	25 0 0
Junior Scholarship in Chemistry and Physics and Histology (Second)	15 0 0
Treasurer's Prize	5 0 0

AT END OF SECOND YEAR :—

Senior Scholarship	50 0 0
Foster Prize	6 0 0
Harvey Prize	6 6 0
Wix Prize	5 0 0
Hichens Prize	7 5 0

AT END OF THIRD AND LATER YEARS :—

Lawrence Scholarship and Gold Medal	42 0 0
Kirkes Scholarship and Gold Medal	30 0 0
Brackenbury Medical Scholarship	39 0 0
Brackenbury Surgical Scholarship	39 0 0
Sir George Burrows Prize	10 10 0
Skyenner Prize	13 13 0
Bentley Prize	6 6 0
Matthews Duncan Prize and Gold Medal	20 0 0

AFTER QUALIFICATION :—

Treasurer's Research Studentship	100 0 0
--	---------

The total value of the Scholarships and Prizes awarded annually is £895.

EXAMINATIONS, 1894-95.

- Lawrence Scholarship and Gold Medal—
J. K. MURPHY.
- Brackenbury Medical Scholarship—
S. GILLIES.
- Brackenbury Surgical Scholarship—
L. GILES.
- Senior Scholarship in Anatomy, Physiology, and Chemistry—
E. C. MORLAND.
- Open Scholarships in Science, Chemistry, and Physics—
A. L. ORMEROD.
- Open Scholarship in Biology and Physiology—
W. L. BROWN.
- Open Scholarship (Junior) in Biology and Physiology—
F. C. BORROW } Æq.
S. R. SCOTT }
- Preliminary Scientific Exhibition—
L. A. WALKER.
- Jeaffreson Exhibition—C. A. S. RIDOUT.
- Kirkes Scholarship and Gold Medal—
NO AWARD.
- Matthews Duncan Medal and Prize.
T. S. PIGG.
- Bentley Prize (Medical)—E. W. GROVES.
- Hichens Prize—NO AWARD.
- Wix Prize—C. C. I. TURNBULL.
- Harvey Prize—
1. H. DAVIES.
2. E. C. MORLAND.
- Sir George Burrows Prize—NO AWARD.
- Skyner Prize—T. J. HORDER.

PRACTICAL ANATOMY.

JUNIOR.

- Treasurer's Prize—F. C. BORROW.
2. { W. H. LEONARD } Æq.
S. R. SCOTT }
4. H. BURROWS.
5. A. R. BAKER.
6. C. S. FROST.
7. H. S. THOMAS.
8. H. WETHERED.
9. C. R. BROWN.
10. L. A. WALKER.

SENIOR.

- Foster Prize—R. RAINES.
2. E. C. MORLAND.
3. { L. A. BAISS } Æq.
G. E. GASK }
5. A. O. B. WROUGHTON.
6. W. T. ROWE.
7. S. A. MILLEN.
8. H. E. WALLER.
9. H. D. EVERINGTON.
10. F. HORRIDGE.

Shuter Scholarship—NO AWARD.

Junior Scholarships—

Anatomy and Physiology—

1. S. R. SCOTT.
2. { F. C. BORROW } Æq.
H. G. WOOD-HILL }

Chemistry and Physics—

- { H. A. COLWELL } Æq.
{ E. C. MORLAND }

EXAMINATIONS, 1895-96.

- Lawrence Scholarship and Gold Medal*—
S. GILLIES.
- Brackenbury Medical Scholarship*—
J. HUSSEY.
- Brackenbury Surgical Scholarship*—
G. V. WORTHINGTON }
H. WILLIAMSON } Æq.
- Matthews Duncan Medal and Prize*—
I. Not awarded. | 2. { G. E. DODSON }
| { T. J. HORDER } Æq.
- Senior Scholarship in Anatomy, Physiology, and Chemistry*—
H. A. COLWELL.
- Open Scholarships in Science, Chemistry, and Physics*—
J. S. WILLIAMSON.
- Open Scholarship in Biology and Physiology*—
C. S. MYERS.
- Open Scholarship (Junior) in Biology and Physiology*—
R. C. BOWDEN }
R. H. PARAMORE } Æq.
- Preliminary Scientific Exhibition*—
J. C. M. BAILEY.
- Jeaffreson Exhibition*—
H. A. KELLOND-KNIGHT.
- Kirkes Scholarship and Gold Medal*—
G. A. AUDEN.
- Bentley Prize (Surgical)*—
T. J. HORDER.
- Hichens Prize*—
F. R. BROOKS.
- Wix Prize*—
Not awarded.
- Harvey Prize*—
1. F. C. BORROW. 2. W. S. DANKS. 3. L. A. WALKER.
- Burrows Prize*.
T. J. HORDER.
- Skynner Prize*—Not awarded.

PRACTICAL ANATOMY.

- | JUNIOR. | SENIOR. |
|--|----------------------------------|
| <i>Treasurer's Prize</i> —A. E. J. LISTER. | <i>Foster Prize</i> —H. BURROWS. |
| 2. T. A. COMPTON. | 2. F. C. BORROW. |
| 3. C. A. S. RIDOUT. | 3. S. R. SCOTT. |
| 4. R. H. R. WHITAKER. | 4. C. S. FROST. |
| 5. G. M. SEAGROVE. | 5. W. H. LEONARD. |
| 6. J. S. WILLIAMSON. | 6. H. S. THOMAS. |
| 7. { J. C. MARSHALL }
A. T. PRIDHAM } Æq. | 7. W. S. DANKS. |
| 9. A. R. TWEEDIE. | 8. T. B. HAIG. |
| 10. A. H. JOHN. | |

Shuter Scholarship—
F. A. ROSE.

Junior Scholarships—

1. R. H. PARAMORE.
2. { A. R. TWEEDIE }
J. S. WILLIAMSON } Æq.

Junior Scholarship in Chemistry (1895)—

1. L. A. WALKER. 2. R. WALKER.

ENTRANCE SCHOLARSHIPS,

OCTOBER

1896.



Open Scholarships in Science.

Biology and Physiology—

C. J. THOMAS.

Chemistry and Physics—

H. F. PARKER } Æq.
E. H. SCHOLEFIELD }

Preliminary Scientific Exhibition—

R. A. S. SUNDERLAND.

Jeaffreson Exhibition—

S. G. MOSTYN.

Junior Open Scholarship in Science.

R. C. ELMSLIE.

ST. BARTHOLOMEW'S HOSPITAL & COLLEGE.

—◆—
THE MEDICAL AND SURGICAL STAFF.

Consulting Physician—Dr. Andrew.

Consulting Surgeons—Sir J. Paget, Bart., D.C.L., LL.D., F.R.S.,
Mr. Luther Holden.

Consulting Ophthalmic Surgeon—Mr. Henry Power.

Physicians—Dr. Church, Dr. Gee, Sir Dyce Duckworth,
Dr. Hensley, Dr. Brunton, F.R.S.

Surgeons—Mr. Thomas Smith, Mr. Willett, Mr. Langton, Mr.
Marsh, Mr. Butlin.

Assistant-Physicians—Dr. Norman Moore, Dr. S. West, Dr.
Ormerod, Dr. Herringham, Dr. Tooth.

Assistant-Surgeons—Mr. Walsham, Mr. Cripps, Mr. Bruce
Clarke, Mr. Bowlby, Mr. Lockwood.

Physician-Accoucheur—Dr. Champneys.

Assistant-Physician-Accoucheur—Dr. Griffith.

Assistant-Surgeon to Obstetric Wards—Mr. Harrison Cripps.

Ophthalmic Surgeons—Mr. Vernon, Mr. Jessop.

Aural Surgeon—Mr. Cumberbatch.

Pathologist—Dr. Kanthack.

Dental Surgeons—Mr. Paterson, Mr. Ackery.

Assistant-Dental Surgeons—Mr. Read, Mr. Ackland.

Administrators of Anæsthetics—Mr. Gill, Mr. Edgar Willett.

Medical Registrars—Dr. Calvert, Dr. Garrod.

Surgical Registrar—Mr. Berry.

Administrator of Electricity—Dr. Lewis Jones.

Casualty Physicians—Dr. Horton-Smith, Dr. Batten.

LECTURES.

Medicine—Sir Dyce Duckworth, Dr. Norman Moore.

Clinical Medicine—Dr. Church, Dr. Gee, Sir Dyce Duckworth,
Dr. Hensley, Dr. T. Lauder Brunton, F.R.S.

Surgery—Mr. Marsh, Mr. Butlin.

Clinical Surgery—Mr. Thomas Smith, Mr. Willett, Mr. Langton,
Mr. Marsh, Mr. Butlin.

Descriptive and Surgical Anatomy—Mr. Walsham, Mr.
Bruce Clarke.

General Anatomy and Physiology—Dr. Klein, F.R.S.

Histology—Dr. Klein, F.R.S.

Chemistry and Practical Chemistry—Dr. Russell, F.R.S.

Materia Medica, Pharmacology, and Therapeutics — Dr.
Brunton, F.R.S.

Forensic Medicine—Dr. Hensley.

Public Health—Dr. Thorne Thorne, C.B., F.R.S.

Midwifery and the Diseases of Women and Children—Dr.
Champneys.

Botany—Rev. George Henslow.

Pathological Anatomy—Dr. Kanthack.

Biology and Comparative Anatomy—Dr. Shore.

Ophthalmic Medicine and Surgery—Mr. Vernon.

Mental Diseases—Dr. Claye Shaw.

Physics—Mr. F. W. Womack.

Chemical Physiology—Dr. Edkins.

Organic Chemistry—Dr. Chattaway.

DEMONSTRATIONS.

Morbid Anatomy—Dr. Calvert, Dr. Garrod.

Diseases of the Skin—Dr. S. West.

Orthopædic Surgery—Mr. Walsham.

Diseases of the Ear—Mr. Cumberbatch.

Diseases of the Eye—Mr. Bowater Vernon, Mr. Jessop.

Diseases of the Larynx—Mr. Bowlby.

Dental Surgery—Mr. Paterson, Mr. Ackery.

Anæsthetics—Mr. Gill.

Practical Surgery—Mr. Bowlby, Mr. C. B. Lockwood.

Practical Anatomy—Mr. Waring, Mr. Bailey.

Assistant-Demonstrators—Mr. Weir, Mr. Furnivall, Mr. Sloane, Mr. Miles.

Operative Surgery—Mr. J. Berry, Mr. H. J. Waring, Mr. Eccles.

Practical Physiology—Dr. Edkins.

Assistant-Demonstrators—Dr. Horton-Smith, Dr. Drysdale.

Practical Pharmacy—Dr. Calvert.

Surgical Pathology—Mr. Bowlby.

Morbid Histology—Dr. Kanthack.

Practical Medicine—Dr. S. West.

Assistant-Demonstrators—Dr. Andrewes, Dr. Fletcher.

Practical Midwifery—Dr. Roberts.

Medical Electricity—Dr. Lewis Jones.

Bacteriology—Dr. Kanthack.

Public Health—Dr. Waldo.

Practical Biology—Dr. Shore.

Assistant-Demonstrators—Mr. Brown, Mr. Horder.

Practical Chemistry—Dr. Chattaway.

Assistant-Demonstrator—Mr. Evans.

Curator of the Museum—Dr. Kanthack.

COLLEGIATE ESTABLISHMENT.

Warden—Dr. SHORE.

Students can reside within the Hospital walls, subject to the College regulations.

Fifteen Scholarships, varying in value from £10 to £150, are awarded annually. See page 536.

Further information respecting Scholarships, Pupils' Appointments, and other details, may be obtained from Dr. SHORE, and at the Museum and Library.

ST. BARTHOLOMEW'S HOSPITAL REPORTS.

VOLUME XXXII.

I N D E X.

	PAGE
ABERNETHIAN Society, Proceedings of	443
Acromegaly, overgrowth resembling	405
Ætiology of chorea	383
Alcoholic paralysis, pathology of	407
Anæmia treated by oxygen inhalations	321
Anæsthesia (palatal and laryngeal) in disseminated sclerosis	173
Andrewes, F. W., diphtheria bacillus in series of sore throats among nurses	145
Antistreptococcic serum in treatment of septicæmia	23
Antitoxin, cases of diphtheria treated by, cause of death and prognosis in	335
Aphorisms from Dr. Gee's ward	29
Appendix, hernia of	93
Arrest of development in lower jaw	403
Atrophy of liver (acute)	425
BAKER, William Marrant, obituary notice	xxxix
Batten, F. E., with Horne, disseminated sclerosis with palatal and laryngeal anæsthesia	173
Bladder, rupture of, in fractured pelvis	199
" villous tumour of	207
" malignant disease of	203, 205
Books presented to Library	334
Brunton, T. Lauder, with Tunnicliffe, acute atrophy of liver	425
Burnett, F. M., treatment of anæmia with oxygen inhalations	321
CARDIAC hypertrophy, œdema, and renal disease	261
Cases from Dr. Church's wards	1
" from Mr. Smith's wards	19
" in Surgical wards	181
" of deformity	400

	PAGE
Cause of death in cases of diphtheria treated by antitoxin	335
Chorea, ætiology of	383
Church, W. S., cases from the wards	I
Clinical aphorisms from Dr. Gee's wards	29
Colic and enteritis simulating intestinal obstruction	61
Collapse and rigor in typhoid fever	107
Congenital absence of radius	400
DESCRIPTIVE list of specimens added to Museum during 1896	451
Development, arrest of, in lower jaw	403
Deformity, cases of	400
Diphtheria bacillus in series of sore throats among nurses	145
" treated by antitoxin, prognosis and causes of death	335
Disseminated sclerosis with palatal and laryngeal anæsthesia	173
Duckworth, Sir Dyce, and Garrod, hepatic cirrhosis, with obstruction in superior vena cava	71
Dunn, W. E. N., double empyema with recovery	213
Dupuytren's contraction, relation of gout and rheumatism to	119
ECCLES, McAdam, hernia of vermiform appendix	93
Ehrlich's reaction in typhoid fever	217
Elephantiasis of one leg in a boy	181
Empyema, fetid	83
" double, with recovery	213
" of gall-bladder	189
Enteritis and colic simulating intestinal obstruction	61
Ether rash	79
FETID empyema	83
Fletcher, H. Morley, ætiology of chorea	383
GANGRENE of leg, traumatic	192
Garrod, A. E., and Sir Dyce Duckworth, hepatic cirrhosis with obstruction in superior vena cava	71
Growth in rectum	21
HÆMATURIA, cases of	210
Heath, cases of deformity	400
Heart, hypertrophy of, œdema and renal disease	261
Hedges, relation of gout and rheumatism to Dupuytren's contraction	119
Hepatic cirrhosis, with obstruction in superior vena cava	71
Hernia of vermiform appendix	93
Historical records of Museum specimens	513
Holmes, H., with Kanthack, cardiac hypertrophy, œdema, and renal disease	261
Horder, Thomas J., cases in Surgical wards	181

	PAGE
Horder, T. J., clinical aphorisms from Dr. Gee's wards	29
Horne, W. J., with Batten, disseminated sclerosis, with palatal and laryngeal anæsthesia	173
Humphry, Sir George Murray, In Memoriam	xxxI
Hydatid disease of arm	187
Hypertrophy of heart, œdema, and renal disease	261
INFECTIVE lesions in lobar pneumonia	309
Intestinal obstruction simulated by enteritis and colic	61
KANTHACK, A. A., with Holmes, cardiac hypertrophy, œdema, and renal diseases	261
„ with Lance, secondary infective lesions in lobar pneumonia	309
„ and May, prognosis and causes of death in diph- theria treated by antitoxin	335
„ descriptive list of specimens added to the Museum during 1896	451
Kidney, rupture of	196
LANCE, H. W., with Kanthack, secondary infective lesions in lobar pneumonia	309
Leucocytosis in scarlet fever	225
Library, books presented to	334
Liver, acute atrophy of	425
Lobar pneumonia, secondary infective lesions in	309
Lockwood, C. B., In Memoriam, Sir George Murray Humphry	xxxI
Lower jaw, arrest of development in	403
MALIGNANT endocarditis (streptococci)	I
May, J. H., with Kanthack, prognosis and causes of death in diphtheria treated by antitoxin	335
McAdam Eccles, hernia of vermiform appendix	93
Mitchell, enteritis and colic simulating intestinal obstruction	61
Morrant Baker, obituary notice of	xxxix
Multiple polypi of rectum	19, 21
Museum, specimens added to, &c.	451
NERVE-INJURY at birth	404
OBITUARY notice of Sir George Murray Humphry	xxxI
„ of William Morrant Baker	xxxix
Obstruction in vena cava superior with hepatic cirrhosis	71
œdema, hypertrophy of heart, and renal disease	261
Osteitis deformans beginning in early life	403
Overgrowth resembling acromegaly	405
Oxygen inhalations in anæmia	321

	PAGE
PALMAR fascia, contraction of, in relation to gout and rheumatism	119
Paralysis, alcoholic, pathology of	407
Paterson, skin-grafting by Thiersch's method	161
„ H. J., Ehrlich's reaction in typhoid fever	217
Pelvis, fracture of, with ruptured bladder	199
Pneumonia with endocarditis	5
„ embolism of right coronary artery	7
„ lobar, secondary infective lesions in	309
Polypi, multiple, of rectum	19, 21
Proceedings of Abernethian Society	443
Profuse diarrhoea causing death in man from tropics	87
Prognosis in cases of diphtheria treated by antitoxin	335
RADIUS, congenital absence of	400
Rectum, growth in	21
„ multiple polypi of	19, 21
Relation of gout and rheumatism to Dupuytren's contraction	119
Renal disease, hypertrophy of heart, and œdema	261
Rigor and collapse in typhoid fever	107
Ruptured kidney	196
SARCOMA of knee-joint	184
Scarlet fever, leucocytosis in	225
Septicæmia treated with antistreptococcic serum	23
Sevestre, leucocytosis in scarlet fever	225
Skin-grafting by Thiersch's method	161
Smith and Worthington, cases from wards	19
Sore throats among nurses, diphtheria bacillus, in series of	145
Specimens added to Museum, list of	451
Streptomycosis or septicæmia treated with antistreptococcic serum	23
Surgical wards, cases in	181
THIERSCH'S method of skin-grafting	161
Tooth, H. H., pathology of alcoholic paralysis	407
Traumatic gangrene of leg	192
Tropics, profuse diarrhoea causing death in man from	87
Tubercular disease of urinary tract	200
Tumour of groin	194
Tunnicliffe, F. W., with Brunton, acute atrophy of liver	425
Typhoid fever, with relapses, very high temperature, and rigors	14
„ rigor and collapse in	107
„ Ehrlich's reaction in	217
URINARY tract, tubercular disease of	200

	PAGE
VERMIFORM appendix, hernia of	93
WARDS, Surgical, cases in	181
West, Samuel, fetid empyema	83
" case of profuse diarrhœa causing death in man from tropics	87
Willett, Alfred, obituary notice of William Marrant Baker	xxxix
" Edgar, ether rash	79
Worthington with Mr. Smith, cases from wards	19



STATISTICAL TABLES

OF THE

Patients under Treatment

IN THE WARDS OF

ST. BARTHOLOMEW'S HOSPITAL

DURING 1895.

BY

THE MEDICAL REGISTRAR,
W. P. HERRINGHAM, M.D. (OXON.), F.R.C.P.

AND

THE SURGICAL REGISTRAR,
JAMES BERRY, B.S. (LOND.), F.R.C.S.



London:

PRINTED BY CHARLES SKIPPER AND EAST,
49, GREAT TOWER STREET, E.C.

1896.

P R E F A C E.

The Classification of Diseases in the Medical Tables is that adopted by the College of Physicians in their Nomenclature of Diseases.

CONTENTS.

	PAGE.
Preface	iii.
Number of Beds	v.
General Statement of the Patients under Treatment during the year	v.
Patients brought in Dead	v.
Occupations of the Male Patients	vi.
Occupations of the Female Patients	viii.

MEDICAL REPORT—

Preface to the Appendix (Medical Tables)	11
TABLE I.—Showing the Total Number of Cases of each Disease under Treatment during the Year 1895, with the Results	12
Abstract of Table I.	30
Index to Medical Cases, showing Diseases and chief Symptoms	31
Index to Register of Medical Post-mortem Examinations	49

SURGICAL REPORT—

Preface to the Surgical Report... ..	62
TABLE I.—Showing the Total Number of Cases under Treatment during the Year 1895, with the comparative frequency and mortality of each Disease at different ages	63
Abstract of Table I.	101
Appendix to Table I.	102
Table showing the Surgical Operations performed	119
Statistics of Anæsthetics	142
Appendix to Table of Surgical Operations performed	143
Sub-Table showing the number of Cases of Erysipelas, Pyæmia, &c.	183
Appendix to the Sub-Table of Erysipelas, Pyæmia, &c.	184
Table of Amputations, with the Percentage of Deaths during the Ten Years from 1886 to 1895 inclusive	187
Index to Register of Surgical Post-mortem Examinations	189

ST. BARTHOLOMEW'S HOSPITAL.

1895.

Number of Beds in	Medical Wards	206
"	"	"	Wards for Diseases of Women	20
"	"	"	Surgical Wards	362
"	"	"	Ophthalmic Wards	25
"	"	"	Unassigned	41

654

On October 1st, these numbers were changed to:—

Number of Beds in	Medical Wards	241
"	"	"	Wards for Diseases of Women	34
"	"	"	Surgical Wards	336
"	"	"	Ophthalmic Wards	25
"	"	"	Unassigned	38

674

GENERAL STATEMENT OF THE PATIENTS UNDER TREATMENT DURING THE YEAR 1895.

Patients remaining in, January 1st, 1895:—

Medical	210	} ... 516
Surgical	306	

Admitted during the year 1895:—

Medical	2,463	} ... 6,774
Surgical	4,311	

} ... 7,290

Discharged:—

Medical	2,038	} ... 6,113
Surgical	4,075	

Died:—

Medical	392	} ... 618
Surgical	226	

} ... 7,290

Remaining in, January 1st, 1896:—

Medical	243	} ... 559
Surgical	316	

Patients brought in Dead	34	} ... 70
Dying in the Surgery or Surgery Ward	36	

OCCUPATIONS OF MALE PATIENTS.

Agents 7	Checkers 3	Firewood cutters ... 5
Angler 1	Cheesemongers ... 5	Fish curer 1
Architect 1	Chemists 6	Fisherman 1
Artist 1	Chocolate maker ... 1	Fish frier 1
Asphalter 1	Chorister 1	Fishmongers 8
	Church cleaner ... 1	Fitters... 2
Bacon dryer 1	Cigar makers... .. 2	Florist... 1
Bag makers 3	Clergyman 1	Footmen 3
Bailiffs 2	Clerks 82	Foremen 10
Bakers 19	Clipper 1	French polishers ... 13
Barmen 22	Clothiers 2	Fret worker 1
Barrel washer 1	Coachmen 20	Fruiters 7
Basket makers 2	Coal merchant 1	Furriers 4
Bat maker 1	Coal trimmers ... 2	
Beadle... 1	Colliers 3	
Billiard marker 1	Compositors 13	Gamekeeper 1
Blacksmiths 18	Conductors 4	Gardeners 19
Blind maker 1	Confectioner 1	Gas fitters 8
Boiler makers 2	Cooks 16	Gas purifier 1
Bookbinders 18	Coopers 9	Gas workers 7
Boot makers 49	Coppersmiths... .. 2	Gatekeeper 1
Bottlers 4	Copyist 1	Gilders 5
Bottle washer 1	Costermongers ... 12	Glass workers 8
Box makers 4	Cow keeper 1	Gold blocker 1
Brass finishers 9	Crate maker 1	Gold digger 1
Brass moulders 5	Custom House officer 1	Goldsmiths 4
Brewer 1	Cycle maker 1	Grain weigher 1
Bricklayers 20		Greengrocers 4
Brick maker 1	Dealers 5	Grinders 3
Brush makers 3	Decorators 2	Grocers 5
Builders 13	Die cutter 1	Grooms 7
Butchers 36	Dispenser 1	
Butler 1	Diver 1	Hairdresser 1
	Dog doctors 2	Harness makers 5
Cabinet makers 38	Drapers 13	Hawkers 30
Cab drivers 21	Draymen 6	Hay binder 1
Cab proprietor 1	Drivers 4	Horse breaker 1
Cab washers 2	Dustman 1	Horse keepers 23
Cap maker 1	Dyer 1	Hotel servant 1
Capsule counter 1		House breaker 1
Card cutters 2	Electrician 1	Housekeeper 1
Caretakers 4	Engine cleaner ... 1	House surgeons 3
Carmen 94	Engine drivers ... 7	
Carpenters 40	Engineers 38	Ice-cream vendor ... 1
Carpet cutter... .. 1	Engine fitter 1	Ice man 1
Carpet planner 1	Engine oil mixer ... 1	India-rubber maker ... 1
Carriage builders 5	Engravers 3	Ink maker 1
Carriers 4	Errand boys 24	Instrument makers ... 4
Carvers 2	Factory hand 1	Ironmonger 1
Case makers 6	Farmers 8	Iron workers 4
Cashiers 2	Farriers 8	
Cattleman 1	Feather dressers ... 2	
Cellarmen 4	Feather sorter ... 1	Jewellers 4
Chair makers... .. 4	Firemen 16	Journalists 2

OCCUPATIONS OF MALE PATIENTS (*continued*).

Kitchen boys... .. 2	Paper dealer 1	Smelter 1
Labourers 291	Paper makers 2	Soda water manufacturer 1
	Paper seller 1	Soldiers 28
	Paupers 2	Solicitors 3
	Paviors 2	Spinner 1
Lamp lighters 2	Pawnbroker 1	Sponge trimmer 1
Lamp maker 1	Pedlar... .. 1	Stationers 2
Laundrymen 3	Perfumer 1	Steel cleaner 1
Law student 1	Pewterer 1	Stevedores 3
Lead workers... .. 2	Piano maker 1	Stick makers... .. 9
Leather cutters 2	Piano tuners 2	Stock keepers 2
Leather dressers 3	Picture-frame maker 1	Stokers 11
Letter sorters... .. 3	Pie maker 1	Storekeepers 3
Lift men 2	Pipe maker 1	Sugar boiler 1
Lightermen 4	Plasterers 7	Surveyor 1
Lion tamer 1	Photographers 4	Sweeps 7
Lithographers 2	Platelayers 5	
Locksmiths 3	Plumbers 21	Tailors 18
Lodge keeper... .. 1	Policemen 5	Tallow chandler 1
Lodging-house keeper 1	Porters 120	Tarpaulin maker 1
	Portmanteau maker... .. 1	Teachers 2
	Postmen 11	Tea worker 1
Machinists 6	Potmen 5	Telegraphists... .. 2
Mail cart drivers 4	Printers 54	Telephonist 1
Manager 1	Provision dealer 1	Thermometer maker... .. 1
Mantle makers 2	Publicans 4	Timekeeper 1
Map colourer... .. 1	Purse maker 1	Tinmen 5
Masons 11		Tobacconists 3
Match makers 2		Tracer 1
Mat maker 1	Rivetter 1	Translators 2
Mechanics 3	Rope makers 2	Travellers 11
Medical students 25		Trunk maker 1
Medical practitioners 7		Turners 4
Merchants 3	Sack makers 2	Type founders 2
Messengers 21	Saddlers 2	Umbrella maker 1
Metal sorter 1	Sail maker 1	Undertakers 3
Metal spinner 1	Sailors... .. 29	Upholsterers 4
Milkmen 4	Salesmen 7	
Millers 3	Saw maker 1	Valet 1
Miners... .. 2	Sawyers 9	Van guards 7
Mosaic worker 1	Scaffolders 3	Vellum binder 1
Musicians 5	Scale cutter 1	Vestryman 1
	Scavengers 2	
	School boys 289	
	Schoolmasters 3	
	Screw makers 2	
	Servants 15	Waiters 12
	Sewermen 2	Warder 1
	Sexton... .. 1	Warehousemen 35
	Ship chandler 1	Watchmakers 4
	Ship keeper 1	Watchmen 6
	Ship's stewards 9	Watermen 3
	Shipwrights 2	Weaver 1
	Shop assistants 13	Wheelwrights 9
	Signalmen 4	Window cleaners 4
	Silversmiths 5	Wire workers... .. 6
	Skewer sharpener 1	Wood carvers 5
	Slaughterers 2	Workhouse master 1
	Slipper makers 2	Writers 5
News vendors 7		
Odd jobber 1		
Offal worker 1		
Office boy 1		
Oilmen 2		
Organ builders 2		
Ostlers 7		
Packers 28		
Packing-case makers 3		
Page 1		
Painters 51		
Pantryman 1		

MEDICAL REPORT.

PREFACE TO THE APPENDIX (MEDICAL TABLES).

An Index of Medical Cases and of the Post-mortem Register for the year is appended to the Statistical Tables.

Owing to the many changes which have this year taken place, it is convenient to adopt the system of classification in use on the Surgical side of the Hospital. The Medical cases, with an exception to be explained later, are this year bound and indexed not according to the Ward in which, but according to the Physician by whom, they were treated. Moreover, the Male cases are now separated from the Female.

Thus of Male Patients :—

Those treated by Dr. Church	are bound in Vol. I.
„ „ Dr. Gee	„ „ II.
„ „ Sir Dyce Duckworth,	„ „ III.
„ „ Dr. Hensley	„ „ IV.
„ „ Dr. Lauder Brunton,	„ „ V.

The Female cases are similarly numbered, and those treated by Dr. Champneys are bound in Vol. VI.

Radcliffe and Isolation are exceptions. It is more convenient to keep all these cases together in one volume, and they are referred to under Radcliffe.

Furthermore, an Alphabetical Index of Patients has been this year added. No such Index is elsewhere to be obtained, it will simplify reference, and it will also enable a patient to be traced forward as well as backward, which is valuable to those who wish to study a disease.

These Indices are embodied in special volumes, one of Male, one Female cases.

TABLE I. (continued).

DISEASE.	Total.		Under 5.		— 10.		— 15.		— 20.		— 30.		— 40.		— 50.		— 60.		Over 60.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
Lateral Sclerosis, Amyotrophic ...	2
Tabes Dorsalis ...	15	4
Multiple Sclerosis ...	3	3
Friedreich's Disease ...	1	1
Tremor ...	1	1
<i>Peripheral.</i>																				
Neuritis ...	16	7
Diphtheritic Paralysis ...	6	2
Syphilitic ...	7	4
Neuralgia ...	5	3
Muscular Atrophy ...	7	4
Spasm ...	3	2
Myalgia ...	2	2
Total ...	252	123	86	22	21

TABLE I. (continued).

DISEASE.	Total.		Under 5.		— 10.		— 15.		— 20.		— 30.		— 40.		— 50.		— 60.		Over 60.		
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	
DISEASES OF THE RESPIRATORY SYSTEM.																					
Catarrh	2	...	1	
Laryngismus	1	...	1	
Laryngitis	24	14	10	...	3	2	1	...	2	...	1	...	1	...	1	
Tubercular	2	2	
Syphilitic	2	...	1	
Phlegmonous	1	
Papilloma of Larynx	1	
Bronchitis	61	31	27	...	3	3	3	6	2	...	4	4	1	2	3	3	4	
With Emphysema	27	18	7	1	1	2	3	7	
With Pleurisy	4	3	1	1	
Emphysema...	6	5	
Bronchiectasis	1	1	
Catarrhal Pneumonia	106	54	23	...	15	14	11	9	6	1	2	2	...	1	
Croupous Pneumonia	67	29	12	...	3	3	...	14	12	...	8	3	...	13	1	
Pneumonia of doubtful nature	9	7	2	
Lung, Cancer of	2	1	1	
Cirrhosis of	1	
Collapse of	2	
Gangrene of	1	
Phthisis Pulmonalis	85	32	18	
Pleurisy	28	20	8	...	1	
Pleuritic Effusion	47	36	10	...	1	

ABSTRACT OF TABLE I.

DISEASES.	Total Number of Cases completed during the Year 1896.	Number of Cases discharged.		Deaths.		Remaining in the Hospital at the end of the Year 1896.
		M.	F.	M.	F.	
GENERAL DISEASES, A	313	134	115	45	19	
Do. B	235	82	127	19	7	
LOCAL DISEASES—						
Diseases of the Nervous System	252	123	86	22	21	
" Circulatory System	184	80	56	34	14	
" Respiratory System	570	326	148	63	33	
" Digestive System	333	134	141	35	23	
" Urinary System	131	61	35	24	11	
" Female Generative System	280	...	269	...	11	
" connected with Pregnancy	62	...	57	...	5	
" of the Cutaneous System	45	16	26	1	2	
CONDITIONS NOT NECESSARILY ASSOCIATED WITH GENERAL OR LOCAL DISEASES—						
POISONS	25	20	2	2	1	
	2,430	976	1,062	245	147	243
		2,038		392		
		2,430				

INDEX

TO THE DISEASES AND CHIEF SYMPTOMS OF PATIENTS DISCHARGED
FROM THE MEDICAL WARDS DURING THE YEAR 1895.

N.B.—The mark (†) signifies that a case terminated fatally ; (‡) that a post-mortem examination was made. The reference to the post-mortem notes, together with an abstract of these, is in each case added to the Ward notes.

ABDOMINAL—

Abscess—Males, IV., 102. Females, VI., 63.
Cancer—Males, IV., 164. Females, VI., 109, 175.
Pain—Males, I., 109 ; II., 20, 126, 162, 164, 227, 262 ; III., 137 ; IV., 91, 151, 168, 197. Females, I., 165 ; II., 131, 174 ; III., 95 ; IV., 16 ; VI., 157, 190, 206, 284.
Tumour—Males, I., 49, 109, 312 ; II., 114, 244 ; IV., 136, 164. Females, IV., 135, 160 ; VI., 236, 243, 258.

ABORTION—Females, II., 50 ; IV., 205 ; VI., 16, 85, 86, 268, 328, 359.

Incomplete—Females, VI., 43, 103, 151, 159, 181, 219, 311, 329, 346, 355.

Induced—Females, VI., 185, 191, 343.

Missed—Females, VI., 55.

Threatened—Females, VI., 96.

ABSCESS—

Abdominal—Males, IV., 102. Females, VI., 63.

Axillary—Females, II., 201.

Cervical—Males, I., 203 ; II., 31, 91. Females, II., 2, 6. *Radcliffe*—Males, 75‡.

Lumbar—Males, II., 234, 236.

Mammary—Females, VI., 22.

Metastatic—Males, II., 11 ; IV., 122 (parotid). Females, II., 109‡ ; III., 12‡, 19.

Peri-nephric—Males, I., 95 ; IV., 194.

Sub-diaphragmatic—Females, II., 192‡.

Superficial—Males, I., 152 ; II., 210. *Radcliffe*—Females, 70.

See also Cerebral, Liver, Mediastinum, Ovary, Pelvic, Para-, Peri-metric, Peritoneal, Peri-typhlitic, Spleen, Vulva.

ACNE ROSACEA—Males, III., 63.

ACROMEGALY—Males, II., 68.

ACTINO-MYCOSIS—Males, III., 135‡.

ADDISON'S DISEASE—Females, III., 223 ? ; IV., 188‡.

ADENITIS—Males, I., 203. Females, I., 15 ; II., 97 ; IV., 123, 145.

AGUE—Males, I., 88, 244 ; II., 173, 243 ; III., 120 ; IV., 152, 174‡ ; V., 9, 16.
Sequelæ of—Males, I., 15.

ALCOHOLISM—

Acute—Males, I., 305.

See Delirium Tremens.

Chronic—Males, I., 144, 177, 234 ; III., 105‡, 184 ? Females, III., 123.

AMNESIA—Males, III., 184. Females, II., 62.

AMYLOID DISEASE—Males, I., 150‡. Females, II., 1‡, 109‡ ; III., 69 ?

- ANÆMIA**—Males, I., 239†; II., 47; III., 28. Females, I., 10, 12, 139, 145, 146, 153, 183, 193, 205, 210; II., 34, 58, 84, 95, 99, 120, 134, 168; III., 3, 10, 38, 45, 64, 70, 77, 91, 139, 172, 175, 188, 220, 221, 222, 223; IV., 47, 78, 123, 131, 173, 181, 184; V., 4.
 With Enlarged Spleen—Males, I., 208; III., 4†.
 Pernicious—Males, I., 48†; II., 47?; III., 4†, 130. Females, III., 63†.
- ANEURYSM**—
 Abdominal—Males, I., 3† (common iliac), 83, 131, 210; II., 279†. Females, II., 103.
 Aortic—Males, I., 26, 90†, 107, 131, 220†; II., 153, 161?, 166, 276†; III., 51?, 60, 64†, 66, 79†, 182?, 220, 243†; IV., 79, 192†. Females, I., 156†; II., 45, 103?, 150?, 150A.
 Cardiac—Males, IV., 11†.
 Cerebral—Females, II., 152†; IV., 118†.
 Embolic—Males, I., 10†.
 Treated with Calcium Chloride—Males, I., 210; II., 153.
- ANGINA PECTORIS**—Males, III., 222.
- ANTITOXIN INJECTED**—
 Of Diphtheria—Males, II., 239†. Females, II., 86; IV., 199.
Rudcliffe—Males, I., 2, 3†, 8, 9, 10†, 11†, 13, 16, 17†, 18†, 19, 20, 22, 24†, 30, 31, 34, 39†, 47, 52, 54, 55, 56†, 62, 63, 66†, 68, 69, 71, 74, 75†, 76†, 77†, 79, 80, 82, 84†, 86, 88†, 90†, 91†, 92†, 93†, 94†. Females, 4, 12, 14, 15, 21, 23, 25†, 32†, 35, 36, 38†, 40, 41, 43, 45, 48, 49, 50, 51, 53†, 57, 58†, 59†, 61, 64†, 65†, 67, 70, 72, 73†, 87, 89†.
 Of Tetanus—Males, II., 133.
- AORTA**—
 Dilatation of—Males, I., 9; III., 106†.
 Pulsation of—Males, I., 116. Females, I., 107.
See Aneurysm.
- APHASIA, AGRAPHIA, APHONIA**—*See* Speech.
- APOPLEXY**—Males, I., 11†; III., 52.
- ARTHRITIS**—Males, I., 178†, 297; III., 105†; IV., 4†. Females, IV., 47, 189.
 Hæmorrhagica—Females, III., 134†.
 After Serum Injection—Males, II., 133.
 Syphilitic—Females, IV., 97.
 Tubercular—Females, I., 144, 181.
- ASCITES**—Males, I., 70, 89, 171†, 236†, 251; II., 202, 203, 236; III., 5, 17, 39, 102†, 122, 139, 163†, 235†; IV., 71†; V., 48†, 50†. Females, I., 11†, 29†, 142, 175, 218†; II., 28, 31, 31A, 119, 140†, 167, 180, 187; III., 51, 52, 62†, 163; IV., 6, 31, 37†, 44, 71, 71A, 75†, 114†; VI., 106, 173.
- ASTHMA**—Males, I., 233, 286; III., 36. Females, II., 194.
- ATAXIA**—Males, V., 19†. Females, II., 83, 129; IV., 111, 195.
See also Tabes Dorsalis.
- ATHETOSIS**—Males, II., 192.
- BONES**—
 Cancer of—Males, III., 46†, 143†; IV., 142? Females, I., 76†.
 Necrosis of—Males, III., 3, 49.
- BRONCHI-ECTASIS**—Males, II., 25†; IV., 134†, 188. Females, IV., 64.
 Treated by Resection of Rib—Males, IV., 134†, 188.
- BRONCHITIS**—Males, I., 25, 28, 33, 43, 61, 67†, 79†, 80, 111, 125†, 142, 159, 172, 206, 233, 267, 274, 286, 301, 312; II., 4, 14, 33, 49, 50, 54, 70†, 75, 86, 92, 102, 103, 104, 109, 125, 140, 159†, 229, 247, 289, 293; III., 8, 24, 31, 36, 57, 67, 196, 213, 224, 226, 242†; IV., 10, 11†, 23, 35, 64, 73†, 85, 89, 139, 170, 250, 257, 261; V., 1, 11, 13, 27, 54. Females, I., 3, 9, 21, 90, 94, 104; II., 18, 22, 27, 35, 61†, 130, 163, 176, 194; III., 13, 36, 41A, 42, 49, 50, 83, 87, 88, 121, 135, 138, 153, 181; IV., 18, 70, 79, 145, 163, 164, 174, 187, 197, 199, 200.

CALCULUS—

- Biliary—Males, I., 41; II., 198; IV., 24?; V., 21†. Females, I., 48; III., 136?; IV., 36†; V., 20†.
 Renal—Males, I., 170, 204, 303?; II., 244, 280; III., 104?; IV., 127? Females, II., 200?; III., 178.

CANCER—

- Abdominal—Males, IV., 164. Females, VI., 109, 175.
 Of Bones—Males, III., 46†, 143†; IV., 142? Females, I., 76†.
 Of Brain—Males, III., 46†.
 Of Dura Mater—Males, IV., 185†.
 Of Gall Bladder and Duct—Males, I., 37†, 96†; III., 42†. Females, I., 216; IV., 36†.
 Of Heart—Females, IV., 121†.
 Of Intestine—Males, III., 56†.
 Of Kidney—Males, III., 46†, 143†; IV., 136? Females, VI., 99.
 Of Liver—Males, I., 44†, 209?; II., 101†, 167†; III., 46†, 81†, 82†, 84†?, 119†, 143†; V., 10, 26†, 48†. Females, II., 54, 167, 196?; IV., 27†, 37†, 114†, 121†, 192†, 193?
 Of Lung—Males, I., 44†; III., 143†; IV., 142?; V., 48†. Females, II., 118; IV., 27†.
 Of Lymphatic Glands (primary)—Males, II., 101†; IV., 98†.
 Of Mediastinum—Males, I., 145†.
 Of Oesophagus—Males, I., 238?, 243; II., 137, 137A.
 Of Omentum—Males, I., 49?; III., 75?
 Of Ovary—Females, I., 76†; VI., 237†, 348†.
 Of Pancreas—Males, III., 84†?, 129†.
 Of Pelvis—Males, I., 44†; IV., 189†. Females, VI., 80, 99, 129, 147, 283†.
 Of Peritoneum—Males, I., 44†; III., 56†; IV., 71†?; V., 48†. Females, IV., 37†; VI., 184, 237†.
 Of Rectum—Males, III., 166; V., 48†. Females, II., 90; VI., 175.
 Of Spleen—Males, I., 44†.
 Of Stomach—Males, I., 49?, 134, 176†, 238?; II., 167†, 306†; III., 56†, 75, 77, 142; IV., 109. Females, II., 128†; IV., 83, 192†; V. 36.
 Of Supra-renals—Males, III., 46†. Females, IV., 121†.
 Of Thigh—Females, III., 204.
See also Uterine Cervix, Uterus, Vagina, Vulva.

CATARRH—Males, I., 23, 285.

CEREBELLAR—

- Abscess—Males, II., 43†.
 Tumour—Females, II., 129.

CEREBRAL—

- Abscess—Males, II., 157†; IV., 137†. Females, III., 12†.
 Aneurysm—Females, II., 152†; IV., 118†.
 Embolism—Males, I., 10†; II., 145†; IV., 4†. Females, I., 14, 26†, 192†, 211.; II., 115.
 Hæmorrhage—Males, I., 19; II., 127†; III., 232†, 236†; IV., 20†; V., 53†; Females, I., 32†; II., 152†; IV., 118†, 175†.
See also Meningeal.
 Mal-development—Females, IV., 35†.
 Syphilis—Males, I., 132; II., 135; III., 89; IV., 94, 133?; Females, III., 130.
 Thrombosis of Sinus—Males, IV., 137†, 229†.
 (or Cranial) Tumour—Males, I., 261†; II., 235, 259, 300; III., 9, 26?, 46†, 101†; IV., 185†; V., 19†. Females, I., 177; II., 63?, 92†; III., 55†; IV., 120†.

CHOLE-CYSTOTOMY—Males, I., 96†.

CHOREA—Males, II., 285; IV., 179, 217, 243, 247; V., 35, 38. Females, I., 33, 196; II., 133, 147, 149, 189; III., 15, 37, 68, 80, 194, 195; IV., 56, 154, 154A, 212; V., 24.

- With Pregnancy—Females, I., 47; III., 118.
 Treated with large doses of Arsenic—Females, I., 70.

- COLIC—Males, I., 12 ; II., 226, 261, 292. Females, I., 17, 42, 71, 187 ; III., 74.
- COLITIS—
 Follicular—Females, IV., 122†.
 Ulcerative—Females, II., 65†.
- COLON—
 Dilatation of—Females I., 220†.
 Typhoid Ulceration of—*See* Typhoid.
- CONJUGATE DEVIATION—Females III., 140 ; IV., 118†.
- CONSTIPATION—Males, I., 12, 180 ; III., 22. Females, I., 17, 116, 121, 128 ;
 II., 174 ; III., 9, 78, 101, 114 ; V., 23.
- CONVULSIONS—Females, II., 40, 55† ; III., 182†.
- CRANIOTOMY—Females, VI., 263.
- CRETINISM—Females, II., 154 (treated with Thyroid Extract).
- CYSTITIS—Males, II., 77 ; III., 173. Females, II., 12†, 83, 136, 192† ; III., 113†,
 209 ; VI., 322.
 Tubercular—Males, IV., 145†.
See also Pyuria.
- CYSTOCELE—Females, VI., 5, 14, 241, 253, 278, 316.
- DEBILITY—Males, I., 5, 97 ; II., 71, 263 ; III., 184, 207 ; V., 14. Females, I., 15,
 25, 27, 28, 98, 102, 107A, 127, 141 ; II., 153, 179 ; III., 183, 203 ;
 IV., 162, 191 ; VI., 123, 269.
- DELIRIUM TREMENS—Males, I., 165† ; II., 27†, 255 ; III., 215 ; IV., 19, 60 ;
 Females, III., 123.
- DEMENTIA—*See* Insanity.
- DERMATITIS—Females, I., 151†.
- DIABETES MELLITUS—Males, I., 4, 17, 163, 186, 289 ; II., 107, 122, 134, 270 ;
 III., 85† ; IV., 78†, 138, 242† ; V., 6. Females, I., 89 ; II., 76†, 195 ;
 III., 126, 193 ; IV., 136, 186.
- DIABETIC COMA—Males, III., 85†. Females, II., 76†.
 Treated with Subcutaneous Injections of Saline Solution—Females II., 76†.
- DIARRHŒA—Males, I., 162, 231, 240, 247, 250†, 257, 274, 283 ; III., 110, 123, 148 ;
 IV., 80, 126, 157, 226† ; V., 2, 7. Females, I., 119, 198, 202 ; II., 30, 68,
 75 ; III., 149, 179, 182†, 189† ; IV., 95, 185 ; V., 19.
 Choleraic—Males, III., 113.
- DIPHTHERIA—
 Treated without Antitoxin—Males, III., 85† ; IV., 220†. Females,
 III., 216† ; IV., 208 ; V., 11, 14†. *Radcliffe*—Females, 42, 44.
 Treated with Antitoxin—*Radcliffe*—Males, 1, 2, 3†, 8, 9, 10†, 11†, 13, 16,
 17†, 18†, 19, 20, 22†, 24†, 31, 34, 39† ?, 47, 52, 54, 55, 56†, 62, 63,
 66†, 68, 69, 71, 74, 75†, 76†, 77†, 78†, 79, 80, 84†, 85, 86, 88†, 90†,
 91†, 92†, 93†, 94†. Females, 4?, 12, 14, 15, 21, 23, 25†, 32†, 35, 36,
 38†, 40, 41, 43, 45, 48, 49, 50, 51, 53†, 57, 58†, 59†, 61, 64†, 65†, 67,
 70, 72, 73†, 81, 83, 87, 89†.
 Sequelæ of—Males, I., 36 ; II., 29† (Hemiplegia).
See Paralysis, Diphtheritic.
- DIPHTHERITIC CONJUNCTIVITIS—*Radcliffe*—Males, 60†.
- DOLICHO-CEPHALUS—Females, II., 23.
- DYSMENORRHŒA—Females, VI., 20, 28, 50, 153, 165, 267.
- DYSPAREUNIA—Females, VI., 48, 294.
- DYSPEPSIA—Males, III., 124 ; IV., 22. Females, I., 6 ; II., 9, 134, 156 ;
 III., 5, 21, 40 ; VI., 12, 280.
- DYSPHAGIA—Females, IV., 146.
See also Oesophageal Cancer, Stricture.

- ECTHYMA CONTAGIOSUM—Females, II., 66†.
- ECZEMA—Males, II., 237†; III., 98. Females, I., 113; II., 125, 159; III., 181.
Radcliffe—Females, 42.
- EMBOLISM—
Cerebral—Males, I., 10†; II., 145†; IV., 4†. Females, I., 14, 26†, 192†, 211; II., 115.
Various—Males, II., 145† (Liver, Intestine), 304†. Females, I., 157† (Popliteal).
See also Infarction.
- EMPHYSEMA—Males, I., 25, 33, 80, 191, 233, 314†; II., 33, 49, 54, 86, 109, 125, 140, 283, 289; III., 31, 67, 242†; IV., 10, 11†, 35, 61, 73†, 84†, 85, 86, 89, 110, 139, 170. Females, I., 104; II., 18, 22, 130; III., 121, 135, 135A†; IV., 18, 75†.
- EMPHYEMA—Males, I., 47, 143, 165†, 183, 199†, 229, 230, 272, 275†; II., 3, 121, 136, 258, 298; III., 1, 135†, 188; IV., 78†, 116, 134†, 135, 175, 211, 230, 256. Females, I., 72†, 152, 195†; II., 67†?, 72, 80, 160†; III., 57?, 117†, 133†, 164†; IV., 24, 101; VI., 140†.
Sequelæ of—Males, I., 190; III., 49.
With Resection of Ribs—Males, I., 47, 143, 183, 229, 230, 272, 275†; II., 121, 258; III., 1, 135†, 188; IV., 175, 211, 230, 256. Females, I., 72†, 152; II., 72, 80; IV., 24, 101.
- ENDO-CARDITIS, Ulcerative or Infective—Males, I., 10†, 76†, 194† (Typhoid); II., 143†, 145†, 304†; III., 167†, 232†; IV., 4†. Females, I., 26†, 157†, 192†; III., 12†.
- ENDO-METRITIS—Females, VI., 15, 58, 235, 240.
Fungosa—Females, VI., 30, 34, 39, 136, 221.
Gonorrhœal—Females, VI., 142.
Purulent—Females, VI., 142.
- ENTERITIS—Males, I., 149, 237; II., 118 (Fibrinous); III., 90, 175; IV., 233†. Females, I., 220†; IV., 7.
Tubercular—Males I., 118†, 120†, 224†, 256†; II., 42†; III., 155†, 197†, 201†; IV., 42†, 155†. Females, I., 62†, 109†; II., 3†, 20†, 104†; IV., 113†.
- EPILEPSY—Males, I., 22, 213, 213A, 295, 304; II., 252, 256; III., 55†, 214; IV., 58. Females, I., 125; III., 33, 123.
Jacksonian—Males, II., 235, 259.
With Status Epilepticus—Males, III., 55†. Females, I., 125.
Sequelæ of—Males, I., 310 (Mania); II., 256. Females, I., 122? (hemiplegia), 125 (Mania).
- EPISTAXIS—Males, II., 186; III., 4†; IV., 4†, 27.
- ERYSIPELAS—Males, II., 104. Females, II., 184; III., 201†; IV., 207.
- ERYTHEMA—Males, I., 65; II., 124, 133, 175, 179, 225; III., 34, 128†; IV., 193†, 205†, 207, 222. Females, I., 51, 79†; II., 16, 21, 135, 186; III., 19, 201, 218; IV., 28, 54†, 157, 189. *Radcliffe*—Males, 29, 33; Females, 37.
Multiforme—Females, III., 146.
Nodosum—Males, II., 2. Females, 1; II., 65†; III., 82, 167, 186; IV., 40, 127, 161, 180; V., 31.
After Serum Injection—Males, II., 133. Females, III., 201. *Radcliffe*—Males, 39†, 86; Females, 12, 70.
Tuberculatum—Females, II., 162.
- EXOPHTHALMIC GOITRE—Females, I., 101, 214; II., 71, 123; III., 2?, 14?, 84; V., 1, 31; VI., 323.
Treated with Supra-renal Extract—Females, I., 101.
Treated with Thymus Extract—Females, V., 1.
- EXOPHTHALMOS—Females, II., 61†.
- FEBRICULA—Males, I., 158; II., 38, 148, 185, 193. Females, II., 86, 111, 136, 171, 172, 181; III., 34, 152; IV., 13, 39, 144.

FEVER—Males, III., 205†. Females, III., 79†.

FISTULA—

Bronchial—Males, II., 3 ; IV., 116. Females, II., 80 ; IV., 135.

Recto-Vaginal—Females, VI., 51.

Utero-Vesical—Females, VI., 259.

Vesico-Vaginal—Females, VI., 153.

FOLLICULITIS, Pustular—Females, III., 107.

FRIEDREICH'S DISEASE—Females, IV., 195.

GALL BLADDER—

Cancer of—Males, I., 37†, 96† ; III., 42†. Females, I., 216 ; IV., 36†.

Dilated—Males, I., 96† ; II., 101† ; III., 84†, 129†, 136.

GASTRIC CRISES—Males, IV., 173. Females, III., 209.

GASTRIC ULCER—Males, I., 187 ; III., 25, 29, 62, 77 ; IV., 183 ; V., 54. Females, I., 23, 30, 50, 88, 91, 103, 134, 145?, 164, 193, 194†, 203 ; II., 10†, 56, 59, 95?, 100?, 120, 138, 141, 177, 192† ; III., 3, 21?, 28, 39, 40?, 56, 139, 171, 191, 196, 199, 220, 221, 225 ; IV., 1, 51, 53, 73, 131, 147?, 190 ; V., 16, 22, 25, 26, 29.

GASTRITIS—Females, III., 94.

GASTR-ODYNIA—Males, II., 89 ; IV., 6. Females, I., 57, 98, 107, 107A, 116, 121, 210 ; II., 44 ; III., 103 ; IV., 147.

GASTRO-ENTERITIS—Males, IV., 154 ; V., 4. Females, I., 129 ; II., 193 ; III., 143† ; IV., 173.

GLYCOSURIA—Males, I., 11† ; IV., 117.

See also Diabetes Mellitus.

GOUT—Males, I., 117 ; II., 33, 267, 289 ; III., 15, 44, 63, 68, 116, 178, 235† ; IV., 195†, 199, 201, 203, 248. Females, III., 103, 150.

HÆMATEMESIS—Males, III., 29, 33, 147, 225 ; IV., 112, 183. Females, I., 23, 88, 103 ; II., 192† ; III., 39, 56, 134†, 191, 196, 199, 220, 225 ; IV., 51, 53, 73, 131, 141, 176?, 190.

HÆMATO-MYELIA—Males, V., 17?

HÆMATURIA—Males, I., 170 ; II., 244, 274 ; III., 104 ; IV., 194, 239 ; V., 3 ; Females, I., 100 ; III., 134†.

Paroxysmal—Males, I., 106 ; II., 18, 55.

HÆMOPTYSIS—Males, I., 77, 83, 99†, 124, 171†, 179 ; II., 76, 87 ; III., 50, 140†, 168†, 182 ; IV., 21, 25, 90, 92, 107, 149†, 188, 195†, 205†, 206, 237, 239. Females, II., 130 ; III., 18 ; IV., 64, 176, 179†, 198†, 204. *Radcliffe*—Females, 67.

HEADACHE—Males, III., 138 ; IV., 163. Females, III., 89.

HEART DISEASE—Males, I., 99†. Females, I., 188 ; II., 114, 115.

Asthenia—Males, IV., 177, 255.

Congenital—Males, I., 182? ; II., 15 ; III., 238. Females, I., 84? ; II., 46, 114?

Dilatation—Males, I., 57†, 94, 137, 205, 245 ; II., 50 ; III., 13, 20, 106, 235† ; IV., 73†, 84†, 170. Females, III., 91, 135, 135A† ; IV., 78.

Fatty Degeneration—Females, I., 29†.

Tachycardia—Males, III., 97.

VALVULAR—

Aortic, Obstructive—Males, II., 103, 204. Females, I., 161, 204, 213.

Regurgitant—Males, I., 181 ; II., 102, 169†, 285 ; III., 91, 96, 183, 217, 222. Females, I., 14 ; II., 126 ; III., 128, 159.

Double—Males, I., 60, 64, 106, 281 ; II., 7, 211†, 216 ; III., 66 ; IV., 45, 95†.

HEART—VALVULAR—*continued*.

Mitral, Obstructive—Males, I., 156; II., 88, 143†; III., 33; IV., 195†; Females, II., 6, 64A; III., 18, 96, 168, 208, 215; IV., 23†, 58, 75†, 198†, 204.

Regurgitant—Males, I., 73, 189, 248, 287; II., 22, 28, 41, 77, 116, 215, 240, 291; III., 63, 126, 185; IV., 113, 141†, 171, 244, 247; V., 43. Females, I., 38, 85, 106, 108, 206†, 211; II., 28, 59, 82?, 162, 190; III., 53, 68, 71, 84, 150, 170, 205, 212; IV., 19, 28, 31, 34†, 100, 203; V., 18†, 21, 28, 30.

Double—Males, I., 184, 193, 225, 293, 300; II., 138, 202, 212; III., 38, 59, 61, 94, 123†; IV., 63, 165, 169; V., 8. Females, I., 115, 189, 215; II., 52, 107, 143, 146, 180, 187; III., 26, 51, 83, 178; IV., 133, 139, 140. *Radcliffe*—Females, 67.

Mitral and Aortic—Males, I., 2†, 83, 105, 135, 154, 169, 171†, 241, 290, 299; II., 1†, 85, 194†, 214, 297†; III., 11, 30, 65; IV., 17, 123, 172†, 176†, 191†, 240; V., 47, 50†. Females, I., 61, 97, 174, 219; II., 16; III., 85, 86, 128?, 129, 132, 174; IV., 19A, 36†, 44, 148.

Mitral and Tricuspid—Males, I., 126†; II., 70†; IV., 36†, 88. Females, I., 53†, 179†; II., 64; III., 51A, 128?, 156, 161; IV., 177†.

Tricuspid—Females, III., 121.

See also Endo-carditis, Ulcerative.

HEMI-ANÆSTHESIA—Males, II., 21, 84. Females, I., 137.

HEMIPLEGIA—Males, I., 10†, 19, 84†, 221; II., 84, 135, 294, 299; III., 89, 154; IV., 4†, 239; V., 29. Females, I., 14, 26†, 122, 137, 201†, 211; II., 69, 115.

Hysterical—Males, II., 21. Females, III., 132.

HYDRO-CEPHALUS—Males, I., 98. Females, I., 131.

HYDRO-NEPHROSIS—Males, IV., 127; V., 31†. Females, I., 60; IV., 41; VI., 170.

HYDRO-SALPINX—Females, VI., 6, 292.

HYSTERECTOMY—

Abdominal—Females, VI., 17A, 150, 155, 229†, 327†, 361.

Vaginal—Females, VI., 21, 52, 220, 289.

HYSTERIA—Males, II., 21, 58, 222; III., 16, 45. Females, I., 165?; III., 132, 162?, 199; IV., 32; VI., 11, 24, 276.

With Contraction of Field of Vision—Males, II., 21. Females, III., 162. Treated with Weir Mitchell's Method—Females, III., 199.

See Neuro-mimesis.

IMBECILITY—Females, I., 168; IV., 35†; V., 5.

IMMERSION—Males, I., 309†.

INFARCTION—*See* Intestine, Kidney, Liver, Lung, Spleen.

INFLUENZA—Males, I., 35, 39, 40, 71, 75, 196, 294?; II., 12?, 39, 48, 51, 53, 67, 80?, 82; III., 32; IV., 39, 56, 66?, 72; V., 49. Females, I., 5, 38, 43, 44, 46, 54, 58; II., 29, 38, 49; III., 45, 46, 47, 51, 58, 60; IV., 10, 11, 15, 46, 49.

Sequelæ of—Females, I., 66?; III., 89, 94; VI., 165.

INSANITY—Males, I., 115, 202; II., 187; III., 150; IV., 128. Females, II., 21, 75, 124; III., 166; IV., 9, 86, 92, 124.

Dementia—Males, III., 145. Females, II., 178.

Mania—Males, I., 310 (Epileptic); II., 207, 256 (Epileptic); III., 233. Females, I., 125 (Epileptic), 217 (? Hysterical); III., 192; VI., 272 (Puerperal).

Melancholia—Males, II., 6.

INTESTINE—

Dilatation of—Females, II., 93.

Embolism of—Males, II., 145†; III., 167†. Females, III., 12†

Hæmorrhage in—Females, IV., 110.

INTESTINE—*continued.*

Int us-susception of—Males, II., 215 ?, 245 ; IV., 168 ? Females, I., 20 †.
 Obstruction of—Males, II., 182 † (Volvulus). Females, VI., 72.
 Tape worm in—Females, II., 79.
See Colon, Enteritis, Gastro-enteritis, Perforation, Rectum.

INTUBATION—*See* Larynx, Œsophagus.

JAUNDICE—Males, I., 38, 41, 92, 99 †, 106, 138 ; II., 90, 114, 178, 198, 242, 278, 282 ; III., 82 †, 84 †, 129 † ; IV., 156, 164. Females, I., 48, 216 ; II., 31, 31A, 53, 74, 122 ; III., 136 ; IV., 71.
 Treated with cold Enemata—Males, II., 90, 114. Females, II., 74.
 With Rigors—Males, I., 149.

KIDNEY—

Cancer of—Males, III., 46 †, 143 † ; IV., 136 ? Females, VI., 99 ?
 Cystic disease of—Males, V., 51 †.
 Floating—Females, II., 6 ; III., 65, 219 ; IV., 41.
 Infarction of—Males, I., 194 † ; II., 304 † ; III., 167 †. Females, I., 192 † ; IV., 36 †.
See Calculus, Hydro-nephrosis, Nephrectomy, Nephritis, Pyelitis, Pyo-nephrosis.

LABOUR—Females, VI., 360.

Induced—Females, VI., 1, 3, 22, 38, 138.
 Obstructed—Females, VI., 22 †, 263.
 With Craniotomy—Females, VI., 263.
 Premature—Females, VI., 6 †, 88.

LAPAROTOMY—Males, II., 168 †, 279 † ; III., 14 † ; IV., 122. Females, I., 194 † ; II., 10 † ; VI., 25, 37, 42, 72, 76, 99, 101 †, 150, 170, 193 †, 237 †, 247, 348 †, 357.

LARYNGISMUS—Females, II., 97.

LARYNGITIS—Males, I., 46, 56, 63, 79 †, 172, 253, 258 ; II., 46, 81 ; III., 27, 48 ; IV., 45. Females, I., 105, 148 ; II., 26, 144 ; III., 32, 160 ; IV., 29, 42, 44, 61, 199. *Radcliffe*—Males, 6, 30, 82.
 Phlegmonous—Females, VI., 140 †.
 Syphilitic—Females, I., 64 ; III., 97 †.
 Tubercular—Males, I., 120 † ; II., 42 †, 207 ; III., 6, 64 †, 218 † ; IV., 42 †, 52, 57, 143 †, 155 † ; V., 25 †, 36. Females, I., 31 † ; IV., 81 †.
 For affection of Larynx in Typhoid, *see* Typhoid.

LARYNX—

Intubation of—Males, II., 46, 239 † ; *Radcliffe*—Males, 1, 63, 66 †, 69, 75 †, 80. Females, 21, 44, 48, 59 †, 70, 73 †.
 Papilloma of—Males, II., 239 †.
 Paralysis of—Males, II., 137 ; III., 51, 66. Females, II., 150.
 Stricture of—*Radcliffe*—Females, 44.

LEAD POISONING—Males, II., 267 ; III., 178.

Colic—Males, II., 176, 230 ; III., 2, 15 ; V., 23.
 Encephalopathy—Males, III., 171 ?
 Paralysis—Males, II., 260 ; III., 2, 88 ; IV., 48 ? ; V., 55.

LEUCOCYTHÆMIA—Males, IV., 47.

LICHEN PLANUS—Females, III., 100.

LIVER—

Abscess of—Males, I., 207 †. Females, IV., 20 †.
 Cancer of—Males, I., 44 †, 209 ? ; II., 101 †, 167 † ; III., 46 †, 81 †, 82 †, 84 † ?, 119 †, 143 † ; V., 10, 26 †, 48 †. Females, II., 54, 167, 196 ? ; IV., 27 †, 37 †, 114 †, 121 †, 192 †, 193 ?

LIVER—*continued.*

- Cirrhosis of—Males, I., 89?, 236†; II., 33, 115†; III., 5, 17, 39, 102†, 122, 163†, 179, 234†, 235†; IV., 112, 261; V., 21†, 24. Females, I., 11†, 29†, 142, 175; II., 24†, 31, 31A, 119, 140†, 196; III., 62†, 198; IV., 6, 17†, 23†, 71, 71A, 176; VI., 302.
- Enlargement of—Males, I., 80, 99†, 182, 184, 209; II., 63†, 129, 140, 169†, 202, 209; III., 84†, 198; IV., 141†, 151, 191†, 223. Females, I., 26†; II., 39, 119; III., 62†, 69, 86, 129, 136, 178. IV., 6, 17†, 44, 71, 176.
- Hydatid Cyst of—Males, II., 139†. Females, II., 202†; IV., 33, 193?
- Infarction of—Males, II., 145†.
- Peri-hepatitis—Males, II., 115†.
- Syphilitic disease of—Males, I., 182?; II., 129?; III., 102†?; IV., 228? Females, II., 39; III., 163.

LUMBAGO—Males, II., 36.

LUNG—

- Cirrhosis of—Males, IV., 134†.
- Collapse of—Males, I., 270†; IV., 15, 252†.
- Fibrosis of—Males, I., 315; II., 78; III., 99. Females, II., 183; IV., 64.
- Gangrene of—Males, III., 140†.
- Infarction of—Males, I., 2†, 76†, 169?; II., 70†; III., 38?; IV., 11†, 172†, 176†, 229†; V., 56†. Females, IV., 198†. *Radcliffe*—Females, 67.
- Pyæmic—Males, I., 21†, 146†. Females, II., 109†; III., 12†.

LUPUS—Males, II., 86. Females, III., 201 (treated with Tuberculin).

LYMPHADENOMA—Males, I., 110; IV., 105†, 145?

MANIA—*See* Insanity.

MARASMUS—Males, II., 44, 253; III., 193†, 195; IV., 216†; V., 43, 46†. Females, IV., 206; V., 10†; VI., 192.

MEASLES—Males, I., 159, 242, 291, 311; II., 239†, 254; III., 211, 213; IV., 197. Females, I., 140, 160†; II., 42, 151†, 158; V., 2. *Radcliffe*—Males, 54, 85. Females, 61, 64†, 70, 73†.

MEDIASTINUM—

- Abscess of—Males, III., 201†.
- Tumour of—Males, I., 108, 145†; III., 51, 73, 73A, 182. Females, II., 150, 150A.
- See* Aneurysm.

MELÆNA—Females, I., 23; II., 192†.

MELANCHOLIA—*See* Insanity.

MELASMA—Males, II., 106. Females, II., 123; III., 175, 223.

MENINGEAL HÆMORRHAGE—Males, I., 11† (Sub-dural). Females, I., 192†.

MENINGITIS—Males, I., 269, 275†; II., 174, 177, 181†, 257?; III., 241; IV., 131, 210, 219. Females, II., 57, 63?; III., 61, 130, 140; IV., 149, 174?; V., 20†, 32.

Cerebro-spinal—Males, I., 42.

Purulent—Males, II., 157†, 248†; IV., 137†. Females, I., 55†; II., 197†.

Tubercular—Males, I., 101†, 224†, 261†; II., 30†; III., 114†. Females, I., 69†, 109†; II., 3†; III., 157†; IV., 22†, 45†, 165†?; V., 8†.

MENORRHAGIA—Females, VI., 27A, 213.

MORPHINISM—Males, IV., 128.

MUSCULAR ATROPHY—

Idiopathic—Males, II., 35; IV., 87. Females, II., 96.

Neural—Males, II., 96?; III., 240; IV., 48?, 132, 185†; V., 55. Females II., 91, 185.

Spinal—Males, II., 61, 290?; IV., 48? Females, IV., 146.

Of doubtful origin—Males, IV., 254. Females, IV., 143.

See also Neuritis.

MUSCULAR SPASM—Males, II., 272.

See also Torti-collis.

MYALGIA—Males, III., 28.

MYELITIS—Males, IV., 130?

Chronic—Males, III., 53. Females, III., 162.

See Paralysis, Paraplegia, Polio-myelitis.

MYXŒDEMA—Females, II., 88†? ; III., 188?, 224.

NEPHRECTOMY—Females, VI., 170.

NEPHRITIS—

Acute—Males, I., 81, 110, 259, 278, 291? ; II., 184, 219, 274?, 304† ; III., 7, 100, 160, 231 ; V., 41, 42. Females, I., 100?, 153 ; II., 125 ; IV., 70, 166†, 168, 196 ; V., 13 ; VI., 1.

Chronic—Males, I., 8, 11†, 30†, 32, 59†, 68, 70, 72, 82, 86†, 140†, 176†, 232, 239†, 251, 314† ; II., 17†, 69, 78, 94, 100, 104, 127†, 143†, 158†, 180†, 187, 195, 204, 214, 305† ; III., 63, 71†, 112, 118, 164, 171, 178, 179 ; IV., 5†, 11†, 20†, 32†, 33, 44†, 46†, 50, 76†, 82, 83, 117, 134†, 195†, 233†, 284. Females, I., 2, 41, 45†, 66, 94, 137, 150, 156†, 179†, 191†, 197†, 218† ; II., 78, 85?, 88†, 119, 162 ; III., 24, 59†, 72†, 150, 155†, 159, 190† ; IV., 66, 108, 112, 118†, 165†, 175†, 211† ; VI., 22, 305, 331†.

NERVE-STRETCHING—Females, II., 170 (Trigeminal).

NEURALGIA—

Of Anterior Crural—Males, I., 185.

Cervico-brachial—Males, II., 149, 201.

Trigeminal—Females, II., 170 ; III., 165.

See Sciatica.

NEURITIS—

Multiple—Males, I., 24 (? syphilitic), 195 ; II., 16, 24, 96? ; III., 105† ; IV., 133? ; V., 33. Females, II., 62, 91, 164 ; III., 17?, 62†, 76, 81 ; IV., 17†, 44, 92, 106, 153.

Solitary, of Ocular Nerves—Males, II., 141 (? rheumatic).

—of Ulnar Nerve—Males, II., 220.

NEURO-MIMESIS—Males, I., 87† ; IV., 148. Females, VI., 280.

NYSTAGMUS—Males, II., 302 ; III., 101†, 103, 241. Females, II., 69, 173 ; IV., 93, 111.

ŒDEMA GLOTTIDIS—Females, VI., 140†.

ŒSOPHAGUS—

Cancer of—Males, I., 238?, 243 ; II., 137, 137A.

Obstruction or Stricture of—Males, I., 238 ; II., 23, 59, 150, 151 ; III., 66, 68?, 152 ; IV., 98†.

—Treated by Intubation—Males, II., 137, 150.

OFFENSIVE EXPECTORATION—Males, II., 3 ; IV., 116, 188. Females, II., 80 ; IV., 135.

ŒPHORECTOMY—Females, VI., 6, 37, 82, 124, 231, 254.

OPHTHALMO-PLEGIA—Males, II., 171.

OPISTHONUS—Males, I., 276 (cervical).

See also Meningitis.

OPTIC ATROPHY—Males, I., 52 ; II., 128 ; III., 181 ; IV., 200. Females, II., 89, 173.

OPTIC NEURITIS—Males, I., 16, 21†, 101† ; II., 43†, 174, 177, 219, 235, 257, 300 ; III., 9, 101†, 114† ; IV., 94, 137†, 210 ; V., 19†. Females, I., 26†, 55†, 177 ; II., 4†, 57, 63, 92†, 129 ; III., 55†, 140, 157†, 216† ; IV., 120†, 149.

OSTEO-ARTHRITIS—Males, I., 64 ; III., 170. Females, II., 36, 43, 117?, 124 ; III., 142, 176, 184, 215.

OTITIS—Males, I., 21†, 72, 119, 133, 150†, 224†, 226, 228, 261†, 275†; II., 31, 43†, 159†, 186, 232; III., 117, 227; IV., 137†, 161, 214, 233†. Females, I., 55†; II., 51, 182†; III., 12†, 117†, 133†, 181; IV., 82.

OVARIOTOMY—Females, VI., 13, 45, 49, 57, 74, 88, 106, 118, 125, 126†, 137, 139A†, 141, 149, 150, 162, 171, 176, 228, 230, 232, 234, 246, 255, 260†, 265, 270, 292, 326, 353, 358.

OVARY—

Abscess of—Females, IV., 113†; VI., 110†, 154†.

Cancer of—Females, I., 76†; VI., 237†, 348†.

Prolapse of—Females, VI., 306, 308.

Tumour of—Females, II., 115; IV., 166†; VI., 13, 20, 45, 49, 57, 74, 88, 106, 118, 125, 126†, 137, 139A†, 141, 149, 150, 162, 171, 173, 176, 226, 228, 230, 232, 234, 246, 255, 260†, 265, 270, 292, 326, 353, 358.

See Oöphorectomy.

PANCREAS—

Cancer of—Males, III., 84†?, 129†.

Cirrhosis of—Males, IV., 78†.

PARALYSIS—

Agitans—Males, II., 34, 113, 277.

Bulbar—Males, I., 282? Females, IV., 146.

Of Cranial Nerves—Males, III., 89; IV., 94, 185†.

Diphtheritic—Males, II., 147, 183; IV., 203†. Females, I., 4, 49; III., 1, 17?

Radcliffe—Males, 55, 94†. Females, 73†.

Facial—Males, I., 133; II., 269; III., 92; IV., 94. Females, III., 110, 130, 157†.

General, of the Insane—Males, I., 9, 115; II., 84. Females, II., 69.

Infantile Spinal—Males, II., 275.

Ocular—Males, II., 141, 174, 177, 300; III., 26, 89, 103, 171; IV., 94, 137†, 185†. Females, II., 92†, 129; III., 157†; IV., 146.

See also Ophthalmoplegia.

Of Sympathetic Nerve—Males, IV., 142.

PARAMETRIC—

Abscess—Females, VI., 197, 300.

Phlegmon.—Females, VI., 90, 108?, 164, 210.

PARA-METRITIS—Females, VI., 69, 90, 257, 296.

PARAPLEGIA—Males, II., 73, 273; IV., 130. Females, III., 113†.

Hysterical—Males, II., 222; III., 16. Females, III., 17?

Spastic—Males, II., 79, 302; V., 17, 18. Females, V., 5.

PAROTITIS—Females, II., 192†; VI., 301.

(Mumps)—Males, II., 19.

PELIOSIS RHEUMATICA—Females, III., 53.

PELVIC—

Abscess—Females, VI., 36, 154.

Cancer—Males, I., 44†; IV., 189†. Females, VI., 80, 99, 129, 147, 283†.

Hæmatocele—Females, VI., 42, 72, 76, 246.

Hæmatoma—Females, VI., 89, 128?

Inflammation—Females, VI., 23.

Pain—Females, III., 30; VI., 29, 40, 53, 94, 124, 161.

Tumour (of doubtful nature)—Females, I., 60; VI., 18, 93, 128, 212, 299.

PELVIS, Contracted—Females, VI., 38, 138, 160 (Nägele's).

PERFORATION—

Of Intestine—Males, I., 20†, 120†, 222†, 317†; II., 168†, 208†; IV., 13†, 162†, 199†. Females, I., 166†; II., 65†; IV., 54†.

Of Stomach—Males, II., 167†. Females, I., 194†; II., 10†, 192†.

- PERICARDITIS—Males, I., 151?, 214†, 248, 292† (tubercular); II., 28, 32, 144†, 248†; III., 11, 116, 185; IV., 153†, 166, 207; V., 56†. Females, I., 108, 219; II., 119, 190; III., 59†, 84, 98†, 117†, 128, 178; IV., 113†; V., 31; VI., 140†.
- PERICARDIUM, Adherent—Males, I., 140†, 171†; III., 94?, 112?, 128†, 235†. Females, I., 115?, 206†.
- PERI-METRIC ABSCESS—Females, VI., 69.
- PERI-METRITIS—Females, I., 92; III., 43, 75; VI., 2, 7, 10, 31, 68, 68A, 69, 75, 78, 83, 84, 90, 95, 97, 104, 117, 127, 131, 139, 182, 207, 209, 250, 256, 262, 272, 273, 274, 285, 301, 312, 317, 325, 351.
Sequelæ of—Females, VI., 195, 198.
- PERINÆUM—Repaired or Ruptured—Females, VI., 5, 14, 51, 70, 77, 81, 100, 122, 144, 146, 158, 166, 180, 188, 199, 199A, 205, 225, 227, 241, 245, 251, 253, 279, 297, 316, 320, 339.
- PERI-OSTEITIS—Males, II., 221. Females, I., 34, 39, 97; IV., 189.
- PERITONEAL ABSCESS—Males, I., 207†; II., 163†; IV., 46†. Females, VI., 139A†. Adhesions—Females, V., 33.
- PERITONITIS—Males, II., 140; IV., 46†; V., 15. Females, I., 220†.
Acute—Males, II., 104, 144†, 203; IV., 233†.
—Septic—Males, I., 20†, 222†; II., 167†, 182†, 208†; III., 14†, 167†; IV., 13†, 99†, 122, 162†, 199†. Females, I., 166†, 194†; II., 65†; VI., 101†, 110†, 139A†.
Chronic—Males, IV., 71†, 114.
Local—Males, I., 160; II., 13. Females, IV., 74.
Tubercular—Males, I., 53, 121, 249?, 292†; II., 163†, 168†; III., 180, 197†, 216; IV., 69?, 145†, 220†. Females, I., 77A†, 78; II., 104†; III., 127?; IV., 85†, 113†, 156†.
- PERITYPHLITIC ABSCESS—Males, I., 27; III., 14†; IV., 99†, 181.
- PERI-TYPHLITIS—Males, I., 123, 157; II., 13?, 123, 131, 191; III., 19, 162; IV., 43, 51, 53, 54, 118, 180, 196; V., 15? Females, I., 145†, 166†, 192†, 209; IV., 63.
Sequelæ of—Males, IV., 238. Females, I., 141.
- PERTUSSIS—Males, I., 66, 67†, 254; II., 25†, 74, 206, 248†; III., 239†, 242†; IV., 67†, 227. Females, I., 51; II., 27, 55†, 66†; III., 124, 145; IV., 89, 164.
- PHLEBITIS—Males, I., 21†.
See Thrombosis.
- PHTHISIS PULMONALIS—Males, I., 7†, 58†, 62†, 76†, 77?, 83, 100, 113, 118†, 120†, 124, 147, 150†, 166, 167, 178†, 179, 190, 199†, 201, 211; II., 42†, 76, 154, 188, 199, 207, 218†, 258, 286, 296†; III., 1?, 6, 40, 41, 47, 50, 57?, 64†, 68, 93, 99, 105†, 122?, 155†, 168†, 218†; IV., 42†, 49†, 62, 64?, 90, 92, 97, 107, 108, 111, 115, 119, 125, 143†, 145†, 149†, 155†, 202†; V., 21†, 25†, 40†. Females, I., 31†, 56, 62†, 77, 77A†; II., 1†, 20†, 41, 66†, 67†, 87†, 109†, 160†, 166; III., 8, 18, 54, 57, 69, 93, 120, 131, 187, 207; IV., 2, 3, 68, 81†, 85†, 113†, 115, 145?, 167†, 182, 211†; V., 3, 9†; VI., 274.
With Pneumonia—Males, III., 155†.
Fibroid—Females, IV., 34†, 179†.
- PITYRIASIS RUBRA PILARIS—Males, III., 132.
- PLACENTA—
Polypus of—Females, VI., 43.
Retained—Females, VI., 159, 248.
- PLEURISY—Males, I., 13, 43, 81, 88, 111, 114, 117, 128, 137, 164, 167, 241, 246, 296; II., 32, 99, 130, 144†, 188, 199; III., 6, 31, 84†, 99, 111, 115, 125, 133, 229; IV., 38, 75, 130, 146, 153, 170, 190†, 233†; V., 56†. Females, I., 90, 132†; II., 116, 119, 121, 183, 192†; III., 8, 141; IV., 64, 129, 198†.
Sequelæ of—Males, I., 315. Females, IV., 5.

PLEURITIC EFFUSION—Males, I., 18, 45, 52, 54, 89, 103, 130, 153, 175, 201, 266, 292†; II., 4, 8, 37, 41, 64, 143†, 154, 155, 170, 175, 200, 205, 228, 248†, 264; III., 58, 69, 71†, 74, 140†, 173; IV., 40†, 41, 55, 77, 97, 101, 104, 106, 121, 144, 150, 158, 166, 213, 229†. Females, I., 22, 77, 77A†, 82; II., 54, 91, 127, 182†; III., 48, 111, 127, 177, 178; IV., 80, 158, 204, 210.

Sequelæ of—Males, II., 152.

PLEUR-ODYNIA—Females, II., 47; IV., 20, 20A., 69, 90.

PNEUMONIA—

Catarrhal—Males, I., 55, 74, 79†, 81, 88, 112, 122, 155†, 161, 188, 228, 232, 252, 254, 255, 260, 262, 264, 265, 268, 273, 306, 307, 308, 316?; II., 29, 65, 66, 83, 98, 105†, 124, 142†, 158†, 160, 199, 206, 238, 246†, 248†; III., 32, 117, 144, 174, 176, 177, 191†, 192, 194†, 199, 200, 202†, 203, 204, 206, 208, 208A†, 210†, 211, 212, 221, 239†; IV., 12, 26, 67, 74, 125, 126, 140, 190†, 203†, 212, 221†, 222, 223, 224, 227, 229†, 231†, 232; V., 44, 45. Females, I., 8, 18, 44, 58, 67, 73, 75, 93, 99†, 117†, 132†, 155, 160†, 186, 195†; II., 55†, 151†, 190; III., 11, 117†, 133†, 140, 145, 169, 217; IV., 8†, 30†, 43, 57, 60†, 62, 76†, 88, 96, 134†, 138, 165†, 170, 201†; V., 14†, 34†. *Radcliffe*—Males, 3†, 5, 10†, 78†, 85, 93†. Females, 38†, 64†, 65†, 73†.

Croupous—Males, I., 78, 85†, 127, 129, 136, 148, 165†, 168, 173, 197, 214†, 216, 217, 218, 263, 271, 284†, 313†; II., 52†, 60, 72, 63†, 108, 111, 117, 121, 132, 144†, 146, 156, 172, 209, 232, 241†, 249, 265, 281; III., 35, 72, 78, 86, 95, 107, 127, 141†, 151, 158, 159, 190, 209, 228; IV., 1, 2, 9, 16, 19, 30†, 31, 65, 68, 70, 81, 96, 120, 129, 147, 159, 160, 161, 167, 178, 234†, 235, 245, 246, 251; V., 20†, 39. Females, I., 36, 83, 138, 154, 167, 200; II., 50, 81, 98, 101, 102, 112, 132, 157, 188, 198, 199; III., 148, 202; IV., 55, 67, 105, 128, 169; V., 12, 17.

—Chronic—Females, II., 17.

—Treated with Ice Poultice—Males, I., 50, 51. Females, I., 138.

—With Abortion—Females, II., 50.

Of Doubtful Nature—Males, I., 152, 169; II., 80, 165; III., 57, 80, 140†; IV., 184, 187†; V., 23. Females, I., 158; II., 5; III., 104, 135A†; V., 15.

Septic—Males, I., 146†; Females, I., 169?; II., 109†, 182†; IV., 23†.

Radcliffe—Males, 75†.

Sequelæ of—Females, IV., 86.

PNEUMO-THORAX—Males, II., 258; III., 3. Females, I., 56, 77; II., 54, 160†; III., 48.

POISONING—

By Hydrochloric Acid—Males, I., 198; IV., 59.

By Petroleum—Males, I., 242.

By Chromate of Potash—Males, III., 121†.

By Oxalic Acid—Males, IV., 93.

By Mercury—Males III., 219. Females., VI., 281?, 291, 329?, 346.

By Carbolic Acid—Females, I., 80, 190†.

POLIO-MYELITIS, Anterior—Males, II., 290?; IV., 48?

PREGNANCY—Females, I., 47; III., 118; V., 27; VI., 45, 48, 88, 148, 190, 287, 313, 323, 332, 335, 350.

Extra-uterine—Females, VI., 37, 72, 76, 89, 101†, 357.

Molar—Females, VI., 8, 8A, 107?

Nephritis in—Females, VI., 1†, 22, 305, 331†.

Vomiting of—Females, V., 27.

PREMATURITY—Females, VI., 19A.

PRURITUS—Females, VI., 252.

PSORIASIS—Females, I., 173; II., 180; III., 7, 126; IV., 202.

Treated with Thyroid Extract—Females, I., 173.

PUERPERAL—

Mania—Females, VI., 272.

Septicæmia—Females, I., 201†?; III., 12†; IV., 23†; VI., 324, 340.

PURPURA—Males, II., 110, 119; III., 30, 205†, 234†; IV., 113, 253. Females, II., 61†, 155; III., 25, 159, 214; IV., 66. *Radcliffe*—Males, 11†.

Hæmorrhagica—Males, III., 44. Females, III., 134†; IV., 50, 132.

PYÆMIA or SEPTICÆMIA—Males, I., 21†, 141, 146†, 309†?; II., 248†; III., 167†; V., 56†. Females, I., 79†, 201†?; II., 6, 109†, 182†; III., 12†; IV., 23†, 189; V., 20†.

See also Ulcerative Endo-carditis, Puerperal.

PYELITIS—Females, II., 12†; IV., 211†.

PYO-METRA—Females, VI., 15.

PYO-NEPHROSIS—Females, II., 12†, 200; III., 182†, 210; VI., 287.

PYURIA—Males, I., 244; II., 88, 233, 244; IV., 194.

RAYNAUD'S DISEASE—Males, I., 65. Females, II., 124.

RECTOCELE—Females, VI., 5, 158, 278.

RECTUM—

Cancer of—Males V., 48†. Females, II., 90; VI., 175.

Hæmorrhage from—Females, III., 220.

Prolapse of—Females, VI., 319.

Stricture of—Females, II., 109; IV., 83; VI., 293.

RESECTION OF RIBS—Females, II., 118.

See also Bronchi-ectasis, Empyema.

RETINAL HÆMORRHAGE—Males, III., 130. Females, II., 163.

RETINITIS ALBUMINURICA—Males, I., 70; II., 127†, 214; III., 63, 179; IV., 5†, 117, 195†. Females I., 2, 89, 137; III., 59†, 159, 190†.

RETINO-CHOROIDITIS—Males, II., 171; Females, IV., 174.

Tubercular—Males, III., 114†.

RHEUMATIC FEVER—Males, I., 34, 73, 102, 142, 189, 192, 193?, 248, 279, 293; II., 56, 112, 197, 215, 224, 291; III., 59, 76, 87, 91, 136, 149, 185, 186; IV., 171, 198, 204, 207, 240, 260; V., 34. Females, I., 85, 95, 108, 118, 161; II., 82, 126, 155, 162, 165, 190; III., 46, 53, 63, 115, 170, 212; IV., 23, 87, 133, 148, 203; V., 6, 31, 35.

RHEUMATISM—Males, I., 31, 212, 215, 287, 299; II., 2, 284; III., 10, 118; IV., 186, 244. Females, I., 123, 176, 189, 219; II., 16, 111?; III., 194, 200; IV., 102, 137, 157, 161, 172.

Gonorrhœal—Males, I., 91; IV., 100.

RHEUMATOID ARTHRITIS—*See* Osteo-arthritis.

RICKETS—Males, I., 258, 264, 266, 267, 276, 298; III., 202†, 203, 204, 208; IV., 52, 67†, 215, 230, 250, 252†. Females, I., 120; III., 125, 145, 158; IV., 60†, 89; V., 34†.

RUBEOLA—Males, III., 54, 70. Females, I., 180; III., 102; IV., 171. *Radcliffe*—Females, 26.

SALPINGITIS, Purulent—Females, VI., 254.

SCARLATINA—Males, I., 221. Females, IV., 77, 104. *Radcliffe*—Males, 6, 30; Females, 7, 28, 53†.

SCIATICA—Males, I., 69; II., 93, 301; IV., 132. Females, I., 59, 135; II., 15; VI., 8A.

SCLERODERMA—Males, IV., 14. Females, III., 96.

SCLEROSIS OF SPINAL CORD—

Disseminated—Females, II., 83, 173; IV., 111.

Lateral Amyotrophic—Males, II., 61, 266.

SEPTICÆMIA—*See* Pyæmia.

SKULL, Fracture of—Males, III., 167‡.

SPEECH, AFFECTIONS OF—Males, I., 9, 36, 282 ; II., 84 ; IV., 5‡. Females, II., 69, 129, 173 ; III., 130 ; IV., 146, 195.

Agaphia—Males, II., 110.

Aphasia—Males, II., 110 ; Females, I., 211 ; II., 115.

Aphonia—Females, III., 183.

SPINAL—

Caries—Males, II., 234.

Curvature—Females, IV., 195.

SPLEEN—

Abscess of—Males, II., 143‡.

Cancer of—Males, I., 44‡.

Enlargement of—Males, I., 182, 208, 298 ; II., 33, 173 ; III., 120, 198 ; IV., 47, 145‡, 220‡. Females, I., 26‡, 29‡, 93, 159 ; II., 39 ; III., 163 ; IV., 114‡.

Infarction of—Males, I., 76‡ ; II., 143‡?, 194‡, 304‡ ; III., 167‡. Females, I., 26‡?, 157‡, 192‡ ; III., 12‡ ; IV., 36‡.

STERILITY—Females, VI., 28.

STOMACH—

Cancer of—Males, I., 49?, 134, 176‡, 238? ; II., 167‡, 306‡ ; III., 56‡, 75, 77, 142 ; IV., 109. Females, II., 128‡ ; IV., 83, 192‡ ; V., 36.

Dilatation of—Males, I., 198A ; II., 97, 134 ; III., 39, 83, 85‡ ; IV., 34. Females, II., 93?, 145 ; IV., 125.

Perforation of—Males, II., 167‡. Females, I., 194‡ ; II., 10‡, 192‡.

See also Gastric Crises, Gastric Ulcer, Hæmatemesis.

SUPRA-RENALS—

Cancer of—Males, III., 46‡ ; Females, IV., 121‡.

Tuberculosis of—Females, IV., 188‡.

SYPHILIS—Males, I., 108?, 115, 132, 247 ; II., 135 ; III., 53, 89, 169 ; IV., 94, 133? Females, I., 24 ; II., 150, 150A ; III., 130 ; IV., 97 ; VI., 336.

Congenital—Males, I., 77, 182? ; III., 7, 139 ; IV., 186. Females, I., 185 ; II., 39 ; III., 163 ; IV., 98.

See also Liver, Syphilitic Disease of.

TABES DORSALIS—Males I., 219, 288 ; II., 62, 95, 128 ; III., 103, 181 ; IV., 103, 173, 200. Females, II., 12‡, 89 ; III., 147, 209 ; IV., 93.

TACHES BLENÂTRES—Males, II., 177 ; III., 134.

TACHYCARDIA—*See* Heart Disease.

TETANUS—Males, II., 133 (Treated with the Antitoxin).

TETANY—Females, I., 81 ; III., 88.

THROMBOSIS—Males, I., 1, 93 ; III., 73, 161, 189 ; Females, I., 13 ; II., 9, 84, 148, 162 ; III., 57, 91, 112, 180, 226.

Of Vena Cava Inferior—Males, V., 32.

Of Cerebral Sinus—Males, IV., 137‡ ; 229‡.

Of Vena Jugularis—Females, IV., 198.

Multiple—Males II., 129 ; IV., 124.

Portal—Males, II., 115‡.

Pulmonary—Males, IV., 229‡ ; Females, II., 20.

See Phlebitis.

TONSILLITIS—Males, I., 174, 250, 280 ; II., 26, 133, 196, 213 ; III., 109, 146. Females, I., 37, 68 ; II., 7, 13, 86 ; III., 9, 90, 106, 197 ; IV., 21, 29, 195, 209 ; V., 33. *Radcliffe*—Females, 27, 46.

TORTI-COLLIS—Males, II., 231. Females, II., 73.

TRACHEA, Stricture of—*Radcliffe*—Males, 75‡.

TRACHEOTOMY--

For Diphtheria—*Radcliffe*—Males, 1, 3†, 8, 9, 10†, 16, 17†, 18†, 19, 20, 22, 24†, 52, 56†, 66†, 69, 71, 74, 75†, 77†, 78†, 80, 84†, 86, 88†, 90†, 91†, 92†, 93†, 94†. Females, 15, 21, 25†, 38†, 44, 48, 49, 50, 51, 57, 58†, 59†, 70, 72, 73†, 87, 89†.

For Laryngitis—Males, IV., 8. *Radcliffe*—Males, 30.

TREMOR—Males, I., 9, 87 ; II., 3†, 113, 302 ; III., 18†, 219 ; IV., 258. Females, II., 8, 69, 173 ; III., 2 ; IV., 111, 120†.

Post-hemiplegic—Males, II., 299.

TUBERCULOSIS—Males, I., 67†?, 95 (of testis), 101†, 224†, 256†, 261†, 292† ; III., 64†, 197†, 201† ; IV., 145†, 218, 220†, 225†. Females, I., 109†, 160†?, 207† ; II., 3†, 66†, 67†, 105 ; III., 22† ; IV., 35†, 94†, 134†?, 165†, 188† ; V., 8†.

Of Urinary Tract—Males, IV., 145†.

See also Meningitis, Phthisis.

TUMOUR OF UNCERTAIN NATURE—Females, I., 163 (of Arm).

See Abdominal, Cerebral, Mediastinum, Pelvic.

TYPHOID FEVER—

FATAL CASES — Males, I., 20†, 194†, 222†, 317† ; II., 27†, 208†, 217† ; III., 157† ; IV., 13†, 162†, 193†, 199†, 205†, 249† ; V., 5†. Females, II., 77† ; IV., 54†.

With Colic Ulceration—Males, IV., 13†, 199†. Females, IV., 54†.

With Delirium Tremens—Males, II., 27†.

With Erythema—Males, IV., 193†, 205†. Females, IV., 54†.

With Hæmoptysis—Males, IV., 205†.

With Hæmorrhage—Males, II., 217† ; IV., 13†, 193†, 199†. Females, IV., 54†.

With Laryngeal Perichondritis—Males, III., 157†.

With Laryngeal Ulceration—Males, IV., 193†.

With Perforation—Males, I., 20†, 222†, 317† ; II., 208† ; IV., 13†, 162†, 199†. Females, II., 77†? ; IV., 54†.

With Peritonitis (without Perforation)—Males, III., 157†.

With Relapse—Males, IV., 205†.

With Septicæmia—Males, I., 194†.

RECOVERIES—Males, I., 1, 6, 14, 29, 139, 181, 200, 223, 226, 227, 235, 277 ; II., 5, 10, 11, 45, 65, 66, 221, 223, 225, 268, 271, 287, 288, 295, 303 ; III., 12, 18, 23, 37, 108, 115, 134, 153, 165, 172, 173, 187, 189, 223, 230, 237 ; IV., 3, 7, 8, 18, 27, 28, 29, 37, 182, 209, 236, 259 ; V., 22, 28, 30, 37, 52. Females, I., 16, 34, 35, 39, 162, 172, 184, 208 ; II., 11?, 21, 37, 48, 70, 135, 137, 142, 169, 175, 186 ; III., 16, 19, 20, 29, 166, 180, 185, 211, 213, 218, 226 ; IV., 9, 25, 26, 38, 65, 150, 151, 194, 205 ; V., 7, 11.

With Abortion—Females, IV., 205.

With Arthritis—Males, I., 181.

With Boils—Males, II., 288.

With Epistaxis—Males, IV., 27.

With Erythema—Males, II., 225. Females, II., 21, 135, 186 ; III., 19, 218.

With Hæmaturia—Females, II., 48.

With Insanity—Females, II., 21 ; III., 166 ; IV., 9.

With Laryngeal Ulceration—Males, IV., 8 (tracheotomy), 37.

With Metastatic Abscess—Males, II., 11. Females, III., 19.

With Nephritis—Males, II., 10.

With Otitis—Females, I., 39.

With Periosteitis—Males, II., 221. Females, I., 34, 39.

With Pleuritic Effusion—Males, III., 173.

With Catarrhal Pneumonia—Males, II., 65, 66.

With Pyuria—Females, I., 35, 39 ; II., 37, 48 ; III., 166.

With Relapse—Males, II., 5, 66, 221, 225 ; III., 37, 187. Females, II., 21, 70 ; III., 218.

With Rheumatism—Females, I., 172.

With Rigors—Females, II., 175 ; III., 180.

TYPHOID FEVER—RECOVERIES—continued.

With Tâches Blenâtres—Males, III., 134.

With Thrombosis—Males, I., 1 ; III., 189. Females, III., 180, 226.

With Vomiting—Females, III., 218.

Sequelæ of—Males, II., 56, 73 (paraplegia).

TYPHUS FEVER—Females, I., 86.

URÆMIA—Males, I., 59†, 140†, 239† (dyspncea) ; II., 69, 305† ; III., 52 ? ; IV., 11† ?, 32†, 44† ?. Females, I., 137, 191†, 197†, 218† ; III., 72†, 206 ? ; VI., 331†.

URETHRA—

Caruncle of—Females, VI., 66, 112, 183, 232, 330.

Hypertrophy of—Females, VI., 105A.

Pouch in—Females, VI., 315.

URINE, Suppression of—Females, IV., 32 (hysterical).

URTICARIA—Females, I., 115. Radcliffe—Males, 11†, 54. Females, 36, 40, 87.

UTERINE APPENDAGES, Inflammation of—Females, VI., 117, 124, 231, 341, 342.

See Hydro-Salpinx, Oöphorectomy, Salpingitis.

UTERINE CERVIX—

Amputation of—Females, VI., 92, 120, 178, 214, 286, 288.

Cancer of—Females, VI., 41, 120, 121, 177, 187, 196, 249, 347.

(Epithelioma)—Females, VI., 13, 21, 65, 71, 92, 102, 113, 130, 178, 186, 214, 220, 286.

Catarrh or Inflammation of—Females, VI., 4, 46, 61, 116, 143, 218, 222, 271.

Cauterisation of—Females, VI., 46, 65, 71, 116, 130.

Curetted—Females, VI., 222.

Dilated—Females, VI., 20, 28, 50, 153, 281.

Erosion of—Females, VI., 32, 245, 307.

Hypertrophy or Elongation of—Females, VI., 73, 144, 145, 146, 180, 205, 216, 225, 227, 288.

Laceration of—Females, VI., 110†.

Polypus of—Females, VI., 98, 111.

Stenosis of—Females, VI., 20.

UTERINE HÆMORRHAGE—Females, VI., 27, 58, 79, 82, 87, 115, 119, 120, 129, 135, 136, 150, 151, 168, 194, 200, 201, 202, 208, 233, 238, 242, 248, 298, 310, 354.

Accidental—Females, VI., 3, 19, 54, 64?, 185, 191, 215, 343, 360.

After Abortion—Females, VI., 134.

Post-partum—Females, VI., 47.

During Pregnancy—Females, VI., 32, 33.

UTERUS—

Cancer of—Females, VI., 4, 37†, 52, 289, 290, 333, 356.

Curetted—Females, VI., 8, 8A, 17, 30, 34, 39, 43, 115, 136, 142, 168, 181, 194, 200, 202, 208, 221, 238, 242, 281, 291, 346, 354.

Fibrocystic Tumour of—Females, VI., 25.

Fibroid Tumour of—Females, III., 223 ; IV., 107 ; VI., 9, 17, 17A, 60, 82, 86, 87, 111, 119, 133, 139 ?, 150, 155, 156, 172, 174, 193†, 223, 229†, 247, 291, 293, 295, 309, 327, 327A, 337, 352, 361.

Impacted, Treated by Taxis—Females, VI., 26, 62.

Enucleated—Females, VI., 169, 179.

Mal-development of—Females, VI., 267.

Mole of, Carneous—Females, VI., 55.

Hydatidiform—Females, VI., 16, 110†, 264.

Polypus of, Fibrous—Females, VI., 91, 132, 211, 217, 266, 277, 304, 318.

Fibro-cystic—Females, VI., 239.

Prolapse of—Females, VI., 73, 144, 145, 146, 180, 205, 216, 227, 261, 279, 314.

Retroflexion of—Females, VI., 308.

Retroversion of—Females, II., 191 ; VI., 55, 59, 294, 351.

Sub-involution of—Females, VI., 298.

VACCINIA—Males, II., 190.

VAGINA—

- Cancer of—Females, VI., 66.
- Dilated—Females, VI., 56.
- Laceration of—Females, VI., 152, 338.
- Occlusion of—Females, VI., 56, 67.
- Prolapse of—Females, VI., 319.

VAGINITIS—Females, VI., 116, 204.
 Senile—Females, VI., 35, 167.

VARICELLA—Males, II., 251. Females, I., 19 ; IV., 84.

VARIOLA—Males, III., 156. Females, II., 161.

VENESECTION—Males, II., 241†.

VISUAL FIELD, Contraction of—Males, II., 21, 84. Females, II., 8 ; III., 162.

VOMITING—Females, V., 27 ; VI., 275.

VULVA—

- Abscess of—Females, VI., 345.
- Cancer of—Females, VI., 44, 44A, 105, 244, 334.
- Cyst of—Females, VI., 303, 321, 349.
- Hypertrophy of—Females, VI. 114, 189.
- Inflammation of—Females, VI., 116.



I N D E X

TO REGISTER OF POST-MORTEM EXAMINATIONS.

MEDICAL, Vol. XXII., 1895,

BY H. H. TOOTH, M.D., F.R.C.P.

ABDOMEN—

- Abscess, of wall—17.
- Fæcal—186.
- Pelvic—163, 200.
- Subdiaphragmatic—291.
- Exploration of—127, 198.
- Fæcal Fistula—70, 198, 200.
- New growth in—214.
- Paracentesis of—141, 262.

ABNORMALITIES—

- Intestines, Appendix Vermiformis, long—195, 317.
- Colon, long—344.
- Diverticulum Ilei—149, 173, 302, 323.
- Jejuni—149.
- Mesocæcum, long—91, 141.
- Œsophagus, Pouching of—260.
- Peyer's Patches, elongated—253.
- Sigmoid Flexure, Pouching of—260.
- Kidneys, Congenital absence of one—317.
- Inequality in size—216.
- Liver, Abnormal Lobulation of—54
- Lung, Abnormal Lobulation of—123.
- Pulmonary Valve, four Cusps—106.
- Toes Webbed—347.

ABORTION—106.

ACETONÆMIA—123, 148, 332.

ADDISON'S DISEASE—310.

ALCOHOLISM—101, 325.

ANÆMIA, Pernicious—117, 88.

ANASARCA—6, 14, 70, 73, 74, 75, 102, 103, 262, 294, 306, 325.

ANEURYSM—

- Anterior Cerebral—156.
- Aorta—*See* Aorta.
- Basilar—7.
- Internal iliac—16.
- Middle Cerebral—91, 156, 157, 267.
- Splenic, Renal and Hepatic Arteries—7.

AORTA—

- Aneurysm, Abdominal—108.
- Ascending Arch—39, 225, 306, 341.
- Descending Arch—104, 334.
- Transverse Arch—257.
- Thoracic—63.

AORTA—*continued.*

- Atheroma of—10, 12, 15, 16, 25, 27, 32, 34, 36, 39, 41, 45, 54, 59, 62, 65, 67, 69
71, 72, 76, 80, 87, 91, 93, 95, 103, 104, 108, 110, 113, 115, 141, 156, 157
225, 226, 228, 234, 236, 238, 241, 257, 260, 273, 282, 285, 296, 320, 325
332, 334.
- Calcified—338.
- Dilated—260.
- New Growth around—143.
- Ruptured—282, 341.

APPENDIX VERMIFORMIS—

- Adherent to Ovary—291.
- Concretion in—317.
- Hernia of—195.
- Perforation of—232, 281.
- Perityphlitis—5, 232.

ARACHNOID—*See* Brain.

ARTERIES—

- Aorta—*See* Aorta.
- Arterio-sclerosis, General—10, 76, 247.
Calcareous—262.
- Axillary, Embolus of—359.
- Anterior Cerebral, Aneurysm of—156.
- Basilar, Embolic Aneurysm of—7.
- Carotid, Embolus of—224.
- Cerebral, Atheroma of—10, 32, 41, 50, 60, 62, 67, 76, 91, 96, 119, 120, 149,
153, 156, 157, 197, 228, 268, 285, 288, 296, 302, 303, 304, 325, 350.
- Middle Cerebral, Aneurysm of—91, 156, 157, 267.
Embolism of—9, 13.
- Coronary, Calcified—228, 282.
Constricted—197, 320, 335.
Embolism of—13.
- Iliac, Common, Embolism of—359.
Internal, Aneurysm of—16.
- Popliteal, Embolism of—224.
- Pulmonary, Atheroma of—6, 15, 20, 64, 91, 93, 141, 238.
Thrombosis of—37, 101, 209.
- Splenic, Renal, Hepatic, Aneurysm of—7.

ASCARIS LUMBRICOIDES—49, 199, 216.

ASCITES—30, 38, 40, 72, 73, 74, 91, 93, 94, 95, 102, 103, 111, 115, 137, 140, 141, 155
164, 184, 211, 271, 294, 312, 325, 337, 338.BILE DUCTS, Dilated—*See* Liver.

BLADDER—

- Calculus—141, 178.
- Cystitis—25, 291, 352.
- Hæmorrhage into—25, 177.
- New growth, Secondary—27.
- Tuberculous ulceration of—203.

BRAIN—

- Abscess—17, 170, 192.
- Anæmia—76, 88.
- Arachnoid, Calcified—273.
Thickened—29, 45, 57, 62, 119, 140, 304, 315, 320, 325, 326.
Tubercle of, without Meningitis—301.
- Dura Mater Calcified—264.
Sub-dural Hæmorrhage—12, 50, 88, 163, 177, 332.

BRAIN—*continued.*

- Hæmorrhage, Cerebral—12, 32, 41, 75, 120, 149, 156, 157, 201, 267, 285, 286
288, 302, 303, 350.
- Old Hæmorrhagic foci—67, 110, 157.
- Sub-arachnoid—60, 281.
- See also Cerebellum and Pons.*
- Hydrocephalus—147, 298.
- Hyperæmia—48.
- Meningitis, Purulent—26, 78, 97, 170, 192, 256, 270, 328.
Simple—43, 213, 258, 276, 313.
Tuberculous—3, 31, 125, 146, 147, 158, 221, 284, 292, 345.
- Microcephalus—29, 141, 266.
- Edema, Sub-arachnoid—6, 14, 17, 67, 86, 110, 119, 197, 280.
- Sclerosis, Symmetrical Cortical—29.
- Softening—7, 9, 13, 51, 52.
- Tumour—42, 83, 147, 163, 207, 278.

BROUGHT IN DEAD—33, 36, 39, 44, 45, 52, 60, 62, 63, 68, 156, 178, 228, 255, 273
296, 304, 323, 334, 343.

CALCULUS—

- Biliary—27, 41, 50, 71, 76, 225, 257, 289.
- Renal—179, 228, 356.
- Vesical—141, 178.

CELLULITIS—Of Neck—106.

CEREBELLUM—

- Abscess—43.
- Atrophy, Right Lobe—178.

CHYLŒDEMA—262.

CHORDÆ TENDINEÆ—*See Heart.*

COLON—

- Abnormalities of—*See Abnormalities.*
- Colitis, Ulcerative—116, 348.
- Constriction of—208.
- Embolism of—274.
- Dilated—290, 348.
- Polypi of—107.
- New growth, Secondary, of—94.
- Perforation of—70, 116, 200.
- Ulceration, Dysenteric, Old—22.
Follicular—175, 200.
Tuberculous—3, 37, 51, 66, 87, 142, 145, 150, 165, 266, 332, 339, 356.
Typhoid—18, 82, 244, 259.
- See also Appendix Vermiformis.*

CONVULSIONS—137?, 159.

CRANIAL BONES—

- Depressed Fracture—274.
- Thickened—91, 110, 128, 141, 148, 153, 156, 285.
- Thin—298.

CYNANCHE CELLULARIS—106.

DERMATITIS, Acute—233.

DIABETES MELLITUS—115, 123, 148, 289, 322.

DIAPHRAGM, Abscess, Sub-diaphragmatic—291.

DIARRHŒA—33, 105, 162, 173, 175, 181, 187, 189, 194, 212, 265.

DIED IN THE SURGERY—79, 128, 182, 302, 303.

DIPHTHERIA—48, 86, 90 ?, 92, 121 ?, 130, 132, 148, 152, 169, 185, 199, 202, 229, 246, 272, 283, 307, 311 ?, 324, 330, 331, 349, 354.

DUCTUS ARTERIOSUS, Pervious—233, 326.

DURA MATER—*See* Brain.

EAR—

Catarrh of Middle Ear—8, 77, 85, 122, 124, 146, 209, 327.

Otitis Media—26, 66, 78, 86, 105, 133, 147, 152, 160, 161, 172, 191, 192, 284, 342.

EMBOLISM—

Axillary and Common Iliac—359.

Basilar—7.

Carotid, Innominate and Popliteal—224.

Middle Cerebral—9, 13.

EMPYEMA—61, 100, 124, 176, 242, 256, 305.

ENTERIC FEVER—18, 19, 82, 231, 243, 244, 252, 259, 264, 276, 277, 299, 300, 316, 357.

EPILEPSY—96.

EYE—

Conjunctiva, Diphtherial Membrane on—229.

Keratitis, Sloughing—160.

Optic Nerve, Atrophy—184.

Dropsy of Sheath—147.

Neuritis—163.

Retinal Hæmorrhage—88, 158.

FÆCAL FISTULA—70, 198, 200.

FALLOPIAN TUBE, Cyst of—262, 290.

FEMUR, Sarcoma of—5.

FIBULA, Osteo-sarcoma of—83.

FORAMEN OVALE, Patent—57, 230.

GALL BLADDER—

Abscess in Wall—71.

Adhesions about—71, 291.

Atrophy of—268, 270.

Cystic Duct Dilated—27.

Obstructed—41, 270, 289.

Dilatation of—41, 127, 236, 257, 289.

Gall Stones—27, 41, 50, 71, 76, 225, 257, 289

New Growth of—27, 34, 50, 208.

GANGRENE—

Extremities, of—126, 224, 359.

Lung, of—124, 165, 170, 248, 333.

HEART—

Aortic Valves, Atheroma of—157, 197, 269.—*See also* Thickening.

Calcified—20, 238, 297.

Fenestrated—110, 214, 266.

Granulations on—7, 9, 11, 17, 25, 140, 211, 237, 358, 359.

Incompetence of—6, 9, 20, 35, 36, 52, 128, 178, 197, 211, 234, 238, 241, 260, 269, 358.

Stenosis—20, 27, 35, 36, 52, 234.

Thickened—6, 16, 20, 27, 35, 36, 52, 67, 70, 74, 80, 128, 234, 241, 285, 294, 296, 297.

HEART—continued.

Atrophy—94, 143.

Auricle—

Left, Ante-mortem Clots in—6, 20, 338.

Right, Ante-mortem Clots in—6, 80, 86, 93.

Septum incomplete—351.

Chordæ Tendinæ, Thickened—20, 128, 281.

Coronary Arteries—

Calcified—228, 282.

Constricted—197, 320, 338.

Dilatation, General, of—9, 15, 55, 74, 95, 151, 218, 338, 351.

Dilatation with Hypertrophy, General—20, 35, 52, 113, 140, 155, 197, 211, 237, 260, 263, 269, 296.

Embolism, Pyæmic, of Wall—281.

Endocarditis, Infective (“Ulcerative”)—7, 9, 13, 65?, 140, 224, 274, 281, 286, 359.

Endocardium, Thickened—46, 128.

Fatty Degeneration—20, 33, 45, 55, 67, 74, 88, 101, 148, 149, 177, 178, 197, 211, 225, 228, 234, 241, 260, 273, 296, 300.

Foramen Ovale, Patent—57, 230.

Mitral Valve—

Adhesion of Cusps—*See* Stenosis.

Atheroma of—10, 32, 34, 41, 54, 67, 69, 70, 76, 91, 111, 238.

Calcification of—6, 20, 80, 236, 237, 247.

Granulations on—7, 9, 17, 23, 27, 65, 140, 224, 243, 274, 358, 359.

Incompetence of—6, 9, 23, 27, 52, 54, 74, 80, 128, 155, 211, 241, 247, 260, 269, 281, 294, 296.

Stenosis of—6, 20, 27, 35, 52, 74, 80, 91, 128, 140, 155, 215, 237, 247, 294, 297, 333, 358.

Thickening of Cusps—16, 128, 211, 281.

Myocarditis—323.

New Growth, Secondary—184.

Obesity of—48, 153.

Pulmonary Valve, Four Cusps to—106.

Tricuspid Valve—

Atheroma of—20.

Granulations on—7, 155.

Incompetence of—20, 74, 80, 93, 197, 260.

Stenosis—74, 80, 215, 294.

Ventricle—

Left, Aneurysm of—15, 36.

Ante-mortem Clot in—333.

Dilated—24, 153, 241, 264, 300, 325.

Dilated and Hypertrophied—9, 10, 36, 59, 128, 149, 156, 215, 238.

Hypertrophied—12, 16, 32, 41, 57, 72, 76, 91, 157, 201, 217, 226, 228, 247, 288, 303, 312, 326, 334, 338, 350, 358.

Induration, Fibroid of—128.

Myocarditis of—323.

Right, Ante-mortem Clots in—15, 338.

Dilated—6, 10, 23, 40, 54, 77, 80, 88, 91, 93, 102, 111, 294, 297, 333.

HERNIA—

Inguinal—207.

Omental—38, 268.

Vermiform Appendix, of—195.

HIP JOINT, Secondary Growth of—83.**HODGKIN'S DISEASE—137.****HYDROCELE—143.****HYDROCEPHALUS—147, 298.****HYDRONEPHROSIS—46, 222, 285, 317, 301.****HYPERPYREXIA—99.**

INTESTINES—

- Abnormalities—*See* Abnormalities.
 - Congestion, passive—80, 111.
 - Distension—18.
 - Duodenum, constricted—71.
 - Embolism of—13, 17 ?, 274, 281, 359.
 - Enteritis—162, 164, 327.
 - Hæmorrhage—244.
 - Lardaceous—188.
 - Obstruction—220.
 - Perforation—18, 19, 82, 145, 231, 252, 259, 277, 357.
 - Peyer's Patches, Pigmented ("Shaven beard appearance")—266, 300, 309, 318.
 - Swollen—147, 162, 248, 300.
 - Tubercle in—29, 146.
 - Ulceration, Cancerous—71.
 - Follicular—116.
 - Tuberculous—3, 37, 46, 55, 66, 77, 81, 89, 142, 145, 146, 165, 167, 172, 180, 183, 221, 230, 275, 284, 301, 339, 345.
 - Typhoid—18, 19, 82, 209, 231, 244, 252, 264, 276, 277, 299, 316, 357.
- See also* Colon, Rectum, and Appendix Vermiformis.

JAUNDICE—50, 69, 71, 73, 91, 127, 141, 203, 270.

JOINTS—

- Hip, Secondary Growth of—83.
- Knee, Hæmorrhage into—177.
- Rheumatoid Arthritis—25.

KIDNEY—

- Abnormality—*See* Abnormalities.
- Abscess, Perinephric—250.
- Atrophy of—262.
- Calculus, Renal—179, 228, 356.
- Cloudy Swelling of—18 ?, 55, 69, 106, 107, 142, 145, 148, 150, 165, 166, 203, 266, 309, 316, 357.
- Congenital absence of—317.
- Congestion, Acute—93, 119, 128, 248.
 - Chronic—6, 23, 52, 73, 74, 80, 111, 155, 197, 211, 257, 269, 294, 333, 338, 341, 358, 393.
- Cysts in—32, 34, 53, 59, 60, 67, 93, 106, 141, 157, 190, 247, 329, 353.
- Fatty—38, 45, 70, 305.
- Hæmorrhage, Punctiform—286.
- Hydro-nephrosis—46, 222, 285, 301, 317.
- Hypertrophy—235, 239.
- Infarction of—7, 13, 27, 67, 73, 224, 243, 274, 281, 359.
- Iron, Free, in—38.
- Lardaceous—4, 167, 339.
- Nephritis, Consecutive—290.
 - Interstitial—10, 12, 16, 32, 35, 41, 54, 57, 60, 62, 65, 67, 76, 101, 106, 110, 113, 120, 151, 153, 156, 157, 164, 170, 178, 192, 201, 215, 217, 223, 225, 226, 228, 234, 232, 235, 288, 303, 315, 320, 326, 334, 335, 338 ?, 347, 350, 352.
 - Tubal, Acute—9, 11 ?, 327.
 - Chronic—22, 40, 55 ?, 59, 70, 86, 97, 102, 103, 123, 132 ?, 140, 169 ?, 190, 240, 243 ?, 258 ?, 262.
 - Tubal with Interstitial—64, 72, 149, 280 ?, 287, 308.
- New Growth, Secondary—34, 83, 210.
- Pyelitis—25, 352.
- Pyonephrosis—212, 290.
- Scars on—281.
- Tubercle of—21, 29, 81, 89, 104, 124, 132, 146, 188, 203, 221, 261, 275, 284, 292, 318.
- Ureters, Calculus in—301.
 - Dilated—290, 301, 352.

LARYNX—

- Epiglottitis, Ulceration, Follicular—182.
 In Typhoid—264.
 Tuberculous—46, 145.
 Laryngitis, Chronic—52, 164.
 Œdema—11, 28, 40, 102, 294, 335.
 Perichondritis—276.
 Ulceration, Simple—11, 71.
 Tuberculous—4, 46, 49?, 51, 55, 65, 104, 117, 145, 153?, 230, 332, 337.
 Vocal Chords Thickened—22, 45.
See also Diphtheria.

LIVER—

- Abnormal Lobulation of—54.
 Abscess—200, 250, 270.
 Adhesions, Perihepatic—17, 27, 51, 94, 141, 142, 147, 153, 164, 165, 196, 203, 335, 347.
 Bile Ducts Dilated—50, 71, 127.
 Capsule Thickened—54, 57, 128.
 Chronic Congestion of ("Nutmeg Liver")—6, 15, 20, 74, 80, 93, 95, 111, 140, 155, 211, 215, 218, 237, 238, 241, 260, 269, 294, 297, 333, 338, 341.
 Cirrhosis—11?, 35, 38, 45, 53, 62, 73, 76?, 102, 120, 141, 164, 182, 257, 268, 271, 280, 289, 325, 332.
 Cloudy Swelling of—9, 55, 69, 106, 174, 244, 259, 264, 266, 309, 315, 316, 357.
 Common Duct Dilated—27, 127.
 New Growth of—71.
 Obstructed—50, 71, 263.
 Fatty—12, 14, 35, 37, 38, 47, 51, 55, 65, 81, 87, 101, 151, 179, 180, 204, 275, 300, 305, 342.
 Hydatid Calcified—151, 250.
 Hæmorrhage into—151.
 Ruptured—151.
 Iron, Free, in—69, 88.
 Lardaceous—4, 188.
 Nævus of—157, 169.
 New Growth of Primary—136.
 Secondary—27, 30, 34, 50, 58, 83, 127, 184, 195, 210, 329.
 Perihepatitis—20, 27, 70.
 Tight Lacing—76, 91, 157.
 Tubercle of—21, 29, 66, 79, 122, 124, 125, 132, 146, 172, 186, 188, 221, 227, 261, 292, 301, 305, 318, 352, 356.

LUNG—

- Abnormal Lobulation of—123.
 Abscess of—17, 166, 342.
 Bronchiectasis—1, 152, 176, 188, 261.
 Bronchitis—90, 191, 243, 253, 299.
 Broncho-Pneumonia—1, 2, 8, 19, 21, 28, 35?, 48, 53, 56, 65, 66, 84, 85, 90, 97, 98, 107, 109, 121, 125, 129, 133, 138, 144, 152, 154, 161, 168, 169, 172, 185, 193, 196, 202, 205, 235, 240, 245, 246, 251, 254, 258, 272, 307, 311, 324, 331, 336, 349, 356.
 Caseous—79, 160, 169.
 Cavities in—4, 23, 37, 45, 46, 51, 55, 67, 77, 87, 117, 122, 124, 142, 145, 150, 165, 167, 188, 206, 248, 250, 261, 263, 266, 289, 305.
 Collapse, Compression—30, 50, 61, 64, 65, 91, 100, 107, 113, 115, 137, 140, 188, 242, 254, 256, 263, 294, 312, 337.
 Collapse, Disseminated—2, 8, 14, 24, 33, 44, 48, 56, 68, 70, 74, 78, 84, 85, 86, 90, 92, 97, 99, 103, 106, 109, 112, 121, 125, 129, 132, 133, 138, 144, 152, 172, 191, 194, 196, 199, 235, 240, 245, 253, 272, 283, 301, 331, 336, 343, 345, 355, 356.
 Congestion—13, 18, 20, 23, 114, 119, 182, 243, 329, 333.
 Emphysema—11, 12, 15, 16, 22, 25, 27, 32, 45, 50, 54, 57, 63, 64, 67, 71, 80, 88, 91, 93, 94, 101, 105, 111, 129, 141, 146, 155, 164, 170, 217, 218, 234, 288, 325, 334, 346, 352.

LUNG—*continued.*

- Fibroid Induration of—23, 57, 63, 67, 147, 150, 176, 182, 261, 263, 290, 295, 297, 332.
 Gangrene—124, 165, 170, 248, 333.
 Hæmorrhage—63, 111, 248, 334.
 Infarction, Hæmorrhagic—6, 10, 15, 80, 155, 209, 260, 269, 333, 338.
 Pyæmic—7, 26, 35, 65, 174.
 New Growth, Secondary—5, 34, 58, 83, 210.
 Œdema—9, 10, 12, 19, 22, 23, 35, 40, 45, 50, 59, 64, 67, 69, 71, 72, 73, 76, 83, 87, 88, 91, 93, 94, 95, 101, 102, 107, 113, 120, 123, 128, 131, 140, 143, 145, 148, 153, 157, 158, 170, 177, 190, 197, 243, 244, 247, 256, 264, 271, 280, 282, 286, 294, 296, 297, 300, 302, 308, 315, 317, 325, 335, 340, 341, 344, 353, 357, 359.
 Pneumonia, Lobar—40, 49, 54, 69, 174, 182, 190, 204, 248, 249, 266, 268, 304, 309, 315, 321.
 Tubercle—3, 4, 29, 37, 45, 46, 51, 55, 63, 65, 72, 77, 79, 81, 87, 89, 104, 117, 118, 122, 124, 125, 132, 142, 145, 146, 147, 150, 165, 167, 186, 203, 206, 221, 227, 230, 261, 262, 263, 266, 275, 280, 284, 289, 292, 295, 301, 305, 310, 318, 332, 337, 339, 345, 352.

LYMPHATIC GLANDS—

- Abdominal, Caseous—21, 37, 55, 89, 146, 158, 180, 196, 244, 262, 266, 311, 316, 337.
 Enlarged—4, 18, 79, 81, 82, 132, 145, 172, 203, 221, 243, 252, 264.
 Melanosis of—184.
 New Growth of, Primary—127.
 Secondary—34, 143, 195, 208, 210, 329.
 Suppurating—19.
 Tubercle of—322.
 Bronchial, Calcified—27, 41, 116, 165, 199, 263.
 Caseous—21, 26, 29, 31, 37, 79, 89, 122, 124, 144, 146, 158, 203, 205, 301, 337, 345.
 Enlarged—2, 3, 4, 20, 28, 36, 56, 57, 70, 79, 83, 107, 128, 132, 142, 144, 160, 161, 169, 172, 176, 196, 202, 205, 206, 230, 235, 242, 243, 245, 246, 249, 253, 256, 258, 261, 272, 277, 297, 299, 309, 318, 331, 339, 356.
 Lardaceous—167.
 Melanosis of—184.
 New Growth of, Secondary—34.
 Tubercle in—66, 77, 125, 142, 261, 266, 284, 292.
 Cervical, Caseous—29, 37, 89, 146, 262, 284.
 Enlarged—68, 79, 81, 90, 106, 107, 122, 130, 132, 137, 142, 152, 203, 246, 249, 272, 337, 349.
 Femoral, Secondary Growth—101.
 Inguinal, Caseous—158.
 Lymphadenoma—137.
 Mediastinal, Secondary Growth in—127, 143, 173.

MARASMUS—24, 47, 179, 181, 183, 219, 255, 279, 293, 319, 355.

MEDIASTINUM—

- Abscess of Middle—81.
 Cellulitis of Anterior—106.
 Glands, Secondary Growth in—127, 143, 173.

MENINGITIS—*See* Brain.

MESENTERY—

- Calcareous Mass in—173.
 Caseous Mass in—77.
 New Growth, Secondary—71, 94.

MORBILLI—121, 246.

NEPHRITIS—*See* Kidney.

NERVE—

- Degeneration in Alcoholism—53, 101?
- Vagus, New Growth involving—173.

NECK, Cellulitis of—106.

NOSE, Ulceration of Septum—4.

ESOPHAGUS—

- Constriction of—143.
- Fibroma in Wall of—335.
- Hæmorrhoids of—141, 164, 195, 271, 289.
- Pouching of—260.
- Ulceration of—116, 128, 130, 145, 268.

OMENTUM—

- Calcareous Mass in—76.
- Hernia—38, 268.
- Tubercle of—167.

OVARY—

- Abscess of—131, 165, 166.
- Cyst of—158, 208, 222, 227, 290, 291.
- Hæmorrhage into—177.
- New Growth of—101.
- Parovarian Cyst—17, 158, 184.

OVERLAID—44, 68?

PANCREAS—

- Atrophy of—115, 148, 322.
- New Growth of—236.

PERICARDIUM—

- Adherent—20, 38, 54, 110, 120, 128, 153, 184, 190, 221, 237, 263, 302, 312, 325.
- Calcification of—20.
- Effusion upon—6, 19, 86, 142, 337.
- Hæmorrhage—39, 177, 341.
- “Milk-spots” upon—6, 27, 34, 94, 101, 102, 104, 113, 157, 188, 197, 269, 326.
- New Growth, Secondary—5.
- Pericarditis—69, 72, 80, 97, 106, 133, 165, 230, 268, 347, 358.
- Tubercle of—263, 337.

PERINÆUM—

- Abscess—19.
- Ischio-rectal Sinus—166.

PERITONEUM—

- Adhesions—19, 30, 34, 51, 70, 74, 118, 180, 200, 210, 214, 262, 309, 348.
- Ascites—30, 33, 40, 72, 73, 74, 91, 93, 94, 95, 102, 103, 111, 115, 137, 140, 141, 155, 164, 184, 211, 271, 294, 312, 325, 337, 338.
- Hæmorrhage into—7, 30, 34.
- Sub-peritoneal—177.
- Peritonitis, Acute—18, 69, 70, 82, 116, 131, 145, 164, 165, 195, 220, 231, 232, 252, 259, 274, 276, 277, 327, 357.
- Chronic—38, 70, 203.
- Pelvic—166.
- Tuberculous—46, 55, 66, 77, 81, 118, 132, 167, 180, 186, 198, 227, 239, 305, 337, 339, 316.
- Secondary Growth on—27, 30, 210, 346.
- Sub-peritoneal Veins dilated—11.

PERTUSSIS—138, 301.

PHARYNX—

Abscess, Retro-pharyngeal—251.

Ulceration, Tuberculous—46, 73.

PHIMOSIS—271.

PHTHISIS—*See* Tubercle of Lung.

PLEURA—

Abscess, sub-pleural—250, 291.

Adhesions—4, 10, 12, 20, 23, 26, 29, 32, 38, 40, 45, 46, 50, 51, 52, 55, 57, 59, 62, 65,

67, 71, 74, 77, 79, 87, 93, 95, 101, 104, 110, 117, 118, 120, 123, 124, 129,

130, 141, 142, 143, 145, 147, 150, 153, 158, 164, 166, 167, 170, 176, 182,

184, 186, 195, 197, 200, 202, 203, 206, 210, 211, 228, 230, 234, 237, 243,

247, 248, 249, 250, 251, 258, 261, 263, 264, 266, 274, 280, 281, 282, 288,

305, 315, 318, 320, 322, 325, 326, 332, 334, 335, 338, 339, 344, 345, 350,

358.

Effusion upon—6, 7, 10, 15, 30, 38, 57, 64, 86, 91, 94, 102, 103, 111, 113, 128,

137, 140, 155, 248, 269, 294, 297, 306, 337.

Empyema—61, 100, 124, 176, 242, 256, 305.

Fibrous Nodules upon—62.

Hæmorrhage into Cavity—63.

Sub-pleural—116, 307.

New Growth, Secondary—143, 195, 210, 346.

Pleurisy, Acute—7, 8, 10, 15, 21, 49, 50, 51, 61, 65, 69, 80, 95, 97, 106, 107,

115, 125, 133, 160, 161, 166, 169, 174, 193, 209, 245, 254, 262, 266, 268

277, 315, 321, 327, 329, 333, 342, 347.

Chronic—113.

Pyo-pneumo-thorax—188, 263, 275.

Thickening of—23, 38, 55, 77, 170, 218, 228.

Tubercle of—66, 104, 262, 318, 337.

PLUMBISM—247.

POISONING—

Carbolic Acid—119, 278.

Potassium Bichromate—216.

POMPHOLIX—107 ?

PONS VAROLII, Hæmorrhage into—120, 285.

PREMATURE BIRTH—319.

PROSTATE, Tubercle of—203.

PSORIASIS—177.

PULMONARY ARTERY—*See* Arteries.

PURPURA—86, 126, 177, 243, 308.

PYÆMIA and SEPTICÆMIA—17, 35, 107, 131, 166, 270, 347.

RACHITIS—

Old—196.

Recent—12, 24, 68, 78, 81, 84, 85, 132, 205, 253, 254, 255, 313, 345, 353

RADIUS—

Enlarged End—78, 84.

Sarcoma of—5.

RECTUM—

Hæmorrhoids—38, 73, 153.

New Growth of—346.

Stricture of 166.

Ulceration of—78.

RHEUMATOID ARTHRITIS—

Knee—25.

Vertebræ—64.

RIBS—

- Beading of—24, 68, 78, 84, 132, 205, 253, 254, 255, 313.
- New Growth, Secondary—101, 210.
- Resection of—100, 176.

SPINAL CORD—

- Hydrorrhachis, Local—258.
- Infantile Paralysis, old—40.
- Meningitis—147, 328.
- Posterior Sclerosis—25.

SPLEEN—

- Abscess—140.
- Accessory—42.
- Adhesions about—7, 70, 141, 142, 153, 164, 165, 196, 203, 305, 335.
- Atrophy of—338.
- Capsule Thickened—118, 271.
 - Fibrous Nodules on—128.
- Congested—6, 20, 36, 74, 80, 111, 113, 155, 197, 260, 269, 275, 294, 297, 333, 358.
- Enlarged—11, 18, 22, 243, 253.
- Hæmorrhage in—103.
- Infarction of—7, 9, 11, 17, 27, 35, 37, 65, 140, 224, 274, 281, 323, 338, 359.
- Lardaceous—167, 188, 339.
- Lymphadenoma of—137.
- Melanosis of—184.
- Tubercle of—21, 29, 66, 79, 81, 89, 124, 125, 132, 146, 147, 172, 180, 188, 258, 284, 318, 337, 345.

STOMACH—

- Congestion of—80.
- Dilatation of—23, 71, 148, 156, 223, 236, 289, 322, 329, 344.
- Hæmatemesis—23.
- Lardaceous—167.
- New Growth of—94, 195, 208, 223, 329, 344.
- Perforation of—195.
- Ulceration of—271, 291, 320.

SUB-MAXILLARY GLAND—

- Abscess—104.
- Lardaceous—167.

SUICIDE—216.

SUPRA-RENAL BODIES—

- Secondary Growth in—83, 184.
- Tubercle of—147, 310.

SYPHILIS, Congenital—219.

TESTIS—

- Epididymis, Tubercle of—203, 261.
- Hydrocele—143.

THROMBOSIS—

- Jugular—26.
- Lateral Sinus—26, 207.
- Longitudinal Sinus—209.
- Portal Vein—141.
- Pulmonary Artery—37, 101, 209.

THYROID GLAND—

- Caseous Mass in—334.
- Enlarged—177, 318.
- Lardaceous—167.
- Tubercle of—318.

TONGUE—

- Ulceration, Simple—98.
- Tuberculous—117.

TONSILS—

- Abscess of—261.
- Enlarged—125, 128, 318.
- Pouching above—97.

TRACHEA—

- Tracheotomy—11, 48, 90, 92, 130, 132, 169, 185, 199, 251, 272, 278, 283, 307, 311, 330, 331, 349, 354.
- Ulceration—142, 251, 257.

TUBERCULOSIS, General—81, 89, 221, 284, 292, 301, 318.

URÆMIA—76, 103, 226, 280 ?, 287 ?, 317, 335, 340.

URETERS—

- Calculus in—301.
- Dilated—290, 301, 352.

UTERUS—

- Abortion—106.
- Abscess of Wall, Pyæmic—166.
- Endometritis—166, 290, 340.
- Fibroid of—25, 42, 51, 222, 290.
- New Growth of—30.
- Polypus of—215.
- Sub-involuted—73, 340.

VERTEBRÆ—

- Curvature, Angular—285.
- Lateral—276.
- Erosion of, by Aneurysm—63.
- Rheumatoid Arthritis of—64.
- Secondary growth of—83, 101.



SURGICAL REPORT.

PREFACE TO THE SURGICAL REPORT.

The general arrangement of the Statistical Tables is the same as in previous years.

The Appendices to Tables I. and II. have this year been considerably enlarged.

DISEASE.	Total.		Discharged.		Died.		Under 5.		— 10.		— 15.		— 20.		— 30.		— 40.		— 50.		— 60.		Over 60.		
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	
																									Discharged.
TUMOURS (continued.)																									
Rodent Ulcer (continued)—																									
Neck ...	1
Scalp ...	1
Multilocular Cystic Tumour of Jaw ...	2	1
Alveolar Carcinoma—																									
Breast ...	41
Breast (recurrent)	16
Glands of Axilla	1
Cystic Duct	1
Intestine	7	2
Jaw ...	1
Kidney ...	1
Liver ...	1
Prostate ...	1
Rectum ...	33	15
Spine ...	1
Stomach ...	7	3
Umbilicus	1
Duct Carcinoma of Breast...	2
Sarcoma—																									
Back ...	2	1

DISEASE.	Total.		Under 5.		— 10.		— 15.		— 20.		— 30.		— 40.		— 50.		— 60.		Over 60.		
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	
TUMOURS (continued.)																					
Sarcoma (continued)—																					
Prostate ...	1	...	1
Synovial Membrane ...	3	...	1
Testis ...	1
Thyroid ...	2
Tongue ...	1
Tonsil ...	1
Cysts—																					
Aerocele ...	1
Breast ...	9
Congenital Coccygeal ...	1
Dental ...	4	...	3
Dentigerous
Dermoid ...	14	...	5
Hygroma ...	6	...	5
Mucous ...	1
Ovarian ...	1
Sebaceous ...	10	...	7
Serous (of Jaw) ...	1
Synovial ...	2
Abdominal Tumour ...	8	...	3
Ateno-Chondro-Fibroma—																					
Parotid ...	2
Adeno-Fibroma—																					
Breast ...	22	...	1
Parotid ...	1	...	1

TABLE I. (continued).

DISEASE.	Total.		Under 5.		— 10.		— 15.		— 20.		— 30.		— 40.		— 50.		— 60.	
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
TUMOURS (continued.)																		
Lymphadenoma	6	3	2	1	2	..	1	1
Lymphangioma—																		
<i>Arm</i>	1	1	1	1
<i>Face</i>	3	2	1	1
<i>Tongue</i>	1	1
Nævus	26	6	20	..	1	12	1	2	2	2	1	2	1	2
Neuro-Fibroma	1	1	1
Osteoma—																		
<i>Forear</i>	1	1	1
<i>Itam</i>	1	..	1
<i>Jaw (Upper)</i>	2	2	2
<i>Phalanx</i>	1	1	1
<i>Scapula</i>	1	1	1
<i>Tarsus</i>	1	1	1
Papilloma—																		
<i>Anal</i>	1	1	1
<i>Bladder</i>	4	3	1
<i>Larynx</i>	3	1	1	1	1
<i>Umbilicus</i>	1	1	1
Polypus—																		
<i>Ear</i>	3	..	3	..	1
<i>Nose</i>	8	4	1	3	2	1
<i>Rectum</i>	3	2	1	..	2	1
Thyroid—																		
<i>Bronchocele</i>	5	1	4	1

DISEASE.	Total.		Under 5.		— 10.		— 15.		— 20.		— 30.		— 40.		— 50.		— 60.		Over 60.	
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF THE LARYNX AND TRACHEA (continued.)																				
Edema of Larynx	2	1
Old Tracheotomy	3	1	1	1
DISEASES OF THE DUCTLESS GLANDS.																				
Thyroid—																				
<i>Cretinism</i>	2	1	1
<i>Graves' Disease</i>	1
<i>Myxœdema</i>	1
Spleen—																				
<i>Enlargement of...</i>	3	2
DISEASES OF THE CHEST.																				
Empyema	4	3	1	2	1	1
" (Old)	1	1
Pleurisy, with Effusion	1	...	1	1
Pneumonia	1	...	1
DISEASES OF THE VASCULAR SYSTEM.																				
Aneurysm—																				
<i>Circoid (of Forearm)</i>	1	1	1	1
" (of Orbit)	2	1	1	1

DISEASE.	Total.		Under 5.		— 10.		— 15.		— 20.		— 30.		— 40.		— 50.		— 60.		Over 60.	
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF THE DIGESTIVE SYSTEM (continued.)																				
Intestines—																				
Hernia—																				
Irreducible—																				
<i>Femoral</i> ...	10	9
<i>Inguinal</i> ...	121	13
<i>Lumbar</i> ...	1	1
<i>Umbilical</i> ...	5	4
<i>Ventral</i> ...	7	6
Irreducible—																				
<i>Femoral</i> ...	16	9
<i>Inguinal</i> ...	12	9
<i>Umbilical</i> ...	5	1
<i>Ventral</i> ...	3	2
Strangulated—																				
<i>Femoral</i> ...	17	3
<i>Inguinal</i> ...	24	18
<i>Umbilical</i> ...	5	2
<i>Hydrocele of Hernial Sac</i> ...	1	1
<i>Hematocoele of Hernial Sac</i> ...	1	1
<i>Abscess of Hernial Sac</i> ...	1	1
<i>Fœcal Fistula</i> ...	1

TABLE I. (continued).

DISEASE.	Total.		Under 5.		— 10.		— 15.		— 20.		— 30.		— 40.		— 50.		— 60.		Over 60.	
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF THE GENITO-URINARY ORGANS.																				
Bladder—																				
<i>Calculus</i> ...	5	2	1	...	1	1	1
<i>Cystitis</i> ...	9	2	6	1	4	1	...	1	1
<i>Tuberculous Disease</i>	7	4	2	1	2	...	1	1	...	3	1
Kidney—																				
<i>Calculus</i> ...	5	3	1	1	1	1	...	1	1
<i>Movable Kidney</i>	4	...	4	1	...	1	1	...	2
<i>Pyelonephrosis</i> ...	3	1	1	1	1	...	1
<i>Renal Colic</i>	6	2	4	1	...	1	1	...	1
<i>Syphilitic Disease</i>	1	...	1	1
<i>Tuberculous Disease</i>	2	1	1	1	...	1	1
<i>Old Nephro-Lithotomy</i>	1	1	1
Prostate—																				
<i>Enlarged</i>	12	9	1	1	...	1	7
<i>Inflamed</i>	1	...	1	1	3
<i>Tuberculous Disease</i>	1	1	1
Spermatic Cord—																				
<i>Encysted Hydrocele</i>	2	2	1	1
<i>Variocoele</i>	40	40	19	...	18	2	...	2	1
<i>Hæmatome of Inguinal Canal</i> ...	1	1	1
Tunica Vaginalis—																				
<i>Hæmatoecle</i>	7	7	1	2	...	2	2
<i>Hydroecle</i>	22	22	2	2	5	2	...	2	3

TABLE I. (continued).

INJURY.	Total.		Under 5.		— 10.		— 15.		— 20.		— 30.		— 40.		— 50.		— 60.		Over 60.			
	M.	F.	Died.	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		
				M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.
Fractures—																						
(Simple)—																						
Clavicle	1	1	1																			
Humerus	6	6																				
Radius	2	1	1																			
Ulna	2	2																				
(Compound)—																						
Clavicle	1	1																				
Humerus	2	1	1																			
Radius and Ulna	2	2																				
Metacarpal	4	3	1																			
Phalanges	5	5																				
(Old)—																						
Clavicle	1	1																				
Humerus	6	5	1																			
Radius and Ulna	9	1	1																			
Radius	2	2																				
Ulna	2	2																				
Dislocations—																						
(Simple)—																						
Scapula (from Clavicle)	1	1																				
Humerus	3	2	1																			
Radius and Ulna	2	2																				

INJURY.	Fatal.		Discharged.		Died.		Under 5.		— 10.		— 15.		— 20.		— 30.		— 40.		— 50.		— 60.		Over 60.			
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.		
																									Discharged.	Died.
INJURIES OF THE LOWER EXTREMITY (continued.)																										
Fractures—																										
(Simple)—																										
Femur	4	2	2	
Neck (Extracapsular)	6	4	
(Intracapsular)	3	1	
Shaft	47	38	9	
Patella	29	19	10	
Tibia	25	21	3	
Fibula	5	5	4	
Tibia and Fibula	61	39	21	
Pott's	12	9	2	
(Compound)—																										
Femur—																										
Shaft	3	1	2	
Tibia	1	1	
Fibula	2	2	
Tibia and Fibula	12	9	1	
(Old)—																										
Femur	1	1	
Neck (Extracapsular)	2	2	
Shaft	8	7	1	
Patella	2	2	
Tibia	2	2	
Pott's	6	5	1	

APPENDIX TO TABLE I.

ACTINOMYCOSIS.

A woman, aged 26, was admitted for actinomycosis of the cheek of sixteen months' duration. About half of one cheek was affected. The disease was treated by large doses of iodide of potassium (up to 30 grs. three times a day), and was completely cured in a few weeks. The diagnosis was confirmed by microscopical examination.

ERYSIPELAS. (See separate table.)

GANGRENE.

A male infant, aged 9 days, was admitted for spontaneous gangrene of both forearms. At birth the arms had been noticed to be swollen and discoloured, but no cause could be found for this condition. On admission, the child was well nourished, and looked otherwise healthy, but both forearms were completely gangrenous and beginning to separate. Both sloughed off spontaneously, and the stumps had almost healed when cerebral symptoms supervened, and the child died on the forty-first day after admission. At the post-mortem, meningitis and a large frontal cerebral abscess were found. There was no other disease of internal organs.

A female infant of 7 months, with signs of congenital syphilis, was admitted with symmetrical gangrene of the skin over the mastoid processes. Two weeks before admission swellings had appeared behind the ears. These had rapidly turned black. On admission a large deep ulcer with black edges was seen in each mastoid region, resembling the ulceration of noma. There was a third similar ulcer in the sub-occipital region. There was no history of varicella or other exanthem. The ulcers spread, and the child died on the fifth day.

A man, aged 67, admitted with gangrene of the leg of two weeks' duration, died next day. He had much albuminuria. There was no post-mortem.

A man, aged 67, with diabetes and gangrene of the leg, died fourteen days after admission.

A man, aged 62, with paraplegia and gangrene of the foot, gradually sank, and died on the twelfth day.

A boot-maker, aged 39, was admitted with spontaneous dry gangrene of the second toe and parts of the first and third toes, of four weeks' duration. The patient was a pale, sickly man, with a feeble circulation, but with no definite disease of heart or arteries; there was no history of hæmaturia; the urine was free from sugar, but contained a trace of albumen. Three years previously the patient had been in the hospital for a similar affection of the opposite foot; the toes had separated, and the gangrene had never recurred. On the present occasion the toes had not yet separated when the patient left the hospital, a month after admission.

A boy, aged 16, was admitted for extensive laceration of both arms by a printing machine. Acute traumatic gangrene set in, and the patient died on the third day.

SEPTICÆMIA AND PYÆMIA. (See separate table.)

TETANUS.

A woman, aged 44, who had cut her thumb with a broken window eleven days previously, was admitted with tetanus of two days' duration. On admission, the patient was very fat; she had a suppurating wound of the interphalangeal joint. There was much stiffness of the neck and jaws, and difficulty in swallowing. The wound was opened up and scraped, but the rigidity gradually increased, and the patient died on the third day after admission.

VENEREAL DISEASES.

A feeble female infant, aged 7 weeks, with congenital syphilis, died of pulmonary collapse five days after admission.

TUMOURS.

Carcinoma.

Of seven patients admitted for carcinoma of the **bladder**, three underwent no operation; one of these was a man of 74 with secondary deposits in the liver; one was a feeble old man of 67 who had had painless hæmaturia for one year, and who was admitted for retention; the hæmaturia subsided after a few days' rest. In both these cases the growth was seen through the cystoscope. The third case was that of a man, aged 49, who had had intermittent painless hæmaturia for twelve months; he refused operation.

A man, aged 45, admitted with a large mass of epitheliomatous **glands** in the neck, died a month afterwards, partly of pneumonia, partly of hæmorrhage from the tumour. At the post-mortem no other primary epithelioma could be discovered.

A woman, aged 61, was also admitted with what appeared to be primary epithelioma of the glands of the groin; the growth was a hard nodular fixed mass as large as a man's fist. No other primary growth could be discovered anywhere. This patient left the hospital without undergoing any operation.

Eighteen men were admitted with malignant disease of the **œsophagus** (one was in the hospital twice). Of these, one underwent tracheotomy and died, three underwent gastrostomy, one dying afterwards. Of the remaining fourteen, ten recovered sufficiently to be able to leave the hospital, and four died.

A carman, aged 54, with a stricture of five months, died of bronchitis and exhaustion on the third day; there was no post-mortem.

An omnibus conductor, aged 58, died on the sixth day after admission of basal meningitis and cerebral abscess; he had also necrosis of the cricoid,

A man, aged 69, admitted for stricture of eighteen months' duration, died of meningitis and pneumonia on the twenty-sixth day. He had been treated with much benefit for several months as an out-patient with Symonds' tubes. At the post-mortem, a large shallow epitheliomatous ulcer was found to have perforated the trachea at its bifurcation; there were secondary growths in the liver.

The fourth case was that of a man, aged 71, who was admitted with a spontaneous fracture of the upper third of the femur. He had recently been under treatment elsewhere "for his liver," which was found to be slightly enlarged. He had slight dysphagia, but could swallow liquids and soft solids without any trouble. On the sixth day his breathing suddenly became very bad, and he died a few hours later. At the post-mortem, the fracture of the femur was found to have been caused by a secondary nodule of epithelioma, an inch in diameter, in the shaft of the bone. There were also secondary nodules in the liver. The primary growth was a large epitheliomatous ulcer involving the lower $4\frac{1}{2}$ inches of the oesophagus, and extending into the pericardium and left auricle. The stomach was full of blood, which had come from the auricle. There was hardly any narrowing of the oesophagus. There was also a small villous papilloma of the bladder, which had caused no symptoms.

Twenty-one men and one woman were admitted for epithelioma of the tongue. Two men were discharged without operation, the disease being too advanced. Upon all the others operations were performed.

A woman, aged 34, with a rodent ulcer of the scalp, was treated with caustics; all the other cases of rodent ulcer were treated by excision.

A labourer, aged 30, from whom a large part of the lower jaw had been removed for multilocular cystic disease four years previously, returned with recurrence in the ascending ramus and in the temporal fossa. His general health was excellent, but no further operation was urged, and he left the hospital.

Of the forty-three women admitted for carcinoma of the breast, two were not submitted to operation. One was a woman, aged 27, with a very large fixed tumour, the other a woman, aged 68, who refused operation.

Of the seventeen cases admitted with recurrent carcinoma of the breast, in two no operation was performed, secondary growths being present in the bones and liver respectively.

A man, aged 38, was admitted with a history of four months' constipation, ten weeks' dysuria and pain in the back; for seven weeks he had passed water only by the help of a catheter. The prostate was found to be very large and hard, and the urine contained blood and pus. Carcinoma of the prostate was diagnosed, and colotomy advised on account of increasing obstruction. Patient left hospital without operation.

Of thirty-three patients admitted for carcinoma of the rectum, eight men and four women underwent no operation; upon ten men and three women inguinal colotomy was performed; from two men and six women the growth was excised.

A man, aged 34, died of carcinoma of the spine. For six months he had complained of increasing pain in the back; on admission he was found to have slight angular curvature of lumbar spine, but no localised tenderness; there was much wasting of the legs. A month later he developed facial erysipelas, and he died two months after admission. At the post-mortem, extensive deposits of alveolar carcinoma were found in the spine, pelvis, skull, and elsewhere; nearly all the growths were suppurating.

Three men and four women were admitted with carcinoma of the **stomach**. Upon two men and one woman no operation was performed ; the men left the hospital ; the woman, aged 54, died of hæmatemesis on the thirteenth day after admission. Upon one man an exploratory laparotomy was performed ; he left the hospital upon the thirty-eighth day after admission. Two women underwent gastro-enterostomy, and the third, who had also ascites and fibroid tumours of the ovaries, and was not known to have any disease of the stomach, underwent ovariectomy ; all three died.

A man, aged 47, was admitted with carcinoma of the **umbilicus**, probably secondary to disease of the stomach ; no operation was performed, and he left the hospital soon after admission.

Sarcoma.

A drayman, aged 49, was admitted for a large sarcoma of the upper end of the **humerus** ; an injury eighteen months ago had been followed by increasing swelling ever since ; secondary growths in the lungs caused the patient to be discharged without operation.

Three male patients were admitted for sarcoma of the **pelvis**. The first, aged 54, had a very large growth of eight months' duration ; he left the hospital in a few days without treatment. The second, aged 46, had a tumour of four months' duration filling up the whole of the iliac fossa ; being unsuitable for any radical operation, he was injected several times with the toxins of erysipelas in increasing doses ; he eventually died of peritonitis, apparently set up by extension of growth through the peritoneum. At the post-mortem, secondary growths were found in the lungs, liver, kidneys, and heart. The third case was that of a schoolboy, aged 14, admitted with a large sarcoma of three months' duration ; he gradually sank and died of pleurisy three months after admission. At the post-mortem, secondary growths were found in omentum and lungs. In all these three cases the ilium was the seat of the growth.

Of three patients with sarcoma of the **chest wall**, one, a man, aged 67, underwent no operation, as the disease was too extensive.

A girl, aged 16, was admitted with a history of seven months' diarrhœa, tenesmus, abdominal pain and loss of flesh. A firm, hard mass could be felt encircling the lower part of the **rectum**. It was thought to be sarcomatous.

A man, aged 53, with a large sarcoma of the **kidney**, left the hospital without operation.

Of the eleven cases of sarcoma of **muscle**, one occurred in the gluteal muscles, four in the thigh, one in the leg, and one in the foot ; the others were cases of recurrence.

A woman, aged 29, was admitted very ill with large tumours of both ovaries, an angular curvature of the spine, and pulmonary phthisis ; malignant disease of ovaries and spine was diagnosed, no operation was performed, and the patient died of exhaustion three weeks after admission. The disease was found to be sarcomatous, primary in the ovaries, secondary in the lumbar spine.

The case of sarcoma of the **peritoneum** was that of a woman, aged 64, from whom a tumour of the omentum had been removed at another hospital fifteen months previously ; she was admitted on account of general distension of the abdomen. She died suddenly on the third day, no operation having been performed. Post-mortem, the whole peritoneum, visceral and parietal was found studded with masses of sarcoma, varying in size from that of a fist downwards. The growth had pressed upon the ureter and caused hydronephrosis.

A harness-maker, aged 73, had been in good health until eight weeks ago, when he had a slight cough followed soon afterwards by dysphagia ; for three weeks he had been unable to swallow any food whatever, and was admitted dying of starvation ; no attempt had previously been made to feed him either by œsophageal tubes or by enemata. He rapidly sank, and died on the second day after admission. Post-mortem, a sarcomatous growth was found completely surrounding and obstructing the lower part of the **pharynx**. There was no ulceration of the mucous membrane. The nature of the growth was confirmed by microscopic examination.

A man, aged 52, was admitted with a rounded mass at the base of the **tongue**, very hard and fixed. There was swelling of the neighbouring epiglottidean fold. Sarcoma was the diagnosis, and no operation was advised.

Cysts.

The case of **aerocele** was that of a man, aged 34, who had always had a small lump in the lower part of his neck. On the day before admission this began to swell, and caused dysphagia and dysphonia. An incision being made down to the swelling, it was found to contain air ; it was not opened, the wound was closed, and the patient made a good recovery, the swelling disappearing.

Of thirteen cases of **dermoid** cyst, in six the tumour was in the neck, in four it was in connection with the orbit, in two with the ovary, and in one case it was in the scrotum ; one patient was admitted twice.

There were six patients with cystic **hygromata**, the tumours being in the neck (two cases), axilla, back of thigh, groin, and scrotum (one case each).

The case of **lymphangioma** of the arm was that of a child, aged 4, who had, on the inner aspect of the upper third of the arm, a soft elastic swelling measuring three inches by two inches. On the skin over it were numerous small vesicles, some of which discharged clear fluid ; it had been first noticed at the age of three months. Parents refused operation.

There were three cases of **papilloma** of the bladder (one patient was admitted twice) ; of these one was a man, aged 22, upon whom no operation was performed, his symptoms being slight ; his hæmaturia subsided in a few days and he left the hospital. The other two were submitted to supra-pubic operation, one dying, one recovering.

A female child, aged 4, was admitted wearing a tracheotomy tube ; tracheotomy had been done four months previously for dyspnoea, the cause of which was not known. All attempts to do without the tube failed ; severe dyspnoea always necessitating its replacement. Sudden dyspnoea eventually proved fatal. At the post-mortem, the larynx was found covered with papillomata, and a groove in the trachea, caused by the end of the tube, had caused fatal blocking of its orifice.

A male infant, aged 9 months, was admitted on account of increasing dyspnoea and hoarseness ; for five months he had been treated by intubation, no exact diagnosis having previously been made. It was intended to perform thyrotomy, but on the evening preceding the day fixed for the operation the child suddenly vomited and coughed up its tube. Severe dyspnoea ensued, and, although tracheotomy was performed almost immediately, the patient died. At the post-mortem, the larynx was found crammed with papillomatous growths ; some were also in the trachea.

Of five patients admitted for **bronchocele** two underwent no operation, in two cases enucleation was performed, and in one case extirpation.

MALFORMATIONS AND DEFORMITIES.

Fourteen male and nine female patients were admitted for **cleft palate** ; of these three males and one female underwent no operation.

In nine male and two female cases, the deformity was complicated with **hare-lip** ; in four male and six female cases there was no hare-lip.

One of the cases was a very remarkable one, the cleft being central with united palate both in front and behind. The cleft was oval, measured $\frac{3}{8}$ inch by $\frac{9}{16}$ inch, and was situated just at the junction of hard and soft palate. It was surrounded by an area of exceedingly thin tissue composed almost entirely of mucous membrane. The extreme tip of the uvula was bifid, but all other parts of the palate and lips were normal. No previous operation had been performed.

Twelve male and eight female patients were admitted for **hare-lip** (six of them were admitted twice) ; plastic operations were performed upon all except two, whose general health was bad. In eleven male and four female cases, the deformity was complicated with cleft palate ; in one male and four female cases, there was no cleft palate.

A girl, aged 10, was admitted with congenital **deformity of both arms** ; on the left side the thumb and little finger were absent, the external condyle was rudimentary, and supination was much restricted ; on the right side the clavicle was natural, the acromion rudimentary, the humerus only three and three-quarter inches long, the radius absent, the carpus deficient, the thumb absent, and the little fingers rudimentary. The fingers were webbed.

DISEASES OF NERVOUS SYSTEM.

Five patients died with **abscess of the brain** ; upon two of these no operation was performed. A girl, aged 18, who had had discharge from the right ear for two years and vomiting for three days, was admitted with much headache, no optic neuritis, perforation of the membrane, a pulse of 96 and a temperature of 100°. The temperature subsequently fell below normal. It was stated that for the last week there had been less discharge than usual from the ear. On the second day she became rapidly worse and died. At the post-mortem a chronic abscess as large as a walnut was found in the right cerebellar hemisphere.

A woman, aged 26, had had four years ago acute necrosis of the tibia and pyæmia ; she had recovered after amputation of the leg, and remained in good health with no symptoms of brain mischief until three weeks before admission, when she complained of headache and pain in the neck. A week later right facial paralysis occurred, and for this she was admitted. At the same time she became deaf on that side for the first time. She had never had discharge from either ear. On admission, she did not seem very ill ; there was partial right facial paralysis and some nystagmus. She was drowsy, but able to talk and take food. Pulse 64 ; temperature 98°. She became rather more drowsy, and on the fourth day was found to have weakness of the right hand, right masseter, and right side of tongue ; also slight optic neuritis. In the next few days she improved a little, but on the ninth day, quite suddenly, while at her tea, she became completely insensible, with increased reflexes, ankle clonus and slow sighing respiration, and she died an hour later. At the post-mortem was found suppurative meningitis, localised to the posterior fossa, and due to a large abscess in the right cerebellar hemisphere, and in the medulla. It had evidently been there for many months, probably years. It had finally ruptured into the sub-dural space.

Of nine cases of **hysteria**, the only one worthy of special mention is the following : A woman, aged 22, was admitted for otitis media and headache ; for fifteen years she had had discharge from the left ear, following scarlet fever. Two years ago the mastoid had been trephined at another hospital

with temporary benefit, and eight months ago, at the same place, the cerebellum had been trephined on account of headache, vomiting and staggering. No pus was found at the operation, but a few days later a little came away from the wound. The symptoms were relieved for a time, but the headache soon returned and has persisted ever since. One brother, and also an intimate friend of the patient's, had died of cerebral abscess. On admission, the chief complaint was of severe occipital headache; there was very slight weakness of left hand, left side of the tongue, and she walked with a staggering gait. Temperature was 98°; pulse 80. There was evidence of old, but no recent optic neuritis. There was no vomiting. The patient was kept under observation for a month, and after a general consultation it was agreed that the case was one of hysteria. The symptoms gradually subsided. Subsequent history of the case after the patient left the hospital confirmed the diagnosis.

A boy, aged 2, died of septic cerebro-spinal meningitis on the fourth day after admission. A chronic abscess of the petrous bone appeared at the post-mortem to have been the starting point.

A cabinet-maker, aged 45, was admitted for chronic œdema of one side of the face and neck; it had existed for ten weeks. Over the affected area were numerous small vesicles, apparently lymphatic. There was little or no pain; there was some otorrhœa. He gradually became worse and died on the fifty-first day after admission. The temperature had been normal until just before death. At the post-mortem one lateral half of the dura mater was found much thickened by chronic inflammation, and there were numerous small points of suppuration. The lateral sinus on the same side was thrombosed.

A man, aged 24, sent to the hospital as a case of acute tetanus, was found to be suffering from general spasmodic contraction of the muscles of various parts of the body. No special treatment was adopted and he recovered completely.

DISEASES OF THE EYE.

A female infant, aged 5 months, admitted for conjunctivitis, died of marasmus and pneumonia on the seventh day.

A girl, aged 2, admitted for keratitis, developed broncho-pneumonia, and was transferred to a medical ward, where she died.

DISEASES OF THE EAR.

A woman, aged 23, was admitted with the following symptoms: Since childhood she had had discharge from both ears; for one month she had been languid and ill, for three days drowsy, with slow speech, frontal headache, and vomiting four or five times a day. On admission, she was in an apathetic condition, seldom speaking. There was no paralysis, no optic neuritis, and no local signs of disease, except a perforation of each membrane and a slight discharge. The pulse varied between 50 and 66, the temperature between 99° and 100°. A consultation was held, and opinions were divided as to whether a cerebral abscess was present or not. The patient remained in this condition about ten days, then began to improve, and left the hospital on the thirty-sixth day, apparently quite well.

A boy, aged 5, had had offensive discharge from both ears for four and a half years; for one week before admission he had had symptoms of meningitis. He was admitted in a semi-comatose condition, and died on the fifth day. At the post-mortem, there was found diffuse purulent meningitis, perforation of the dura mater, and caries of the temporal bone, but no thrombosis or cerebral abscess.

DISEASES OF THE DUCTLESS GLANDS.

A woman, aged 46, admitted for hæmorrhoids, was found to have **myxœdema** and to be unfit for any operation. She was transferred to a medical ward, where she eventually died.

DISEASES OF THE VASCULAR SYSTEM.

A slaughterer, aged 45, was admitted with a large popliteal **aneurism** of four months' duration, and uræmia, due to acute nephritis. No operation was performed; the aneurism gradually consolidated, and the patient left the hospital on the fifty-eighth day, quite cured, both of the aneurism and of the nephritis.

A gardener, aged 57, was in the hospital for a few days; two years and five months previously the innominate artery had been tied at another hospital for aneurism of the subclavian and axillary arteries. He was found to be quite well, and there were no signs of any recurrence of the disease.

DISEASES OF THE DIGESTIVE SYSTEM.

A woman, aged 32, was admitted with a large **cyst** in the right side of the abdomen. It had been noticed for a few days only; it lay immediately below the liver and could easily be felt also behind in the loin. There were no urinary symptoms. Aspiration yielded twelve ounces of rather viscid, clear whitish fluid, with much cholesterin. The tumour was considered to be probably a dilated gall bladder, but as it was not causing any inconvenience to the patient, no further operation was performed.

Hernia.

The number of patients (144) admitted for reducible hernia is greater than in the previous year (119). The cases of irreducible hernia (36) show a similar increase in number (27 last year). The number of strangulated herniæ (47) is almost exactly the same as in the two preceding years (45 and 46).

A laundress, aged 57, with a large irreducible femoral hernia, was admitted with the view of having an operation for radical cure. For three months she had had some abdominal pain and occasional vomiting, attributed to increase in the size of the hernia. Soon after admission the patient developed bronchitis, and the operation was postponed. On the thirteenth day after admission she became very much worse, and died rather suddenly. At the post-mortem there was found a primary carcinomatous ulcer of the stomach measuring three inches by three and a half, with secondary deposits in the gall bladder and peritoneum. The hernia consisted wholly of omentum. This patient had never had hæmatemesis, had never complained of pain after food, and during her stay in the hospital had vomited only once.

A fat woman, aged 63, was admitted with a large irreducible umbilical hernia of four years' duration. A year ago the skin over the hernia became inflamed, one month ago a definite ulcer formed. Two days before admission intestinal contents first began to come through the bottom of the ulcer. On admission, intestine was visible on the floor of the ulcer, but it was shut off by adhesions from the general peritoneal cavity. On the third day after admission the patient suddenly became collapsed, and died in a few hours. At the post-mortem it was found that part of the herniated and perforated small intestine had slipped back into the abdomen, and thus set up fatal peritonitis.

A male infant, aged 1 hour, was admitted with an extensive protrusion at the umbilicus of most of the large intestine and some of the small. The child gradually sank and died on the second day.

Intestinal Obstruction.

An iron-moulder, aged 56, was admitted with acute intestinal obstruction. Four days previously, shortly after a heavy meal, he had been suddenly seized with violent abdominal pain and vomiting. Vomiting and constipation continued until his admission. His abdomen at this time was tender, but not greatly distended. He passed flatus occasionally. He was treated by enemata, and on the seventh day after admission his bowels acted. He left the hospital on the twenty-eighth day, quite well.

A woman, aged 46, was admitted with a history of fourteen days' absolute constipation. Her general condition seemed good, and the abdomen was only moderately distended. Shortly after admission she became collapsed, and a few hours later she died. There was no post-mortem.

Of nine children admitted for **intus-susception**, two were not subjected to operation. A girl, aged 2, had had for twelve hours abdominal pain, vomiting, and passage of blood and mucus per rectum; a definite tumour could be felt in the left hypochondrium. A large enema of warm water reduced the intus-susception, and the child made a rapid and complete recovery.

A male infant, aged 6 months, was admitted for intus-susception. Five days previously there had been a sudden attack of abdominal pain, followed next day by the passage of blood per rectum. Constipation was present. On admission the abdomen was distended, and the child seemed to be in pain. The child vomited several times. A rectal examination under chloroform revealed nothing abnormal in the abdomen. The bowels were then opened several times, so no operation was performed. The child died on the second day after admission. At the post-mortem, an unreduced ileocolic intus-susception was found high up and far back in the right lumbar region.

Of the ten patients admitted for **inflammation of the vermiform appendix**, one was in the hospital no less than five times during the year, and had been in on several previous occasions. On this patient, and on seven others included in the medical tables, operations were performed. (See Appendix to Table II.) Three male patients, aged 8, 12, and 38, recovered without operation; the symptoms were slight, and subsided soon after admission without any suppuration having occurred.

DISEASES OF THE GENITO-URINARY ORGANS.

A man, aged 58, admitted with **cystitis**, gradually sank and died four weeks later. At the post-mortem, advanced chronic interstitial nephritis was found.

A man, aged 40, was admitted with tuberculous cystitis. For nine months he had had frequent and painful micturition, for two months hæmaturia; tubercle bacilli and much blood and pus were present in the urine. He was treated with frequent injections of iodoform solution into the bladder, and left the hospital after ten weeks' treatment, much improved. Many months later he was known to be in good health, and to have lost practically all his symptoms.

A man, aged 38, was admitted very ill with hæmaturia and stricture; he died on the third day, and at the post-mortem the bladder and both kidneys were found to be dilated and in a state of acute suppuration.

Of twelve patients admitted for **enlargement of the prostate**, one was treated by castration and died, one by supra-pubic cystotomy and recovered; this patient was admitted four months later on account of the fistulous opening, which closed after a fortnight's rest and washing out of the bladder. Upon the remaining nine patients no operation was performed. Two died;

one was a feeble man of 65, with an ischio-rectal abscess ; he died on the third day ; there was no post-mortem. The other was a man, aged 62, who was admitted for retention, which was relieved by catheter ; he died on the second day and was found at the post-mortem to have greatly dilated kidneys.

A sailor, aged 29, with **acute prostatitis** of a fortnight's duration, was admitted with symptoms of acute septicæmia, of which he died two days later. At the post-mortem, suppuration in the cellular tissue of the pelvis and infarcts in the lungs were found.

A man, aged 31, who had had one testis removed three years ago for **tuberculous disease**, was admitted with a large swelling of the prostate, supposed to be tuberculous ; there was also tuberculous disease of the remaining epididymis and a perineal fistula. No operative treatment was recommended.

A bricklayer, aged 43, from whose left **kidney** several stones had been removed in the hospital nine months previously, was re-admitted very ill with albuminuria, hæmaturia, diarrhoea, vomiting, a sub-normal temperature, and a considerable swelling in the left loin. He was passing about ten ounces of urine daily. These symptoms had existed seven weeks. The patient died on the third day. At the post-mortem, the left kidney was found much dilated, full of pus, and containing a stone as large as a walnut. In the ureter, near its vesical end, were three calculi as large as peas. The opposite kidney was much hypertrophied and in a state of recent parenchymatous inflammation. There was no calculus or distension of this kidney.

Of seventy-nine patients admitted for **stricture of the urethra**, twenty underwent urethrotomy ; of the whole number, seven died ; one after internal urethrotomy, one after external urethrotomy ; five died upon whom no operation had been performed. A brush-maker, aged 38, was admitted for retention ; he had also phthisis ; he was being put under chloroform preparatory to the passage of a catheter, when his heart failed ; at the post-mortem, extensive disease was found both in lungs and in kidneys. A photographer, aged 57, admitted for stricture and cystitis, became maniacal soon afterwards, and died on the tenth day ; at the post-mortem, much old and recent peritonitis were found, as well as two stones in the bladder. A cabinet-maker, aged 67, with stricture of fifty years' duration, was treated by continuous dilatation ; he died two months after admission, of pneumonia ; at the post-mortem he was found to have chronic interstitial nephritis and some dilatation of bladder and kidneys. A case-maker, aged 69, was admitted very ill with albuminuria and a tight stricture ; he died of uræmia on the fourth day, and at the post-mortem the kidneys were found to be much dilated and with very little cortex. A man, aged 65, was admitted with a tight stricture of many years' standing and a temperature of 102°. He was treated with catheters and improved, his temperature becoming normal ; on the twenty-sixth day, thrombosis of the left axillary vein occurred, followed two days later by vomiting, shivering, drowsiness, delirium, hæmaturia and albuminuria. The temperature again rose gradually until the patient died thirty-one days after admission. The post-mortem showed suppurative pyelonephritis and old and recent pericarditis ; the left innominate, internal jugular, subclavian, axillary and femoral veins were in a state of suppurative thrombosis ; there were infarcts in spleen and kidney ; springing from the first part of the arch of the aorta was a sacculated aneurism $1\frac{1}{2}$ inches in diameter, partly filled with suppurating blood clot, the source of the infarcts. The suppuration of the aneurism was evidently secondary to the pyæmic condition started by the disease of the kidneys and bladder. A woman, aged 45, who had had one kidney removed eight years previously, for pyonephrosis, was re-admitted with uræmia and insanity ; she was sent to an asylum.

Five children were admitted for **noma** ; of these two died ; one was cauterised with nitric acid ; the other was a feeble, unhealthy female infant of 18 months ; she had had measles one month previously, and for four days had had signs of noma ; the brawny swelling and redness extended upwards as high as

the umbilicus. Temperature 105°. Incisions were made into the inflamed parts, but the child died on the third day ; the post-mortem showed extensive cellulitis within the pelvis and septic broncho-pneumonia.

DISEASES OF THE ORGANS OF LOCOMOTION.

Of forty-six patients admitted for **caries of the spine**, two died. A boy, aged 14, admitted with caries of the dorsal spine, a large psoas abscess and a high temperature, died three months later of exhaustion and amyloid disease ; the post-mortem showed very extensive disease. A boy, aged 18, admitted with caries of the dorsal spine and extensive suppuration, died of exhaustion six months later. The post-mortem showed that the suppuration extended continuously from the neck to the thigh.

A very remarkable case of **softening of the tibia** was in the hospital for a few weeks. The patient was a woman, aged 37, who six years previously had fallen and struck her right leg against a wall ; three months later the right knee became stiff and painful ; two years later, after a confinement, the knee became much worse, the joints of the fingers also swelled. On admission, the right knee was semiflexed, but under an anæsthetic considerable over-extension could be produced, the movement taking place not at the joint, but apparently a little distance below it. The limb was put in plaster for six months and then in a leather splint. Amputation was advised, but refused. The patient subsequently went to another hospital, where the limb was amputated ; it was then found that about one-third of the upper end of the tibia was completely devoid of earthy matter and could be bent like a piece of indiarubber ; the rest of the bone appeared to be normal, and there was a very abrupt line between the soft and hard portions of the bone. (See St. Thomas's Hospital Museum.)

A feeble woman of 58, admitted for neuralgia of a thigh stump, died suddenly a few days afterwards. Post-mortem showed the cause of death to be chronic interstitial nephritis and emphysema of the lungs.

Of ninety patients admitted for **tuberculous disease of the hip**, only one died ; this was a boy, aged 3, who was admitted for early hip disease ; an operation for the radical cure of hernia had been performed upon him six weeks earlier, and tubercle of the peritoneum had then been noticed. After admission an abscess formed, and on two occasions this was opened and scraped out. Tuberculous peritonitis progressed, and the boy died of meningitis five months after admission.

A female infant, aged 20 months, admitted with acute **suppurative arthritis of both elbows**, died six days later with pericarditis, abscesses in the lungs and other signs of pyæmia.

A girl, aged 12, admitted for pneumonia, developed acute suppurative arthritis of the **knee** a few days later. She was treated by free incisions and drainage, and made an excellent recovery.

DISEASES OF THE CUTANEOUS SYSTEM.

Only one patient died ; this was a boy, aged 14, who was admitted with **pityriasis rubra** of one month's duration. He died one month later of suppurative pyelitis.

A woman, aged 32, was admitted for ulceration of the lower lip and much swelling of the cheek ; this had begun as a vesicle or pustule a few days previously. The patient's child had been vaccinated a fortnight before ; the disease was concluded to be **vaccinia**, and the patient made a speedy recovery.

A similar case was that of a paper-sorter, who, when first admitted, was thought to have anthrax, but this was shown also to be vaccinia of the face.

There were two curious cases of **phagedœnic ulceration** of a somewhat doubtful nature. A hawker, aged 18, who had had ordinary lupus of the face and neck for eighteen months, was admitted with phagedœnic ulceration of the lips and cheek. The lupus had run the usual slow course until six weeks before admission, when the ulceration rapidly extended. On admission, a large part of both upper and lower lips and much of one cheek had been completely destroyed, so that all the teeth of that side were exposed; the patient was pale and thin; there was no evidence of any syphilitic taint. Under the influence of iodide of potassium and good feeding, the ulcer rapidly healed, and the patient left the hospital on the thirty-ninth day quite well, but with much deformity of the cheeks.

A compositor, aged 31, was admitted with a phagedœnic ulcer on the front of the shoulder and root of the neck. It had begun two months previously as a pimple, which broke and then gradually extended. On admission, there was a shallow ulcer about five inches in diameter with a well-marked, sharply cut edge; there was a history of a syphilitic sore eleven years previously. There was no evidence of tubercle. The patient was in the hospital eight months, during which time the ulcer continued to spread although healing up partially now and then. It was scraped and cauterised many times and treated by iodide of potassium, mercury and various other drugs.

ABSCESS.

Three male and three female patients died. An emaciated male child, aged 3 months, with an axillary abscess, died of marasmus.

A watch-maker, aged 56, admitted with an axillary abscess and cellulitis following facial erysipelas, developed pneumonia, and died on the twentieth day.

A cabinet-maker, aged 63, admitted with an ischio-rectal abscess, developed septicæmia and albuminuria, and died three weeks after admission. There was no post-mortem.

A female child, aged 5 months, was admitted with a swelling in the neck of six weeks' duration; three weeks ago an abscess had been opened in the neck and a sinus had been present ever since; for two weeks there had been dyspnoea. On admission, there was considerable dyspnoea, and arrangements had been made to lay the sinus open and explore the swelling; the child, however, grew suddenly much worse and died. The post-mortem showed the presence of a considerable but very deep-seated abscess pressing upon the side and front of the pharynx.

A female, aged 8 months, died a few days after admission for an abscess of the thigh of two weeks' duration. The post-mortem showed tuberculous laryngitis and pneumonia.

A woman, aged 24, was admitted with a pelvic abscess, apparently due to uterine inflammation a year previously. The patient gradually sank, and died several weeks after admission.

BURNS AND SCALDS.

Thirty-four patients were admitted for burns; of these seven died. A man, aged 72, who accidentally set fire to his coat-tails, was admitted with an extensive burn of the back, and died in ten hours. A boy, aged two, with an extensive burn of trunk, face and arms, died in six hours. Three girls, aged 3, 9 and 15, admitted with extensive burns caused by clothes catching

fire, died within a few hours. A woman, aged 33, with burns of face, arms, chest and legs, caused by the upsetting of a lamp, became gradually comatose and died three days after admission ; there was no post-mortem. A girl, aged 6, admitted with a recent burn of chest and arm, died suddenly, on the forty-fifth day, five days after a plastic operation had been performed upon the ulcerated surface ; there was no post-mortem.

Of thirteen patients admitted for scalds, none died.

INJURIES OF THE HEAD.

Three patients were admitted for **gunshot wounds**. A girl, aged 17, made an excellent recovery from a pistol wound of the face ; the bullet entered just behind the angle of the jaw on the right side, traversed the palate, and emerged just below the left orbit ; she left the hospital on the twenty-fourth day, quite well.

An insane clerk, aged 34, fired five shots from a revolver into his temple ; none of the bullets penetrated the skull, and but little harm was done ; the wounds healed quickly, and he went to an asylum on the twenty-third day.

The third case ended fatally after an operation to extract a bullet from the brain. (See Trephining.)

Of fifty patients admitted for **concussion**, none died.

Of sixteen patients admitted for **fracture of the base** of the skull, eight died.

A woman, aged 60, fell down stairs, and died two days later ; the post-mortem showed much laceration of the brain.

A male infant, aged 15 months, fell from a window, and died within an hour.

A boy, aged 8, fell twenty-five feet on to his head ; he was admitted unconscious, bleeding from the nose, with conjugate deviation of eyes to the left, and with a hæmatoma in the occipital region. On the third day, spasmodic movements of the arms and legs and other signs of cerebral irritation set in. On the fifth day, the temperature, hitherto normal, began to rise. The child gradually became weaker, optic neuritis developed, the convulsive movements continued, and the child died on the twelfth day, never having regained consciousness. The post-mortem showed a fracture across the middle fossa, purulent basal meningitis, and slight laceration of the brain.

A boy, aged 9, was knocked down, and a wheel passed over his head ; he died in a few hours.

A solicitor, aged 35, threw himself from a window, and died half an hour after admission ; besides an extensive fracture of the base, he was found to have ruptured his spleen, and to have fractured all the ribs on one side of the chest.

The other three fatal cases died after operations. (See Trephining.)

A woman, aged 32, admitted bleeding from both ears after a fall upon the head, made a good recovery, and left the hospital on the seventh day.

A woman, aged 46, who had fallen downstairs, made a good recovery after being unconscious several days, and having had bleeding from the ear, facial paralysis, and convulsions. She left the hospital at her own request on the twenty-fourth day, apparently quite well.

A window-cleaner, aged 22, fell from a second-floor window, and was admitted with a compound fracture of the frontal bone, without depression, and a fracture of the base, he was bleeding from ear and nose and into the orbit; after several days of unconsciousness, with symptoms of cerebral irritation, he made an excellent recovery, leaving the hospital on the forty-first day, quite well.

A carpenter, aged 36, who had fallen forty feet from a scaffold, was admitted unconscious and bleeding from the ear, facial paralysis supervened later, drowsiness was re-placed by great irritability, and the patient eventually made a partial recovery, being sent to an asylum on the thirty-ninth day. When last seen, five months later still, he had been discharged from the asylum, but he still had facial paralysis, and his mental condition was not quite satisfactory.

A boy, aged 16, and a man, aged 42, both of whom had bleeding from the ear and facial paralysis, made good recoveries, leaving the hospital on the forty-second and thirty-ninth days after admission.

A boy, aged 9, with bleeding from the ear and symptoms of cerebral irritation, made an excellent recovery, leaving the hospital on the thirtieth day.

A man, aged 23, admitted with bleeding from the ear, probably due to a fractured base, discharged himself from the hospital on the third day.

Four patients were admitted for **simple fracture of the vault of the skull**.

A boy, aged 14, admitted with a simple depressed fracture of the vault, had no bad symptoms of any kind, and left the hospital on the thirty-seventh day, quite well.

A boy, aged 2, with a similar injury, made an equally good recovery, no operation being performed in either case.

A carman, aged 50, fell from the shafts and struck the back of his head, he walked at once to the hospital, and was found to have a small contused wound at the back of the head, but no signs of any cranial or intra-cranial injury. Later he had a "fit," and returned to the hospital, he then gradually became unconscious, the right arm and leg became paralysed, and he died shortly after admission to the ward, and six hours after the accident. At the post-mortem a linear fracture of the vault was found beginning at the lambdoid suture, one inch to the left of the middle line, thence running horizontally forwards to end just above the root of the zygoma on the same side; there was no depression of bone; there was a large extra-dural extravasation of blood and slight laceration of the surface of the brain.

The fourth case died after an operation. (See Trephining.)

Eight patients, all males, were admitted for **compound fracture of the vault without depression**, none were trephined, and all made good recoveries. One of these was a man, aged 50, who had fallen from a ladder; a linear fracture of the frontal bone was associated with complete blindness of one eye and paralysis of the third nerve on the same; he left the hospital on the twenty-second day, quite well except for the eye symptoms, which persisted.

Of five patients admitted for **compound depressed fracture of the vault**, four recovered after trephining (or elevation of bone); the fifth patient was a boy, aged 7, who had been hit on the back of the head by a brick; there was a small depressed fracture; no operation was deemed advisable, and the patient made an excellent recovery, leaving the hospital on the twenty-first day, quite well.

A man, aged 38, died of traumatic **extra-dural hæmorrhage**; he had fallen from a second-floor window, and died within an hour. At the post-mortem there was no fracture of the skull and no visible laceration of the brain, but there was a large hæmorrhage (about four ounces) within the sub-dural (arachnoid) space. No cause for the hæmorrhage, other than injury, could be discovered.

INJURIES OF THE ABDOMEN.

Of thirteen cases of injury to the **kidney**, only one ended fatally; the patient was a boy, aged 15, upon whom a heavy bale of paper had fallen, he was admitted collapsed, and died next day; at the post-mortem, he was found to have rupture of one kidney, laceration of the spleen, fracture of the pelvis and many ribs, and injuries to the lungs. A girl, aged 6, who fell off a wall on to a scraper, was admitted eleven days later on account of hæmaturia and *melœna*; the hæmaturia lasted three weeks, and the patient then made a good recovery; in this case it was thought that there had been a severe injury to the duodenum as well as to the kidney.

A butcher, aged 21, was admitted in a state of collapse, having fallen twelve feet and struck the left loin. There was considerable pain and tenderness over the pelvis and loins, and much hæmaturia. This persisted almost continuously for six weeks, and at one time was so profuse that the propriety of an operation upon the kidney was considered. The hæmorrhage eventually ceased spontaneously, and the patient left on the sixtieth day with a slight trace of albuminuria, but otherwise quite well.

Two patients died of rupture of the **liver**; a *van-boy*, aged 16, who had been run over, died in seventeen hours, and post-mortem was found to have also injured his spleen and kidney, and to have broken many ribs; another boy, aged 17, who had been crushed between buffers, died on the following day; in this case, also, numerous ribs were broken, and there was much blood in the peritoneal and both pleural cavities.

INJURIES OF THE CHEST.

A man, aged 44, recovered after a pistol wound of the chest wall; the bullet had lodged in a rib.

An Italian asphalter, aged 27, was accidentally shot with a revolver; the bullet passed through the forearm and entered the upper part of the chest; the patient was paraplegic on admission, this condition gradually increased till the patient died on the seventh day. At the post-mortem the bullet was found to have traversed the right lung and to have buried itself in the body of the fifth dorsal vertebra. It had partially penetrated the spinal canal and contused the spinal cord.

A labourer, aged 37, was run over across the chest by an omnibus wheel; he had fracture of many ribs, and died a quarter of an hour after admission; there was no post-mortem.

INJURIES OF THE NECK.

Two women, aged 19 and 20, were admitted for **bullet wounds** of the neck; in the first case the bullet buried itself in the side of the neck rather low down; in the second case the bullet entered the cheek, penetrated the pharynx, and passing downwards was lost in the neck. In neither case was any attempt made to extract the bullet; in both cases no bad symptoms occurred, and the patients made excellent recoveries.

Four patients were admitted for suicidal **incised wounds** of the throat. One was a man, aged 47, in whose case the air passages were not opened; he made a good recovery. A shoe-maker, aged 68, had cut deeply through the crico-thyroid membrane; a tracheotomy tube was inserted, but the man died on the seventh day of congestion and œdema of the lungs.

A man, aged 86, inflicted a small superficial wound through skin and sternohyoid muscles only ; he developed pneumonia and died on the seventh day ; the wound remained healthy, and there was no suppuration in any part of the neck.

A man, aged 52, died seventeen hours after cutting a large piece clean out of his larynx with a dinner knife.

INJURIES OF THE BACK.

Of five patients admitted for fracture-dislocation of the cervical spine, one recovered. He was a boy, aged 15, who fell three feet vertically on to his head ; he was unconscious for three quarters of an hour ; he was admitted with the signs of a severe contusion or slight fracture of the cervical spine. There were no nervous symptoms ; after a month's rest in bed, he recovered sufficiently to be sent to Swanley, but a recurrence of pain and the formation of a bony swelling about the lower part of the neck necessitated re-admission, and further rest for another five months, after which he left the hospital quite well, but still wearing a poroplastic apparatus.

A woman, aged 50, who had fallen downstairs and become completely paraplegic at once, died on the fourteenth day ; the fracture was through the third cervical vertebra, and the most prominent feature of the post-mortem was suppurative pyelitis.

A boy, aged 14, became entangled in machinery ; on admission there was complete paralysis of all the limbs and absence of deep reflexes ; he died in four hours. The post-mortem showed a fracture-dislocation of the third and fourth cervical vertebrae and complete crush of the cord.

A gas-fitter, aged 27, fell twenty feet, striking the back of his neck across a railing ; he was completely paraplegic on admission, and died in eighteen hours. There was no post-mortem.

A builder, aged 45, admitted completely paraplegic after a fall, died in twelve hours ; no post-mortem.

Two patients recovered after fracture of the dorsal spine.

A watchman, aged 60, fell twelve feet on to his back, and was admitted with paraplegia and loss of knee jerks ; the latter had returned by the eighth day ; he developed cystitis, and then dementia, and was finally sent to an asylum on the sixtieth day, still partially paraplegic, and with exaggerated knee jerks and ankle clonus.

A man, aged 44, who jumped from a window during a fire, was admitted with partial paraplegia ; he developed cystitis and increase of reflexes ; he gradually recovered, and was discharged after three months able to get about on crutches, but with reflexes still exaggerated. Six months later he was re-admitted for a short time, and was found to be still partially paraplegic.

A sign-writer, aged 49, fell from a scaffold and struck the lower part of the back. He walked to the hospital, then suddenly became paraplegic. Both motion and sensation gradually returned in the course of the next few days, and the patient discharged himself five days after admission.

INJURIES OF THE PELVIS.

A man, aged 29, was admitted with fracture of the pelvis and rupture of the urethra, caused by a heavy weight falling upon him ; a catheter was tied in until the hematuria had ceased, which was at the end of a fortnight. He made a good recovery and left the hospital on the fiftieth day after the accident.

A carpenter, aged 67, who had been crushed between a train and a platform, died a month later ; at the post-mortem an extensive fracture of the pelvis was found and chronic interstitial nephritis.

Six patients, aged 10, 14, 22, 24, 29 and 36, were admitted for **rupture of the urethra** alone. Three were treated by catheter only; two by immediate perineal section; the sixth did not come to the hospital until the fifth day after the injury; extravasation of urine was already present, and was treated by free incisions. All six made a good recovery.

INJURIES OF THE UPPER EXTREMITY.

A woman, aged 73, admitted with a fractured clavicle, died on the eighteenth day, of chronic interstitial nephritis.

An engineer, aged 55, who had been knocked down and run over, was admitted with a dislocated shoulder; he was very fat; soon after admission he began to vomit; this continued until the fourth day, when he died. The post-mortem showed a fatty liver and old pleurisy, but no injury or other disease of abdominal viscera.

INJURIES OF THE LOWER EXTREMITY.

Four patients, whose ages varied from 12 to 52, with punctured wounds of the knee joint, recovered without any complication.

A hawker, aged 67, admitted for extra-capsular fracture of the femur, died on the forty-sixth day. The post-mortem showed advanced interstitial nephritis.

Nineteen men and ten women were admitted for simple fracture of the patella; of these three women and one man had already sustained a previous fracture of the other patella. Four male patients were treated by wiring operations, the others by mechanical appliances only.

A printer, aged 75, admitted for simple fracture of the tibia, gradually sank and died on the forty-sixth day, apparently chiefly from old age.

A washerwoman, aged 67, admitted for a simple fracture of tibia and fibula, died of pneumonia on the sixteenth day; the post-mortem showed chronic interstitial nephritis.

A carpenter, aged 81, with a simple **Pott's fracture** and a fracture of the forearm, died of bronchitis on the eighth day; no post-mortem.

A railway servant, aged 44, with an extensively crushed thigh, caused by the passage of a railway truck over it, died of shock in three hours.

A man, aged 47, both of whose legs had been crushed by a train, died of shock on the second day.

A porter, aged 49, crushed both legs in a lift and lost much blood; some local gangrene occurred in the left leg, but the right leg, which at first was cold and pulseless, and looked the worse of the two, recovered its circulation. The patient died on the third day. At the post-mortem, a fatty liver was found and some simple ulceration of the stomach; complete rupture of both anterior and posterior tibial arteries was found in the leg that did not become gangrenous.

A man, aged 60, was admitted with **dislocation of the hip** and fracture of the femur and ribs, caused by having been knocked down and dragged by a tramcar. The dislocation was reduced, but a large bedsore developed over the sacral region, which had been much bruised. The patient gradually sank and died on the forty-third day.

Two male patients, aged 26 and 33, and two female patients, aged 8 and 30, with dorsal dislocation of the hip, made good recoveries after reduction.

TABLE II. (continued).

AGE AND SEX.

OPERATIONS.	TOTAL.		Discharged		Died.		Under 5 Years.		-10.		-20.		-30.		-40.		-50.		-60.		-70.		Over 70.			
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.		
REMOVAL OF TUMOURS (continued.)																										
Bronchocele	1	2	1	2
Epithelioma—																										
Back	1	..	1
* Bladder	..	1	..	1
Cheek	6	..	6
Ear	1	..	1
Eyelid	1	1	1	1
Floor of Mouth	3	1	3	1
Glands	7	1	7	1
Jaw	1	..	1
Leg	..	1	..	1
Lip	7	..	7
Palate	4	..	4
Scalp	..	4	..	4
Scrotum	3	..	3
Tonsil	1	..	1
Rodent Ulcer—																										
Face	5	3	5	3
Leg	1	..	1
Neck	..	1	..	1
Sarcoma—																										
Back	..	1	..	1
Chest Wall	..	2	..	2
Chin	1	..	1

* For other cases see Cystotomy.

OPERATIONS.	AGE AND SEX.																								
	TOTAL.		Discharged		Died.		Under 5 Years.		- 10.		- 20.		- 30.		- 40.		- 50.		- 60.		- 70.		Over 70.		
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	
OPERATIONS ON TONGUE (continued.)																									
For Epithelioma (continued)—																									
Excision of one Lateral Half (Through the Mouth)—	7	...	7
Without Removal of Glands...	2	...	2
Without Removal of Glands (With Splitting of Cheek)—	2	...	2
Without Removal of Glands	3	...	3
Without Removal of Glands (With Removal of part of Jaw)—	1	...	1
Without Removal of Glands	1	...	1
Local Removal
For Innocent Tumour—																									
Local Removal of Adenoma..
Local Removal of Lymphan- gioma ...	1	...	1
Excision of Chronic Ulcer ...	1	...	1
Excision of Tuberculous Ulcer	1	...	1
OPERATIONS ON BURSE, FASCLE AND TENDONS.																									
Burse Removed ...	3	5	3	5
Ganglia Incised or Removed	...	6	...	6

AGE AND SEX.

OPERATIONS.	TOTAL.		Discharged.		Died.		Under 5 Years.		-10.		-20.		-30.		-40.		-50.		-60.		-70.		Over 70.		
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	
																									M.
ABDOMINAL SECTION (continued.)																									
For Hydatid of Liver ...	3	2	2	1	1	1
Cholecystomy ...	1	4	...	4	1	1
Suture of Ruptured Stomach...	1	1	1
For Plastic Operation on Fibrous Stricture of Pylorus...	1	1	2
Gastrostomy ...	4	...	2	...	2
Gastro-Jejunostomy	2
For Acute Intestinal Obstruction— <i>Strangulation by Band</i> ...	2	3	...	1	2	3
<i>Cause uncertain</i> ...	6	1	2	1	5	2
Intus-susception ...	9	1	6	1	3	3
For Appendicitis ...	2	2	...	1	2	1
Enterotomy*— <i>For Faecal Fistula</i>	1	1
" <i>Fibrous Stricture of Colon</i> ...	1	1	2
" <i>Carcinoma</i> ...	1
Intestinal Anastomosis ...	1	...	1
For Extra-Uterine Gestation	...	6	...	5	1	1
For Drainage of Pelvic Abscess	...	1
Removal of Uterine Appendages...	2	...	2

* Other cases under Operations for Hernia and Intus-susception.

STATISTICS OF ANÆSTHETICS.

During the year 1895 Anæsthetics were administered 5,485 times.

Chloroform	2,553	times.
Nitrous Oxide Gas	1,507	,,
Ether	242	,,
Gas and Ether	1,150	,,
Ether and Chloroform	33	,,
Total	5,485	,,

There were two deaths.

1. A man, aged 38, was admitted with stricture and retention, he had also phthisis. Chloroform was administered to him twice ; on the first occasion there was much struggling ; on the second, he died suddenly while a catheter was being passed. At the post-mortem, the lungs, liver, kidneys and heart were all found to be unsound.

2. A man, aged 28, who was somewhat debilitated by the effects of suppuration, was being put under chloroform preparatory to the grafting of a large ulcer of the thigh. He died suddenly, and at the post-mortem nothing abnormal was discovered.

APPENDIX TO TABLE II.

OPERATIONS ON THE EYE.

There were no deaths during the year.

PLASTIC OPERATIONS.

A boy, aged 16, was admitted with an **artificial anus**. Right lumbar colotomy had been performed at another hospital six months previously for acute intestinal obstruction. A small hard lump could be felt in the right iliac region. An attempt was made to close the artificial anus by a plastic operation, but failed, the wound breaking down a few days later. The patient gradually became more and more emaciated, and died of the original disease six months after admission and five months after the operation. At the post-mortem, a myxo-sarcoma of the ascending colon was found with dissemination over the peritoneum.

The other plastic operation upon an artificial anus was to improve the spur of a colotomy wound, the colotomy having been done for carcinoma of the rectum.

One of the plastic operations upon the **palate** was for the closure of an artificial cleft that had been left after the removal of a fibro-angioma of the upper jaw.

Of the eleven operations upon contracted scars, seven were upon old burn scars. A girl, aged 6, upon whom a plastic operation had been done for a burn of the arm on the fortieth day after the accident, died suddenly five days later; there was no post-mortem.

A boy, aged 3, died after an operation for **ectopia vesicæ**; the first part only of the operation had been performed, a bridge of skin having been dissected up from the abdominal wall, and an ivory plate inserted beneath it; it was intended to transplant this later. On the third day after the operation nystagmus was noticed, followed later by loss of knee jerks, alteration in speech, and difficulty in swallowing. These symptoms gradually increased, respiration became wholly thoracic, and the child died on the tenth day after the operation. The post-mortem showed that the lower lobes of both lungs were collapsed, but there was no other evidence of disease. All parts of the brain and its membranes were perfectly healthy, and the wound itself looked healthy.

One of the plastic operations for **hare-lip** consisted in the removal of a wedge-shaped portion from the lower lip of a girl, aged 16, to remedy the unsightly prominence produced by an old hare-lip operation. The other operations were all for the closure of the cleft in the upper lip.

A plastic operation was performed with much success upon a case of extreme **hypospadias**; the patient, aged 27, had been brought up as a girl until the age of 17.

A woman, aged 62, was admitted for **incontinence of fœces**, following an excision of the rectum and coccyx that had been performed abroad several months before for fibrous stricture; some improvement followed the plastic operation.

A plastic operation was performed upon a congenital **recto-vaginal fistula**; the patient, aged $3\frac{1}{2}$ years, had been operated upon for imperforate anus soon after birth. The second operation failed to improve the condition.

Rhinoplasty was performed three times.

A boy, aged 16, with partial destruction of the nose from congenital syphilis was improved by a small plastic operation to enlarge the contracted nostrils.

A woman, aged 25, was admitted with depression of the bridge of the nose due to old disease of the nasal septum; an attempt was made to form a new bridge by turning up portions of bone from the upper jaw on each side. The improvement was not great.

A somewhat similar case was that of a woman, aged 38, who was admitted for a flattened nose: a plastic operation had been performed already with much success for the restoration of the nose after syphilitic destruction three years ago; the primary disease had occurred five years ago. There was also much destruction of the palate. In this case no attempt was made to interfere with the bones, but small flaps were turned in from the cheeks to raise the depressed soft parts of the nose. The operation was followed by some cellulitis of the face, which, however, soon subsided, and the patient's appearance was, on the whole, improved by the operation.

A boy, aged 17, was admitted with **stenosis of the naso-pharynx**, following diphtheria at the age of 5. The soft palate was united to the back of the pharynx, leaving an opening only just large enough to admit a probe; the opening was enlarged and a wedge-shaped piece of soft palate was removed. The opening was kept patent by drainage tubes, but considerable contraction, nevertheless, occurred subsequently.

A woman, aged 42, with a **urethro-vaginal fistula**, caused by a confinement nine months ago, was improved by two plastic operations; nine years ago she was in the hospital for a vesico-vaginal fistula due to the same cause; on that occasion the fistula was closed by operation, and remained closed.

A woman, aged 32, with a very large **vesico-vaginal fistula**, was in the hospital nearly six months, underwent four plastic operations, and was eventually discharged greatly improved with only a small fistulous opening still remaining.

EXCISION OF BONES AND JOINTS.

A child, aged 3, was admitted with severe double talipes varus. Fitzgerald's operation had already been performed on the right foot without much benefit. The right **astragalus** was excised, and a month later a wedge of astragalus and os calcis removed from the left foot; the position of the latter not having been sufficiently improved, the remainder of the astragalus, together with the external malleolus, were subsequently excised. The wounds ran the usual aseptic course, and the child was discharged four months after admission with both feet in good position.

A hawker, aged 67, had had for three weeks a painful swelling of the left **clavicle**; two days before admission he felt a sudden crack in the bone, and this was followed by increased pain and swelling and inability to move the arm properly. On admission, there was a prominent swelling two inches in diameter springing from the inner half of the bone, which was fractured at the seat of the tumour. The inner two-thirds of the clavicle were excised, together with some glands. The patient made a good recovery from the operation and left the hospital twenty-six days later, five weeks after admission; but there was already recurrence in loco and in the axilla.

The **coccyx** was removed from a woman, aged 37, on account of ankylosis in a bad position.

Excision of the **elbow** was performed twice during the year, once for tuberculous disease and once for ankylosis; both patients were girls, aged 17. Excision of the **hip** for tuberculous disease was performed eleven times; all the patients recovered; in three cases of male children, aged 2, 10 and 13, an abscess was present at the time of operation, but the skin was unbroken; in one of these there was dislocation of the joint, and during the operation the sciatic nerve was partially divided, but immediately re-united. In the remaining eight cases, sinuses were present at the time of operation; in most of them the disease had existed for a long time, in one amyloid disease was present, in another there was necrosis of the whole head of the bone; nearly all the patients were greatly improved by the operation, and most of them left the hospital with sinuses almost healed. The ages were 5, 6, 9, 9, 11, 12, 16 and 18.

Excision of the **knee** was performed six times. For tuberculous disease four times, upon male patients, aged 13, 13 and 32, and upon a girl, aged 8; in one case only were sinuses present at the time of alteration; in three of the cases ivory pegs, in one steel pins, were used to unite the bones; all the patients made excellent recoveries. For ankylosis of the knee in a bad position, excision was performed once, upon a girl, aged 11, with an excellent result.

The sixth case was that of a married woman, aged 25, who was admitted with what was thought to be tuberculous disease of the knee. Four years ago the knee became painful and swollen; it gradually got worse, and for several months the patient had been unable to walk. On admission, she was a delicate, anæmic woman; the right was greatly swollen, the swelling having almost exactly the shape of the synovial membrane, and being very prominent, soft and well defined. No fluid could be detected in the joint; there was no swelling in the popliteal space, and there were no signs of suppuration. After a consultation, excision of the knee was performed; almost the whole of the synovial membrane was found to be the seat of a soft sarcomatous growth, which had eroded the patella and femur in a characteristic manner. The disease was completely removed by the operation, but the patient died of shock a few hours later. Microscopic examination showed the tumour to be an oval-celled sarcoma. The post-mortem showed that there were no secondary deposits.

Excision of part of a **metacarpal** bone was performed once for caries, and once for old dislocation.

Excision of the head of a **metatarsal** bone was performed five times for hallux valgus, twice for tuberculous caries, and once for deformity.

Excision of one or more **ribs** was performed twenty-six times. Eighteen times a portion of one rib only was removed for empyema; the ages of the patients varied from 8 months to 38 years; sixteen patients recovered; two infants, aged 8 and 13 months, died; of these the former had also meningitis.

Two or more ribs were removed four times for empyema or its sequelæ; all these patients recovered; one of them was a porter, aged 22, who had had a cough for two years, and signs of phthisis for at least one year; he was admitted into a medical ward with empyema, and was aspirated several times; three months after admission a portion of the seventh rib was excised; suppuration continuing, portions of all the ribs from the third to the ninth inclusive, were removed; this wound healed well, and the chest wall fell in considerably, but the discharge from the pleural cavity had not ceased when the patient was discharged, on the sixty-seventh day after the last operation.

For bronchiectasis, portions of the fifth and sixth ribs were removed from a man, aged 46, to allow the chest wall to fall in, and the cavities to shrink

The pleura was not opened, but the lung was punctured with a trocar; nothing was found. The operation was considered successful, as the man improved very greatly, his condition up to that time having been stationary or getting worse.

A man, aged 48, died after a similar operation for bronchiectasis and cirrhosis of the lung.

In the case of a girl, aged 11, with cancer of the lung, a portion of one rib was removed, and the patient recovered sufficiently to leave the hospital.

A man, aged 19, with actinomycosis of the lung, died after excision of a rib (Nearly all these cases of resection of ribs were in the Medical Wards.)

A small part of the **scapula** was removed with a recurrent sarcomatous tumour springing from the fascia over that bone; the growth had already been removed three times previously in the course of four years; the patient made a good recovery.

Excision of the **upper jaw** was performed four times for carcinoma.

A farm labourer, aged 59, was admitted for a tumour of the inner part of the orbit, which had been visible for four months; for three years the nostril had been obstructed. The cheek was reflected, the nose freely opened, and the growth removed with part of the upper jaw. The growth, which proved to be a cylindrical-celled carcinoma, was found to be very extensive, involving the frontal sinus, antrum and nose. The patient made a good recovery, and left the hospital on the thirty-sixth day after the operation. Six months later (in the following year) he was re-admitted, and the fistulous opening into the nose was closed by a plastic operation.

A woman aged 60, was admitted with an extensive epithelioma of the hard palate of four months' duration. A large portion of both upper jaws was removed. Convalescence was delayed by an attack of facial erysipelas, but the patient made a good recovery, and left the hospital, quite well, on the eightieth day after the operation.

An oilman, aged 67, with an extensive epithelioma of the palate and upper jaw of six months' duration, made an excellent recovery, after free removal of the whole upper jaw and malar bone, leaving the hospital three weeks later.

A packing-case maker, aged 42, was admitted with a foul perforating epithelioma of the hard palate, extending into the antrum; it had existed twelve months; there was some enlargement of glands near the angle of the jaw. Excision of the upper jaw was performed, the orbital plate and glands being left. The patient made an excellent recovery, leaving the hospital on the thirty-second day. When seen again eight months later he was quite well; no recurrence had taken place, and the swelling of the glands had subsided.

The nature of the tumour in all these four cases was confirmed by microscopical examination.

Excision of the wrist was performed once for tuberculous disease, upon a boy, aged 16.

OPERATIONS ON BONES.

In the six cases of **osteotomy** for ankylosis of the hip, the infra-trochanteric operation was performed five times with excellent results.

In the sixth case, the operation was done through the neck of the femur; the patient was a boy, aged 3, who had had symptoms of hip disease for one year. When admitted, there was a large abscess, and the hip was flexed beyond a right angle. The abscess was opened, and much caries of the bone

was found. Four months later, the temperature being normal, the wound healed, and the position of the limb as before, the neck of the femur was divided with an Adam's saw. This was followed by some cystitis and further suppuration about the hip. The patient left the hospital five months after the operation, with a small sinus not quite healed.

All the cases of osteotomy for genu valgum did well ; in one case of a girl, aged 18, osteoclasia had previously been attempted, but the bone could not be broken.

In two cases of great deformity, Ogston's intercondyloid operation was performed, in all the others the section of the bone was made above the condyles.

Among the cases of **sequestrotomy** the following was the most remarkable. A barman, aged 18, was admitted for gonorrhœal phimosis of seven weeks' duration. He was circumcised ; a week later he complained of pain just above the knee, this increased, and the thigh became swollen and very tender. The temperature remained normal, and there was no evidence of suppuration. Two months after admission an exploratory incision was made, which revealed *quiet necrosis of the whole shaft* of the femur without suppuration. The whole shaft was removed, and the patient made an excellent recovery, leaving the hospital six weeks after the operation, the wounds being soundly healed, and the patient able to walk with crutches.

Trephining the mastoid for otitis media was performed nine times. Five patients made good recoveries ; in two other cases the skull was subsequently trephined for intra-cranial complications (these cases are described later under that heading) ; another patient was a man, aged 18, who had had for eight years discharge from both ears, for five days before admission he had had pain in the head, rigors, and a high temperature ; he was admitted with tenderness over the mastoid and in the neck. The mastoid was trephined, but the patient died of pyæmia on the seventh day after the operation and the twelfth from the onset of acute symptoms ; at the post-mortem, suppurative thrombosis of the lateral sinus and of the whole length of the internal jugular vein was found ; there were also abscesses in the lungs.

The ninth case was that of a boy, aged 11 ; ten days before admission he first complained of pain and swelling behind the ear ; three days later an incision was made, and much foul pus let out. When admitted, he was complaining chiefly of occipital pain. He was conscious, but appeared to be in a dazed condition. His temperature was 97.8° , and his pulse slow. There was no optic neuritis or paralysis of any kind. The mastoid was immediately trephined, the cranial cavity being opened ; an extra-dural abscess was discovered and freely opened. The patient was not much relieved ; he became very restless, vomited frequently, and died on the fifth day. At the post-mortem, a large **cerebellar abscess** was found immediately under the area of operation.

Trephining the skull for chronic cephalalgia was performed twice, with good result. One case was that of an acrobat, aged 31, who had suffered from constant headache after a blow on the head two years before ; there was no optic neuritis ; he was trephined over the vertex, and the headache was relieved.

The other case was that of a woman, also aged 31, who had had exactly similar symptoms for four years, following a severe blow on the head. There were no signs of cerebral tumour ; there was a tender spot on the occiput. She was trephined over this spot, and made an excellent recovery, being completely relieved of her headache.

For **cerebral abscess** the skull was trephined five times.

A girl, aged 17, with history of five months' discharge from the ear, one month headache, and vomiting. On admission much offensive discharge from

the ear, patient very ill, with temperature varying from 106.2 to 97°. The mastoid was trephined, some foul pus let out, and the patient improved. Subsequently she became worse, the lateral sinus was opened, and foul pus again let out; an abscess in the temporo-sphenoidal lobe was also opened. The patient gradually sank, and died three days after the last operation. The post-mortem showed, besides the cerebral abscess and suppurative thrombosis of lateral sinus, pyæmic abscesses of the lungs and spleen, and meningitis.

A governess, aged 24, was admitted with the following history: Seventeen years ago, scarlet fever; four months ago, polypus removed from right ear; discharge from ear ever since; twelve days pain in the ear, gradually increasing; three days shivering and vomiting; since that time she had been getting drowsy and has shivered and vomited several times. On admission, drowsy with pain and swelling over mastoid; knee jerks absent. Temperature 102°, pulse 114. She was trephined over the mastoid, but no pus was found. Five days later she had slight paralysis of left facial and hypoglossal nerves, and anæsthesia of left cheek; left pupil small and re-acting sluggishly, some paresis of left arm. Temperature between 101° and 99°, pulse 64. Drowsiness increasing. She was then trephined over the left temporo-sphenoidal lobe, and an ounce of pus let out. Just before the operation there was a rigor with temperature 104°. She improved considerably, but then gradually relapsed, and ten days later she was trephined again, higher up, and more pus let out. In the next month she gradually became weaker, and died on the seventieth day after admission. At the post-mortem, large portions of the temporo-sphenoidal and parietal were found to be in a sloughing condition, and there was some localised meningitis.

A plumber, aged 18, had had foetid discharge from both ears for seven years; for three days there had been vomiting, shivering and pain in the head, and his speech became slow. On day of admission, several general convulsions. On admission, intense frontal headache; a tender spot behind and above right ear; knee jerks increased; no paralysis. Pupils equal, no optic neuritis, pulse 80 to 112, temperature 99.6° on admission, 104° a few hours later, then gradually rose till death. On the third day after admission optic neuritis appeared. Next day mastoid was trephined, no pus found. Skull then trephined over temporo-sphenoidal lobe and large extra-dural abscess opened. The subjacent dura mater was in a sloughy condition. Retraction of the head and other signs of meningitis set in, and the patient died two days later. At the post-mortem, diffuse suppurative meningitis was found, but no cerebral abscess.

An engineer, aged 21, was admitted with a history of chronic otorrhœa of five years' duration. He was otherwise in good health until eight days ago, when he had severe pain at back of head and neck. He shivered and vomited. On admission, drowsy, head much retracted, no vomiting, no optic neuritis; pupils normal, slight weakness of left external rectus; left facial paralysis; staggering gait. Three days after admission, his condition being worse, he was trephined on right side, first over cerebellum, then over temporo-sphenoidal lobe, but no pus was found. Two days later, patient being much worse, he was trephined again on left side, but nothing found. Death occurred next day. At the post-mortem, no abscess was found, but there was much extravasated blood beneath the base of the brain and in the right frontal lobe; this blood was derived from an aneurism of the anterior communicating artery, which had ruptured.

A boy, aged 17, who had had chronic otorrhœa from both ears for several years, received a blow on the head nine days before admission. It stunned him slightly, but caused no wound. Two days later there was some swelling of face and head, and later he became delirious. On admission, conscious but drowsy and irritable. Temperature 102°, pulse slow. Left temporal region swollen and exquisitely tender. Incision down to the bone let out

some pus. Three days later more pus let out, and bare bone felt. Eight days after admission he had slight paralysis of left arm; three days later the drowsiness increased, and his temperature having been falling since admission, he was trephined over the left frontal bone; more than an ounce of pus was let out from beneath the dura mater. The diploc was infiltrated with pus. After the operation, temperature gradually rose again, and he died on eighteenth day after admission. Post-mortem, general suppurative meningitis, much suppuration in the frontal lobe, and extensive necrosis of frontal bone; the suppuration was not connected directly with either ear.

For **gunshot wound**, trephining was performed once; the patient was a merchant, aged 62, who shot himself with a revolver, first in the temple, then in the palate; when admitted, shortly after the accident, he was sensible, and could speak fairly well; there was a good deal of twitching of both sides of the face, but no paralysis. The aperture in the skull was at once enlarged with bone forceps, the bullet felt with probe and finger, and extracted. On the following day there was paralysis of the opposite arm and side of face, and some anaesthesia. The temperature was for three days between 99° and 102°, then lower. Patient became restless and delirious, died on the seventh day. At the post-mortem, the bullet was found to have passed through the right corpus striatum into the lateral ventricle, from which it had been extracted.

For **recent injury**, five patients were trephined; a boy, aged 15, with a compound depressed fracture, made an excellent recovery.

A man, aged 52, who had been struck by a falling plank, was trephined for a compound depressed fracture of the frontal; he died of meningitis on the ninth day; the post-mortem showed extensive fracture of the base, involving the ethmoidal cells and much laceration of brain.

A man, aged 75, with fracture of the base, was trephined for intra-cranial hæmorrhage, but died in a few hours; post-mortem, extensive hæmorrhage into arachnoid space.

A man, aged 28, fell eighteen feet on to his head; he was admitted drowsy, but able to walk. Some paralysis of left arm and leg followed, and he became very irritable. There was a hæmatoma on the left side of the head. On the fifth day he was much worse, and was trephined in the region of the hæmatoma. A very extensive fracture of the vault and base was found, and the patient died a few hours later. At the post-mortem, it was found that the right side of the brain had been much lacerated.

A boy, aged 17, was found insensible at the foot of some stone steps; the insensibility deepened, and a blood extravasation on the head became larger. He was trephined, and an extensive fissured and depressed fracture was found; much blood was let out from within the dura. The brain was much lacerated; death occurred one hour after the operation. The post-mortem showed also much laceration of the base of the brain.

For **suppurative thrombosis** of lateral sinus, trephining was performed upon a man, aged 29. The history was, for many years otorrhœa from both ears, for fourteen days he had been dull and ill, ten days pain in the head; seven days ago there was a fresh discharge of pus from the left ear; for six days he had had two rigors daily, for five days frequent vomiting. On admission, left frontal and temporal headache, much tenderness over internal jugular as far down as the sternum. No paralysis of any kind. Pulse 96, temperature 100°. Next day, five rigors since admission, temperature 105.4°. He was then trephined over lateral sinus, which was found to contain foul pus; jugular vein also tied. Temperature fell after operation, but rose again to 101°; signs of pneumonia set in, and patient died on third day after operation, and fourth after admission. No post-mortem.

For **Jacksonian epilepsy** trephining was performed three times; two patients recovered and one died. A boy, aged 14, nineteen months ago had received a blow on the head rendering him unconscious for a few minutes; eighteen months ago he suddenly lost power of speech for a few minutes and had spasm of right hand, arm and leg; for a year there were no further symptoms, but in the last six months there had been numerous fits, affecting chiefly the right arm and seldom causing loss of consciousness. There was no headache, no vomiting, no optic neuritis. He was trephined, and a tuberculous mass one inch in diameter and a quarter of an inch thick was removed from the surface of the left cerebral hemisphere; it contained a little thick cheesy pus. For seven days the temperature remained normal, and patient did well, then symptoms of meningitis set in, followed by hernia cerebri, and death on the thirty-ninth day after operation. No post-mortem.

Another boy, aged 14, who had fallen on to his head eleven years ago, and had epileptic fits for four years, increasing in number and severity. There appeared to have been an old depressed fracture. He made an excellent recovery and had only a few slight fits after the operation; he left the hospital on the thirty-seventh day afterwards.

Another boy, also aged 14, had injured his head ten years ago; at that time there had also been some necrosis of the skull. For five years he had had slight epileptic attacks without loss of consciousness; a few days after admission he began to have a series of severe fits involving leg and arm, and for the first time attended by loss of consciousness, he had fifty fits in four hours. Patient, after the operation, never had a bad symptom, and when seen four months later he had had no recurrence of fits.

Linear craniectomy for idiocy was performed three times.

Two boys, both aged 4, left the hospital on the fourteenth and eighteenth days after operation; in each case a piece of bone measuring six inches by three quarters of an inch was removed; there was no improvement in the mental condition; in neither case was there any undue tension of the membranes. Upon a third male microcephalic idiot, aged 12, the operation was done upon both sides of the head with an interval of five weeks between the operations. Both wounds healed soundly by first intention, but after the second the child gradually became weaker and weaker and eventually died with thrombosis of the inferior vena cava on the fifty-second day after the last operation. Post-mortem, it was found that the openings in the skull had been filled up by dense fibrous tissue. The brain showed considerable congenital atrophy, especially of the middle and posterior lobes. It was considerably smaller than the cranial cavity.

Three male patients, aged 4, 30, and 40, made excellent recoveries after elevation of compound depressed fractures of the skull.

Wiring of an ununited fracture of the humerus was performed upon a butcher, aged 32, three months after the injury; the case did very well, but the patient was re-admitted three months later with a fresh fracture of the same bone in the same place.

A man, aged 36, with an ununited fracture of the radius of ten months' duration, made a good recovery after a similar operation.

The **patella** was wired four times for recent fracture; the patients were men, aged 22, 23, 34, and 39, and in each case the fracture was simple and transverse; the operation was performed on the second, fifteenth, ninth, and third day respectively after the accident. In the second case the operation was a subcutaneous coronal wiring; the other three were open operations, silver wire being passed through holes drilled in the fragments. In all cases the wounds healed by first intention, except the third, in which a small sinus remained; in this case the wire had to be removed on the thirty-seventh day after the operation. The patients left the hospital sixteen days (at his own request), thirty-one days, fifty-three days, and forty-four days respectively after the operation.

Bone grafting was performed upon a miner, aged 52, who was twice in the hospital for ununited fracture of the ulna. Two and a half years ago he had broken both bones of the forearm; a year before admission both bones had been wired, the radius successfully, the ulna not. The old wire was removed from the ulna, the ends refreshed, and two inches of fibula from an amputated limb was fitted into the gap; the wound healed well, and the ulna was apparently soundly united when the patient left the hospital seven weeks after the operation. Eight months later he was re-admitted; it was then found that the inserted piece of bone had united with the upper, but not with the lower fragment. The half united piece of bone was then removed and re-placed by two and a half inches of femur from the freshly amputated hind leg of a young lurcher dog. The wound healed by first intention, and the patient left the hospital on the forty-eighth day after the operation; the union appeared then to be fairly firm.

OPERATIONS ON JOINTS.

A married woman, aged 30, was admitted with tuberculous disease of the knee of nine months' duration; a sinus led directly into the joint, and there was extensive caries of the patella; the temperature was normal. The patella was excised and the synovial membrane freely removed. The suppuration, however, continued, and after various minor operations, amputation was performed at the lower third of the thigh. The patient made a good recovery and left the hospital on the thirty-eighth day after the amputation, and one hundred and forty-second after admission.

AMPUTATIONS FOR INJURY.

No death occurred after any primary amputation for injury.

After secondary amputation, two patients died. A man, aged 24, who had been crushed by a lift, was admitted with a compound fracture of the femur with much laceration of soft parts. On the third day, temperature began to rise, on the twelfth profuse secondary hæmorrhage occurred; the wound was opened up, but the exact source of hæmorrhage could not be found; it was packed lightly with gauze, and no further hæmorrhage ensued. Gangrene following, the limb was amputated through the middle of the thigh on the fifteenth day, but the patient, who was very feeble at the time, died three hours later. Examination of the limb showed ulceration of the popliteal artery by the lower fragment. No post-mortem.

The other fatal case was that of a flower seller, aged 30, who had been run over by a van, and was admitted with an extensive laceration of the leg; an attempt was made to save the limb by thorough antiseptic cleansing, but two days later the flaps were found to be sloughing, and cellulitis set in; numerous incisions were made. On the sixth day, the patient being very ill at the time, amputation was performed above the knee. The patient gradually sank and died on the day after the operation. No post-mortem.

A boy, aged 16, who had been caught in machinery, was admitted with a compound fracture of left tibia, simple fractures of humerus and right fibula, and separation of lower epiphysis of femur and upper epiphysis of right tibia. He developed gangrene of the leg, and amputation at the knee was performed on the thirty-sixth day. Some suppuration occurred in the thigh, followed by secondary hæmorrhage, but the patient eventually made an excellent recovery.

AMPUTATIONS FOR DISEASE.

Out of fifty-two amputations for disease, two deaths occurred.

A woman, aged 27, was admitted for a sarcoma of the upper part of the thigh. Five years ago she had first noticed pain in the calf, and the muscles of that region wasted; for three years she had had anæsthesia of the toes, gradually

increasing upwards ; in the last few months there had been increased difficulty in sitting and in getting about ; for five months she had noticed a swelling which had steadily increased in size. On admission, the patient was pale and looked ill ; at the back of the thigh, near the glutei, was a tumour as large as a man's fist, apparently in the muscles. A small piece removed for microscopic examination showed the tumour to be spindle-celled sarcoma. Amputation by lateral flaps was performed just below the trochanters, the common femoral artery and vein being tied at the beginning of the operation. Death occurred from shock a few hours afterwards ; there was no post-mortem. The growth involved the sciatic nerve and hamstring muscles nearly as high as the tuberosity of the ischium.

A man, aged 67, died on the twenty-third day after amputation through the lower third of the thigh for a very foul epitheliomatous ulcer of the leg ; the leg had been the seat of a chronic ulcer for thirty years. There was no post-mortem.

Fifty cases recovered after amputation for disease.

A man, aged 43, recovered after amputation at the shoulder for a large periosteal sarcoma of the upper end of the humerus ; pain had been present for ten months, swelling for three.

A clerk, aged 49, was admitted with extensive tuberculous suppuration about the axilla, and several sinuses ; the latter were opened up and scraped several times, but the suppuration continued, the shoulder joint became involved, and the patient's general condition became so bad that amputation at the shoulder was performed eight months after admission. The patient made a good recovery, leaving the hospital thirty-nine days after the operation with the wound almost healed.

Two women, aged 29 and 35, with large myeloid sarcomata of the femur of two years' and three years' duration respectively, made excellent recoveries after amputation through the upper third of the thigh.

A man, aged 18, made a good recovery after amputation through the middle of the thigh for periosteal sarcoma ; he had first noticed pain four weeks, and swelling three weeks before admission. On admission, there was a small, soft elastic swelling just above the inner side of the knee ; it was thought to be an abscess, but when incised only clear fluid escaped. Further incision showed the growth to be a sarcoma, and amputation was performed through the middle of the thigh, just six weeks after the first onset of symptoms. The tumour was a periosteal sarcoma as large as a goose's egg. The patient left the hospital on the thirty-ninth day after the operation.

A boy, aged 15, made a good recovery after amputation of a useless leg, which had been the seat of elephantiasis for many years.

A man, aged 58, made a good recovery after amputation through the thigh for an epithelioma engrafted on a chronic ulcer of forty years' standing.

A man, aged 58, admitted for gangrene of the foot and glycosuria, recovered after amputation through the lower third of the thigh. The glycosuria was supposed to be due to diabetes mellitus ; it disappeared before the patient left the hospital.

A man, aged 65, two months before admission, scratched his little toe ; three weeks ago it became black and dry ; he was admitted with dry gangrene of the toe and part of the foot ; there was no albuminuria or glycosuria ; the gangrene slowly spread, and the temperature began to rise until the ninth day, when amputation was performed through the lower third of the thigh ; the popliteal artery was found to be thrombosed ; the temperature fell at once, and although some sloughing of the posterior flap took place, the patient made an excellent recovery, and went to Swanley sixty-five days after the operation.

Amputation through the knee was performed upon a man, aged 24, for a pulsating sarcoma as large as a man's fist, situated in the muscles of the leg behind the inner ankle; it had been noticed seven months. The patient made an excellent recovery.

A man, aged 47, was admitted with an ulcer of the leg of twelve years' duration; it had lately increased in size and became very prominent; microscopic examination showed it to be a sarcoma, and amputation was performed just below the knee, the patient making a good recovery.

A girl, aged 16, was admitted for a perforating ulcer of the foot; Syme's amputation was performed, and the ulcer recurred in the stump; eventually, however, it healed.

Of ten amputations of the toes, one was performed for congenital webbing, the others for hammer toe.

OPERATIONS ON THE BREAST.

Of the twenty-three cases of **adeno fibroma**, one occurred in a man, aged 35; it had been growing for four months. A woman, aged 47, was admitted with a very hard tumour nearly two inches in diameter, which had been first noticed eleven months before; it was slightly adherent to the skin, but not to the muscle; the nipple was not retracted; the axillary glands could not be felt. The tumour was thought to be scirrhus, and the breast was removed, together with the axillary glands. The tumour was encapsuled, of dense, almost stony hardness; microscopically it was found to be an almost pure fibroma with mere traces of gland tissue. The patient made a good recovery.

The six cases of **simple cyst** of the breast removed locally, occurred in women, aged 39, 46, 46, 46, 48 and 53. In three cases the breast was amputated for **cystic disease**; a woman, aged 46, with a very large cyst; another, aged 63, with a large cyst completely filled with rough papillary growths, some of which had undergone gelatinous degeneration; it had been growing eight months; the third was a case of multiple involution cyst in a woman, aged 39. The glands were not removed in any of these cases.

Thirty-seven women underwent amputation of the whole breast for carcinoma, in thirty-four cases the axillary glands being also removed; in several cases very extensive removal of the skin was performed, the wound being closed by the help of a plastic operation, performed at the same time.

Two patients died. A woman, aged 37, with extensive disease of ten months' duration; there was much ulceration of skin; the operation was undertaken chiefly for the relief of pain; the wound itself did well, and the patient was relieved for a time, but she died two months later with recurrence in the breast and with numerous secondary nodules in the liver.

A woman, aged 48, was admitted for a small lump in the breast which had been noticed for two months; a small portion was removed for microscopic examination and found to be carcinoma; a few days later an extensive operation was performed, the whole of the breast and axillary glands and part of the pectoralis major being removed. The patient was much collapsed, never recovered consciousness, and died next day. At the post-mortem, nothing was found to account for death.

Five women underwent partial amputation of the breast for carcinoma; there were no deaths.

Sixteen women underwent operations for the removal of carcinoma that had recurred in the breast or glands, or both; one died; she was a woman, aged 59, from whose breast a scirrhus tumour had been removed locally two and a half years before; she was admitted with a recurrent tumour as large as an egg.

The whole breast and the affected axillary glands were freely removed, and the wound drained. On the second day after the operation, the temperature began to rise, and there was slight erythema about the axilla. The patient gradually became comatose, vomited frequently, and died on the eighth day after the operation. At the post-mortem, most of the wound was found to have healed by first intention; there was slight chronic interstitial nephritis, but it seemed probable that septic infection had been the chief cause of death.

REMOVAL OF TUMOURS.

A woman, aged 57, had had for four months a slightly painful swelling in the region of the left masseter. On admission she was in good health; involving the whole of the ascending ramus of the lower jaw was a smooth, oval, firm swelling of the size and shape of half an egg; it was situated beneath the masseter; it did not expand the bone on its inner surface. When cut into it was found to contain clear fluid; the exact nature of the cyst was never clearly ascertained. There was no evidence of its being either hydatid or dentiferous. A portion of the bony cyst wall was removed with beneficial result.

There were three cases of large fibro-angiomaticous tumours of the nasopharynx, in male patients, aged 15, 18 and 21; the symptoms in all cases were blocking of one nostril and epistaxis; the duration of symptoms was three months, two years and eight years respectively. In the first case the upper jaw was removed and the tumour then cut away with scissors; in the other cases the soft palate was split, part of the hard palate chiselled away, and the growth then removed, in the one case with an *écraseur*, in the other with scissors and elevator. In all the cases there was much hemorrhage at the time of operation, ceasing as soon as the tumour was extracted. All three made excellent recoveries, leaving the hospital on the forty-third, sixth and thirty-seventh days after the operation. Microscopically, in each case the tumour consisted of dense fibrous tissue with numerous large blood-vessels.

A large, thick *nævus*, about two and a half inches in diameter, was removed, together with a large part of the breast, in which it was situated, from a girl, aged 16 months; there was very severe hæmorrhage, but the patient quickly recovered.

An *exostosis* was removed from the iliac fossa of a man, aged 23; it had caused much pain and inconvenience. The operation was followed by some sloughing of the skin; on the forty-ninth day after the operation, he was being put under chloroform for skin grafting, when he died under the anæsthetic.

Bronchocele operations were performed three times. A cystic adenoma and a cyst were removed by intra-glandular enucleation from a man, aged 40, and a woman, aged 46; primary union in both cases. A woman, aged 46, who had had a bilateral parenchymatous goitre for many years, and had had much dyspnoea, underwent extirpation of the right lobe; she made an excellent recovery and was greatly relieved.

An *epithelioma* was removed locally through the urethra from a woman, aged 53; she had had pain for three months and hæmaturia for two months. An ulcer, an inch in diameter, was seen on the anterior wall of the bladder, near the urethra. It was removed chiefly by scraping with a Volkmann's spoon; there was not much hæmorrhage. The patient left the hospital on the thirty-second day after the operation, much relieved.

An *epithelioma* was removed from the ear of a man, aged 64; it had originated around a gouty tophus.

In one case of *epithelioma* of the floor of the mouth, in a man, aged 70, part the alveolus of the jaw was also removed; the patient made a good recovery.

A woman, aged 67, with an epithelioma of the scalp, was four times in the hospital during the year, and underwent as many operations for its removal.

There were two cases of removal of sarcoma of the chest wall; one was a melanotic sarcoma, the other had originated in an old *nævus*.

A man, aged 48, with a large, sloughing **fibro-sarcoma of the scapula**, underwent local removal of the tumour, developed cellulitis of the arm, then pneumonia, and died on the forty-eighth day after the operation.

A man, aged 21, from whom a **sarcoma of the synovial membrane of the knee** had been removed in the hospital in 1892, was in again twice during the year and small local recurrent tumours were removed.

A woman, aged 56, who had had tracheotomy done two months previously for a **sarcoma of the thyroid gland**, was re-admitted with dyspnoea; after the tracheotomy the tumour had diminished so much in size that the wound had been allowed to close up. There was a large, irregular, hard tumour involving the right lobe of the gland rather more than the left. An attempt was made to enucleate the tumour, but had to be abandoned on account of hæmorrhage. A portion of the growth was therefore cut away, with considerable relief to the breathing; the wound healed, and the patient left the hospital on the twenty-second day after the operation, much relieved.

A myeloid **sarcoma of the shaft of the tibia** was removed locally from a woman, aged 20. It had been noticed seven months; it was situated five inches from the upper end of the bone.

OPERATIONS ON THE TONGUE.

The classification of operations on the tongue has this year been somewhat altered and extended, especially with reference to the exact nature of the operation and to the removal of lymphatic glands.

Twenty-one operations were performed for the removal of **epithelioma** (inclusive of two cases of mere local removal); in twelve of these cases the glands were removed at the same time. There was one death; a pale, thin man, aged 53, who was admitted with an ulcer of six months' duration, situated on the posterior half of the tongue, extending as far back as the anterior pillar of the fauces. One lateral half of the tongue was removed through a sub-maxillary incision, the glands being removed at the same time; one of these had to be dissected off the jugular vein, to which it was adherent. The patient did well for ten days, then developed facial erysipelas, the temperature running up to 103°; after a week it had fallen again to normal. A second attack of erysipelas subsequently occurred, and the patient was apparently recovering from this, when he died rather suddenly on the forty-fifth day after the operation. At the post-mortem, the pericardium and left pleura both contained turbid fluid and lymph. The wound in the mouth had healed, and no growth could be found in the neck or elsewhere. The whole tongue was removed three times from men, aged 41, 55 and 61 respectively; all made good recoveries. The operation most often performed (seven times) was that of removal of one lateral half of the tongue together with the glands; the ages of these patients were 43, 43, 45, 48, 62, 66, 73; the last but one of these cases had been in the hospital seven years previously, when an epithelioma had been removed locally from his tongue. All seven made good recoveries, although an attack of pleurisy occurred in one case. The two patients from whom one lateral half of the tongue was removed without removal of the glands were aged 61 and 71; the former had a small ulcer of six months' duration, and the glands were believed not to be affected; the latter was a man, aged 71, with emphysema of the lungs and a doubtful enlargement of one gland.

In two cases the cheek was split ; in one case the patient was 61, and had extensive disease of twelve months' duration, situated far back at the side of the tongue ; in the other, the patient was aged 76 ; the disease had begun two years previously, seventeen months ago it had been removed, and five months ago recurrence was first noticed ; there was extensive disease of the side of the tongue fixing it to the jaw. In both these cases the glands were also affected, but it was not thought advisable to remove them. Excision of the anterior half of the tongue without removal of glands was performed upon three men, aged 40, 59 and 69 ; the disease had existed three, four and six months respectively, and involved the tip of the tongue. Part of the lower jaw was removed in one case, that of a man, aged 66, with an epithelioma of two and a half months' duration, involving the under surface of the tip and floor of the mouth ; it was adherent to the jaw, the alveolar part of which was removed. All these patients made excellent recoveries.

A woman, aged 33, was admitted for a growth as large as a hazel nut at the back of the tongue, just in front of the epiglottis. It was removed locally ; microscopical examination showed it to be a simple adenoma with no trace of thyroid tissue. A similar growth had been removed from the same situation eight years before.

A man, aged 44, was admitted for an ulcer in the middle of the dorsum of the tongue ; it had begun eleven months previously as a lump, which burst two months later ; for years this part of the tongue had been irritated by an artificial tooth plate ; there was phthisis at the apex of one lung ; microscopic examination of an excised bit having confirmed the diagnosis of tubercle, the ulcer was removed locally.

OPERATIONS ON BURSÆ, FASCIÆ AND TENDONS.

There was no death, nor any serious complication after any of the fifty-five operations of this class.

OPERATIONS ON THE LARYNX AND TRACHEA.

Tracheotomy for diphtheria was performed forty-nine times, with twenty-five recoveries ; of children under the age of 2 years two recovered (aged 12 and 19 months), and nine died (aged 5, 9, 11, 15, 16, 17, 18, 20, and 22 months).

A man, aged 61, with epithelioma of the œsophagus, who had had dysphagia for eight months, ptosis three months, and dyspnœa four days, underwent tracheotomy on the day after admission ; he gradually sank, and died three days later. He had a large mass of growth in the neck, and secondary growths in the lungs and elsewhere.

A woman, aged 56, was admitted with dyspnœa, due to sarcoma of the thyroid gland. The swelling had been first noticed ten weeks before. Both lobes of the gland were considerably enlarged, very firmly fixed to the larynx, but not to surrounding parts ; the right vocal cord was paralysed. A severe attack of dyspnœa necessitated tracheotomy on the seventh day after admission—a portion of the isthmus being at the same time removed. Microscopical examination showed round-celled sarcoma. Some temporary diminution in the size of the tumour followed the operation, and the patient left the hospital a month later, much relieved. She was subsequently re-admitted, and part of the tumour was removed. (See Removal of Tumours.)

A child, aged 16 months, was admitted with a history of having had diphtheria three months previously ; tracheotomy had then been performed, but the child had never afterwards been able to dispense with the tube, and for this reason he was admitted. Under chloroform, a finger was introduced into the larynx from below, and a foreign body felt. It was pushed upwards with

a probe and extracted through the mouth. It proved to be a dress-hook measuring $\frac{1}{2}$ by $\frac{1}{3}$ inch. A few days later the child was able to breathe freely without the tube, and he was discharged quite well seventeen days after the operation.

Gas was being administered in the surgery to a man, aged 52, preparatory to breaking down some adhesions about the ankle; symptoms of asphyxia supervened, and were relieved by prompt tracheotomy; primary suture of the tracheal wound was then performed, and the man made a good recovery.

A woman, aged 25, was admitted for multiple papillomata of the larynx of fifteen months' duration. Tracheotomy was performed, Hahn's canula inserted, the larynx then split open and the growths removed freely. The patient made an excellent recovery.

OPERATIONS ON THE VASCULAR SYSTEM.

The femoral artery was tied in Hunter's canal for popliteal aneurism in a man, aged 27; the patient made an excellent recovery.

A porter, aged 35, was admitted with a large arterio-venous aneurism of the left orbit; it had followed an injury twelve years ago; ten years ago the left common carotid had been tied, but pulsation soon returned, and the aneurism continued to grow slowly. On admission, it was found that pressure upon the right carotid checked the pulsation of the aneurism, so that artery was tied. The operation was followed by some temporary improvement, but the pulsation quickly returned, and the man left the hospital in the same condition as before.

A similar but much smaller aneurism of the orbit, in a woman, aged 24, was treated by excision of the aneurism after many surrounding vessels had been tied. The patient made a good recovery, and was greatly improved.

The case of ligature of the internal jugular vein has been described in full under trephining for thrombosis of lateral sinus. Ligature of varicose veins of the leg was performed forty-seven times without a bad result of any kind.

OPERATIONS ON GENITO-URINARY ORGANS.

Nephrotomy was performed eight times; there was one death.

A butcher, aged 33, who had been stabbed in the loin, was admitted with profuse hæmaturia, which continued until the thirteenth day, when considerable hæmorrhage occurred also from the external wound. Urine had also, in the last two days, come from this wound. The kidney was then exposed through a lumbar incision, and was found to have been cut nearly in two by the original stab. Pressure forceps were applied to a bleeding point, and left on for twenty-four hours. The hæmaturia soon ceased, the wound healed, and the patient left the hospital on the sixtieth day, quite well.

A woman, aged 39, who had had hæmaturia three years ago, had been confined six months ago, and had ever since complained much of pain in the back, was admitted with a large pyonephrosis; no tubercle bacilli were found in the urine. A lumbar incision was made, and six ounces of pus evacuated. No calculus was found. She improved greatly after the operation, and left the hospital six weeks later with a sinus in the loin.

A man, aged 32, had had occasional hæmaturia for twenty-seven years, after a blow upon the loin. Two years ago a pint of pus had been evacuated from the kidney; no stone was found. The wound had never healed, and on account of the fistula and occasional rigors, headache and pain, he was re-admitted. An exploratory nephrotomy revealed a contracted and apparently useless kidney. No further operation was performed, and the patient left the hospital a month later with the sinus still discharging slightly.

A woman, aged 54, had in the last three years had numerous attacks of pain in the loin, accompanied by vomiting and headache, but no hæmaturia; a tumour could be felt at times in the loin; an incision through the loin showed the pelvis of the kidney to be dilated to the size of a hen's egg. This was opened, but no stone was found. The kidney was also unnaturally moveable so it was stitched to the abdominal wall; the patient was quite freed from pain, and left the hospital six weeks later with a small sinus still open.

A woman, aged 60, who had had for eighteen months pain in the loin, four months swelling of legs, but no renal colic, hæmaturia or other renal symptoms, was admitted with a large mass in the right side of the abdomen. Abdominal section showed it to be a kidney from which brownish fluid was exuding into the peritoneal cavity. A lumbar incision was then made, and pus and calculous matter let out. The cavity in the kidney was then stitched to the abdominal wound and drained in front and behind. The patient was much collapsed, and died two days later. The post-mortem showed a mass of carcinoma which involved also the psoas and ascending colon; there were secondary deposits in the liver.

A woman, aged 23, had had renal pain on and off for eight years; six months before admission a calculus had been removed from the kidney; the wound healed, but re-opened a few weeks later, and a sinus persisted ever since. An exploratory nephrotomy showed the kidney to be much dilated and suppurating. Nephrectomy was recommended, and the patient left the hospital to return a few weeks later (in the following year).

A traveller, aged 41, who had been operated upon for stricture a few weeks previously, returned on account of pain in the back and pyuria; an exploratory nephrotomy revealed nothing abnormal; the pain ceased.

An exploratory nephrotomy was performed upon a woman, aged 31, with an abdominal tumour of doubtful nature; the case is more fully described under cholecystotomy, which was subsequently performed.

Nephrectomy was performed twice by the abdominal method and four times by the lumbar; there were no deaths.

A woman, aged 27, had had for six months a painless tumour in the abdomen, gradually increasing in size; there were no urinary symptoms of any kind. The patient looked quite healthy; in the lower part of abdomen, chiefly in the left iliac fossa, was a large globular, prominent, firm, elastic swelling, not felt in the loin, and not extending into the pelvis. Abdominal section showed a sarcoma of the lower part of the left kidney, which was situated lower than usual. The kidney was removed, and the patient made a good recovery, leaving the hospital on the thirty-fifth day after the operation. A month later, however, she returned with paraplegia and other signs of recurrence, and in this condition she again left the hospital, three days later. The tumour weighed six pounds, and measured seven inches in diameter; microscopically, it was a round-celled sarcoma.

A girl, aged 16, was admitted with a large abdominal swelling, thought to be an ovarian cyst. It had been growing for four years, and occupied the whole of the right side of the abdomen. Abdominal section showed it to be a hydronephrosis; it was completely removed, the peritoneal edges were sutured, and the wound drained from the loin; the patient made a good recovery.

A woman, aged 40, was admitted with a large, irregular and very hard swelling of the right kidney, first noticed fifteen months ago; she suffered considerable pain; she had been under observation for a year and was getting worse; the tumour was removed through a lumbar incision, and proved to be composed mainly of dense fibrous tissue; the pelvis was not dilated, and there was no stone; the tumour weighed seventeen ounces, and was a very rare form of syphilitic disease. The patient made an excellent recovery.

A healthy-looking man, aged 24, was admitted on account of attacks of renal pain with hæmaturia and pyuria. The kidney was exposed by a lumbar incision, found to be tuberculous, and removed. The patient did well, and left the hospital two months later with a small sinus.

A woman, aged 38, who had previously undergone nephrorraphy for painful and dilated kidney, was re-admitted on account of pain; the kidney was removed by lumbar operation, and was found to be much dilated and atrophied. The patient left the hospital two months later with the wound almost healed.

A woman, aged 54, who had undergone nephrorraphy for moveable kidney five months previously, was re-admitted with a sinus in the loin; at the operation the pelvis of the kidney had been found somewhat dilated. The sinus was enlarged and drained, but did not heal. Nephrectomy was then performed; the kidney tissue was found to be much atrophied; the patient did well, and was discharged forty-two days later with the wound almost healed.

Nephro-lithotomy was performed three times, with one death.

A woman, aged 59, who had had hæmaturia ten years, and a noticeable renal swelling two years, was admitted very ill with a large right pyonephrosis. An incision was made through the loin, and several large calculi removed. She was much collapsed, and died three days later. At the post-mortem, there was thrombosis of the external iliac vein and extensive mitral and aortic disease.

The man, aged 36, upon whom nephro-lithotomy had been performed during the preceding year, was re-admitted with a sinus; this was enlarged, and another calculus was removed; the sinus had almost healed when he left the hospital a month later.

The woman, aged 23, mentioned under nephrotomy, was admitted with a seven months' history of renal pain and pyuria; an incision through the loin revealed a dilated kidney, containing about four ounces of purulent urine and a small calculus; the latter was removed, and the kidney drained.

Nephrorraphy was performed only once during the year; the patient, aged 54, was admitted for attacks of renal pain accompanied by vomiting; the kidney was explored and found moveable, with pelvis dilated to the size of a hen's egg; this was opened, but no stone was found; the kidney was fastened to the muscles by deep stitches, and the patient left the hospital forty-seven days later with the wound almost healed; she was re-admitted later. (See under Nephrectomy).

Lithotrity was performed four times upon male patients, aged 41, 26, 9 and 5; the first three made good recoveries; in the last case the operation was difficult and incomplete, and supra-pubic lithotomy was subsequently performed. (See Lithotomy.)

Lithotomy was performed by the supra-pubic method twice.

One patient was the boy just mentioned, aged 5, upon whom lithotrity had been unsuccessfully performed twenty-eight days earlier; he had had symptoms for two years; two stones were removed, weighing together 30 grains; the bladder was not sutured and no drain was used; the patient made an excellent recovery, and the wound had completely healed when the patient left the hospital on the fortieth day after the operation.

The other case was that of a boy, aged 16, who had had pain and occasional hæmaturia for six months; an oxalate stone an inch in diameter and weighing half an ounce was removed; the bladder was not sutured, the skin wound was sutured above and below only, and a drainage tube was left in for one day only. The wound was quite closed by the thirty-ninth day, and the patient left the hospital on the forty-ninth.

Supra-pubic cystotomy for conditions other than calculus was performed six times, with one death.

A paper-seller, aged 63, was admitted very ill with retention of urine, due to **enlarged prostate**, and stricture of many years' duration; supra-pubic cystotomy was performed, and the edge of the bladder wound sewn to that of the skin; the patient made a slow but good recovery, and he left the hospital fifty days later; four months later he was re-admitted for the fistula, which then closed spontaneously.

Three men had **carcinoma** of the bladder; one, aged 72, who had had hæmaturia two years and incontinence of urine four months, made an excellent recovery after drainage of the bladder, and left the hospital twenty-eight days later still wearing the tube; another, aged 60, who had had intermittent and usually painless hæmaturia for nearly three years, was admitted with cystitis. Both wounds in skin and in bladder were sewn up, except at the lower angle, where a drain was inserted temporarily; the bladder was drained also by the urethra. The wound healed in a fortnight, and the patient left the hospital thirty-one days after the operation, greatly relieved; the third, aged 47, had had frequency of micturition eleven months, hæmaturia ten months, very severe for three weeks; the abdominal wound and bladder were both sutured except at the lower angle, which was temporarily drained; a catheter was kept in the urethra for a fortnight. The patient made an excellent recovery from the operation, and left the hospital much relieved about six weeks later. He was re-admitted six months later (next year) with more hæmaturia.

In all three cases the growth was very extensive, and no attempt was made to remove or otherwise interfere with it locally.

Two men had **villous papilloma** of the bladder. A bookbinder, aged 42, with six years' history of hæmaturia, was re-admitted and operated upon a second time, the growth being scraped and cauterised with thermo cautery. Eleven months later he was re-admitted for retention, but was otherwise in good health.

A wire-worker, aged 52, had had hæmaturia for eight years; three years ago he had had a villous tumour removed in the hospital by supra-pubic operation; after this he was free from symptoms for one year. Recently he had had rigors and high temperature. The bladder was opened, and several ounces of villous growth scraped away; there was much bleeding stopped with hot water and cautery. After the operation he passed very little urine, and he died on the fourth day. At the post-mortem, it was found that almost the whole growth had been removed, but there was pelvic peritonitis which had evidently existed before the operation. There was no dilatation of either ureter or kidney.

Castration was performed nine times, with one death. A man, aged 69, who had had for four years difficulty in micturition; for two years had used the catheter and had had two attacks of cystitis and one of orchitis, was admitted with cystitis and enlargement of the prostate. Both testes were removed, together with part of the scrotum; the operation was followed by rigors, collapse and death in forty-seven hours. The post-mortem showed some chronic interstitial nephritis and a prostate which was uniformly enlarged, and measured two and half inches in diameter.

The other cases of castration were for **hæmatocoele** in a man, aged 68; **retained testis** in a boy, aged 14; **sarcoma** of three weeks' duration in a stoker, aged 23; **syphilitic disease** in patients, aged 28 and 39; **tuberculous disease** in patients aged 48 and 54 years, and 16 months: all did well.

A boy, aged 14, underwent an operation for **transplanting a retained testis**, and excising the sac of a hydrocele; he made a good recovery.

Ligature of varicocele was performed by the open method ten times, and subcutaneously twenty-six times; there was no serious complication in any case.

In one of the cases of **excision of the tunica vaginalis** for hydrocele' in a man, aged 50, a good deal of orchitis followed the operation.

Of the fourteen cases of **internal urethrotomy** for stricture, one died ; he was a man, aged 41 ; he had had gonorrhoea twenty years ago and symptoms of stricture for one year ; only the smallest catheter could be passed through the stricture, which was situated in the penile urethra ; there was no albuminuria ; the operation was followed by high temperatures, then drowsiness and albuminuria, and death on the seventh day, probably from suppurative pyelitis, but there was no post-mortem.

In two of the cases external urethrotomy was performed as well as internal.

Of the ten cases of **external urethrotomy** for stricture (including the two just mentioned), one died ; this was a man admitted with a large abscess of the scrotum, and a stricture ; he died on the day after the operation, and at the post-mortem was found to have a prostatic abscess and pelvic cellulitis as well.

Vaginal hysterectomy for carcinoma was successfully performed four times upon women, aged 42, 43, 46 and 46.

A woman, aged 46, was admitted with a very large uterine **fibroid** ; an attempt was made to remove it through the vagina, but the operation had to be abandoned. Great collapse ensued, and the patient died next day. At the post-mortem, fibroids were found weighing one hundred and fourteen ounces, there was a cyst in each ovary, and the tubes contained pus.

OPERATIONS ON THE RECTUM AND ANUS.

Among one hundred and twenty-seven operations of this class there was no death. Secondary hæmorrhage occurred on the sixth and sixteenth days respectively after operations for ligature of piles ; both patients were women, aged 54. The first had also a fistula, which was incised at the same time ; the other had also an attack of cutaneous erysipelas, which preceded the hæmorrhage ; both made good recoveries. The same complications occurred also in some of the cases of excision of the rectum. **Excision of the rectum** was performed upon two men and seven women.

A man, aged 48, was admitted with an extensive carcinoma, chiefly on the posterior wall, and extending further upwards than the finger could reach. The rectum was excised by Kraske's method, the coccyx and about an inch of sacrum being also removed, together with some enlarged pelvic glands. The operation was severe, and was followed by some recurrent hæmorrhage, from which the patient quickly recovered. Considerable stricture of the rectum followed, and for this a linear proctotomy was done three months later ; at this time there was already local recurrence of the disease. Colotomy was advised, but refused, and the patient left the hospital four months after the operation.

A cooper, aged 58, who had had pain, diarrhoea, and other symptoms for nearly three years, was admitted with a carcinomatous growth involving three-quarters of the circumference of the rectum ; it was chiefly on the posterior wall, and a finger could be got well beyond it. Excision of the rectum was performed with a good result ; some secondary hæmorrhage occurred on the seventh day, but soon stopped.

Six women underwent excision of the rectum for carcinoma, and one for sarcoma.

A woman, aged 24, who had had pain and hæmorrhage for seven months, was admitted with a growth posteriorly involving two-thirds of the circumference of the bowel and just within reach of the finger. A woman, aged 50, with twelve months' pain, diarrhoea, and slight hæmorrhage ; growth on

posterior and lateral walls three inches up—some fixity of rectum—on tenth day after operation some secondary hæmorrhage. A woman, aged 58, with five months' pain, diarrhœa, and hæmorrhage; much stricture at the anus; two and a half inches of rectum excised, together with most of vaginal septum. A woman, aged 40, with three months' pain and hæmorrhage; a comparatively small growth on anterior wall; a small recto-vaginal fistula followed the operation. A woman with two months' pain; a lump size of walnut on posterior wall; and a woman, aged 56, with three months' pain and hæmorrhage; a comparatively small growth just within the anus. All these cases made good recoveries.

One woman, aged 39, underwent excision of the rectum for melanotic sarcoma; for six months she had been passing blood; there was a tumour as large as a walnut extending two inches up the rectum; she made a good recovery.

MISCELLANEOUS OPERATIONS.

A feeble boy, aged 3, died of bronchitis ten days after admission for **cancrum oris**.

A girl, aged 3, also died; she was admitted very ill with **cancrum oris** following measles; three weeks before, the whole of one cheek was affected; it was scraped and cauterised with nitric acid on three occasions. The cheek became perforated, and the child died of broncho-pneumonia on the eleventh day after admission.

A feeble, emaciated girl, aged 21 months, had had for fourteen days ulceration of the mouth, and for one day swelling of the cheek; there was no history of measles or any other illness; the whole of one cheek and parts of the lip and opposite cheek were greatly swollen and ulcerated. She was treated for a week with antiseptic lotions, then the cheek was scraped and cauterised; various caustics and the actual cautery were applied on several occasions during the next ten days, the ulceration continuing to spread, and the cheek becoming perforated. Finally, after the fifth application of the cautery, healing began, and the child made a good recovery, although with such destruction of the cheek that nearly all the teeth on one side were exposed to view.

Another female child, aged 4, with **cancrum oris**, six weeks after measles, recovered after a single scraping.

Of three cases of **noma pudendi**, two, aged 3 weeks and 13 months, recovered after scraping and cauterisation; the third, aged 3 months, a feeble infant with **noma** of three weeks' duration, was scraped and cauterised with nitric acid, but died eleven days after admission of broncho-pneumonia.

A girl, aged 24, with a large **nævus of the rectum** and a history of twenty-two years' hæmorrhage, was re-admitted and treated by actual cautery with some benefit.

Two men, aged 22 and 23, were admitted with pustules, resembling **anthrax**, on the forearm and face respectively; one was treated by excision, the other by erasion; both rapidly recovered; neither case was proved definitely to be anthrax.

Tuberculous glands were excised or erased in forty-six cases without a death or any other serious complication.

Among forty cases of removal of **adenoid vegetations**, there was one death; the patient was a delicate child, aged 4, who had had, almost since birth, a discharge from one ear and nasal obstruction; adenoids and tonsils were both removed; the temperature remained normal for a week; on the

eleventh day the abdomen became tense and tender, and vomiting set in; she died next day, and at the post-mortem, general peritonitis was found, probably tuberculous in its nature. There were numerous caseous tuberculous glands in different parts of the body. It subsequently transpired that these vague abdominal symptoms had been present for several weeks. (The death recorded under removal of enlarged tonsils refers to the same case.)

A female infant, aged 3 months, made a good recovery after removal of a meningocele; the tumour was slightly pedunculated and as large as a horse-chestnut, and situated in the centre of the supra-occipital bone; the skin was reflected, the base tied with silk, and the tumour cut away; the child made a good recovery, and left the hospital on the twenty-third day after the operation.

A female infant, aged 9 weeks, was admitted with a very large occipital and cervical meningocele; it was tapped several times, but the child gradually sank and died; on post-mortem examination of the tumour, which was about half as large as the head, it was found that it communicated freely with a large cavity inside the skull; about one-third of the whole cyst was within the skull. There was much flattening of pons medulla and cerebellum, and the third ventricle was considerably dilated.

A carpenter, aged 67, who had been crushed between a train and a platform, was admitted with extensive bruising due to fracture of the pelvis; he gradually sank and died on the thirtieth day; transfusion of saline solution was performed a few hours before his death, without any benefit.

OPERATIONS FOR HERNIA.

Herniotomy for strangulation was performed twenty-eight times, with ten deaths.

Of the fatal cases, in two the operation was done on women for femora hernia.

A woman, aged 52, with a history of four days' vomiting and absolute constipation, was admitted in a state of collapse, with a cold skin, dry tongue and barely perceptible pulse; there was a small femoral hernia, of the existence of which the patient herself was not aware. An incision was made into the hernia and a drainage tube inserted; the patient gradually sank, and died on the third day. At the post-mortem, it was found that one and a half inches of ileum were involved; the parts were firmly adherent round the neck of the sac, and there was no peritonitis.

A laundress, aged 53, with three days' vomiting, constipation and pain, was admitted with a tense femoral hernia as large as a hen's egg; at the operation the gut was found much congested and was reduced. For four days the patient did fairly well, but with rising temperature; she then rather suddenly became delirious, and died on the fourth day after the operation. At the post-mortem there was no perforation and no peritonitis, but some chronic interstitial nephritis.

Four men died after herniotomy for inguinal hernia.

A man, aged 56, with four days' vomiting, constipation and pain, was admitted with an inguinal hernia as large as a foetal head. At the operation, the cœcum was found in the sac, as well as several inches of small intestine, in a very bad condition; the gut was incised, the contents evacuated, the incision sewn up, and the bowel returned into the abdomen; death occurred four days later; at the post-mortem there was perforation of the gangrenous portion of small intestine.

A man, aged 50, with two days' pain, vomiting and constipation, had had much taxis before admission; he died on the day after the operation. At the post-mortem there was no peritonitis, nor any rupture or gangrene of intestine, but the tissues in the iliac fossa had been extensively torn, probably by the injudicious taxis applied by the patient's friends.

A man, aged 65, with three days' pain and constipation, and two days' vomiting, was admitted with a hernia of the cœcum, which was found to be gangrenous; resection of the bowel by Murphy's method was performed (described more fully under Enterectomy), but the patient died two days later.

An undertaker, aged 47, who had had a rupture for twenty years, irreducible for seven years, painful and larger for two days, and who had been vomiting for one day, was admitted with an enormous scrotal hernia, tense and without impulse; it was translucent; it was punctured, and four and a half pints of serous fluid withdrawn, and the inguinal canal then incised without opening the sac or reducing the hernia. No relief following, the abdomen was opened on the following day, and a coil of small intestine opened. The man died next day, and at the post-mortem it was found that half the small intestine and seven inches of large intestine were in the sac, and had been badly strangulated, although it was not gangrenous; the constriction had been relieved. There was no peritonitis. The contents of the sac weighed seven pounds.

Two men and two women died after herniotomy for **umbilical hernia**.

A fat gas-fitter, aged 42, had had an irreducible umbilical hernia for two years; for six days he had had pain, for four days constipation and vomiting; in the last few hours the vomiting had been fœcal; the gut was returned at the operation and the ring sewn up. After the operation the patient did not vomit for ten hours, then vomiting returned and continued, until patient died, twenty-four hours after the operation; at the post-mortem, five inches of small intestine were found to have been badly strangulated, but there was no perforation of the intestine, nor any peritonitis.

A man, aged 68, had had a hernia for fifteen years, never reducible; there had been constipation for three days, vomiting two days; the hernia was large; he gradually sank and died the day after the operation. Post-mortem, no perforation, no peritonitis.

A woman, aged 60, was admitted with an irreducible umbilical hernia; four days after admission vomiting began; herniotomy and radical cure were performed within a few hours, but she died collapsed on the following day; at the post-mortem, the cœcum was found to have ruptured from distension; there was great narrowing of the transverse colon, which had been dragged upon by the omentum in the sac.

A woman, aged 50, admitted with a large umbilical hernia and a history of constipation four days, and vomiting two days, died four weeks after herniotomy and resection of small intestine (described more fully under Enterectomy).

Eighteen patients recovered after herniotomy.

After herniotomy for **femoral hernia**, two men, aged 52 and 81, with strangulation of a few hours and two days respectively; in the latter case the vomiting was fœcal.

Ten women, aged 30, 38, 40, 40, 43, 52, 61, 62, 63 and 64, with strangulation of nine hours, thirty hours, a few hours, two days, three days, four hours, three days, one day, two days, and (?) five days, made good recoveries; in the fourth of these ten cases, an abnormal obturator artery was divided; the proximal end was with difficulty secured, and no further trouble ensued.

After herniotomy for **inguinal hernia**, four men recovered, aged 24, 34, 39 and 66; strangulation had lasted two days, a few hours, fifteen hours, and sixteen hours respectively.

After herniotomy for umbilical hernia, two women, aged 24 and 65, with strangulation of one and two days respectively, recovered.

A chair-maker, aged 45, recovered after an operation for femoral hernia that had been reduced *en masse*. On the day preceding admission a femoral hernia of sixteen years' duration came down, patient pushed it back with some difficulty, and immediately afterwards felt pain in abdomen. A few hours later vomiting set in; the bowels were not opened. On admission, he was a healthy-looking man, in much pain, and vomiting frequently; no external hernia could be felt anywhere, but there was an indistinct fulness in the left iliac fossa. The abdomen was at once opened in the left linea semilunaris, and a knuckle of intestine as large as a horse-chestnut was found lying close to the internal ring and constricted by the neck of the sac; it was freed, and the patient made an uninterrupted recovery.

The operation for radical cure of reducible hernia was performed one hundred and twenty times upon ninety-three male and twenty-seven female patients; two patients died.

A stout woman, aged 50, who had been operated upon twice previously for radical cure of inguinal and umbilical hernia, was admitted with a large ventral hernia near the linea alba. A third operation for radical cure was performed, the ring, which was one and a half inches in diameter, being closed. The patient did well until the fifth day, when she was suddenly seized with rapid breathing and collapse, and died in a few hours. At the post-mortem, a simple ulcer of the duodenum was found to have perforated; it was a quarter of an inch in diameter, and situated at the anterior and upper part of the duodenum, close to the stomach; there was also some dilatation of the stomach.

The other case was that of a female infant, aged eight hours, admitted with a hernia of the size of a cricket ball, into the umbilical cord; the sac was thin and translucent; the neck was about as large as a little finger; there was some cyanosis; the hernia was reduced by taxis, but on the following day the sac was found to be sloughing, so it was cut away, and the edges of the opening united; the child became more cyanosed, and died the same day.

In five other cases there were minor complications. A woman, aged 28, aborted at two and a half months on the sixteenth day after radical cure of inguinal hernia. A man, aged 24, after operation for inguinal hernia, developed pneumonia; the wound had healed by first intention, and the temperature had been normal after the first three days; on the twenty-third day after the operation the patient developed pneumonia; empyema followed; he was transferred to a medical ward, where he eventually recovered. A man, aged 41, with inguinal hernia; on the fourteenth day, after the wound had apparently healed, it re-opened, and a focal fistula ensued; this, however, closed spontaneously a few days later, and the patient left the hospital on the forty-first day after the operation with the wound quite healed. A man, aged 65, became insane after an operation for inguinal hernia, on both sides, but the insanity had become less severe before the patient left the hospital, on the forty-first day after the operation.

A boy, aged 3, was operated upon for inguinal hernia; at the operation miliary tubercles were found in the wall of the sac; the patient made a good recovery from the operation, and left the hospital; a few weeks later, however, he was re-admitted with hip disease and tuberculous peritonitis, and of these he died a few months later. (See Appendix to Table I.)

The operation for radical cure of irreducible non-strangulated hernia was performed twenty-six times upon twelve male and fourteen female patients; one patient died. She was a woman, aged 76, who had had an irreducible femoral hernia for many years; she was admitted very ill, with vomiting, abdominal pain and distension; although the hernia showed no obvious signs of strangulation, it was thought right to explore it. A mass of non-strangulated

omentum was found and removed. No improvement followed, and the patient died five days later. At the post-mortem, the illness was found to be due to diffuse suppurative inflammation of the pancreas, with local peritonitis; there was also an incomplete obstruction of the small intestine by a band of old adhesions, and there were numerous gall stones.

There were only two other cases with complications; a man, aged 38, in whom the operation for irreducible omental inguinal hernia was followed by an attack of jaundice and albuminuria; there was no suppuration, and he made a good recovery, leaving the hospital on the twenty-fourth day after the operation, with the wound healed.

A man, aged 35, after an operation for femoral hernia, had a slight attack of lobar pneumonia, which, however, soon cleared up; an operation for varicose veins was then done upon him, and he left the hospital fifty-one days after admission.

COLOTOMY.

Inguinal colotomy was performed twenty-four times, with fourteen deaths; several of these deaths were due, however, more to the original disease than to the operation. For malignant disease it was performed seventeen times, with eleven deaths; for fibrous stricture and obstruction of uncertain nature seven times, with three deaths.

The fatal cases were as follows :—

A labourer, aged 66, with carcinoma of the rectum, was admitted with intestinal obstruction of three days; he was much distended; colotomy was done at once, the bowel being opened at once and a tube tied in. The temperature remained normal for ten days, then began to rise, and the patient gradually sank and died on the sixteenth day. There was no post-mortem.

A carpenter, aged 34, admitted for carcinoma of the rectum of six months' duration; there was slight obstruction; sixteen days after admission colotomy was performed, the bowel being opened two days later. The patient died on the following day. At the post-mortem, general peritonitis was found, due to the bowel having become partially separated from the abdominal wall. There were secondary nodules in the peritoneum.

A chemist, aged 67, was admitted with obstruction of two weeks' duration; no previous history of any intestinal trouble; abdomen much distended, colotomy performed at once, bowel opened immediately, no tube inserted. The patient died two days later of peritonitis, apparently starting in the neighbourhood of a greatly-distended cœcum, the serous coat of which had given way. There was a small tight carcinomatous stricture of the sigmoid flexure.

A man, aged 68, who for one year had had alternately diarrhoea and constipation, and for eight days complete constipation, abdominal distension, and occasional vomiting, walked to the hospital. He was found to have carcinoma of the rectum. On the following day, colotomy was performed, the bowel being opened at once and a tube tied in. He gradually sank, and died on the sixth day after the operation. The post-mortem showed peritonitis, which had started from leakage at the wound.

A woman, aged 38, was admitted with incomplete intestinal obstruction; a fortnight after admission the abdomen was opened in the middle line, a carcinomatous stricture found in the sigmoid flexure and left inguinal; colotomy then performed, the bowel being opened at once and a tube inserted. The patient made a good recovery from this operation, but twenty-four days later enterectomy was performed, which proved fatal (case more fully described under Enterectomy).

In the five preceding cases the intestine was united to the abdominal wall by numerous sutures, no glass rod being passed through the mesentery.

In the three following cases a glass rod was put through the mesentery, and few sutures employed :—

A shoe-maker, aged 68, who had had symptoms of carcinoma of the rectum for one year, was admitted with a large fixed carcinoma just within the anus ; little obstruction ; inguinal colotomy was performed six days after admission, a glass rod being passed through the mesentery, and the bowel opened three days later. Patient was relieved by the operation, but gradually became weaker, and died two months later. At the post-mortem, the whole pelvis was found to be occupied by a mass of suppurating carcinoma, originating in the rectum ; the primary growth had perforated the bowel ; there were no secondary growths.

A marble mason, aged 55, was admitted with a fixed mass of carcinoma of the rectum of at least fifteen months' duration ; he had already had four attacks of obstruction, from which he had recovered. On the ninth day after admission, colotomy was performed, a metal director being passed through the mesentery, and few sutures used. On the following day the bowel was opened and a little fecal matter came away ; a few hours later about eighteen inches of small intestine prolapsed through the wound ; they were immediately re-placed by the house surgeon, and on the following day the patient seemed none the worse ; he, however, developed general peritonitis, and died on the fifth day after the operation. His temperature was never at any time above 99°. The post-mortem showed that the peritonitis had started at the wound, and that there had not been any excessive distension of the colon above the stricture.

A carpenter, aged 53, with carcinoma of the rectum of eight months' duration, but without much obstruction, underwent colotomy six days after admission. A glass rod was passed through the mesentery ; the operation was difficult and tedious, lasting eighty minutes. Patient restless afterwards ; bowel opened on third day without much relief ; next day the patient died, apparently of peritonitis. There was no post-mortem.

A woman, aged 45, was admitted with a tight stricture of the rectum one and a half inches from the anus ; there was a history of syphilis thirty years ago, and of vomiting, diarrhoea, and painful defæcation for five months. Inguinal colotomy was performed, and the bowel opened seven days later. The colotomy wound did very well and the patient was much relieved, but she continued to have much severe pain about the sacrum and in the legs. A month later secondary nodules began to appear on various ribs, and patient gradually sank and died on the eighty-sixth day after the operation. At the post-mortem, a mass of carcinoma was found to occupy nearly the whole pelvis, and to have destroyed most of the sacrum and neighbouring parts of the ossa innominata. There were secondary growths also in the liver and spleen. Both ureters were involved in the growth, and were much dilated, uræmia having been the immediate cause of death.

In two cases the operation was done for malignant disease, originating outside the rectum.

A woman, aged 43, with carcinoma of the uterus and breast of a year's duration, was admitted with incomplete obstruction. Eighteen days after admission the obstruction being worse, colotomy was performed, the bowel being opened at once ; no glass rod was used. Patient was relieved for a few days, then pain returned, and she died three weeks afterwards. No post-mortem.

A compositor, aged 57, was admitted for a large abdominal tumour. For eight years he had been subject to constipation. Two and a half years ago, at another hospital, the abdomen had been opened and the tumour examined, but

not removed, as it seemed fixed to the pelvis. Since that time he had had several attacks of retention of urine. On admission, he was found to have a large firm tumour occupying the whole of the pelvis, and extending up nearly to the umbilicus. It was easily felt per rectum; the urethra was ten and a half inches long. Constipation increasing, colotomy was performed, a glass rod being put through the mesentery, and the bowel opened four days later; on the following day, however, the man died of peritonitis, the bowel having become detached from the abdominal wall. At the post-mortem, several ounces of fœces were found in the peritoneal cavity; the colon above the colotomy wound was greatly distended with fœces. The tumour was a huge spindle-celled sarcoma, unconnected with any bone, and had originated in the prostate; there were secondary growths in liver and pleura. There was no ulceration of either rectum or bladder.

Three women died after colotomy for fibrous stricture of the rectum.

A woman, aged 48, who had had constipation and diarrhœa for four months, was admitted with peritonitis and much distension of the abdomen. Colotomy was performed, the bowel being opened at once and a tube tied in; no glass rod used. The operation was difficult, the colon being much bound down by adhesions. Much fluid was found in the peritoneal cavity. Patient was relieved for a couple of days, then distension again increased; the abdomen was opened and washed out on account of the peritonitis, but patient died twenty hours later, on the fifth day after the first operation. At the post-mortem, there was a tight stricture of the rectum, due to inflammation around carcinomatous tumours of both ovaries. The immediate cause of death was peritonitis.

A woman, aged 29, was admitted for pain in chest, vomiting, alternating diarrhœa, and constipation and ascites; these symptoms had lasted eight months. A tight stricture of the rectum necessitated colotomy; no glass rod used; peritonitis and secondary growths in the peritoneum were discovered at the operation. The bowel was opened on the sixth day, but the patient died on the following day. At the post-mortem, carcinoma of the stomach was found to be the primary disease; this had set up chronic peritonitis, which in turn had led to fibrous stricture of the rectum and the transverse colon.

A woman, aged 54, who had suffered for 33 years with fibrous stricture of the rectum, was admitted very ill with symptoms of obstruction. The abdomen was opened as for left inguinal colotomy, and what was thought to be distended sigmoid flexure was sewn to the abdominal wall; the patient continued to vomit, and on the third day the viscus was opened; a large quantity of thin acid fluid escaped, and the patient died a few hours later. At the post-mortem, it was found that the pyloric end of the stomach had been dragged down to the iliac fossa by peritoneal adhesions, and that this had been opened instead of the large intestine; there was an abnormal attachment of great omentum to the abdominal wall, concealing the sigmoid flexure.

Nine patients **recovered** after ten inguinal colotomies.

A man, aged 50, with a carcinomatous stricture of the rectum, did very well and left the hospital on the twenty-sixth day after the operation; a glass rod was put through the mesentery, and the bowel opened on the sixth day.

An engine-driver, aged 59, with carcinomatous stricture of the rectum, also did well and left the hospital on the twenty-fifth day; a glass rod was used, and the bowel opened on the fifth day.

A shopman, aged 66, with two years' history of carcinoma of the rectum, made an equally good recovery, leaving the hospital on the twenty-seventh day; a glass rod was used, and the bowel opened on the sixth day.

A woman, aged 62, who had had symptoms of carcinoma of the rectum for ten months, and whose rectum had been excised at another hospital nine

months ago, was admitted with recurrence ; colotomy was performed, no glass rod was used, and the bowel was opened on the fourth day ; she left the hospital on the fortieth day.

A woman, aged 30, with carcinoma of the rectum of seven months' duration, made a good recovery ; no glass rod was used, and the bowel was opened on the fifth day. Three minor operations were afterwards performed upon the wound to improve the spur, which at first did not prevent the fœces from passing through the rectum. Patient left the hospital on the seventy-fifth day after the first operation.

In none of the five preceding cases was there much abdominal distension.

A carpenter, aged 46, with carcinoma of the rectum, was admitted with some abdominal distension, but no vomiting. Colotomy was performed, no glass rod was used, and the bowel was opened at once, a tube being tied in. On the third day the tube was removed ; on the fifth the bowel was found to be slipping back into the abdomen ; within a few hours the abdomen was opened in the middle line, washed out and drained, as it was thought that the peritoneum was being infected by fœcal matter : the colon was again stitched to the abdominal wall, and the patient made a good recovery, leaving the hospital on the forty-ninth day.

A woman, aged 43, was admitted with fibrous stricture of many years' duration ; linear proctotomy had been performed sixteen months before, but the stricture had returned ; colotomy was performed on the tenth day after admission ; no glass rod was used, and the bowel was opened on the fourth day. She left the hospital on the seventieth day after the operation.

A man, aged 55, with three months' constipation and three days' vomiting, was admitted with much distension. Right inguinal colotomy was performed at once ; no glass rod was used, a tube was tied into the cœcum and removed on the sixth day. The patient made a good recovery, and left the hospital on the seventy-first day after operation. The cause of the obstruction was never clearly ascertained, but was probably malignant.

A woman, aged 62, had suffered from constipation for many years ; for three months she had been much worse, with loss of flesh and occasional vomiting. On admission, there was complete obstruction, and much distension ; no definite cause could be found. Left inguinal colotomy was performed at once, but without satisfactory relief ; eight days later, the obstruction continuing, the cœcum was opened, stitched to the abdominal wall, and a tube tied in for four days. The obstruction was then relieved, and the patient made a good recovery, leaving the hospital on the ninety-first day after the first operation.

Median colotomy. A man, aged 70, was admitted with twelve months' abdominal pain, distension, and constipation. For seventeen days he had been vomiting, and for ten days there had been absolute constipation ; he was very ill, and greatly distended. The abdomen was at once opened in the middle line, and a malignant stricture found at the lower part of the sigmoid flexure ; it was pulled up into the wound with much difficulty, and opened there. The gut gave way during the manipulation, and the patient died a few hours later with fœcal extravasation ; at the post-mortem it was found that the gut had been opened below the stricture.

Lumbar colotomy was not performed at all during the year.

ABDOMINAL SECTION.

Four patients underwent operations for **tuberculous peritonitis**.

A boy, aged 2, who had been ill six months, and had been in the hospital five months, and was much exhausted by the disease ; no fluid was found but numerous tubercles in the peritoneum ; the operation was little more than an

exploratory one, the wound being closed up again very soon. When the wound was dressed on the sixth day, it was found that it had failed to unite, being apparently infected by the tubercle within the peritoneal cavity; fistulous openings formed in the intestines, and the child died on the twelfth day after the operation.

A man, aged 44, was admitted with chronic intestinal obstruction; the abdomen was opened and much peritonitis, apparently tuberculous, was found. The appendix, which was involved in the inflammation, although apparently not the cause of it, was removed, about two quarts of clear serous fluid were removed from the peritoneal cavity. The patient did well for nine days, then suddenly became collapsed, and died in a few hours. No post-mortem.

A boy, aged 6, appeared to be quite well until seven days before admission, when he fell and struck his abdomen. Four days later he complained of pain, and the abdomen began to swell. On admission, there was much distension, and the boy looked ill. He had no pain, but the temperature was slightly raised. Two months later a deep-seated swelling was found in the right iliac fossa; this was opened and found to be a localised intra-peritoneal abscess; five ounces of pus were let out. There was no faecal matter in this pus, but on the following day fæces came through the wound. The faecal fistula gradually closed in the course of the next few months, and the patient eventually made a good recovery and left the hospital just one year after admission.

A girl, aged 6, was admitted very ill with suppurative tuberculous peritonitis of six weeks' duration; much pus had been discharged from the umbilicus before admission. The abdomen was opened and drained, and the child made an excellent recovery, leaving the hospital two months later with the wounds soundly healed.

For perforated gastric ulcer the abdomen was opened in three cases.

A nurse, aged 33, who had suffered for about four weeks with slight pain after food and other symptoms of dyspepsia, but without vomiting or hæmatemesis, was suddenly seized with signs of perforation. The abdomen was opened four hours after the onset of symptoms, a small perforated ulcer in anterior wall of stomach near the cardiac end was quickly found and sutured. The abdomen was very thoroughly washed out with hot water. The whole operation lasted one hour and ten minutes. The patient lived three days, the temperature gradually rising to 102°. At the post-mortem, the wound in the stomach was found soundly closed. A very small amount of lymph was the only sign of peritonitis.

A woman, aged 48, who had had no previous symptoms of any gastric trouble, was suddenly, while at work, seized with violent abdominal pain and collapse. Several hours later she was admitted to a surgical ward and laparotomy performed twelve and a half hours after onset of symptoms. The peritoneal cavity contained free gas and gastric contents. A large perforation was discovered in the stomach, an attempt was made to close it by stitching omentum over it, direct closure by suture being impossible. At the time of operation the disease was thought to be malignant, as there was much induration around the perforation. The abdomen was washed out. The patient died twenty-four hours later. At the post-mortem, a large and very old simple ulcer was found on the anterior wall of the stomach near the lesser curvature; this had been united by adhesions to the liver; the rupture of some of the adhesions had caused the fatal perforation; the closure of the opening in the stomach had been incomplete.

A woman, aged 18, who had suffered from indigestion for nearly two years, was suddenly seized with abdominal pain and collapse. On the following day she came to the hospital with peritonitis. The abdomen was opened in the middle line above the umbilicus, about twenty-four hours after the onset of symptoms. A perforated gastric ulcer was found and sutured with much difficulty. The abdomen was irrigated and closed. The patient was greatly

collapsed after the operation and died a few hours later with a temperature of 105·8°. At the post-mortem, an ulcer as large as a sixpenny piece was found on the anterior wall of the stomach, near the middle of the lesser curvature. The sutures had not completely closed it. A similar ulcer was found at the corresponding situation on the posterior wall, but adhesions had prevented this from perforating.

The abdomen of a woman, aged 48, was opened and washed out for peritonitis on the fourth day after a colotomy; the peritonitis existed before the colotomy was done; the patient died twenty hours afterwards. (Case more fully described under Colotomy).

A boy, aged 13, was admitted with signs of acute peritonitis; the illness had begun with vomiting and general abdominal pain nine days before. A few hours after admission the abdomen was opened in the middle line, nothing found wrong in right iliac fossa, but in region of descending colon there was a considerable localised **intra-peritoneal abscess**. The peritoneum was irrigated and drained. A faecal discharge from the wound followed, but the boy recovered and left the hospital 132 days after admission.

The case of the man, aged 46, whose abdomen was opened and washed out on account of infection by faecal matter **after a colotomy**, has already been described under colotomy.

For **hydatid of the liver**, abdominal section was performed five times, with two deaths.

A woman, aged 26, had noticed a painless swelling in the region of the liver for about one month. The peritoneal cavity was opened, the cyst incised and irrigated, and its cut edges sewn to those of the wound in the abdominal wall. The cavity was drained; the patient made a good, although slow recovery. For many weeks afterwards pieces of membrane came away at intervals; the patient left the hospital five months after the operation with a sinus which had not quite healed, and which was in the same condition five months later still.

A man, aged 25, had had a slowly increasing lump in the abdomen for five years; the peritoneal cavity was opened and the cyst treated as in the preceding case, the cyst wall was extremely dense and adherent to the liver tissue; no attempt was made to remove it; the contents of the cyst were numerous small cysts with clear fluid and much caseous material. The cyst wall gradually came away in pieces during the next few weeks, and the patient left the hospital on the forty-fourth day after the operation, with a sinus not quite healed.

A labourer, aged 45, first noticed the swelling thirteen years ago; eight years ago he had been in the hospital and the hydatid of the liver had been opened and drained successfully; for five years he had been aware of a fresh swelling in the same situation. A large hydatid with very thick wall was opened by abdominal section, and several pints of fluid and cysts evacuated. No attempt was made to remove any part of the cyst wall. After the operation, there was a considerable discharge of bile from the wound. The patient made a good recovery and left the hospital on the fifty-ninth day after the operation with a sinus nearly healed: a month later it was still not quite healed.

A soldier, aged 28, who had been in Asia, was admitted with a large hydatid of the liver. The swelling had been noticed about eight months, and had been tapped twice before admission. Rapid increase in size had followed the last tapping. Slight hæmoptysis had been present for three months. The abdomen was opened, the cyst stitched to the abdomen wall, and then opened at once. Several pints of turbid fluid and the inner wall of the cyst were removed. The cavity was drained, and the wound was quite healed in seven weeks. Since admission, the patient had been much troubled with cough and

occasional hæmoptysis; physical signs of tumour of the right lung having developed, an incision was made through an intercostal space near the angle of the scapula into a hydatid of the lung; the cyst wall was removed with forceps. After the second operation there was considerable fever with rapid pulse and respiration, and the patient died a month after the last operation, and four months after admission. There was no post-mortem.

A woman, aged 30, had had for twelve months a swelling in the region of the liver; it had caused no pain or inconvenience. The abdomen was opened over the tumour, which was found to be a hydatid of the liver. The cyst and its fibrous capsule were with much difficulty separated from the liver; there was profuse hæmorrhage, and a portion of the cyst wall had to be left behind. The patient died of shock a few hours afterwards. At the post-mortem, much blood was found in the peritoneal cavity; the hæmorrhage had occurred chiefly from large branches of hepatic veins.

Cholecystotomy was performed once for empyema of the gall bladder, twice for gallstones, once for a thickened gall bladder, and once for carcinoma with gallstones; the last case was the only fatal one.

A woman, aged 31, was admitted with a swelling in the right loin of doubtful nature; after a preliminary nephrotomy which revealed nothing abnormal, an incision was made, a few days later, in front over the gall bladder, which was stitched to the abdominal wall, opened and found to contain pus, no calculi were found. She made a good recovery, and left the hospital two months later with the wound healed. The same patient was re-admitted a few months later with a fresh attack of inflammation and re-opening of the old wound; the gall bladder was re-opened (through the peritoneal cavity) and several fragments of stone, together with one large rounded smooth stone, were removed. The patient left the hospital twenty-five days later, quite well, with the wound healed.

A woman, aged 55, had had jaundice for five years, increasing on the whole, but with periods of intermission lasting generally about a month. For the last six months it had been continuous and deeper. An enlarged gall bladder could be felt, although not easily; the liver was rather large and hard. On the ninth day after admission a severe paroxysm of pain occurred. The abdomen was opened, the gall bladder, which was deeply seated, was brought to the surface and opened, and a faceted gallstone as large as a pea was removed. The gall bladder was stitched to the skin and drained. The jaundice diminished, the pain disappeared, and the patient left the hospital on the forty-ninth day after the operation, greatly relieved. Seven months later she was known to be in good health, but the sinus was still open.

A woman, aged 40, was admitted for pain over the region of the gall bladder; the latter was opened and found to have no stone but a very thick wall. The wound healed, and the patient made a good recovery.

A clerk, aged 47, was transferred from a medical ward on account of deep jaundice and a distended gall bladder. He had been ill for nine weeks. Cholecystotomy was performed and twenty-five gallstones removed. His condition was not improved, so eleven days later an attempt was made to perform cholecystenterostomy, but the operation had to be abandoned. The patient died a few hours later, and at the post-mortem a primary columnar-celled carcinoma of the cystic duct was found, involving also the hepatic duct.

Rupture of stomach. A van-guard, aged 18, was admitted in a state of collapse, having been struck in the stomach by the pole of a van. Six and a half hours after the accident, the abdomen was opened, and a large rent in the anterior wall of the stomach, one and a half inches from the pylorus and nearer the lower border than the upper, was discovered and sewn up with Lambert's sutures. The abdominal cavity, which contained a large meal of

meat and cauliflower, was thoroughly washed out and drained. The patient never recovered from the shock, and died four hours later. At the post-mortem it was found that the sutures had completely closed the rent.

Pyloroplasty was performed upon a man, aged 54, who was transferred from a medical ward with stricture of the pylorus, produced by swallowing hydrochloric acid three months previously. He was extremely emaciated, and was vomiting frequently. Just above the umbilicus the pylorus could be felt as a hard, moveable, tender lump. The abdomen was opened, the pylorus divided transversely and sewn up vertically, then covered with an omental graft. The pyloric opening, before the operation, was about a quarter of an inch in diameter. Patient was afterwards fed by the rectum. He gradually sank, and died four days after the operation. At the post-mortem, there was no leakage or peritonitis. The lumen of the pylorus had been considerably increased. The stomach was somewhat contracted, and the mucous membrane superficially ulcerated. The stricture was of a simple fibrous nature.

Gastrostomy was performed four times, with two deaths.

A man, aged 49, who had suffered for five months with dysphagia, and in whom an œsophageal bougie stopped fourteen and a half inches from the teeth, was transferred from a medical ward. He had recently had rigors, hoarseness of voice, and paralysis of one vocal cord. The first stage of a gastrostomy was performed, but the man died on the second day, before the stomach was opened. The post-mortem showed a tight stricture, apparently of a simple nature, opposite the left bronchus: there was scarring above and below this point.

A man, aged 43, was transferred from a medical ward on account of stricture of the œsophagus; symptoms had existed for nine months. He had been in the hospital before, and had been treated with œsophageal catheters for four months with much benefit. On admission he was very weak and ill. Nothing could be passed through the stricture. Gastrostomy was performed, the stomach being opened on the third day, but the patient died on the fourth. At the post-mortem, an epitheliomatous stricture was found at the lower end of the œsophagus, and a large secondary mass in the abdomen.

A man, aged 53, had had dysphagia for fifteen months, and vomiting after meals for three months before this. When admitted he was in good condition, with a stricture, thought to be malignant, opposite the cricoid cartilage. Nothing abnormal could be felt in the neck from the outside. Gastrostomy was performed, the stomach opened on the fourth day, and the patient made an excellent recovery, leaving the hospital on the forty-third day after the operation. He was known to be in good health and at work six weeks later.

A man, aged 41, with increasing dysphagia of six months' duration, and a swelling in the neck of two months' duration, had lost much weight recently, and had become hoarse. An epithelioma of the œsophagus could be felt in the neck. Gastrostomy was performed six days after admission, the stomach being opened five days later; he was fed by the artificial opening four days later, and left the hospital on the fourteenth day after the first part of the operation.

Gastro-jejunosomy was performed twice.

A woman, aged 32, had been quite well until eight months before admission, when she first felt pain in the abdomen; three months later, she first noticed a swelling, which had been increasing ever since, and she had had frequent vomiting, especially after food. On admission she was weak and emaciated. In the right hypochondriac and epigastric regions was a hard moveable swelling. The abdomen was opened in the middle line, a loop of jejunum

passed through a hole in the transverse mesocolon, and united to the stomach by an inner row of interrupted silk sutures and an outer continuous suture. The operation lasted one and a quarter hours. After the operation, the patient continued to vomit, became gradually weaker, and died on the twelfth day. The temperature had never been above 100.2° , and for the last six days had been normal. At the post-mortem, the union was found to be good, there was no leakage and no signs of peritonitis; the stomach was greatly dilated, and there was extensive carcinomatous disease of the pylorus, with secondary deposits in the mesentery and liver.

A woman, aged 67, had had for five months vomiting and pain after food, for six weeks a lump in the abdomen, and she had lost much flesh. At a consultation, gastro-enterostomy was advised, although the case was not thought to be a very hopeful one. The abdomen having been opened through the left rectus muscle, the stomach and jejunum were opened and united by a double row of fine silk sutures. The whole operation lasted forty-five minutes. After the operation the patient was fed partly by rectum, partly by mouth. Vomiting occurred occasionally, and the patient gradually sank and died on the fifth day after the operation. At the post-mortem, a little localised peritonitis was found, two of the stitches having given way. The opening between stomach and intestine easily admitted a fore finger. There was no constriction of the colon. The pylorus was the seat of a small carcinoma surrounded by much chronic inflammation; there was no dilatation of the stomach.

For acute intestinal obstruction the abdomen was opened six times; in five cases the cause was a band; all these patients died; the sixth case recovered; in this the cause of the obstruction was uncertain.

A schoolboy, aged 14, had been constipated for seven days, and had had faecal vomiting for thirty-six hours. The abdomen was opened immediately, and a strangulation of small intestine found, due to a Meckel's diverticulum, attached to the anterior abdominal wall. It was divided, but death occurred three hours later. At the post-mortem, part of the small intestine was found to be gangrenous.

A stone carver, aged 44, had had pain, constipation and vomiting for four days. On admission, he was collapsed, and had hiccough and faecal vomiting. The abdomen was opened immediately, and several inches of small intestine found strangulated by a band of uncertain origin, which was divided. The distended intestines were punctured with a trocar, and half a pint of this faecal matter evacuated. The stomach was also washed out. The patient survived four days. At the post-mortem, several inches of small intestine were found to be gangrenous. No trace of the band could be found.

A woman, aged 37, had had vomiting and constipation for four days. Ovariectomy had been performed upon her at another hospital, fourteen years previously. Abdominal section was performed at once, a band found and divided; the distended intestines were incised and then sutured. The patient died in a few hours, and at the post-mortem the ovarian stump was found to have been adherent to the mesentery in such a way as to cause strangulation of about two feet of ileum. The constriction had been relieved by operation, and the patient appeared to have died from paralysis of the bowel. There were no signs of recent peritonitis, and very few adhesions from old peritonitis.

A woman, aged 43, was admitted with a history of pain and constipation for four days, vomiting for three. On admission, she was in fairly good condition, complaining only of abdominal pain. Nothing could be felt in the abdomen. In the right inguinal region was a small, soft irreducible swelling with no impulse. On the following day, the symptoms of obstruction continuing, the swelling was explored, and found to be a cyst connected with the round ligament. The abdomen was then opened, and a band found strangulating the

ileum. Two coils of intestine adherent to each other gave way during the separation; the openings in the intestine were sewn up. The operation lasted two hours. The operation was followed by collapse, restlessness, and death on the following day. At the post-mortem, the wounds in the intestine were found closed; there was some chronic interstitial nephritis, but no peritonitis.

A woman, aged 63, who said she had always been subject to constipation, had been suddenly seized, four days before admission, with abdominal pain following immediately by vomiting; since that time there had been vomiting and constipation and distension of the abdomen. On admission, there was faecal vomiting, but no collapse. Abdominal section was performed at once, several inches of small intestine were found almost gangrenous, and gave way during the manipulation. The holes were sewn up. A band was found deep down in the pelvis and divided. The patient died in a few hours. At the post-mortem, numerous bands of old adhesions were found in the pelvis; one of these was attached to the mesentery of the ileum; another part of the ileum was adherent to the uterus. The post-mortem showed slight peritonitis and several feet of small intestine greatly congested and almost gangrenous.

A washerwoman, aged 58, was suddenly seized, two days before admission, with violent abdominal pain and vomiting; constipation ensued. The abdomen was opened on the day after admission, but nothing found except some congestion of about twelve inches of ileum; she made a good recovery, but was re-admitted in the following year with a small ventral hernia, which was then cured by operation.

*Eight children were operated upon for intus-susception.

A male infant, aged 10 weeks, was suddenly seized, on the day before admission, with abdominal pain and vomiting, followed by the passage of blood and mucus. On admission, nothing could be felt in abdomen or rectum, but on opening the abdomen, an ileo-cæcal intus-susception was found extending to the splenic flexure; it was reduced with difficulty, and the child died within twenty-four hours from the onset of symptoms. The post-mortem showed nothing more.

A male infant, aged 3 months, a few hours before admission, passed some blood and mucus. On admission, it looked ill; an intus-susception was felt on the left side of the abdomen and also per rectum. Injection of milk and water caused the tumour to disappear, and the bowels acted several times; on the following day the intus-susception returned and was again reduced in the same way. On the fourth day after admission it returned again and could not be reduced: on the same day the abdomen was opened, and a large ileo-cæcal intus-susception found, which extended as far as the rectum. It could not be reduced, and the peritoneum was torn in the attempt. The intestine was opened above the intus-susception, emptied, and then sewn up again. The child died a few hours later, a little more than four days after the first onset of symptoms. At the post-mortem, it was clear that the intus-susception had never been completely reduced.

A male infant, aged 6 months, had had, for about twenty-four hours, constipation, vomiting, and passage of blood and mucus; a tumour was felt above the umbilicus, but not per rectum. Air, and then oil, were injected with no relief. The abdomen was then opened and the intus-susception reduced. The operation lasted fifty minutes, and the child died in a few hours. The post-mortem showed that the intus-susception had started at the ileo-cæcal valve.

A male infant was admitted with symptoms of intus-susception of thirty-six hours' duration. A well-defined swelling was felt in the left iliac region; injection of milk and water caused it to disappear; it returned a few hours later, and was again injected. The symptoms persisting, the abdomen was

* One of these was in a medical ward and is not included in Table I.

opened and some congested intestine was seen ; this was taken to be a reduced intus-susception, as no other tumour could be felt. The child died thirty hours after admission. At the post-mortem, a large intus-susception was found extending down into the rectum, and lying behind the distended small intestine. It seemed clear that it had never been completely reduced.

A schoolboy, aged 12, died after an enterectomy for intus-susception of three days' duration (case described fully under Enterectomy).

A female infant, aged 9 months, transferred from a medical ward, died after an enterectomy for intus-susception of three days' duration (also described fully under Enterectomy).

A female infant, aged 4 months, was admitted very ill with an intus-susception that could be felt in the rectum, and a history of five days' abdominal pain, vomiting, and passage of blood and mucus. No tumour could be felt through the abdominal wall. The abdomen was immediately opened and the intus-susception drawn up with difficulty from the pelvis ; reduction was impossible, the serous coat giving way. The intus-susception was cut away, a ligature being placed around the lower and a tube in the upper end of the intestine. Much trouble was experienced in returning the intestines to the abdomen, they had to be punctured to let gas out. The child died soon after the operation.

A schoolboy, aged 12, had had for four days abdominal pain and vomiting. There was no collapse or abdominal distension. After a consultation with one of the physicians, the abdomen was immediately opened and three intus-susceptions found, one at the ileo-cæcal valve, and two higher up. None of them appeared to be acutely strangulated. They were all easily reduced, and the patient made an excellent recovery.

Operations for **appendicitis** were performed ten times upon nine patients ; in one case the operation was done in a quiet interval after acute attacks, the appendix being successfully removed. All the others were cases of acute appendicitis with abscess ; of the latter, in six the abscess was opened and drained by a comparatively small operation through peritoneal adhesions, the appendix itself not being removed—all these cases recovered ; in three cases the abdominal cavity was freely opened, washed out and drained. In these three cases more or less general peritonitis was already present before operation ; in one of these cases the appendix was removed ; all three died in less than twenty-four hours.

A cook, aged 20, was admitted with an abscess connected with the appendix. Vomiting and constipation had been present for two days, and there was a tender swelling in the right iliac fossa. On the day of admission, an incision was made just above and parallel to Poupart's ligament, and a small abscess found deep down behind the cæcum. A fecal concretion was removed, and the wound drained ; during the operation the general peritoneal cavity was accidentally opened and immediately sewn up again. The patient made an excellent recovery, and left the hospital on the thirty-ninth day, quite well. She was seen again a year later, and was still quite well.

A man, aged 20, who had suffered a year previously from typhlitis, was admitted with a tender swelling in the right iliac fossa ; pain and constipation had been present for a week, and he had had a rigor. On the day of admission an incision was made into the peritoneal cavity ; nothing was found, and it was sewn up again ; another incision was then made just above Poupart's ligament, and some foul pus and fecal matter let out. No attempt was made to remove the appendix, the cavity was drained, and the patient made an excellent recovery, leaving the hospital on the forty-ninth day, quite well and with the wound healed.

A man, aged 21, was admitted very ill with acute appendicitis of many days' duration. In the iliac fossa was an abscess of considerable size, which was immediately opened by an incision through the abdominal wall and adherent

peritoneum ; several ounces of foul pus were evacuated, and the cavity drained. A fecal fistula persisted for many weeks, but eventually healed, and the patient left the hospital seven months after admission. Fifteen months later he was seen again ; he was then at work as a labourer, and had had no further trouble of any kind.

A cabinet-maker, aged 16, was admitted with a large acute abscess in the right iliac fossa ; it had been present for twenty-three days. A year previously he had had a similar attack, which had passed off without external suppuration. On the day of admission, an incision was made parallel to Poupart's ligament, and ten ounces of foul pus were let out ; the general peritoneal cavity was not opened ; the cavity was washed out and drained, and the patient made an excellent recovery, leaving the hospital on the eighteenth day with a sinus almost healed.

A man, aged 36, had had for eight weeks a swelling in the right iliac fossa, increasing lately. There was a brawny, ill-defined swelling ; on the tenth day after admission an incision was made without opening the general peritoneal cavity, and four ounces of greenish pus let out. The man made a good recovery, leaving the hospital three months after admission, but with a sinus still open.

A trunk-maker, aged 17, who had been in the hospital several times before (twice this year), and had been operated on before, was re-admitted with a fresh abscess. The abscess was opened without wounding the general peritoneal cavity, and the patient did well, leaving the hospital with a minute sinus still open.

The same patient was re-admitted five months later, and in a quiet interval, after an attack of appendicitis, the appendix was removed. It was found to be thickened to the size of a little finger, but it was not distended by fluid or concretion ; there was no suppuration. The patient made an uninterrupted recovery and left the hospital on the sixteenth day after the operation, quite well.

A warehouseman, aged 23, was transferred from a medical ward with acute peritonitis due to the bursting of a perityphlitic abscess. He had been quite well until seven days previously, when he was suddenly seized with pain in the right iliac fossa, followed by vomiting. The pain continued and became general over the whole abdomen. There was much tenderness and rigidity of abdominal muscles. While straining at stool, collapse suddenly occurred, a few hours before the operation ; the abdomen was opened, found to contain free pus, and was washed out, but the patient died a few hours later. At the post-mortem, the remains of an abscess as large as a goose's egg was found at the upper part of the pelvis ; it was connected with a perforated appendix and had ruptured into the general peritoneal cavity.

A schoolboy, aged 9, was quite well until seven days before admission, when he complained of abdominal pain and began to vomit ; the abdomen became distended ; constipation was present, but not complete. On admission, he looked ill and had a pinched face ; the abdomen was tender all over, but chiefly in the upper part. Within a few hours of admission, the abdomen was opened in the middle line below the umbilicus, and some foetid pus let out ; this pus appeared not to be quite free in the peritoneal cavity, but to be more or less localised. The intestines in the upper part of the abdomen being much distended, another incision was made above the umbilicus, the intestines there found to be free from pus or lymph. The intestine was opened and a tube inserted, but the boy became very restless soon after the operation, and died in about sixteen hours.

A packing-case maker, aged 18, was admitted for slight pain in the right iliac fossa, some constipation, but no distension of the abdomen ; the symptoms had lasted two days. On the second day after admission, he was suddenly seized with severe abdominal pain, vomiting and collapse. The abdomen was

opened at once in the middle line ; much purulent peritoneal fluid escaped, at first non-odorous, then foul ; the incision was enlarged, the intestines brought out, and a perforated appendix found, and a concretion free in the abdominal cavity ; the appendix and concretion were removed, and the abdomen washed out and drained ; the patient was greatly collapsed, and died seven hours later.

Besides the above cases, the appendix was removed in another case, described under tuberculous peritonitis.

Enterotomy, as a separate operation, was performed four times ; in several other cases it was performed as a part of another operation, described under intus-susception, acute intestinal obstruction, &c.

A stableman, aged 53, was admitted with an extra-capsular fracture of the neck of the femur, caused by a fall upon the hip. Three days later, his abdomen became greatly distended and he had vomiting and constipation. A coil of small intestine was brought out through an abdominal incision and opened, but the patient died a few hours later. At the post-mortem, no cause for the intestinal obstruction could be found.

The case of the man, aged 47, upon whom enterotomy was performed after an operation for strangulated hernia ; that of the woman, aged 36, upon whom it was performed for obstruction after removal of an extra-uterine gestation, and that of the woman, aged 32, after ovariectomy, have been described under their respective headings.

Enterectomy was performed eight times ; six times by means of Murphy's button, once by Maunsell's method, and once by both methods simultaneously in a case of double enterectomy.

An enormously stout woman, aged 50, with a large umbilical hernia, was admitted for the third time with strangulation, she had been constipated for four days and had been vomiting two days. Herniotomy was performed, and fourteen inches of small intestine resected. The gut was greatly thickened and distended, so a Murphy's button of the largest size was used. The patient survived the operation twenty-eight days, dying eventually of a large fœcal abscess and peritonitis. At the post-mortem, the button was found impacted a few inches below the region of resection. It had set up ulceration and perforation of the intestine.

A man, aged 65, who had been ruptured thirty years, and had had three days' pain and constipation and two days' vomiting, was admitted in fairly good condition with a large strangulated scrotal hernia ; there was considerable inflammation of the skin. Herniotomy was immediately performed ; the contents of the sac were found to be cœcum and ileum ; on the former was a patch of gangrene about two inches in diameter ; six inches of ileum and three of colon were excised, the ends being joined by a one-inch Murphy's button. The coats of the bowel were much thickened and fatty, and there was much difficulty in fixing the button ; the two halves could not be firmly approximated. The operation lasted one and a half hours. The patient gradually sank and died two days later. At the post-mortem, peritonitis was found, due to leakage from incomplete closure of the button.

A delicate boy, aged 12, was admitted with symptoms of acute intestinal obstruction of three days' duration ; for several years he had been subject to occasional attacks of abdominal "cramp," no blood or mucus had been passed per anum. The abdomen was slightly distended ; close to the right anterior superior iliac spine could be felt a hard, tender cylindrical swelling. Abdominal section showed this to be an intus-susception of several inches of ileum about four inches above the ileo-cœcal valve. The intestine, being already gangrenous, was resected, Murphy's button being used. The operation lasted seventy minutes. For a day or two the boy did fairly well, but he then became very feeble, the wound showed no signs of repair, and on the fifth day, sudden

collapse was quickly followed by death. At the post-mortem, the button was found to be still holding the cut ends of intestine ; no leakage had occurred, but only very slight union had taken place. In the duodenum were two simple ulcers, one of which had recently perforated and was the immediate cause of death.

A female infant, aged 9 months, was admitted with the usual symptoms of intus-susception of three days' duration. The tumour could be felt by the rectum. The abdomen was opened immediately, and the intus-susception found irreducible and gangrenous ; resection was done by Maunsell's method, but the child died a few minutes after the operation was finished.

(The four preceding cases appear in the statistical tables under operations for hernia and intus-susception.)

A woman, aged 22, with a fœcal fistula of fourteen years' standing, the result of old perityphlitis, and who had undergone several operations already, was re-admitted. Several inches of small intestine were resected, and a Murphy's button inserted ; the operation was very complicated, and lasted one and a quarter hours ; the patient died collapsed twelve hours later. The post-mortem showed a communication between one of the sinuses and the peritoneal cavity ; there was an enterolith as large as a walnut that had evidently lain in the small intestine for many years.

A man, aged 57, was admitted with chronic intestinal obstruction. A small, hard lump could be felt in the region of the cœcum. There was no abdominal distension or vomiting. Fourteen inches of intestine, including the cœcum and two inches of ileum were resected, and a Murphy's button inserted. The operation lasted one and a half hours. The patient survived five days. About three inches of the part removed formed a dense, hard mass, which was at first thought to be malignant, but microscopic examination showed it to be only chronic inflammatory tissue. At the post-mortem, it was found that the man had had a chronic ulcer of the stomach, which had quickly set up local peritonitis and enteritis, thus leading to the stricture of the colon. The proximal end of the intestine grasped by the button had sloughed from the pressure of the button.

A woman, aged 65, was admitted with intestinal obstruction of eight days' duration (vomiting and constipation). The abdomen was much distended. Laparotomy was performed, and a small malignant growth discovered in the upper part of the descending colon ; thirteen inches of gut were resected and a Murphy's button inserted ; silk sutures were also used. The operation lasted over two hours, and the patient died collapsed an hour and a half later. At the post-mortem, thin fœces were found oozing from the intestine, at the seat of operation.

A woman, aged 38, was admitted for intestinal obstruction ; she had for years been constipated, but much more in the last two months ; she had had a similar attack two years ago. For eight days she had been constipated, for one day she had been vomiting. She was relieved by enemata, but a fortnight after admission vomiting recurred. The abdomen was opened, and a malignant stricture found in the sigmoid flexure. A left inguinal colotomy was performed at once. Twenty-four days later, the patient's general condition being good, a double enterectomy was performed ; the stricture was excised and a Murphy's button inserted ; at the same time the region of the colotomy wound was excised and the ends of the intestine joined by Maunsell's method. The patient did well for two days, then had severe abdominal pain and gradually sank and died on the fourth day. The post-mortem showed peritonitis due to fœcal extravasation at the area of the upper resection, where there was an opening large enough to admit the tip of the little finger ; there was also slight leakage at the site of the Murphy's button. There was a considerable accumulation of fœcal matter in the intervening nine inches of intestine.

The only other case in which Murphy's button was used during the year was one of **intestinal anastomosis**. This operation was performed only once, upon an ink-maker, aged 32, who was admitted for diarrhœa, vomiting and abdominal pain of seven months' duration. There was a large tender swelling in the region of the cœcum. The abdomen being opened, the tumour was found to be carcinoma of the ascending colon, and irremovable. Intestinal anastomosis was effected between the ileum and ascending colon by means of Murphy's button. The insertion of the button, after the abdomen had been opened, took exactly four and a half minutes. After the operation he was somewhat collapsed for a few hours and then made an uninterrupted recovery; on the second day he took liquid food by the mouth, on the eleventh solid food; on the twenty-second day he was allowed to get up, on the thirtieth the button passed, and on the thirty-first he went home. Four months later he died at home of extension of the disease, and at the post-mortem it was found that the anastomotic opening was of satisfactory size.

Abdominal section for **extra-uterine gestation** was performed six times, with one death.

A woman, aged 37, pregnant about six months. A fœtus weighing $1\frac{1}{4}$ lbs. was extracted and lived half an hour. The placenta, of the size of a man's fist, was left in the pelvis, and the wound treated without drainage. The patient died on the second day; at the post-mortem, several ounces of blood-stained fluid were found in the pelvis.

A woman, aged 36. The sac had ruptured, producing collapse; a great mass of clot was turned out and the end of the Fallopian tube removed. On the second day afterwards, symptoms of intestinal obstruction necessitated re-opening of the wound and enterotomy. The patient recovered from this and six weeks later an attempt was made to close the fistulous opening; it had not quite closed, however, when the patient left the hospital three months after the first operation.

A woman, aged 31. A small fœtus was seen among the clot. Wound closed. Patient did well.

In the three other cases of women, aged 23, 27, and 41, the fœtus was not actually seen; all the patients did well.

A woman, aged 22, had a **pelvic abscess**, which was opened and drained, but the patient died of peritonitis. At the post-mortem it was found that it had originated in an ulcer of the ileum.

The **uterine appendages** were removed in two cases from women, aged 40 and 43, with uterine fibroids and double hydro-salpinx respectively.

A woman, aged 29, recovered after removal of a suppurating tube and ovary.

A woman, aged 37, died after removal of a suppurating tube, which burst during the operation. There was no post-mortem.

Uterine fibroids were removed by abdominal section in two cases. In the case of a woman, aged 32, a pedunculated tumour larger than a child's head was removed by cutting through the pedicle, close to the uterus. The tumour was partly cystic, and had been noticed seven months; the patient did well.

In the case of a woman, aged 44, a large fibroid was also removed locally, together with the ovary and tube on one side. The patient died; at the post-mortem the cause of death was not clear; it was probably due to general unsoundness of heart, liver and kidneys.

Abdominal hysterectomy was performed seven times. Four women, aged 42, 41, 39 and 47, recovered; in the first of these the stump was treated by the extra-peritoneal method, in the last two by the intra-peritoneal.

A woman, aged 38, was treated by intra-peritoneal method: vomiting, restlessness, abdominal pain followed the operation, and the patient died on the third day. The temperature was never as high as 100°, but the pulse was 140. The post-mortem showed peritonitis.

A woman, aged 48, underwent hysterectomy with intra-peritoneal treatment of the stump for a large tumour, probably fibro-sarcoma. The patient died on the third day, and at the post-mortem pus was found in the pelvis, some of the stitches having given way. Both stump and wound looked healthy.

A woman, aged 44, with a fibroid of four years' duration, underwent intra-peritoneal hysterectomy and removal of an ovarian cyst; she died of shock a few hours later, and the post-mortem did not throw any further light on the cause of death.

After **ovariotomy**, thirty patients recovered and two died.

A woman, aged 32, was admitted with fibroid tumours of both ovaries, and chronic peritonitis with much ascites. Both ovaries were removed; the patient died on the sixth day. At the post-mortem, a large malignant ulcer of the stomach was also found.

A woman, aged 28, had had for ten months pain and abdominal swelling. An exploratory abdominal section was performed and eight pints of blood-stained fluid removed; a fixed, papillomatous, malignant mass was found in connection with the ovary. Further operation not being considered advisable, the wound was closed; the patient made a rapid recovery from this operation. Seventeen days later, at the urgent request of the patient, a radical operation was performed, and the whole of the diseased ovaries removed, together with some of the affected peritoneum. For a few days the patient did very well, but on the seventh day the patient had some abdominal pain and a quick pulse. On the following day the abdomen became distended; the small intestine was opened and much fluid feces escaped, but the patient died a few hours later. At the post-mortem, it was found that all the malignant disease had been removed and that the intestinal obstruction was caused by adhesion of part of the small intestine to the wound, and consequent kinking.

Abdominal section as a **merely exploratory** operation was performed thirteen times.

A man, aged 43, was admitted with a very hard lump, about three inches in diameter, in the lower part of the left rectus abdominis. It lay behind the rectus, and overlapped it slightly on either side. It had been noticed three months. Abdominal section showed that intestine was adherent to it, and as it was thought to be malignant and irremovable, nothing further was done; the wound healed quickly, and the man left the hospital a week or two later.

A labourer, aged 47, had had for six weeks a hard, moveable, nodular mass in the hypogastric and epigastric regions. For one year he had been losing flesh; there was no pain, and no gastric or intestinal symptoms. An exploratory operation revealed a mass of cancer in the gastro-hepatic omentum, connected probably with disease of the pylorus. It was not a suitable case for further operation. The wound quickly healed, and the patient left the hospital on the twenty-fourth day after the operation.

A man, aged 69, was admitted with chronic constipation and acute obstruction, which had lasted one day. Fœcal vomiting was present, and there was much distension of the abdomen; he had double inguinal hernia. The abdomen was opened, but no cause of obstruction could be found. After the operation, the obstruction was partially relieved, but the patient died five days later. At the post-mortem, a small, tight, carcinomatous stricture was found in the sigmoid flexure, firmly adherent to the brim of the pelvis.

A painter, aged 47, was admitted with a large tumour of the liver, supposed to be malignant; as there was, however, some doubt as to the diagnosis, a small exploratory incision was made over the swelling. The tumour being

found to be malignant and irremovable, the abdomen was closed. After the operation the patient gradually became weaker, his temperature became more and more sub-normal, and he died on the thirteenth day. There was no post-mortem.

A woman, aged 32, was admitted with a large abdominal tumour; an exploratory operation showed very extensive colloid carcinoma of the ovaries. Nothing more was done, and the patient gradually sank and died in five days.

The case of the woman, aged 28, in whom an exploratory laparotomy was followed a few days later by removal of malignant disease of the ovary, has been described under ovariectomy.

A boy, aged 16, was admitted for a large swelling in the loin; it had been noticed one month. An exploratory laparotomy was performed, and a pint of pus removed by aspiration. The wound was closed, and another incision made through the loin; the patient made a good recovery. The swelling was a perinephritic abscess.

A painter, aged 45, had suffered from abdominal pain for four months; for five days it had been much worse, with constipation and slight vomiting. When admitted, the abdomen was distended and tympanitic, and he was thought to be suffering from some form of intestinal obstruction. An exploratory laparotomy showed a very large retro-peritoneal swelling, clearly irremovable. The patient died two days later, and at the post-mortem a large aneurism was found at the junction of the thoracic and abdominal portions of the aorta; it had leaked and caused a huge extravasation of blood behind the peritoneum. (See Medical Statistics.)

A carpenter, aged 55, had had for three months increasing abdominal pain, chiefly in the hypogastric and left lumbar regions. An exploratory abdominal section revealed a pulsating tumour as large as a cricket ball in the region of the cœliac axis. It was doubtful whether the pulsation was inherent in the tumour, or transmitted. The tumour was thought to be either an aneurism or a new growth. Nothing more was done. The patient made a good recovery, and left the hospital seven weeks after admission, quite free from pain.

A woman, aged 30, was admitted with pain in the back, and a doubtful tumour in the abdomen. An exploratory incision showed nothing but some enlarged lumbar glands. The patient made a good recovery.

A boy, aged 10, fell on to a spike of a railing. He was admitted in a state of great collapse, with a wound in the right groin. The abdomen was immediately opened; much blood was found, but no injury to any viscus was discovered. The boy died two days later of acute peritonitis, and at the post-mortem it was found that the spike had transfixed the cœcum, penetrated the inferior vena cava, and passed deeply into the muscles. The wound of the cœcum was quite small, but had set up the fatal peritonitis.

A hawker, aged 56, was admitted with a swelling in the groin and symptoms resembling those of strangulated hernia. An incision into the swelling showed nothing but blood; an exploratory abdominal section showed nothing abnormal within the abdomen. The patient made a speedy recovery. The case was probably one of mere contusion of the abdomen.

A child, aged 5, slipped from a tramcar and was dragged a considerable distance. He was admitted on the following day with much inflammation of the scrotum and abdominal tenderness. A sub-acute peritonitis followed, and on the thirty-third day the abdomen was opened on account of symptoms of obstruction. Numerous adhesions were found, and some were divided. For the next fortnight the patient did well, but he was then removed from the hospital by his parents, against advice. It was suspected that the case might have been of tuberculous origin.

APPENDIX TO SUB-TABLE OF CASES OF ERYSIPELAS, PYÆMIA, &c.

ERYSIPELAS—Cutaneous.

Admissions.

Of thirty-one patients, none died.

Occurring in Hospital.

A man, aged 48, with a foul epithelioma of the cheek, and a woman, aged 22, after a burn from a hot air bath, developed erysipelas in the hospital and recovered.

A man, aged 34, with extensive carcinoma of the spine and suppuration in various parts of the body, had an attack of erysipelas while in the hospital, and died some weeks later.

After Operations.

A man, aged 49, after amputation at the shoulder for suppurative arthritis, and three female patients, aged 15, 54 and 60, after operation for cleft palate, ligature of piles and partial excision of both superior maxillæ for carcinoma ; all recovered.

A man, aged 53, after removal of the tongue and glands for epithelioma, developed erysipelas on the twentieth day, and when apparently convalescent twenty-five days later, died suddenly. At the post-mortem, pericarditis and pleurisy were found to be the immediate cause of death.

Phlegmonous.

Admissions.

Eight male and one female patient died.

A male infant, aged 14 days, died on the day after admission for idiopathic cellulitis of the scrotum and thighs ; the disease had existed one week. The post-mortem showed pulmonary collapse and pelvic cellulitis.

A male infant, aged 3 months, admitted with erysipelas of the scalp, died on the sixth day. The post-mortem showed partial collapse of both lungs.

A man, aged 36, admitted for cellulitis and gangrene of the leg, died on the following day. The post-mortem showed chronic interstitial nephritis.

A man, aged 51, was admitted very ill with acute cellulitis of the neck following tooth extraction a fortnight before. The cellulitis extended into the chest, and the patient died on the fifth day. The post-mortem showed that the whole of the cellular tissue of the neck and the superior mediastinum was infiltrated with fœtid pus, but there was no localised abscess.

A man, aged 51, admitted with cellulitis of the scrotum and a perineal abscess due to stricture, died on the eighth day. At the post-mortem it was found that the suppuration had extended to the prostate and thence to the pelvic cellular tissue and peritoneum.

A man, aged 62, admitted with extensive cellulitis of the thigh, died of septicæmia on the following day. The post-mortem showed that the disease had originated in a very foul ischio-rectal abscess, which had burrowed into the thigh.

A man, aged 63, who had been a heavy drinker, was admitted with cellulitis of the arm of fifteen days' duration. Incisions were made and pus let out, but the man gradually sank and died on the fortieth day. The post-mortem showed pleurisy and chronic interstitial nephritis.

A woman, aged 45, was admitted with cellulitis of the arm of four weeks' duration, which had followed a suppurating wound of the hand. The patient died of pneumonia on the fifth day. No post-mortem.

Occurring in Hospital.

Three men, aged 33, 36, and 44, who developed cellulitis in the hospital after admission for suppuration in the hand, contused wound of scalp, and compound fracture of skull; all recovered.

A woman, aged 30, who had been run over, was admitted with an extensive lacerated wound of the leg. Cellulitis set in on the second day, incisions were made, but the patient grew steadily worse. On the seventh day amputation through the thigh was performed, but the patient gradually sank, and died three days later. No post-mortem.

After Operations.

A woman, aged 38, and a boy, aged 7, who developed cellulitis after a plastic operation for syphilitic destruction of the nose, and a tenotomy for talipes; both recovered.

A man, aged 48, who developed cellulitis of the arm on the seventeenth day after a local removal of a sloughing sarcoma of the back, died thirty-one days later. At the post-mortem, pleurisy was found to be the chief cause of death.

PYÆMIA AND SEPTICÆMIA.

Admissions.

Of six male and four female patients admitted with pyæmia and septicæmia, only one recovered. This was a woman, aged 27, with chronic pyæmia following parturition. In the case of two male patients, aged 18 and 51 years, and two female patients, aged 18 and 20 months, the disease was a complication of otitis media, cellulitis of the neck, noma vulvæ, and suppuration in the elbow (cases described elsewhere under those headings).

A girl, aged 2, was admitted with pyæmia following acute necrosis of the tibia, which had begun five days before admission. The child had, on admission, pneumonia and abscesses on both arms. It developed pericarditis, and died on the seventh day after admission. The post-mortem showed also pulmonary infarcts and slight pleurisy.

A man, aged 18, with pyæmia and double empyema of ten days' duration, died on the twelfth day after admission. No post-mortem.

A negro lion tamer, aged 23, was admitted with septicæmia, caused by bites and scratches inflicted by a lion upon the thigh and arm two days previously. He died of pneumonia on the sixth day after admission.

A man, aged 42, was admitted with great swelling of the thigh and some fluid in the left knee. The history was, that a week previously he had shivered and felt ill; he kept at work until the day before admission, when he first noticed swelling of the left thigh. There was no history of any injury. On admission his temperature was 101° . The patient rapidly grew worse; on the day after admission, an incision was made down to the femur, but no pus was found; the patient died a few hours later. At the post-mortem, all the muscles of the thigh were found infiltrated with sero-purulent fluid, and there was an abscess in the lung. The condition was evidently one of acute septicæmia, but no disease of bone or joint or any other source of inoculation could be found.

The ninth case was that of a man, aged 62, who died of septicæmia and erysipelas complicating ischio-rectal abscess (described under Erysipelas).

Occurring in Hospital.

Two men, aged 63 and 65, and a woman, aged 30, died of septicæmia after admission for ischio-rectal abscess, stricture of urethra, and lacerated wound of the leg (last case described under Erysipelas).

After Operations.

The only case was that of a woman, aged 59, who died, probably of septicæmia, after an amputation of the breast for scirrhus. (See Breast Operations).

DELIRIUM TREMENS.

The only case was that of a man, aged 32, who developed the disease after admission for a simple fracture of the tibia and fibula. He recovered.



TABLE OF AMPUTATIONS, WITH THE PERCENTAGE OF DEATHS DURING THE TEN YEARS
from 1886 to 1895 inclusive.

OPERATIONS.	CASES UNDER TREATMENT.										PERCENTAGE OF DEATHS.										Total Number of		Average Per-centage of Deaths.	
	1886, 1887,		1888,		1889,		1890,		1891,		1892,		1893,		1894,		1895,		Cases.	Deaths.				
	1886,	1887,	1888,	1889,	1890,	1891,	1892,	1893,	1894,	1895,	1886,	1887,	1888,	1889,	1890,	1891,	1892,	1893,			1894,	1895,		
PRIMARY—																								
Thigh ...	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	15	6	40
Knee-Joint	1
Leg ...	1	3	2	1	1	2	1	2	3	2	15	4	26.66
Ankle-Joint ...	1	1	3	1	33.33	
Shoulder-Joint
Arm ...	2	2	...	2	1	6	1	1	15	2	13.33	
Forearm ...	3	2	4	1	2	3	17	2	11.76	
SECONDARY—																								
Thigh	4	1	3	2	1	2	13	5	38.46	
Leg ...	2	1	1	1	2	7	1	14.28	
Arm	1	1	1	...	1	1	5	1	20.00	
Forearm ...	1	...	2	2	5	
Shoulder-Joint

* Foot also amputated.

OPERATIONS.	CASES UNDER TREATMENT.										PERCENTAGE OF DEATHS.							Total Number of Cases.	Average Percentage of Deaths.							
	1886.	1887.	1888.	1889.	1890.	1891.	1892.	1893.	1894.	1895.	1886.	1887.	1888.	1889.	1890.	1891.	1892.			1893.	1894.	1895.				
FOR DISEASE—		
Hip-Joint	3	...	1	8	37.5
Thigh ...	12	25	15	13	7	13	14	10	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	143	9.79
Knee-Joint	...	1	1	1	...	2	2	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	10	10.00
Leg ...	8	5	7	7	4	6	6	11	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	67	4.47
Ankle-Joint	9	11	6	1	6	...	4	5	4	5	4	5	4	4	4	4	4	4	4	4	4	4	4	4	52	9.60
Shoulder-Joint	1	1	1	3	1	...	3	1	...	3	1	...	3	1	...	3	1	...	3	1	...	9	11.11	
Arm ...	4	2	...	2	5	4	2	1	3	4	25	27	3.70
Forearm	5	5	2	4	...	4	1	1	2	1	20	24	4.16

INDEX.

TO REGISTER OF POST-MORTEM EXAMINATIONS.

SURGICAL, 1895.

BY THE SURGICAL REGISTRAR.

NUMBER OF SURGICAL POST-MORTEM EXAMINATIONS MADE
DURING THE YEAR—196.

- ACTINOMYCOSIS, Lung—167.*
- ABDOMINAL CONTUSION—272.
- ABDOMINAL SECTION—*See* Operations—Laparotomy.
- ABNORMALITIES (Congenital)—
- Bladder, Band in—283.
 - Extroversion—139.
 - Brain, Atrophy—46.
 - Meningo-encephalocele—21.
 - Heart, Malformation.
 - Intestine, diverticula—183, 230, 296.
 - Meningo-encephalocele—21.
 - Neck, Hygroma—235.
 - Thyroid, absence of one Lobe—236.
 - Accessory—288.
 - Bilobed—304.
- ABSCESS—
- Axilla—45.
 - Brain—5, 107, 111, 114, 154, 176, 192.
 - Intra-peritoneal—101, 187.
 - Ischio-rectal—55, 266.
 - Kidney—2.
 - Lung—73, 300.
 - Neck—255.
 - Ovary—62.
 - Prostate—55.
 - Psoas—220.
 - Subcutaneous tissue—*See* Cellulitis.
 - Tunica Vaginalis—55.
- AMPUTATIONS—*See* Operations.
- ANEURYSM—
- Anterior communicating Artery—296.
 - Aorta—241, 253, 281.
 - Ankylosis of Knee—16.
- AORTA—
- Aneurysm—241, 253, 281.
 - Atheroma—25, 100, 127, 150, 241.
 - Kinking—103.
 - Rupture—29.
- APPENDICITIS—*See* Vermiform Appendix.
- ARM, Gangrene—114, 190.
- ARTERY, Femoral, Rupture—239.
- ARTHRITIS, Suppurative—
- Elbow—200.
 - Hip—107.
- ATHEROMATOUS ARTERIES—24, 25, 100, 127, 150, 241.

* The figures refer to the pages of the Surgical Post-mortem Register, kept in the Library.

- BACK—Sarcoma—11.
- BILE DUCTS, Carcinoma—69.
Dilatation—183.
- BLADDER—
Congenital Band in—283.
Dilatation—78.
Hypertrophy—16, 166.
Inflammation—16, 37, 89, 146, 227, 241.
Papilloma—212, 270.
Tubercle—283.
- BRAIN—
Abscess—5, 107, 111, 114, 154, 176, 192.
Atrophy—46.
Bullet Wound—195, 279.
Laceration of—1, 20, 24, 185, 216, 285.
Meningo-encephalocele—21.
Sarcoma—137.
- BREAST—
Carcinoma of—131, 221, 265.
- BURNS AND SCALDS—219, 245.
- CALCULI—
Biliary—69, 258.
Intestinal—62.
Ureteric—182.
- CANCERUM ORIS—*See* Stomatitis.
- CARCINOMA—
Bile Ducts—69.
Breast—131, 221, 265.
Femur—270.
Gall Bladder—258.
Kidney—207.
Liver—67, 131, 174, 207, 228, 270, 290.
Lung—49.
Lymphatic Glands—49, 59, 67, 69, 290.
Esophagus—49, 67, 111, 174, 270.
Ovary—41, 236.
Peritoneum—258.
Rectum—13, 210, 282, 290, 302.
Rib—290.
Sigmoid Flexure—57, 194, 233.
Spleen—290.
Stomach—76, 132, 155, 228, 258.
Thyroid—23.
Vertebrae—290.
- CARIES, Spine—104, 220.
- CELLULITIS AND ERYSIPELAS—11, 37, 55, 122, 149, 266, 289, 295.
- CHLOROFORM, Death from—16, 211.
- CHOROID PLEXUS—
Cyst in—304.
Tubercle—16.
- CLAVICLE—
Fracture—150, 159, 171, 231.
- COLON—
Stricture—132, 283.
Simple Ulceration—233.
- COLOTOMY—*See* Operations.
- CUT THROAT—*See* Neck.
- CYSTITIS—*See* Bladder.

- DERMATITIS EXFOLIATIVA—2.
 DIABETES—100.
 DIAPHRAGM, Rupture—171, 244.
 DISLOCATION, Elbow—171.
 DUODENUM—
 Diverticula—183.
 Congestion after burn—219.
 Ulceration—143, 256, 275.
- EAR, Inflammation of middle—5, 73, 83, 154, 176, 192, 202, 213, 223, 296, 298.
 ELBOW—
 Suppurative Arthritis—200.
 Dislocation—171.
 ENTERECTOMY—*See* Operations.
 ENTEROLITH—62.
 ENTEROTOMY—*See* Operations.
 ERYSIPELAS—*See* Cellulitis.
- FEMORAL ARTERY—Rupture—239.
 FEMUR, Fracture—198, 229, 239, 252, 270.
 FIBROMA OF MESENTERY—115.
 FIBULA, Fracture—25, 91, 292.
 FISTULA, Fœcal—62, 254.
 FOOT, Diabetic Gangrene—100.
 FRACTURES—
 Clavicle—150, 159, 171, 231.
 Femur—198, 229, 239, 252, 270.
 Fibula—25, 91, 292.
 Humerus—54, 65, 171.
 Jaw (Lower)—229.
 Pelvis—29, 127, 171, 201, 229, 231.
 Radius—171.
 Rib—8, 29, 65, 85, 91, 148, 159, 171, 201, 225, 231, 244, 250, 269, 270, 285.
 Scapula—65, 239.
 Skull (Base)—1, 27, 124, 143, 148, 159, 185, 216, 250, 260.
 Spine—29, 148, 159, 161, 171, 248.
 Sternum—148, 171.
 Tibia and Fibula—25, 91, 292.
- GALL BLADDER—
 Adhesions about—155.
 Carcinoma—258.
 Sarcoma—224.
 GALLSTONES—*See* Calculi.
 GANGRENE—
 Arm—114, 190.
 Foot—100, 226.
 Labia—88, 249.
 Leg—99.
 Intestine—106, 173, 254.
 Testis—302.
 GOÏTRE—*See* Thyroid.

GUNSHOT WOUND—
 Brain—195, 279.
 Lung—80.
 Palate—195.
 Spine—80.

HÆMO-PERICARDIUM—*See* Pericardium.

HÆMORRHAGE—
 Intra-cranial—1, 20, 24, 27, 185, 238, 235.

HÆMO-THORAX—*See* Pleura.

HEART—
 Congenital malformation—299.
 Dilatation—16.
 Sarcoma—88.
 Valvular disease—163.

HERNIA—
 Inflamed—145, 254.
 Old radical cure—256, 303.
 Reducible—3, 253, 256.
 Irreducible—183, 258, 259.
 Strangulated—87, 101, 129, 157, 169, 173, 183, 247, 259, 267, 299

HERNIAL SAC, Fibrous plates in—253.

HERNIOTOMY—*See* Operations.

HIP—
 Suppurative Arthritis—107.
 Tubercle—303.

HYDRO-NEPHROSIS—*See* Kidney.

HYDRO-PERICARDIUM—*See* Pericardium.

HYSTERECTOMY—*See* Operations.

ILEUM—
 Diverticulum—230, 291.
 Perforation of Ulcer—187.

INTESTINAL OBSTRUCTION—198, 230, 281, 301.
 Strangulation (by band)—94, 113, 183, 189, 193.

INTESTINE—
 Calculus—62.
 Diverticula—183, 230, 291.
 Duodenal Ulcer—143, 256, 275.
 Fœcal Fistula—62, 254.
 Fibrous Stricture—132, 283.
 Gangrene—106, 173, 254.
 Rupture—29, 57.
 Sarcoma—224.
 Simple Ulceration—187, 233.

INTUS-SUSCEPTION—19, 93, 106, 135, 170, 215.

JAW BONES, Fracture—229.

KIDNEY—
 Abscess—2.
 Carcinoma—207.
 Cyst (simple)—25, 49, 57, 189.
 Hydro-nephrosis—54, 78, 146, 218, 290.

KIDNEY—*continued.*

- Hypertrophy—182.
- Infarcts—241.
- Moveable—3.
- Nephritis, acute—212, 241.
 - chronic interstitial—3, 29, 55, 89, 99, 100, 120, 127, 150, 153, 166, 169, 189, 193, 227, 243, 252, 253, 265.
- Pyelitis—2, 16, 54, 161.
- Pyo-nephrosis—163, 182.
- Rupture—8, 27, 201, 231, 244.
- Sarcoma—43.
- Tubercle—16, 283.

KNEE, Ankylosis—16.

LAPAROTOMY—*See* Operations.

LARYNX—

- Inflammation—206.
- Papilloma—179, 240.
- Œdema—*See* Œdema of Glottis.
- Tubercle—288.

LEG, Gangrene—99.

LIVER—

- Carcinoma—67, 131, 174, 207, 228, 270, 290.
- Cirrhosis—49.
- Fatty—272.
- Hydatid—87, 222.
- Rupture—8, 29, 85, 201, 225, 229, 244, 269.
- Sarcoma—43, 97, 180.

LUNG—

- Actino-mycosis—167.
- Abscess—73, 300.
- Bronchitis—35, 67.
- Bullet Wound—80.
- Carcinoma—49.
- Collapse—21, 34, 45, 46, 139, 149, 179, 213, 274, 303.
- Emphysema—25.
- Laceration—65, 85, 171, 201, 225, 231.
- Œdema—11, 54.
- Pneumonia—25, 59, 78, 83, 88, 95, 174, 235, 243, 249, 268, 288.
- Sarcoma—39, 43, 180.
- Tubercle—16, 137.

MARASMUS—21.

MENINGITIS—5, 45, 83, 107, 111, 114, 174, 191, 202, 223, 260, 298, 303.

MENINGO-ENCEPHALOCELE—*See* Brain.

MESENTERY, Fibroma—115.

NECK—

- Cellulitis—122.
- Cystic Hygroma—235.
- Wound—119, 243, 293.

NECROSIS—

- Palate—35.
- Skull—176, 191.
- Vomer—35.

NEPHRITIS—*See* Kidney.

NOMA PUDENDI—88, 249.

ŒSOPHAGUS—

- Carcinoma—49, 67, 111, 174, 270.
- Fibrous Stricture—209.

OPERATIONS—

- Adenoids, removal—274.
- Amputation, Breast—131, 221, 265.
- Appendix, removal—103.
- Cholecystotomy—69.
- Colotomy—13, 41, 57, 132, 180, 194, 210, 224, 282, 291, 302.
- Crauiectomy—46.
- Enterectomy—19, 62, 101, 120, 145, 151, 261, 275.
- Enterotomy—9, 170, 198, 267.
- Excision, Knee—237.
- Extroversion of Bladder (plastic operation)—139.
- Gastro-jejunostomy—155, 258.
- Gastrostomy—67, 209.
- Hernia, radical cure—256.
- Herniotomy—87, 101, 129, 157, 169, 173, 183, 193, 217, 259, 267, 299.
- Hysterectomy—165, 217, 287.
- Laparotomy—23, 93, 94, 97, 113, 115, 125, 135, 141, 187, 189, 193, 205, 214, 215, 222, 233, 236, 246, 277, 281.
- Nephrotomy—163.
- Lithotomy—199.
- Ovariectomy—76, 115, 301.
- Pylorus (plastic operation)—273.
- Sarcoma of Back removed—11.
- Tongue removed—295.
- Tracheotomy—179, 206, 240. (*See also Medical Index.*)
- Trephining, Mastoid—5.
 - Skull—5, 20, 24, 176, 191, 216, 223, 296.

OSTEO-ARTHRITIS—*See Arthritis.*

OSTEOMA, Pelvis—211.

OTITIS MEDIA—5, 73, 83, 154, 176, 192, 202, 213, 223, 296, 298.

OVIOTOMY—*See Operations.*

OVARY—

- Abscess—62.
- Carcinoma—41, 236, 301.
- Cystoma—153.
- Sarcoma—97, 158.

PALATE—

- Necrosis—35.
- Bullet Wound—195.

PANCREAS, Suppuration—183.

PAPILLOMA—

- Bladder—212, 270.
- Larynx—179, 240.

PELVIS—

- Exostosis—211.
- Fracture—29, 127, 171, 201, 229, 231.
- Sarcoma—39, 43, 137.

PERICARDIUM—

- Blood in—253.
- Calcareous plate in—78.
- Inflammation—73, 208, 241, 295.
- Laceration—171.
- Serous effusion—35.

PERITONEUM—

- Adhesions—49, 183, 193.
- Carcinoma—258.
- Inflammation—9, 13, 23, 41, 43, 55, 57, 76, 89, 101, 125, 132, 141, 145, 155, 157, 180, 187, 189, 210, 217, 233, 236, 246, 254, 256, 261, 274, 277, 282, 287, 301.
- Sarcoma—39, 224, 251.

PHARYNX, Sarcoma—95.

PLEURA—

- Hæmo-thorax—80, 91, 159.
- Pleurisy—9, 11, 13, 39, 73, 89, 167, 213, 294, 295.
- Pneumo-thorax—65, 80, 171.

PLEURISY—*See* Pleura.

PNEUMO-THORAX—*See* Pleura.

PROSTATE—

- Abscess—55.
- Enlarged—146, 166.
- Inflammation, acute—37.
- Polypus—243.
- Sarcoma—180.

PYÆMIA AND SEPTICÆMIA—5, 37, 73, 137, 208, 266, 294, 300, 304.

PYO-NEPHROSIS—*See* Kidney.

PYLORUS, Fibrous Stricture—273.

RADIUS, Fracture—171.

RECTUM—

- Carcinoma—13, 210, 282, 290, 302.
- Fibrous Stricture—41, 132, 291.

RIB—

- Carcinoma—290.
- Fracture—8, 23, 65, 85, 91, 148, 159, 171, 201, 225, 231, 244, 250, 269, 270, 285.
- Sarcoma—137.

SARCOMA—

- Back—11.
- Brain—137.
- Bone, Pelvis—39, 43, 137.
- Rib—137.
- Vertebræ—137, 158.
- Gall bladder—224.
- Heart—43.
- Intestine—224.
- Kidney—43.
- Liver—43, 97, 180.
- Lung—39, 43, 180.
- Ovary—97, 158.
- Peritoneum—39, 224, 251.
- Pharynx—95.
- Prostate—180.
- Spermatic cord—180.
- Synovial Membrane—237.
- Stomach—97.

SCAPULA, Fracture—65, 239.

SEPTICÆMIA—*See* Pyæmia.

SIGMOID FLEXURE, Carcinoma—57, 194, 233.

SKULL—

- Fracture—1, 20, 24, 27, 124, 143, 159, 185, 216, 250, 260, 285.
- Necrosis—176, 191.

SPERMATIC CORD, Sarcoma—180.

SPINAL CORD, Bullet Wound—80.

SPINE—

- Bullet Wound—80.
- Carcinoma—290.
- Fracture—29, 148, 159, 161, 171, 248.
- Lateral Curvature—272.
- Sarcoma—137, 158.

SPLEEN—

- Carcinoma—290.
- Contusion—8.
- Infarct—241.
- Rupture—201, 231, 250.

STERNUM, Fracture—148, 171.

STOMACH—

- Carcinoma—76, 132, 155, 228, 258.
- Rupture—205.
- Sarcoma—97.
- Ulcer—23, 151, 214, 277, 292.

STOMATITIS, Ulcerative—35, 268.

STRICTURE—*See* Urethra.

STUMP—3.

SUFFOCATION—240, 255.

SUPRA-RENAL—

- Adenoma—25.
- Rupture—269.

SYNOVIAL MEMBRANE, Sarcoma—237.

SYPHILIS, Congenital—34.

TESTIS—

- Atrophy—91.
- Gangrene—302.

TETANUS—203.

THIGH, Abscess—288.

THROMBOSIS—

- Iliac Vein—163.
- Inferior Vena Cava—46.
- Innominate Vein—241.
- Lateral Sinus—5, 75, 202.
- Renal—46.

THYROID GLAND—

- Absence of one lobe—236.
- Accessory—288.
- Adenoma—146.
- Bilobed—236.

TIBIA—

- Acute Periostitis—208.
- Fracture—25, 91, 292.

TONGUE—Removal—295.

TRACHEOTOMY—*See* Operations.

TREPHING—*See* Operations.

TUBERCLE—

- Bladder—283.
- Choroid Plexus—16.
- Hip—303.
- Kidney—16, 283.
- Larynx—288.
- Lung—16, 237.

TUNICA VAGINALIS, Abscess—55.

ULCER—

- Duodenum—143, 256, 275.
- Stomach—23, 151, 214, 277, 292.

- URETER, Calculus in—182.
 URETHRA, Stricture—16, 54, 55, 78, 89, 218, 241.
 URETHROTOMY—*See* Operations.
 UTERINE APPENDAGES—
 Extra-uterine foetation—125.
 Hydro-salpinx—254.
 UTERUS, Fibroid—41, 251, 274.
- VEIN, Thrombosis—
 External Iliac—163.
 Inferior Cava—46.
 Innominate—241.
 Lateral Sinus—5, 73, 202.
 Renal—46.
- VENA CAVA INFERIOR—
 Laceration—29.
 Punctured Wound—246.
 Thrombosis—46.
- VERMIFORM APPENDIX, Ulceration—9, 103, 141.
- WOUND—
 Inferior Vena Cava—246.
 Intestine—246.
 Neck—119, 243, 293.
 Thigh—304.
 Thumb—203.
 (Bullet Wound)—
 Brain—195, 279.
 Lung—80.
 Palate—195.
 Spine—80.



LONDON :
PRINTED BY CHARLES SKIPPER AND EAST,
49, GREAT TOWER STREET E.C.

Printed
in
Great Britain



