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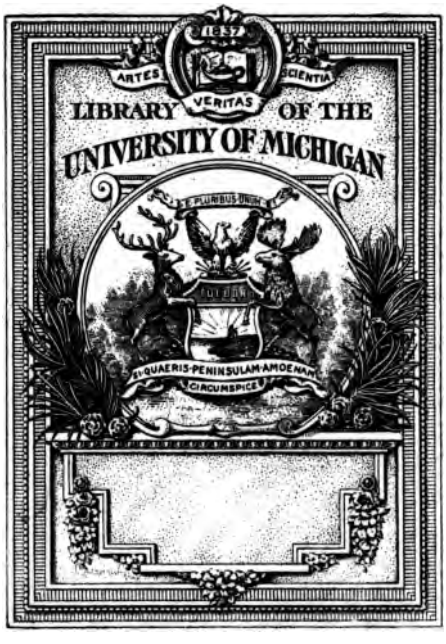
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OF THE

EPIDEMIOLOGICAL SOCIETY

OF

LONDON.

NEW SERIES. VOL. VII.

SESSION 1887-88.

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CONTENTS.

EPIDEMIOLOGICAL SOCIETY OF LONDON.

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ROBERT LAWSON, LL.D., Inspector-General of Hospitals (*retired*).

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France, Italy, and Spain ...	B. A. WHITELEGGE, M.D.
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Surg.-Gen. C. A. GORDON, C.B., M.D., Q.H.P.
G. B. LONGSTAFF, M.B.
Sir W. GUYER HUNTER, K.C.M.G., M.D., M.P.
JOHN MACCOMBIE, M.D.
Surg.-Major R. PRINGLE, M.D.
E. C. SEATON, M.D.
HERMANN WEBER, M.D.

LIST OF MEMBERS.*

(P) *President;* (T) *Treasurer;* (S) *Secretary.*

HONORARY MEMBERS.

Elected.
1888. Abbate Pacha, Cairo.
1867. Acland, Sir Henry W., K.C.B., M.D., F.R.S., Oxford.
1888. Amado, J. J. da Siloa., M.D., Lisbon.
1888. Bacelli, Guido, M.D., Rome.
1888. Billings, J. S., M.D., War Dept. Surg.-Gen.'s Office, Washington.
1888. Brouardel, S., M.D., 195, Boulevard St. Germain, Paris.
1888. Carsten, B., M.D., The Hague.
1888. Colin, Léon, M.D., 17, Boulevard St. Germain, Paris.
1868. Guipon, D. J., M.D., Laon Aisne, France.
c. 1888. Hirsch, A., M.D., 113, Pottsdamer Strasse, Villa 2, Berlin.
1885. Humphreys, Noel, General Register Office, Somerset House, W.C.
1888. Hunt, Ezra M., M.D., Trenton, New Jersey, U.S.A.
1888. Koch, R., M.D., 36, Klosterstrasse, Berlin.
1888. Larrey, F. H., M.D., 91, Rue de Lille, Paris.
c. 1888. MacLaurin, H. A., M.D., Macquarie Street, Sydney, N.S.W.
1888. Meijer, van Overbeck de, M.D., Utrecht.
c. 1888. Moleschott, J., M.D., Rome.
1888. Pettenkofer, Max von, M.D., Munich.
1888. Proust, A., M.D., 9, Boulevard Malesherbes, Paris.
1852 Richardson, B. W., M.D., F.R.S., 25, Manchester Square, W.
1850. Simon, Sir John, K.C.B., D.C.L., F.R.S., 40, Kensington Square, W.

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* Members are requested to inform the Secretaries of any corrections when necessary.

Elected.

c. 1883. Sonsino, Prospero, M.D., 11, Via San Lorenzo, Pisa.
1863. Stark, J., M.D., Bridge of Allan, Stirlingshire, N.B.
1888. Sternberg, Geo. M., M.D., John Hopkins University, Baltimore, U.S.A.
1888. Stokvis, B. J., M.D., 26, Sarphatstraat, Amsterdam.
1888. Vallin, E., M.D., 5, Avenue de Messine, Paris.

ORDINARY MEMBERS.

1887. Abraham, P. S., M.D., 11, Nottingham Place, W.
1878. Airy, Hubert, M.D., Local Government Board, Whitehall, S.W.
1882. Armstrong, Henry E., Town Hall, Newcastle-on-Tyne.
1888. Ashby, Alfred, M.B., Town Hall, Reading, Berks.
1884. Atkinson, F. E., Whitefriars, Settle, Yorkshire.
1881. Axe, J. Wortley, F.R.C.V.S., Veterinary College, Camden Town, N.W.
1882. Barry, F. W., M.D., Local Government Board, Whitehall, S.W.
1888. Bately, John, M.D., High Street, Gorleston, Great Yarmouth.
1888. Blaxall, Francis, R.N., M.D., Local Government Board, Whitehall, S.W.
1870. Buchanan, George, M.D., F.R.S., 24, Nottingham Place, W. (P. 1881-83. T. 1871-81.)
1887. Butterfield, Harris, Sevenoaks, Kent.
1888. Caiger, Frederick, M.D., Northern District Hospital, Winchmore Hill, N.
1886. Cameron, James, M.D, Guildford House, Hendon, W.
1882. Carpenter, Alfred, M.D., Duppas House, Croydon.
1885. Cobb, R., Surg. (*Gone abroad.*)
1868. Cock, Fred., M.D., 1, Westbourne Park Terrace, W.
1875. Collie, Alexander, M.D., Eastern District Hospital, Homerton, E.
1887. Collins, G. W., 66, Adelaide Road, N.W.
1884. Comyn, J. I., Dep. Surg.-Gen., 32, Dawson Place, W.
1871. Corfield, W. H., M.D., 19, Savile Row, W. (S. 1871-72.)
1885. Cornish, W. R., Surg.-Gen., C.I.E., 8, Cresswell Gardens, South Kensington, S.W.
1838. Corrie, A. T., F.R.C.S., Staff-Surgeon, H.M.S. *Rupert*, Hull.
1879. Cory, Robert, M.D., 73, Lambeth Palace Road, S.E.
1863. Crawford, Sir Thomas, K.C.B., M.D., Director-General Army Med. Dept., Craig's Court, S.W.
1887. Cresswell, Francis, Winchmore Hill, N.
1884. Cullimore, D. H., M D., 27, Welbeck Street, W.
1888. Dick, J. N., C.B., R.N., Director-General, Navy Medical Department, The Admiralty, Northumberland Avenue, S.W.
1863. Dickson, Walter, M.D., R.N., Fairfield, Belvedere Road, Upper Norwood. (President 1885-87.)
1886. Donovan, Justin F., M.D., R.N., Malta.
1880. Duka, Theodore, M.D., 55, Nevern Square, S.W.
1879. Ewart, Joseph, M.D., Dep. Surg.-Gen. Ind. Med. Dept. (*retired*), Montpelier Hall, Brighton.
1884. Faulkner, Alex., Surg., Messrs. Grindley & Co., 55, Parliament Street, S.W.
1875. Fayrer, Sir Joseph, K.C.S.I., M.D., LL.D., F.R.S., Surg.-Gen. Ind. Med. Dept. (*retired*), 53, Wimpole Street, W. (P. 1879-81.)
1887. Fletcher, W. F. E., M.B., Greetby Hill, Ormskirk.

Elected.
1888. Fosbroke, G. H., M.D., Bidford, Stratford-on-Avon.
1880. Gordon, C. A., C.B., M.D., Q.H.P., Surg.-Gen., 25, Westbourne Square, W.
1885. Gresswell, D. Astley, B.A., M.B., 5, Oakley Square, N.W.
1884. Hanbury, Sir James, K.C.B., Surg.-Gen. (*Gone abroad.*)
1875. Hare, C. J., M.D., Berkeley House, 15, Manchester Square, W.
1885. Harris, A. E., L.R.C.P., 14, The Avenue, Sunderland.
1884. Harris, F. W. H. D., Station Hospital, Madras.
1882. Hobley, S. Halford, 49, Bellevue Road, Southampton.
1887. Home, W. E., R.N., Surgeon H.M.S. *Alexandra.*
1883. Hopwood, E. O., M.D., London Fever Hospital, Islington, N.
1887. Hunt, J. Percival, Sheerness Hospital. Kent.
1880. Hunter, Sir W. Guyer, K.C.M.G., M.D., M.P., Surg.-Gen. Ind. Med. Dept., 21, Norfolk Crescent, W.
1887. Hyslop, T. B., County Asylum, Prestwich, Manchester.
1887. James, C. A., 24, Cazenove Road, Stoke Newington, N.
1863. Jenner, Sir William, Bart., K.C.B., M.D., F.R.S., 63, Brook Street, W. (P. 1866-68.)
1888. Karkeek, P. G., Isca, Torquay, Devon.
1887. Kelly, C., M.D., Gratwicke, Ellesmere Road, Worthing.
1881. Kynsey, W. R., Colombo, Ceylon.
1863. Lawson, Robert, Inspector-Gen., 20, Lansdowne Road, Notting Hill, W. (P. 1871-73.)
1886. Lingard, Alfred, 49, Lambeth Palace Road, S.E.
1884. Ligertwood, Thomas, M.D., Dep. Surg.-Gen., Royal Hospital Chelsea, S.W.
1880. Longstaff, G. B., M.B., Southfield Grange, West Hill Road, Wandsworth, S.W.
1887. Low, R. Bruce, M.D., Local Government Board, Whitehall, S.W.
1882. Manifold, M. F., Surg.-Gen., Twyford Lodge, 215, Upper Richmond Road, Putney, S.W.
1885. Martin, Sydney, M.D., 60, Gower Street, W.C.
1878. MacCombie, John, M.D., South-Eastern District Hospital, Deptford, S.E.
1880. McKellar, A. Oberlin, St. Thomas's Hospital, S.E.
1885. McLeod, K., A.M., M.D., Surgeon-Major, Messrs. Grindley and Co., 55, Parliament Street, S.W.
1885. Moir, John, Hack Road, Victoria Docks, E.
1883. Moore, Sandford, M.B., Surg.-Major, 33, Clarendon Park West, Kingstown.
1872. Murray, John, M.D., Surg.-Gen. Ind. Med. Dept. (*retired*), 17, Westbourne Square, W. (P. 1877-79.)
1875. Murphy, Shirley Forster, Hon. Secretary, 41, Queen Anne Street, Cavendish Square, W. (S. from 1877.)
1883. Nash, W., M.B., Surg.-Major Army Med. Dept., Craig's Court, S.W.
1885. Page, David, M.D., Local Government Board, Whitehall, S.W.
1883. Paget, Charles E., Hon. Secretary, Kendal, Westmorland. (S. from 1883.)
1858. Paine, H. J., M.D., Elmsfield, Newport Road, Cardiff, South Wales.
1884. Parkes, Louis, M.D., 61, Cadogan Square, S.W.
1883. Parsons, H. Franklin, M.D., Local Government Board, Whitehall, S.W.
1880. Payne, J. F., M.D., 78. Wimpole Street, W.
1887. Penny, J. C., M.D., Dep. Insp.-Gen., Glen Cairn, Kew Gardens.

Elected.

1884. Pringle, R., Surg.-Major, M.D., 2, Aberdeen Terrace, Blackheath, S.E.
1885. Redmayne, Hugh, Ambleside, Westmorland.
1880. Reid, Sir John Watt, K.C.B., M.D., Admiralty, Northumberland Avenue, S.W.
1875. Reynolds, J. Russell, M.D., F.R.S., 38, Grosvenor Street, W.
1875. Roberts, F. T., M.D., 102, Harley Street, W.
1873. Saunders, C. E., M.D., County Asylum, Hayward's Heath, Sussex.
1882. Scriven, J. B., Brigade-Surgeon Ind. Med. Dept., 95, Oxford Gardens, Notting Hill, W.
1873. Seaton, E. C., M.D., 35, George Street, Hanover Square, W.
1887. Shaw, C. Knox, 2, Pevensey Road, St. Leonards-on-Sea, and 112, Harley Street, W.
1884. Simpson, W. J., M.D., Bengal Club, Calcutta.
1878. Smee, A. H., The Grange, Hackbridge, Surrey.
1885. Smith, D. B., Dep. Surg.-Gen., East Cliff, Woolston, near Southampton.
1888. Smith, Frederick, Veterinary Surgeon, Montreal, Cargate Avenue, Aldershot.
1882. Smith, W. R., M.D., F.R.S.E., 74, Great Russell Street, Bloomsbury, W.C.
1880. Spear, John, Local Government Board, Whitehall, S.W.
1872. Squire, William, M.D., 6, Orchard Street, W. (S. 1873-77.)
1880. Sweeting, Richard, Western District Hospital, Fulham, S.W. (T. from 1887.)
1888. Sykes, J. F. J., M.B., 171, Camden Road, N.W.
1886. Taylor, M. W., M.D., Kenmure House, 202, Earl's Court Road, S.W.
1888. Thomson, Theodore, M.D., Health Offices, Sheffield.
1871. Thorne, R. Thorne, M.B., F.R.C.P., 45, Inverness Terrace, W. (T. 1881-87. S. 1879-81. P. from 1887.)
1888. Turnbull, A., R.N., M.D., Fleet Surgeon, 23, Matheson Road, West Kensington, W.
1876. Turner, George, M.D., Rosslynn, Broxbourne, Herts.
1888. Walford, Edward, M.D., Cardiff.
1854. Weber, Hermann, M.D., 10, Grosvenor Street, W.
1882. Whitelegge, B. A., M.D., 13, Belgrave Square, Nottingham.
1883. Willoughby, Edward F., M.D., Bratton Lodge, Green Lanes, N.
1888. Wilson, J. Mitchell, M.D., 51, Hall Gate, Doncaster.
1887. Yarrow, G. E., M.D., 87, Old Street, E.C.
1883. Young, Keith Downes, A.R.I.B.A., 17, Southampton Street, Bloomsbury, W.C.

Corresponding and Non-Resident Members.

N.R. 1867. Allbutt, T. Clifford, M.D., 6, Park Square, Leeds.
C. 1875. Arkhangelsky, D., M.D., St. Petersburg, Russia.
N.R. 1875. Armistead, William, M.B., Great Shelford, Cambridge.
C. 1852. Barker, R. H., Hungerford, Berks.
C. 1852. Bartolomé, Mariano Martin de, 334, Glossop Road, Sheffield.
C. 1856. Barton, E. H., New Orleans, U.S.A.
C. 1853. Beneke, F. W., Marburg, Germany.
N.R. 1883. Bovill, Edward, M.B., C.M., Motihari, Champaran, India.

Elected.

c. 1860. Brionde, A. de, Haute Loire, France.
N.R. 1864. Buckle, Fleetwood, M.D.
N.R. 1866. Caddy, J. T., M.D., Dep. Inspec.-Gen., 82, Redcliffe Square, S.W.
N.R. 1881. Carter, H. Vandyke, M.D., Surg.-Major Ind. Med. Dept.
c. 1851. Cartledge, Benjamin, Vet. Surg., Sheffield.
N.R. 1883. Castor, G. F., M.B., Colonial Surgeon, Cape Coast Castle, West Coast of Africa.
c. 1871. Christie, James, M.D., Zanzibar.
N.R. 1873. Close, C. Stratherne, Surgeon-Major Army Med. Dept.
N.R. 1875. Coffin, R. J. M., Alwington House, Baron's Court, West Kensington, S.W.
c. 1875. Colvill, W. H., Surg.-Major, Bagdad.
N.R. 1880. Condon, J. H., M.D., Surg.-Major Ind. Med. Dept., Cawnpore, N.W.P. India.
c. 1851. Cooke, R. T. E. B., The Haven, Esplanade Gardens, Scarborough.
N.R. 1885. Corney, Bolton G., Suva, Fiji.
c. 1856. Cox, Wm. Isidore, Hawkesbury-Upton, Chippenham, Gloucestershire.
N.R. 1885. Crawford, D. G., Surg. I.M.S., Mymensingh, Lower Bengal.
c. 1871. Cuningham, J. M., M.D., Surg.-Gen. Ind. Med. Dept., Sanitary Commissioner of Govt. of India.
c. 1860. Curtis, Josiah, Boston, U.S.A.
N.R. 1876. Day, E. J., Dorchester.
N.R. 1883. De Renzy, A.C.C., C.B., Surg.-Gen. Ind. Med. Dept. (*retired*), 9, Sydenham Villas, Bray, Co. Wicklow, Ireland.
c. 1878. Delmas, Louis H., M.D., P.O. Box 524, Havana, Cuba.
c. 1853. Dempster, Charles Carroll, M.D., Dep. Insp.-Gen. (*retired*).
N.R. 1852. Dickinson, Wm. Lindow (*address unknown*).
c. 1867. Dickson, E. Dalziel, M.D., Physician to British Embassy, Constantinople.
N.R. 1871. Domenichetti, Richard, M.D., Trinity Lodge, Louth, Lincolnshire.
N.R. 1864. Donnet, J. J. L., M.D., R.N., 5, Park Road, Bognor, Sussex.
N.R. 1876. Downes, A. H. J., M.D., Chelmsford, Essex.
N.R. 1863. Ducket, C. A., M.D., 12, St George's Terrace, Great Yarmouth.
N.R. 1871. Dunlop, James, M.D., 18, Carlton Place, Glasgow, N.B.
N.R. 1871. Dyke, Thomas Jones, F.R.C.S., Merthyr Tydvil, South Wales.
N.R. 1882. Fasken, William A. D., M.D., Surg. Ind. Med. Dept., Messrs. Grindlay and Co., 55, Parliament Street, S.W.
N.R. 1875. Fox, J. Makinson, M.D., The Grove, Lymm, Cheshire.
N.R. 1883. Furnell, M. C., M.D., Surg.-Gen., Shearman's Gardens, Madras.
c. 1851. Galland, —, M.D., Malta.
N.R. 1889. Gipps, A. G. P., Surgeon H.M.S. *Dreadnought*, Mediterranean Station.
c. 1863. Hall, E. Swarbreck, 58, Campbell Street, Hobart Town, Tasmania.
N.R. 1852. Hardwicke, Junius, Clifton Lodge, Rotherham.
.R. 1876. Hardwicke, H. Junius, M.D., Burton Lodge, Wostenholm Road, Sheffield.
N.R. 1872. Harris, Henry, M.D., Trengweath, Redruth, Cornwall.
1852. Harrison, J. B., M.D., Higher Broughton, Manchester.

Elected.

N.R. 1880. Harrison, R. Charlton, M.D., 13, Sandringham Gardens, Ealing, W.
N.R. 1887. Harvey, W., M.D., St. Eveste, Newton Abbott, Devon.
N.R. 1855. Haviland, Alfred, International Club, Trafalgar Square, W.C.
C. 1852. Hebra, Ferdinand, M.D., Vienna.
N.R. 1881. Henderson, G. C., M.D., Kingston, Jamaica.
N.R. 1882. Herring, John F., St. Mary's House, Atherstone, Warwickshire
N.R. 1875. Hime, T. W., M.D., 54, Horton Road, Bradford, Yorkshire.
C. 1853. Hirsch, August, M.D., 113, Pottsdamer Strasse, Villa 2, Berlin.
N.R. 1871. Hogg, F. R., M.D., R.H.A. (*address unknown*).
N.R. 1875. Home, Sir Anthony D., K.C.B., V.C., M.D., Surg.-Gen., India.
N.R. 1866. Home, A. H., M.D., Hullerhirst, Steventon, Ayrshire.
C. 1851. Hoskins, S. E., Guernsey.
N.R. 1881. Irwin, Chamney Graves, M.D., Dep.-Surg.-Gen., Edinburgh, N.B.
N.R. 1855. Jacobson, T. E., Sleaford, Lincolnshire.
C. 1886. Janssen, —, M.D., Inspecteur en Chef d'Hygiène, Brussels.
C. 1851. Kellay, —, M.D., Beyrout, Syria.
N.R. 1883. Kirker, Gilbert, R.N., M.D., R.N. Hosp., Gosport.
C. 1857. Leas, Jose Fernandes da Silva, Cape de Verde.
C. 1875. Lent, —, M.D., Cologne.
C. 1886. Le Roi de Méricourt, L., M.D., 5, Rue Cambacères, Paris.
N.R. 1879. Lilburne, James, M.D., Dep. Inspec.-Gen., Duncrivie House, Milnathort, N.B.
C. 1851. Litt, W., Veterinary Surgeon, Shrewsbury, Salop.
N.R. 1873. Little, James, M.D., 14, Stephen's Green North, Dublin.
N.R. 1885. Lloyd, W. Harris, M.D., Dep. Inspec.-Gen. (*gone abroad*).
C. 1857. Lowtzoff, —, St. Petersburg, Russia.
N.R. 1881. Lyons, R. T., M.D., Surgeon-Major, Rampart Barracks, Fort William, Calcutta.
C. 1887. McLaurin, H. A., M.D., Sydney, New South Wales.
C. 1870. Mallet, —, M.D., Warsaw.
N.R. 1865. Mapother, E. D., M.D., 6, Merrion Square North, Dublin.
N.R. 1867. Marshall, John, 13, Liverpool Street, Dover.
N.R. 1860. May, George, jun., 43, Castle Street, Reading, Berks.
C. 1854. Moffatt, Thomas, M.D. (*address unknown*).
C. 1886. Moleschott, Jacob, M.D., Rome.
N.R. 1872. Moore, John Wm., M.D., 40, Fitzwilliam Square West, Dublin.
N.R. 1884. Moore, W. J., C.I.E., Surg.-Gen. (*address not known*).
C. 1851. Monzon, Rafael, Barbacaos, New Grenada.
C. 1853. Neumann, —, M.D., Berlin.
C. 1851. Noble, Daniel, M.D., 258, Oxford Road, Manchester.
N.R. 1881. Oldham, Chas., Brigade Surg. Ind. Med. Dept., Shurdington Road, Cheltenham.
C. 1872. Poggio, Don Ramon Hernandez, Cuba.
C. 1854. Purple, S. S., M.D., New York, U.S.A.
N.R. 1879. Ransom, W. H., M.D., F.R.S., Nottingham.
N.R. 1875. Ransome, Arthur, M.D., F.R.S., Devisdale, Bowdon, Cheshire.
N.R. 1884. Ray, George, Surg.-Gen., 76, Jermyn Street, S.W.
N.R. 1874. Reid, Walter, M.D., Fleet-Surgeon, Royal Naval Hospital, Haslar.
N.R. 1866. Robertson, James, M.D., Alfred Place, Melbourne.
C. 1886. Rochard, G., M.D., Paris.
N.R. 1863. Rowe, T. Smith, M.D., Cecil Street, Margate.

Elected.

N.R. 1879. Ross, J. T. C., C.I.E., Surgeon-General Ind. Med. Dept., The Grove, Ryde, Isle of Wight.
N.R. 1881. Russell, E. G., M.B., B.Sc., Surg. Ind. Med. Dept., Gauhàti, Assam, India.
N.R. 1875. Russell, J. B., M.D., 1, Montrose Street, Glasgow.
N.R. 1859. Sanderson, J. Burdon, M.D., F.R.S., 50, Banbury Road Oxford.
C. 1851. Sankey, F. F., Malta.
C. 1879. Sarell, Richard, M.D., Pera and Therapia, Constantinople.
C. 1873. Schleisner, —, M.D., Copenhagen.
C. 1874. Schlimmel, —, M.D., Teheran.
C. 1852. Schürmayer, Professor, Heidelberg, Germany.
N.R. 1858. Scratchly, G., New Orleans, U.S.A.
C. 1851. Sharpe, Richard, Beverley Road, Hull.
N.R. 1869. Shaw, James, Insp.-Gen. (*address not known*).
C. 1853. Sigmund, C. L., Vienna.
N.R. 1867. Smith, R. Wagstaff, Mount Rundell, Harborne, Birmingham.
C. 1886. Sonsino, Prospero, 11, Via San Lorenzo, Pisa.
N.R. 1854. Spinks, C. N., Sankey Street, Warrington.
C. 1851. Spooner, W. C., Vet. Surg., Southampton.
C. 1876. Sternberg, Geo. M., M.D., United States Army, Port Point, San Jose, San Francisco, California.
N.R. 1885. Sturridge, P. F., Kendal, Westmorland.
N.R. 1880. Tatham, John, M.D., Town Hall, Salford, Manchester.
C. 1860. Taylor, H. Sharp, 15, Quarry Street, Guildford.
C. 1855. Tholozan, J. Désiré, M.D., Teheran.
C. 1888. Thompson, Ashburton, M.D., Sydney, New South Wales.
N.R. 1853. Todd, J. George, Evenwood, West Auckland, Durham.
N.R. 1885. Tomes, Arthur, Surg. Ind. Med. Service, Bengal.
N.R. 1873. Townsend, Stephen Chapman, C.B., Surg.-Gen. Ind. Med Dept., Sanitary Commissioner for Central India.
C. 1851. Tribes, E., Nismes, France.
C. 1855. Tribuchet, Adolphe, Paris.
C. 1853. Valerj, —, M.D., Rome.
C. 1855. Virchow, Rudolf, Professor, 10, Schellings Strasse, Berlin.
C. 1856. Von Iffland, —, Beaufort, New Quebec.
N.R. 1879. Watson, Alexander, M.D., R.N., Dep. Insp.-Gen., Army and Navy Club.
C. 1853. Watson, John, M.D., Jamaica.
N.R. 1887. Welch, Henry, M.D., Blackpool, Lancashire.
N.R. 1880. Whishaw, J. C., M.D., Surg.-Major Ind. Med. Dept., Lucknow.
N.R. 1880. White, J. Berry, M.D., Surg.-Major Ind. Med. Dept. (*address not known*).
C. 1861. Whitelaw, Wm., M.D., Kirkintilloch, Dumbartonshire.
N.R. 1854. Wiblin, John, Southampton.
N.R. 1865. Williams, C., 48, Prince of Wales's Road, Norwich.
N.R. 1884. Williamson, R. E., M.B., Brookfield, Lymm, Cheshire.
N.R. 1872. Woodall, John (*address not known*).
N.R 1875. Wortabet, John, M.D., St. John's Hospital, Beyrout.

TRANSACTIONS
OF THE
Epidemiological Society of London.

ON THE PROGRESS OF PREVENTIVE MEDICINE DURING THE VICTORIAN ERA.

By R. THORNE THORNE, M.B.Lond., F.R.C.P., President.

Inaugural Address of Session 1887–88.

(*November 9th*, 1887.)

Her Majesty the Queen ascended the throne on June 20th, 1837. She has now reigned for fifty years, and there are but few phases of the great and all but unhindered progress which has been so marked a feature of the Victorian Era that have had a greater effect for good upon her people than that which has had to do with the improvements affecting public health. Review of the advance thus made in all its branches would cover too large a field for me to deal with in this address, but I shall venture to lay before you some account of that progress, in so far as it has been identified with the development of Preventive Medicine and Epidemiology in this kingdom; feeling sure of your interest in an endeavour to place on record, in this Jubilee year, some account of a movement which, whilst it has effected a saving of life, and a prevention of disease on a scale the magnitude of which it would be difficult to define, has done so much to elevate the condition of man, and to bring health and happiness to an untold number of British homes.

In the year of the Queen's accession one of the greatest hindrances to progress in the prevention of disease was removed by the passing of the Act for the Civil Registration of Deaths, as well as of births and marriages. There had, indeed, been some partial registration of deaths since the date of Queen Elizabeth; the London weekly Bills of Mortality, first commenced in 1593, had been uninterruptedly continued from 1603 to 1831; but the system gradually became less rather than

more efficient. Even before 1593 certain records had been kept which chronicled the periodic devastations of this country by famines and epidemics; black death, sweating sickness, and plague being prominent amongst the latter. Later on came other records, medical and historical, supplying information, more or less inexact and incomplete, as to large and often sustained mortalities from small-pox, scarlet fever, influenza, and what appeared to be typhus and diphtheria, as well as from other allied epidemic diseases. The last of these epidemics was due to cholera, which made its first appearance in the United Kingdom in October 1831, and from which, in the following year, many thousands perished. But no registration of the causes of death existed at the time, and hence, although 52,547 cholera deaths were heard of from various sources as having occurred in the United Kingdom during that epidemic, yet the actual extent of cholera fatality remained unknown. Such statistical information as was available up to 1837 was all but useless for scientific purposes; death-rates were calculated on no certain data, and their significance was correspondingly open to question. The first requisite to a proper understanding of the etiology of disease was wanting, and epidemics were looked upon as enveloped in inscrutable mystery, and as far beyond human control.

But, in 1837, better machinery for the compilation of vital statistics was provided; and when Dr. William Farr entered the office of the Registrar-General of England, in 1839, those first steps were taken to secure such classification and grouping of deaths from various causes and in various localities, on sex, age, and condition of life, as have so largely contributed "to demonstrate the principal springs" of health and mortality. For no class of diseases was this more needed than as regards that group which appeared in the form of epidemics, concerning which, as Dr. Farr tells us, man could only "whisper of telluric and meteoric influences"—unless, indeed, some "comet was at hand to account for the phenomena". There was, in short, the grossest ignorance as to the group of so-called epidemic, endemic, and contagious diseases, the prevalence of which had formed epochs of chronology, affected the stability of cities, states, and governments, and brought wide-spread misery to communities and individuals.

The first publication of Abstracts of Deaths began to throw some light on the behaviour of epidemics, and the records indicated that, with a population which in 1831 had numbered some fourteen millions in England and Wales, infectious diseases were causing large mortalities. Thus, in the ten

quarters from July 1st, 1837, to December 31st, 1839, we can trace the march of an epidemic of small-pox which destroyed at least 30,819 persons in England and Wales; and it became evident, from the facts learned as to the incidence of the disease, that the rate of mortality increased with the density of the population, and that the extension of the infection was differently disturbed by the protection which some persons derived from vaccination, and by the artificial diffusion of the malady which accompanied the practice of small-pox inoculation. In 1840 scarlet fever was the reigning epidemic, the deaths from this cause having steadily increased from 5,802 in 1838 to 19,816. Diarrhœa, dysentery, and so-called cholera formed a group which was also causing a large mortality, and the distribution of the three affections was such as to make it in practice difficult to draw a line between them and those forms of diarrhœal disease which assumed epidemic characters. Attention was also, in 1840-41, attracted to the heavy mortality from "typhus", a term under which all the fatal attacks of continued fever were at that time registered. Already, the dothinenteria of Louis, the enteric fever of our day, was referred to in British statistical returns as in all probability different from typhus, though there is good reason for believing that at that date much of our "fever" mortality was in reality due to true typhus. Thus, in 1841, the excess of the "typhus" death-rate in towns over that obtaining in the country was 56 per cent. This fact, indeed, led to a further examination of the difference in the general death-rate of towns and country districts, and it was found that, whereas the annual mortality per cent. of population was 1·8 in country districts, it was as high as 2·6 in town districts; and in 1841 the Registrar-General pointed out that, whereas the mean duration of life in Surrey was 45 years, it was not more than 37 in the metropolis, and only 26 in Liverpool. Pulmonary consumption was also now ascertained to be one of the diseases which exhibited a striking excess in the case of town life. In fact, the general mortality was found to be increased 44 per cent. by the then condition of English towns and cities, and much that led to this excess of death was rightly held to be capable of remedy.

Some account of the principal diseases which I have thus incidentally referred to, namely, small-pox, typhus, enteric fever, scarlatina, diphtheria, phthisis, and cholera, will, in the main, suffice for the purpose of illustrating the progress which I am desirous of recording. They are typical of that group of diseases which is amenable to the control of preventive medicine and to public health measures; they hold

a prominent place in the history of scientific and epidemiological research during the period under consideration; and I propose briefly to review the course of each of them during the past fifty years.

I.—Small-pox.

Jenner's great discovery of vaccination—a process by which man can be protected against small-pox without himself becoming, as had been the case in variolous inoculation, a source of danger to others—was made known to the world towards the close of last century, but it was not until 1840 that the first Vaccination Act was passed which inaugurated the system of public vaccination, under which "all persons" could claim to be vaccinated at the public cost, and which has been the means of saving so great an amount of life in this country. It was also reserved for this era to extend and elaborate that system by the Acts of 1853, 1858, 1867, and 1871, and to promote and superintend the execution of those Acts by a department of the Government and by local officers. The diminution of small-pox occurring in sequence to these measures may be judged of by the following statement:—During the period 1838-42, towards the end of which the advantages of the Act of 1840 were in operation, the rate of mortality from small-pox in England and Wales was 57·2 per 100,000 living; by the end of 1849 it had fallen to 29·7, and during the period 1850-54 to 27·4. It was at this time that the Vaccination Act of 1853 came into operation. That Act embodied the principle of making vaccination compulsory on all infants within a few months of birth; and the Epidemiological Society, in a report which was printed by order of Parliament, and laid before the House of Commons, assisted in securing the adoption of this new departure, which has so largely contributed to the prevention of death from a loathsome disease. During the course of the three next quinquennial periods the small-pox rate fell further to 19·8, 19·0, and 14·4 per 100,000 respectively; the latter portion of the last period, namely, that for 1865-69, being in part influenced by the operation of the provisions embodied in the comprehensive Act of 1867.

So far, the operation of vaccination performed in one way or another could be credited with a success without parallel in the history of preventive medicine. But during the next five years—1870-74—the small-pox rate rose to 42·7 per 100,000; and although it again fell as low as 8·3 during the period 1875-79, and to 6·5 for 1880-84, yet the sudden

leap upwards in 1870 afforded incontestable proof that the practice of vaccination, as it was then largely carried out in this country, had not secured for the inhabitants generally that extent of immunity from small-pox which had been hoped for; and it became evident that there was yet much to learn as to the circumstances under which an operation apparently so simple, and yet so dependent for its full efficiency on the observance of important details, could best be carried out.

One principal point elicited as the result of the epidemic 1870-72 was, that a large proportion of the deaths from small-pox had occurred amongst persons over 15 years of age who were stated to have been vaccinated; and examination of the returns went to show that a large amount of imperfect and insufficient vaccination had been practised at a period when the then adult population had, as infants, been submitted to that process.

Other considerations were also raised by that epidemic. Small-pox, both in this country and abroad, was ascertained to have been of a malignancy heretofore unknown to the present generation, and it was assumed by some that a process of vaccination which had been sufficient to protect against fatal small-pox of the ordinary virulence had failed in the presence of an infection of exceptional potency such as that which had been imported under the circumstances of the great Franco-German war.

How far such theory of an exceptional potency of infection can be held to account for the results that ensued, may be open to doubt. But other possible explanations merit attention. Thus, there is the surmise of a special receptivity on the part of those attacked. The protection which vaccination had afforded against small-pox in this country had been steadily, and to an increasing extent, given to infants during several generations, and together with its extension there had been a gradual failing of that power of resistance to attack which is believed to be inherited by the offspring of parents who have themselves suffered from a specific infectious fever, in much the same way as the transmission of physical qualities takes place. And in this way it has been explained that, just as measles in 1875 wrought such havoc amongst the inhabitants of the Fiji Islands who had had no opportunity of acquiring any immunity against the natural force of the poison by reason of heredity, so small-pox, under the circumstances of 1871, broke in upon the descendants of a people who, by reason of vaccination, had had no opportunity for transmitting to their children such resistance to attack from

small-pox as had formerly been brought about by attacks of the same disease in their ancestry.

And again. When vaccination was first made gratuitous, many adults must then have resorted to it for the first time, and thus people acquired the protection attaching to a primary vaccination at an age which gave them a practical immunity against small-pox for the remainder of their lives. But as years passed on, primary vaccination during infancy became more and more general, and long intervals of time often elapsed between the performance of that operation and exposure to the poison of small-pox. The bearing of these and kindred considerations upon the epidemic of 1871-72 led to the belief that we were not yet fully acquainted with all the circumstances under which the full effect of vaccination in its conflict with small-pox could be acquired.

At this stage, Dr. Buchanan, medical officer to H.M. Local Government Board, commenced an investigation into the influence of vaccination as a protective against small-pox, the results of which are embodied in a series of reports which form some of the most important chapters in the history of this subject, and are characteristic of the labours by which progress in this department of the science of preventive medicine has been advanced during recent years.

At the outset of this inquiry it became evident that vaccination had led to a vast saving of life during infancy and childhood. Examining into the mortality from small-pox in children under ten years of age in London, Dr. Buchanan ascertained that this mortality "among the unvaccinated was about a hundredfold the mortality from small-pox among the vaccinated";* and that if in the year 1881, which was then under consideration, the small-pox mortality amongst the vaccinated children had been at the same rate as amongst the unvaccinated, London would during that one year have been confronted with 12,000 more deaths from small-pox than actually occurred. This saving of 12,000 young lives in one twelve months was effected by the current vaccination available; but it was further shown that "the power of a thorough vaccination to protect against death from small-pox is at least ten times greater than the power of much that passes for vaccination"; and that as regards the children dying in London of small-pox within some ten years of alleged vaccination, the mortality in this class obtaining "private" vaccination was more than twice as great as in the class seeking vaccination at the hands of public officials, who

* Report of the Medical Officer of the Local Government Board for 1881. [C.—3337.—I.]

are under the requirement to perform the operation according to a recognised standard of efficiency. In short, vaccination had not failed to prevent death from small-pox during the early period of life, except in so far as the application of the prophylactic had been imperfect.

In one sense the failure, at that date, to apprehend the need for such vaccination as we have now learnt gives the highest form of protection against small-pox is not to be wondered at. The extraordinary and continuous decline in small-pox mortality during the quinquennial periods already referred to, from a rate of 57·2 per 100,000 in 1838-42 to 14·4 in 1865-69, which had followed on the extension of the system of public vaccination, was enough to lead to an undue exaltation of the influence of vaccination as such; and it required the subsequent rise to 42·7 per 100,000 during the period 1870-74 to convince those who were engaged in the study of preventive medicine that there was more to be learnt about vaccination before its full protective value could be enjoyed by the public. Much of the needed information has already been obtained. Thus, whilst no difference can be found in the protective value of lymph, whether taken direct from the calf or after it has passed through the human subject, it is known that the full effect of the operation of vaccination can only be secured by careful selection of the lymph used, and by the performance of the operation in such a way as to secure at some four or five points of insertion well-foveated scars, which shall, in the aggregate, cover at least half a square inch of surface. And, further, it is becoming more and more evident that the protective value of even an efficient vaccination is not always so enduring as many had, at first, been inclined to think; that its full effects are not unfrequently limited to a comparatively short term of years; and that, for the retention of its full protection, it calls for repetition, especially in the young, probably within not less than ten years of the first operation.

This conclusion may to a large extent be drawn from the results of Dr. Buchanan's investigations, and it is prominently brought out by the statistics published in the recent report of the Vaccination Committee of the Epidemiological Society, and which go to indicate the need for an even earlier repetition of vaccination.* From that report we learn that, during the first five years of life, when liability to death from small-pox is at its highest, the influence of recent vaccination, efficient in point of foveation, number, and total area of the

* Report of the Vaccination Committee. *Transactions of the Epidemiological Society*, vol. v, Session 1885-86.

resulting scars, is pre-eminent in its protective power against fatal attack; that, indeed, at all ages up to twenty years "small-pox patients having 'good' vaccination at the date of their attack, die, according to their age, from $\frac{2}{80}$ to $\frac{5}{80}$ only the extent of unvaccinated patients at corresponding ages." The value of an efficient vaccination is thus strikingly indicated.

But if the power of vaccination as such, and without proper regard to quality and frequency of its application, has in the past been somewhat over-estimated, there is steadily accumulating evidence that the proper use of this extraordinary prophylactic has a potency which borders on the marvellous. Some who profess that they do not believe in its influence for good would allege that the all but uninterrupted diminution in small-pox mortality which has followed on the extension of vaccination has been brought about by an improvement, synchronously effected, in the general sanitary circumstances to which the population have year by year been exposed. But, under the same circumstances of treatment, unvaccinated small-pox patients die nowadays at the same, if not at a higher, rate than they died before the discovery of vaccination, while vaccinated small-pox patients die at a rate varying between one-fifth and one-fortieth of the rate of the unvaccinated; the difference between the one-fifth and the one-fortieth being determined by the degree of vaccination.*

Comparison of the results of exposure in hospital to the infection of typhus on the one hand, and of small-pox on the other, are of especial interest in this connection. In the London Fever Hospital there were, during the ten years 1861-70—the latest period during which typhus was received into that institution—no less than 179 attacks, and 42 deaths from typhus amongst the nurses and staff of the hospital;

* An unknown essayist, writing in *The World*, No. 127, 5th June 1755, supplies important information as to the deadly character of small-pox uncontrolled by any such protective as vaccination; and he, for the purposes indicated in his essay, contrasts it with the same disease as modified by inoculation. He states that, prior to the introduction of small-pox inoculation, London, thanks to the fatal ravages of small-pox, was tolerably roomy; people preferred to stay at home in the provinces rather than come to London and catch the disease. . . . "But now, thanks to inoculation, this danger has disappeared, and London is most inconveniently crowded." . . . "This inconvenience has, in a great measure, been hitherto prevented by the proper number of people who were daily removed by the small-pox in the natural way, one at least in seven dying, to the great ease and convenience of the survivors, whereas, since inoculation has prevailed, all hopes of thinning our people in this way are at an end, not above one in three hundred being taken off, to the great incumbrance of society."

whereas, according to the recent report of the Vaccination Committee of the Epidemiological Society, out of a total of 734 nurses and attendants in the three small-pox hospitals of the Metropolitan Asylums Board, whence complete information was procurable, not a single re-vaccinated person took small-pox. Of these 734 members of the staff, 79 (mostly former patients) had recently suffered from small-pox; of the remainder, 645 were re-vaccinated before entering on their duties; and 10 were, for one and another reason, not re-vaccinated. Every one of the latter contracted small-pox, whereas not one of the 645 re-vaccinated persons suffered from the disease.

Or again. During 1882 fourteen nurses were engaged at the Newcastle-upon-Tyne infectious hospital in attendance on cases of typhus. Of these, nine contracted the disease, and two died. In an adjacent pavilion nine other nurses were in attendance on small-pox patients. Of the nine, all but one, who had recently had small-pox, were re-vaccinated before coming on duty. None of these contracted small-pox, but one of them did catch typhus. So, also, during the same year, the Medical Officer of Health of Gateshead reported that "every nurse who has been more than a fortnight in the typhus wards has suffered from typhus; on the other hand, the only officers who took small-pox were two kitchen girls whom I neglected to vaccinate."

In each of these three groups of cases the two sets of nurses and attendants were drawn from the same class; they were fed alike, and subjected to the same sanitary circumstances. They differed in one respect only. One group of persons were fully exposed to the poison of small-pox, and being vaccinated they escaped; the others, unprotected by any prophylactic against the poison of typhus, suffered heavily, either by disease or death, from the infection to which they were exposed.

But evidence as to the value of re-vaccination is no longer limited to experiences such as I have referred to, for the story of an experiment on a gigantic scale is to be found in the report of the German Vaccination Commission.* There we find that, since the establishment of compulsory re-vaccination at the age of twelve years, "in Germany, as a whole, small-pox has diminished to a degree never before known, so far as any records reach"; whereas "in all neighbouring countries small-pox is, as usual, still very prevalent"; "that the German large cities suffer scarcely at all from

* On the German Vaccination Commission. *Transactions of the Epidemiological Society*, vol. v, 1885-86.

small-pox, which continues to demand its victims in all large foreign cities"; that "the German army is almost free from small-pox, while other armies still suffer severely"; and, lastly, that "not a single death from small-pox has occurred in the Prussian army since 1874", whereas both the neighbouring "Austrian and French armies still show considerable losses in this respect."

Reference to the knowledge recently acquired as to small-pox would be incomplete were I not to advert to the disturbing influence which the establishment of hospitals for the isolation of that disease has had upon the incidence and mortality from small-pox in the metropolis and in other towns. The investigations of Mr. W. H. Power into this matter are well known. They have been submitted to the consideration of a Royal Commission, and the whole subject has been further dealt with in Dr. Buchanan's recent reports on the proceedings of the Medical Department of the Local Government Board.* It is now generally admitted that aggregations of small-pox patients do cause an increase of small-pox in their neighbourhoods; and Mr. Power's contention that a hypothesis of the conveyance of the contagium through the general atmosphere can alone explain the circumstances of the diffusion, now meets with the almost unanimous support of competent scientific judges.

Advance in our knowledge of the natural history of small-pox, of its more obscure methods of diffusion, and of the circumstances under which the full protective effects of vaccination are to be acquired against this terrible scourge, has characterised the past fifty years; and although the record of a steadily diminishing small-pox death-rate was at one point marred by the circumstances of the period 1870-72, yet even that experience will have tended to a useful end, if only the striking lessons it has taught are but applied to the further control of this eminently preventable disease.

II.—Typhus Fever.

Review of Typhus, and of the influence of sanitary measures upon it in this country, are greatly hindered by the circumstance that it was not until 1869 that the different forms of continued fever were separated in the mortality returns of the Registrar-General. But there is abundant

* Reports of the Medical Officer of the Local Government Board: On the Use and Influence of Hospitals for Infectious Diseases, 1882 [C.—3290]; Annual Report for 1884 [C.—4156]; Annual Report for 1885 [C.—4844—I]; and Annual Report for 1886 [C.—5171].

evidence to show that typhus was widely prevalent during the earlier portion of the half-century we are considering, in many of our large cities, where poverty, over-crowding, and the resulting depressing influences were at work. In 1837 the disease was prevalent in several parts of the United Kingdom. In the period 1846-48 a typhus epidemic of terrible magnitude followed on an extensive failure of the potato crop in Ireland, which had brought about a vast amount of wretchedness and famine; and from Ireland the disease was imported into and spread in several of the large cities of England. About this date typhus was first discriminated in hospital returns from relapsing and from enteric fever; and the epidemic prevalence of the disease in London in 1848 is shown from the fact that 786 cases of typhus came under treatment in the London Fever Hospital during that year. In 1856 the typhus admissions into the London Fever Hospital reached 1,062. Another epidemic commenced in Ireland in 1861, and in the following year the disease was again widely prevalent in London. Indeed, the records of the London Fever Hospital show that during the period 1862-70 a long-continued prevalence of typhus was maintained, the admissions being 14,589 in all.

Since 1869 there has been an almost continuous decrease of typhus deaths in England, the fatal attacks falling from 4,281 in that year to 318 in 1885; the corresponding rates per thousand living being 0·19 and 0·01. And, in seeking the causes of this vast improvement, some reference to the incidence of the disease in London and Liverpool may be opportune. The conditions that bring about epidemic typhus are now but too well known. The disease is essentially associated with over-crowding and destitution; the depressing influence resulting from those circumstances being amongst the most powerful of its predisposing causes. Both in London and in Liverpool its prevalences have been mainly on those localities where houses have been so densely massed together on area as to preclude free movement of air about them, and where there has been such overcrowding of persons in dwellings as has ensured the fouling of the air within them by the concentrated emanations of living bodies. The incidence of the disease has also been essentially on the pauper and badly housed elements of the community.

In London there were 716 typhus deaths in 1869; but since that date this mortality has undergone rapid and almost continuous decrease, until in 1885 there were only 28 such deaths; the decrease in the rate per thousand living during the same period having been from 0·23 to 0·01. But

even these death-rates do not tell the whole story of the disappearance of this disease; for of the deaths which have in recent years been attributed to typhus in the metropolis, the majority have not been true instances of that disease.

In considering the circumstances under which the blot of our metropolitan typhus record has been so largely effaced, I would call to mind some of the conditions which affected the housing of the people during the earlier part of the past half-century. Writing in 1849, Sir John Simon, K.C.B. (then Mr. Simon), drew public attention to the existence of localities within the City of London which were so irremediably bad that no mere provision of wholesome water-supply and drainage could bring healthiness to their inhabitants. These localities exhibited complicated turnings, narrow inlets, close parallels of houses, and high barriers preventing light and movement of air. There were "courts and alleys hemmed in on all sides by higher houses, having no possibility of any current of air, and (worst of all) sometimes so constructed back-to-back as to forbid the advantage of double windows or of back doors, and thus to render the house as perfectly a *cul-de-sac* out of the court, as the court is a *cul-de-sac* out of the next thoroughfare."* The inherent vice of these houses was the lack of means for through-ventilation, and amidst it all there was "a dense population of human beings with an atmosphere hardly respirable from its closeness and pollution." "Typhus", it is added, "prevails there . . . . not as an occasional visitor, but as a habitual pestilence." Passing on to 1865, Mr. Simon, then medical officer of the Privy Council, in dealing with the question of the house accommodation for the poorer classes in towns and cities, generally adverts again to the sources and distribution of metropolitan typhus, and in his report he condemns especially the then "tenement-houses", the evils of which combined to constitute "one monstrous form of nuisance", where overcrowding reached a proportion that "no obtainable quantity of ventilation" could keep the air of the dwelling free from hurtful contamination, and where the houses, large but densely peopled, were "often without a span of court-yard, either front or back", and where the influence was so degrading, that to children "born under its curse" it must often have been "a very baptism into infamy". At this date some 2,000 cases of typhus were annually received into the London Fever Hospital; the subjects of this preventable fever being largely derived from localities such as have been indicated.

* John Simon, *Public Health Reports*, vol. i, p. 57. London, J. and A. Churchill.

But great changes as regards the circumstances of the housing of the poor and working-classes of London were already in progress; and, since then, most of the old typhus-haunts have been demolished. Great thoroughfares, bringing with them light and air, have been cut through dense aggregations of houses; courts and alleys have been opened out, their exposure to the public gaze often leading to their being altogether swept away; and a vast number of unhealthy dwellings have been demolished. Healthy dwellings have also replaced the unhealthy ones; and a large expenditure of money has been incurred in attaining these objects.

I have not found it possible so to arrange the various proceedings that have been taken in this direction as to trace their influence, in point of time, upon the reduction of the London typhus mortality; but I would note that between 1856 and 1887 a sum of over 14 millions sterling has been devoted by the Metropolitan Board of Works to measures tending almost exclusively to the destruction of unwholesome house property, and to the opening up of wide thoroughfares and breathing spaces in crowded parts of the metropolis. And this sum does not include over two millions spent on the Thames Embankments; neither does it take any account of outlay in the erection of wholesome dwellings to replace unwholesome ones—this latter work having been almost altogether undertaken by bodies and individuals having no access to the public purse. Thus, the Company of which Sir Sydney Waterlow is Chairman has, during the past twenty-five years, provided model-dwellings for 25,000 persons; some £440,000 has been spent within the City precincts in artizans' and labourers' dwellings, and such bodies as the Artizans' and Labourers' and General Dwellings Company, and the Peabody Trustees, have laboured in the same direction. Indeed, an intimate relation must be admitted to exist between these efforts and the disappearance of London typhus.

The same inference may be drawn from the circumstances of typhus in Liverpool, though the data are somewhat different. In 1840 no less than 40,000 of the inhabitants of that city lived in cellars—"dens into which sunlight never penetrated, and vile-smelling"; and it was well asserted that from such dwellings no amount of disinfection could banish typhus; whereas, on the other hand, the so-called "open courts" remained on the whole healthy, and this even in districts "smitten with fever". In 1843 it was found that no less than 55,534 of the inhabitants lived in courts nearly one-

third of which were closed at one end, so that no through-ventilation was possible; and such preliminary measures as were adopted, or in contemplation, for dealing with this serious condition received a fatal check when, in 1847-49, an already densely over-crowded city had to face the great immigration of a destitute population seeking to escape the miséries of the Irish Famine. Writing in 1866, Mr. Simon refers to a munificent beginning of work, for the purchase and destruction of dwellings "unfit for habitation" in Liverpool, where £100,000 was then being devoted to this purpose. But the work to be performed was a gigantic one; and, judged by the needs of that city, the progress was but slow. In 1882 Dr. Stopford Taylor reported that there still remained, in illegal occupation, 945 cellars in streets, and 165 in courts; and, even as recently as 1884, Mr. E. T. Stephens, a member of the Liverpool Town Council, in his evidence before the Royal Commission on the Housing of the Working Classes, referred to a report made in 1865, in which the then Medical Officer of Health spoke of an overcrowded group of streets in which the fever mortality was between two and three times as great as for the borough generally; and the witness added, "I can see no substantial improvement in the character of those streets since the date of that report"—twenty years ago. He further stated that there remained between 15,000 and 16,000 unsanitary dwellings in Liverpool. Another witness said that no less than "70,000 persons" in the borough still needed "healthful dwellings". And in the same year, Dr. Stopford Taylor, in dealing with the conditions of Liverpool typhus, recorded a number of instances which emphasise every phase of that misery which attaches to this disease in its relation to destitution amongst a people living under aggravated conditions of overcrowding and aerial filth.

The following are typical illustrations:—

"Margaret Carroll, age 11, 9 The Bower, 9 Court, Gore Street. The child has been about fourteen days ill. She lies on rags, in a state of great filth. The house is almost bare of furniture, and the people appear to be destitute. There are two or three other starved-looking children, and the mother wears only a single garment. The father, a strong, able-bodied man, tremulous with drink, volunteers the opinion that the child is suffering from want of food, and not from fever. He also states that he has been drinking hard for the last fourteen days, and considers that drink is more easily got than food."

"Matilda Crosby, aged 35, 1/13 Eaton Street. The poor creature in this filthy and abominable court has been five or six days ill. The room is so dark as to necessitate the use of wax matches to examine the patient. The room is foul and filthy, almost bare of furniture, and the other inmates are clothed with rags. Two or three half-naked children bear evidence of chronic starvation."

"Melaney, 6/1 Hughson Street. The intense fœtor of the room in which the sick people lay necessitated waiting outside until the window was opened. The occupants of the bed, which is about 3 feet by 6, were five in number, and all exceptionally filthy. The mother is a fish-hawker, and the fish were in a basket under the bed. The family appeared to be suffering from chronic starvation, and two members had fever. The upper rooms of the house were sub-let."

"Lizzie Thompson, aged five, 7/2 Rankin Street. The child has been sick about nine days. There is another child, aged about four years, who has recently been ill, and is now convalescent. The mother states that during the last two weeks the income of the family has been 4*s.* 6*d.* per week, and they have no other money nor relief. Her face bears the imprint of chronic starvation, and the surroundings confirm her story."

"Mumford, 1/13 Upper Frederick Street. Three persons, viz., the father and two children, are sick in this house, which is miserably furnished, filthy, and foul-smelling from the dirty skins and clothing of the inmates. The mother states that for seventeen weeks the income of the family, derived from the earnings of the father, has been 7*s.* per week. In reply to the inquiry as to how they were living, the mother replied, with some emphasis, 'they were not *living*, but *starving*.'"

But, gradually, a number of these conditions have been ameliorated, and within recent years even more substantial efforts have been made to cope with them. Amongst these I would mention the provision of healthy homes by the Liverpool Labourers' Dwellings Company, Limited; and the fact that, under a Sanitary Amendment Act of 1864, there had been expended up to the close of last year, and for the purposes in question, a sum of nearly £199,000; and an expenditure of £141,000 for works under Cross's Act, in the demolition of crowded areas and in the erection of wholesome dwellings. The precise effect of these several measures on the diminution of typhus cannot be traced, for it was not until 1877 that typhus became a separate entry in the borough records. But it deserves notice that, whereas in 1877 there were 204 such deaths, they fell to 96 in 1880; and though during the three years 1881-83 typhus again became prevalent, and caused 1,030 deaths, this was followed by a marked diminution and a record of only 71 fatal attacks in 1885, and of 45 in 1886; this latter being the lowest number ever yet recorded in any one year.

At this stage allow me to recall the fact that, as far back as 1849, Mr. Simon referred to back-to-back houses in connection with typhus. And he by no means stands alone in condemning the vicious system of so constructing dwellings as to deprive them once for all of means of through-ventilation. Confining my remarks on this point to two sources of information which have been prominent in connection with typhus prevalences, I would quote the opinion of the experienced Health Officers of Glasgow and of Liverpool. In his evidence before the recent Royal Commission on the Housing of the

Working Classes, Dr. Russell declared that "these back-to-back houses are the curse of Glasgow". In Liverpool they have been described by successive officers as "blind houses"; "houses only open to the front"; "houses back-to-back, and only one room deep"; or, more recently still, as "straight-up" houses. And they are never mentioned except in terms of reprobation, as violating the first principles of health, and as having a marked connection with the persistence of typhus.

Having regard to facts such as these, the Local Government Board have embodied in their model code of bye-laws as to new buildings, clauses requiring that every new domestic building shall have, both to the front and to the rear, a defined minimum amount of open space; and the Board make it a practice to refuse confirmation of such codes as do not embody this principle. And the Royal Commission of 1884 have also made a recommendation to the effect that a similar requirement should be enforced, even under the difficulties of the metropolis, where the cost of building-land is so excessive.

Thousands of lives have been sacrificed to the greed and ignorance under which houses have been aggregated together in disregard of the requirements of health. Millions have been spent in endeavouring to undo the mischief wrought by past generations in this respect; and if we consider alone the relation of typhus to that uncleanliness of air which results from the concentrated emanations of human bodies, we have abundant ground for demanding that a check shall be placed on all who, for one motive or another, would interfere with that free movement of air around our future dwellings which is so essentially necessary for the prevention of disease.

III.—Enteric Fever.

During the early part of the half-century we are considering, many able workers were engaged in research which had for its object to determine whether the "fever" mortality of this country was due to one or more distinct and separate diseases. English, French, and German pathologists had alike noticed the special lesions which we now know to be pathognomonic of enteric fever; and in Glasgow the fact was early recognised that two altogether different eruptions were met with in the "fever" wards. Much the same conclusions were being arrived at in the United States. In this country, where both typhus and enteric fever were commonly prevailing side by side, it was not until 1840 that it was made clear that the rose-

spot eruption and the intestinal lesion of Peyer's patches were characteristic of enteric fever, and incompatible with true typhus. In that year the late Dr. A. P. Stewart, in a masterly description, indicated the more prominent differences between the two fevers, declaring that their characters were "so marked as to defy misconception". Besides which, he went so far as to point out that there were differences in the circumstances under which the two diseases occurred.

But this doctrine of the non-identity of the two diseases was by no means universally accepted, and it remained for Sir William Jenner, as the result of his researches, published between 1849 and 1853, to clear up all further doubt on the subject. And he did more. He showed conclusively that the two diseases by no means necessarily prevailed together; that the one did not communicate the other; and, above all things, he made it clear that they depended on different causes.

Accurate observation of the symptoms, natural history, and pathology of disease has ever been at the foundation of progress in preventive as in curative medicine; but there probably never was an occasion when so much depended on correct diagnosis as when these two diseases were once for all dissociated from each other. So long as one "fever" was alone recognised, the fatality which on the one hand attached to our jails, camps, and city tenements, where misery and excessive crowding obtained, was confused with that which, on the other hand, occurred alike in the mansions of the wealthy—to which sewer-air had by ill-contrived arrangements been laid on—and in scattered rural districts, where the need for the proper disposal of excreta was not yet recognised. This confusion of the two diseases barred the way to progress in preventive medicine; for the causation of that which seemed but one disease, and which yet appeared under such widely differing circumstances, long defied detection. But, when once the distinction was made, the road was clear. The remedy needed for the prevention of the one disease had little or nothing in common with that for which the other called; and whilst the removal of conditions favouring that form of filth which comes of an air laden with the accumulated emanations of a crowded and destitute people rid our towns of typhus, it was found necessary, for the prevention of enteric fever, to cope with the varying circumstances under which poisoning by means of the excreta of populations could operate, either through water and food consumed, or through air breathed. Medical science having once indicated the directions in which remedial measures were needed, the work of prevention could be intelligently pushed forward; and

I may truly say that its rapid advance, and the widespread benefits resulting from it, have been largely due to the impulse given by the skilfully discovered differentiation of these two continued fevers.

It was not until 1869 that enteric fever and simple coninued fever were separated, for the purpose of death registration, from the general body of "fever" deaths. From that date the records concerning them are distinct; but in any reference to them before 1870 they must be dealt with collectively.* In 1869, the number of fatal attacks from enteric and simple continued fever in England and Wales was 13,967, and in the years that immediately followed but little diminution is to be observed in their fatality. During the five years 1876-80 the average annual number of deaths had fallen to 8,657, and in the period 1881-85—and this notwithstanding a steady increase in population—it had fallen to 6,671. Or, comparing the annual death-rate per thousand living in 1869 and in 1885, I find that they were 0·39 and 0·17 respectively in the case of enteric fever, whilst for simple continued fever they were 0·24 and 0·02 respectively—a sufficiently marked decrease in both cases.

Before the period for which these comparisons can be made, it had been shown, by means of reports and otherwise, how urgently our towns and cities stood in need of proper means of drainage and water-supply. In 1849, Mr. Simon pointed out, as regards the City of London, how often a cesspool was to be found "actually within the four walls of the inhabited house; the latter reared over it, as a bell-glass over the beak of a retort, receiving and sucking up incessantly the unspeakable abomination of its volatile contents"; and it is notorious how generally wells were resorted to which were sunk in a filth-laden soil, and how even public water-services were so contrived as to give a minimum supply with the maximum amount of trouble, and often with a large share of danger. These and kindred matters were shown to be prevailing on a wide scale during the period in which the General Board of Health instituted its long series of public inquiries. And, again, after Mr. Simon had for nine years held office as medical officer to H.M.'s Privy Council, he still found it necessary continuously to advise—1st, that, by appropriate structural works, all the excremental produce of the population shall be so promptly and so

* With reference to the interpretation to be put on the term "simple continued fever", see Dr. Longstaff's paper in the *Proceedings of the Epidemiological Society*, New Series, vol. iv, on "The Seasonal Prevalence of Continued Fevers in London."

thoroughly removed that the inhabited place, in its air and soil, shall be absolutely without fœcal impurities; and, 2nd, that the water-supply of the population shall be derived from such sources, and conveyed in such channels, that its contamination by excrement is impossible.* And, as regards enteric fever, he went on to show—by reference to Dr. Buchanan's well-known report on the results gained in various parts of England, by works designed to promote public health—how remarkable had been its diminution since the execution of efficient sanitary works. To quote a few examples only. Comparison between the enteric fever annual death-rate per 10,000 people living after the execution of such works, with that for periods which had preceded them, showed that there had been a fall at Merthyr Tydvil from $21\frac{1}{3}$ to $8\frac{2}{3}$, at Croydon from 15 to $5\frac{1}{2}$, at Ely from $10\frac{2}{5}$ to $4\frac{1}{2}$, at Penrith from 10 to $4\frac{1}{2}$, and at Stratford from $12\frac{1}{2}$ to 4.

In the meantime another great advance had been made. Although it had long been an accepted doctrine that filth was in some way related to the production of disease, yet but little was known, during the earlier part of the present reign, of the actual means by which conditions of filth affected health; and many of the works which had been carried out, and which had led to results such as I have referred to, were primarily adopted for the prevention of nuisance. But it had now come to be regarded as the result of etiological research that the great potency of filth for mischief lay in the fact that it formed a nidus for the multiplication and spread of definite and specific contagia of disease; and it is largely to the apprehension of this important principle that the vast expenditure for sanitary works which has characterised the past quarter of a century has been due.

But even in places where works aiming at the safe disposal of the solid and liquid refuse of populations, and at the provision of wholesome water-services, had been carried out, periodic, and at times large, outbreaks of enteric fever still recurred, and it remained for those engaged in epidemiological research to discover those more obscure channels through which the infection of this fever was at times conveyed to man.

In May 1858, Dr. Michael W. Taylor wrote a paper in the *Edinburgh Medical Journal* on "The Infection of Fever by Ingesta", in which he suggested that an outbreak of what we know now to have been enteric fever was due to specific contamination of milk, derived from a dairy where the disease prevailed. This report received but little publicity.

* Ninth Report of the Medical Officer of the Privy Council, 1866.

It was followed in 1870 by a report from Dr. Ballard, then Medical Officer of Health for Islington, who had succeeded in tracing a prevalence of the same fever to the distribution of a particular milk-service. Within less than a semicircle of a quarter of a mile radius, 168 cases of that disease occurred within ten weeks, and the occurrence could not be explained by any of the ordinarily recognised means with which the spread of that disease was then known to be associated. Gradually a suspicion arose that there was some connection between the outbreak and the use of milk from a certain dairy; but the idea was still a novel one, and it was only after further exhaustive investigation that Dr. Ballard was at last in a position to demonstrate that connection as the true explanation of the disease. Out of 140 families supplied from the dairy in question, there were no fewer than 70 attacks, with 30 deaths, the disease picking out, as it were, its victims from the homes of the dairy customers living widely apart, in different streets and squares, and showing a marked incidence on large consumers of milk. And, finally, it was ascertained that water from a tank in direct communication with some old drains had been used for dairy purposes. The report in which Dr. Ballard announced the discovery he had made was, at the time, characterised as a masterpiece of medical logic, and it is worthy of being remembered on such an occasion as this as forming one of the most striking and important episodes in the history of preventive medicine in its application to enteric fever and other infectious diseases.

In 1873 there followed the admirable report by Mr. Netten Radcliffe and Mr. W. H. Power, on a wide-spread epidemic of the same disease in West London. It was traced to the use of milk from a large London dairy, and the ultimate conclusions arrived at made it practically a certainty that the milk in question was "infected with enteric fever material", and that at the particular farm from which it was derived the water used "for dairy purposes contained excremental matters from a patient suffering from enteric fever immediately before and at the time of the outbreak."

Numerous instances of the same sort were gradually brought to light, and there were soon strong indications to show that milk formed a specially favourable vehicle for the propagation and distribution of the contagion of this and of other diseases. Outbreaks of enteric fever have since, in the same way, been traced to the use of cream; and, later on, the occurrence of infectious diseases as the result of the use of ice and iced creams prepared with materials containing the specific

poison of disease, went to prove that the process of freezing did not suffice for the destruction of the contagia in question.

And, more recently, the frequent occurrence in this country of enteric fever in connection with milk-supplies has led observers to consider how far it is possible that this affection may have a starting-point in some bovine disease; whilst Dr. James F. Allen, Medical Officer of Health to the Corporation of Pietermaritzburg, has gone so far as to contend that his South African experiences warrant him in the conclusion that there is a disease in cows and calves allied to that of enteric fever in man, and he holds that there are strong grounds for believing that enteric fever may be produced in man by means of milk from cows suffering from the corresponding bovine disease. The subject is an important one, and it is sure to receive the careful consideration of epidemiologists.

In the meantime a new channel for the dissemination of enteric fever had been discovered. In 1873, Dr. Blaxall, during the course of an inquiry into an outbreak of enteric fever at Sherborne, had found that, during intermissions in a public water-service, foul matters had facilities for getting into water-mains; and in the following year Dr. Buchanan was able to show conclusively that a localised spread of enteric fever in Caius College, Cambridge,* had been mainly due to the suction of specifically contaminated air and other matters into the water-pipes of a particular portion of the college during intermissions in the water-service, and to the subsequent mingling of this air with the water used for drinking purposes. And late in the same year I had occasion to trace an epidemic of the same disease in Lewes to a precisely similar cause. In this instance a wide-spread diffusion of the disease took place, and no less than 486 attacks, with some 40 deaths, resulted before the mischief could be checked by the substitution of a constant for an intermitting water-service. Since that date the conditions under which intermitting water-services become dangerous to the public health have been very generally recognised, and, as a consequence of investigations such as I have referred to, additional measures for the prevention of enteric fever have become practicable.

Year by year evidence was accumulating to show how great was the potency for mischief of even minute portions of the specifically diseased evacuations of enteric fever patients when such matter was placed under circumstances

* Report of the Medical Officer of the Privy Council and Local Government Board. New Series, No. II, 1874.

favourable to its multiplication and its communication to man. One of the most striking instances of this potency occurred in the epidemic of enteric fever which I had to investigate, in 1879, in the wide area over which the Caterham Water Company distributed their supply.* In that case 352 cases of enteric fever took place at Caterham, Redhill, and certain intervening places, some 200 of the earlier cases being traced directly to the use, during a particular fortnight, of water derived from a deep well, in which a man suffering from that disease had been employed, under circumstances which left no possible doubt that his excreta had got access to the well-water. In this case it was estimated that, after all possibility of further pollution had come to an end, no less than 1,861,000 gallons of water had been pumped from the well during the fortnight in question; and Dr. Buchanan, in dealing, in his annual report for 1881,† with the question of the amount of specific pollution which might suffice to render a potable water dangerous, showed that, in this case, the water could have contained no such amount as one grain of excremental matter per gallon; and he brought forward considerations tending to prove that an amount of specifically polluting matter so infinitesimal in quantity as to be altogether beyond detection by chemical analysis was fully potent for mischief.

It may, indeed, now be claimed as one of the most settled principles of preventive medicine that all excremental filth, irrespective of its quantity, must be regarded as potentially noxious to man; and that, for the prevention of such a disease as enteric fever, "the one essential is cleanliness." Measures based on this principle have already done much to diminish the amount of sickness and death which were formerly the result of enteric fever. But much still remains to be done, and it will be the duty of epidemiologists to continue the study of the etiology of this disease, and the detailed history of its occurrences, with a view of learning whether there are not still undiscovered means by which its contagium becomes communicated to man.

IV.—Scarlatina.

Scarlatina is a disease the mortality from which in England and Wales has, from time to time, exhibited very

* Annual Report of the Medical Officer of the Local Government Board for the year 1879. [C.—2681—I], 1880.

† Annual Report of the Medical Officer of the Local Government Board for the year 1881. [C.—3337—I], 1882.

extensive fluctuations. The first decennial summary of the Registrar-General showed that during 1851-60 the deaths at all ages from this disease amounted to 88 per 100,000 living; in the next decennial period, 1861-70, the rate had risen to 97; but during the ten years 1871-80 there was a substantial diminution, the rate standing at only 72. Since 1878, when the rate was 75 per 100,000, it has fallen year by year without interruption, until, in 1886, it was in the proportion of only 17 per 100,000 living. There is, perhaps, no disease concerning which it can be said with less certainty that diminutions year by year in its fatality foretell a permanent lessening in its prevalence than is the case in regard of scarlatina. Not only do different outbreaks vary very greatly as regards mortality, but the epidemic prevalences tend to occur in cycles; and an abatement extending over a few years has been known to be followed by a wide and fatal diffusion of the infection. And not only so, but the more recent diminution in the amount of fatal scarlatina may be in noteworthy part matter of diagnosis; thus, whereas many deaths which are now registered as due to diphtheria were formerly returned under the heading of scarlatina, or scarlet fever, the difference between the two diseases is year by year becoming more recognised, and the fatal attacks are not confused under one name to the extent that was formerly the case.

But, after making every allowance, there remains the important fact, that ever since the decennial period 1861-70 there has been a very general and fairly steady diminution in the fatality of scarlatina in this country, until, in 1885, the rate of death from that cause was less than a quarter of that which formerly prevailed; and it is impossible not to regard so long-continued and marked an abatement as an indication that some of the means conducing to the spread of this very fatal fever have been materially restricted.

And to those who are engaged in the study and practice of that branch of medicine which has prevention for its main object, the fact is the more satisfactory, because it must be admitted, with respect to scarlatina, that the great saving of life indicated in the statistics quoted has, in the main, been brought about by epidemiological research, and by the resulting intelligent apprehension on the part of the public of the methods by which the specific contagion of this disease may be multiplied and is communicated to man. Scarlatina has no such relation to works of water-supply and drainage as have those other preventable diseases of which enteric fever is a type, and such effective action as has been taken

to check its ravages has been based on a growing scientific knowledge of the etiology of the disease.

Up to a comparatively recent date all the more generally adopted means of prevention were in one way or another included within such processes as had to do with the isolation of the sick from the healthy, and the disinfection or destruction of such articles as were known to act as vehicles for the infection. And it may be recorded with satisfaction that, apart from such measures as are now adopted by private individuals, many public bodies have come to regard it as a duty to take such action as lies in their power to stay the diffusion of this disease. Thus, several hundreds of the Sanitary Authorities of England and Wales have provided their districts with isolation hospitals, and with means of disinfection; and measures have been initiated under the auspices of the Education Department of the Privy Council with a view of staying the dissemination of infection through the agency of elementary schools.

But a record of some of the principal phases by which our knowledge of scarlatina has from time to time been added to will go to show that, stage by stage, we have had to deal with newly discovered conditions favouring the spread of the infection—conditions lying beyond the limits of the ordinarily recognised means of personal intercommunication; and, also, that we have now arrived at a critical point in the natural history of this disease—a point at which it is evident that we shall, in the future, have largely to rely on the skilled co-operation of those who devote themselves to the study of disease in the lower animals. I believe that Dr. Michael W. Taylor, of Penrith, was the first to record an occurrence of scarlatina in connection with a milk-service. He did so in August 1870; the history which he gave being that of a prevalance of the disease in Penrith, where the customers of a certain dairy were so largely attacked that "there was only one house containing children, which continued intercourse with the dairy to the end, that escaped the hurtful influence." It was ascertained that an attack of scarlatina at the dairy had preceded the general outbreak; that the dairyman's wife, who was nursing her sick child, at times milked the cows; that the milk was brought from the byre into the infected dwelling before it was given out for sale; and that a large proportion of those attacked could have had no communication with the dairy except as the result of the consumption of the milk issued from it. Gradually similar experiences were recorded by other observers, and sixteen instances in which scarlatina

is believed to have followed on the distribution of special milk-services were brought under the notice of the International Medical Congress of London in 1881; the evidence implicating the milk being, in a majority of instances, of the most convincing character.

It thus became a gradually accepted doctrine that scarlatina could be, and largely was, communicated through the agency of milk, and in a majority of instances there was recorded the belief that the specific contamination of the milk had been directly derived from some antecedent case in the human subject. But there remained a certain proportion of cases in which this means of contamination could not be traced; and when, in '1882, Mr. W. H. Power reported on a scarlatina outbreak which had coincided with the distribution of a special milk-service in certain districts of London,* he expressed himself as satisfied "that it was practically out of the question that the milk had become infected in any of the commonly believed ways that require a human subject as the source of infection." And, further, he brought forward a series of facts, which went to show that many of the circumstances connected with milk outbreaks of scarlatina generally accorded rather with some cow-condition than with the theory of milk-infection through any human agency. And he formulated certain positive indications as to the nature of the relation between infectivity of milk and ailment of cows, "indications which arise as soon as there is seen to be probability that the milk", in a given case, "was infected when it left the animal." These views received support, from the circumstance that Dr. Klein, experimenting, in view of these considerations, with human scarlatinal material, was able to transmit to the cow a distinct disorder, and to further transmit it from the cow to other animals.

So matters remained until 1885, when a wide distribution of scarlatina in Marylebone and other London districts was traced to several separate milk-services, derived from a Hendon farm, under circumstances that induced the Local Government Board to instruct Mr. W. H. Power to investigate and report upon the subject. The conclusions which were arrived at were not long since brought under the consideration of the Epidemiological Society, and they are calculated to have a most important influence in the future of sanitary medicine as applied to the prevention of this disease, which

* Report of the Medical Officer of the Local Government Board for 1882. [C.—3778—I], 1883.

still annually destroys some 10,000 to 12,000 lives in England and Wales.

Mr. Power's report* demands the careful and detailed study of all who are interested in the etiology of scarlatina; but on this occasion I can only refer to its more salient features. The several prevalences of scarlatina were conclusively shown to have been brought about by the use of the milk from the Hendon farm; it was quite impossible to connect the means by which the milk had received its infection with any pre-existing scarlatina in the human subject; and it was clear that it was the milk of certain cows—and of those cows only—that had to do with the diffusion of the disease.

During the course of his 1882 inquiry Mr. Power had expressed the belief that there existed grounds for associating the infection of the milk with some parturient condition in the cow; and in the 1885 epidemic the scarlatina was found to be related, in point of time, to the arrival at the implicated farm of three recently calved cows, and to the use of their milk whilst they were suffering from an ailment at first sight unimportant, but which turned out to be a specific contagious and transmissible disease, which had followed in the process of calving.† And, further, when inoculation was made into calves, either directly from the diseased products of the affected cows, or by means of sub-cultures of micro-organisms connected with these substances, a disease was produced which, to quote Dr. Buchanan, had "unmistakable affinities in its local phenomena with the Hendon cow disease, and in its constitutional manifestations with scarlatina in the human subject." The cow disease was ascertained by Dr. Klein to be characterised by the presence of a definite micro-organism in the affected tissues; and both the blood and tissues of ordinary human scarlatina have been found to exhibit organisms identical in their morphological characters and pathological properties with those which had been found in the case of the Hendon cow.

Summarising the information available on this subject, Dr. Buchanan, in his last report, states as follows:—"1. The disease in man and in the cow alike is characterised by closely similar anatomical features. 2. From the diseased tissues and organs of man and cow alike the same micrococcus can be separated, and artificial sub-cultures be made

* Report of the Medical Officer of the Local Government Board for 1885. [C.—4844—I], 1886.

† Annual Report of the Medical Officer of the Local Government Board for 1886. [C.—5171], 1887.

from it. 3. These sub-cultures, no matter whether established from man or cow, have the property, when inoculated into calves, of producing every manifestation of the Hendon disease, except sores on the teats and udders—no doubt for the reason that the milk apparatus is not yet developed in calves. 4. But—and this I have from Dr. Klein's later observations while this report is in preparation—the sub-cultures made from human scarlatina and inoculated into recently calved cows can produce *in those cows*, along with other manifestations of the Hendon disease, *the characteristic ulcers on the teats;* ulcers identical in character with those observed at the Hendon farm. 5. The sub-cultures, established either from the human or the cow disease, have an identical property of producing in various rodents a disease similar in its pathological manifestations to the Hendon disease of cows and scarlatina in the human subject. 6. Calves fed on sub-cultures established from human scarlatina obtain the Hendon disease. 7. Children fed on milk from cows suffering under the Hendon disease obtain scarlatina. . . . . The above combine, I think, to form a mass of evidence to show that the Hendon disease is a form, occurring in the cow, of the very disease that we call *scarlatina* when it occurs in the human subject."

Here, indeed, we have a chain of circumstances linked together with the scientific accuracy which is typical of that form of research which has, at various stages, led to the identification of disease in man with definite micro-organisms, and which forcibly calls to mind the keen forethought of those who in the early days of the Epidemiological Society laid it down as a leading principle that its "objects" should include "a knowledge of epizootics, or epidemic diseases among the lower animals, whether contemporaneous or not with such diseases in the human family."

V.—Diphtheria.

Diphtheria is so often regarded as having certain affinities with scarlatina, that some consideration of its history may appropriately be included in this place; besides which, it is a disease which has the more interest for us because such study as it has received in this country has been carried out during the Victorian Era.

Diphtheria is known to have been epidemic on the continent of Europe during the 16th and 17th centuries; and, according to Sir John Simon, it was prevalent in England during the two middle quarters of the past century, there

being also, later on, record of certain small local epidemics. But it was not until the summer of 1855 that the disease received much notice in this country. At that date an extensive epidemic prevailed in Boulogne, many English residing there being attacked. In September of the same year it was recognised at Launceston, Cornwall. In July 1856 it appeared in Lincolnshire; in 1857 and 1858 it was manifested in many parts of England; and from that date it has been an almost continuous and, generally speaking, an increasing cause of death amongst us.

In 1859 the disease was made the subject of a somewhat extensive inquiry by the Medical Department of the Privy Council; it was regarded as distinct from scarlatina, prevailing mainly, but by no means exclusively, in places either marshy or otherwise damp, and on cold, wet clay soils; it was distinguished by certain characteristic neuroses; and, though no definite connection could at that date be traced between the disease and any affection of the lower animals, it was reported by Dr. Greenhow to have been found prevailing simultaneously with certain forms of disease affecting the mouth, lips, nose, etc., of certain lower animals, such as cattle and horses.

The etiology of the disease was, subsequently to that date, matter of frequent inquiry, such investigation as was made being, however, largely limited to single outbreaks. But, in 1880, Dr. Airy was instructed by the Local Government Board to make a somewhat comprehensive inquiry into the circumstances of new appearances of the disease in various parts of England and Wales, and some of the conclusions at which he arrived are as follows.

Whilst admitting that the disease was largely spread from person to person, especially amongst the young, Dr. Airy was much struck by the failure, in a great majority of the cases investigated, to trace any cause to which the several outbreaks could with any probability be ascribed; a failure which could hardly have occurred if it were the case that communication, direct or indirect, with previous human diphtheria were a necessary or even a habitual condition of a new outbreak of the disease; and he, in this connection, recalls the opinion of Dr. Burdon Sanderson, who, in 1859, reported that "the circumstances were frequently such as to shut out even the possibility of personal communication." Bearing upon this point, it is also noteworthy that diphtheria operates more frequently in sparsely populated country districts than it does in densely populated towns, where the facilities for intercommunication are at their greatest. Dr. Airy

further reported that he had found appearances of the disease to be far more frequent in October than in any other month; that it had prevailed more frequently on clayey than on sandy soils; that it had a preferential incidence on families that had shown liability to throat-affections; and, taking note of the whole of the circumstances which had come under his notice, together with the discoveries of Oertel and Hüter, he held that nothing short of a living organism capable of infecting milk and air, and of being transmitted by means of wind currents, could fully account for all that he had met with.

In many respects Dr. Airy's inferences were confirmatory of those of previous observers; and, amongst other things, he gives some support to the views which I brought under the notice of the Epidemiological Society in 1878, and in which I endeavoured to show, from the history of diphtheria, that there were grounds for believing that certain infectious diseases were not exclusively self-propagating, but did at times arise independently of antecedent cases in the human subject. The following are the passages in which I especially dealt with this subject.

"In isolated districts, and in houses situated at times many miles away from other habitations, and in some instances lying in lonely spots among mountain ranges, where a visit to or a visit from the nearest town or village would be a circumstance too important to be forgotten, I have met with instances of what appeared to me to be nothing more than a simple inflammation of the throat, at times so trivial that it has passed all but unnoticed, and yet it has led, by transmission through other persons, to cases of well-marked and severe diphtheria. The first attacks have often happened in children whose former movements could apparently be traced with the strictest accuracy; they have occurred under circumstances which did not appear to admit of previous infection; and it has been difficult to interpret their occurrence except on the supposition that in some way they have arisen independently of prior cases. With regard also to the well-marked attacks of diphtheria to which they seemed to give rise, all other sources of infection could be excluded with a degree of certainty rarely to be met with. And as to those first affected, whose cases appeared to be earliest in a series which led up to attacks of well-marked diphtheria, it has more than once happened that they were children in whom so-called 'sore-throat' was a common affection, and whose fauces, when examined, exhibited a loss of tissue indicative of former throat-attacks."

"I have stated that instances such as these have been met with in connection with outbreaks of diphtheria, and it may therefore be inferred that the early cases were merely mild attacks of that disease, the poison of which had been received in some unascertained method; but every one of these instances occurred in persons living miles away from the apparently very limited infected localities, which in some cases were mere hamlets, and this source of error seemed to be one which could unquestionably be excluded. And, during the same investigations, I have noticed the fact that, over an area of some miles around the district in which genuine diphtheria was prevailing, there existed, prior to, as well as during, the diphtheria epidemic, numerous instances of sore-throats which, so far as an examination of the patients was concerned, in every way resembled the early cases above referred to, and yet which gave no indication of being infectious; sore-throat being, in fact, in the surrounding district a prevailing ailment. And I have hardly been able to refrain from drawing the conclusion that conditions very similar to those under which genuine diphtheria was epidemic in a limited district obtained, and had obtained, before genuine diphtheria was anywhere seen, over a wide area around the immediately infected locality; and that these conditions, leading to a somewhat general predisposition to simple and apparently non-infectious sore-throat, had, further, probably under somewhat modified circumstances, tended at certain points to produce an affection capable of putting on the property of infectiveness, which thus lead to the transmission of the disease in a distinctly communicable form to others."*

"If this be a correct interpretation of the circumstances related, it would appear to indicate the possible occurrence of what may perhaps be looked upon as *the progressive development of the property of infectiveness.* And if the contagia of the acute specific diseases do—as has been suggested by more than one observer—belong to the vegetable world, I knew no grounds for refusing to believe that organisms capable of producing a minor and an uncommunicable disease in particular stages of their growth may, in other stages of their growth, or in the course of their subsequent development, become capable of producing a major disease communicable from person to person; the affair being essentially one of soil. This is not at all a

* In this connection Dr. Gresswell's paper on "Diphtheria and its Liability to Recrudescence" deserves study. (See *Transactions of the Epidemiological Society*, vol. v, 1885-86.)

question of the development of a living organism out of matter independently of antecedent life, but merely the production, by means of a process of evolution, of that which gives to an already existing organism that property by which it becomes infective—a property which it may perhaps lose directly it is deprived of the circumstances which favoured its development, in much the same way as special characteristics may be artificially developed in higher plant-life, and be as easily lost again."

Since I wrote the above, in 1878, circumstances have arisen which will make it necessary in the future, in seeking for antecedent infection, to look to the possibility of such infection being derived from the lower animals. But, apart from such consideration, additional evidence has, I think, been forthcoming in favour of the opinion that the contagium of diphtheria is often characterised by a want of stability when compared with that of other contagia, such as small-pox, and that it does affect the human subject in a form which is capable both of progressive development and of relapse in point of type, according to the circumstances of its surroundings.*

But whilst diphtheria may be regarded as not necessarily dependent in its origin on an antecedent human case of the same affection, yet its epidemic spread is largely due to personal intercommunication, and this especially amongst children between the ages of 3 and 12 years: an age incidence that necessarily involves the question of attendance at elementary schools. Numerous investigations have been made, which show the marked influence of school attendance and of age as a cause of diphtheria spread. In an outbreak on which I reported to the Local Government Board early in 1877, and in which no less than 170 diphtheria attacks had occurred in a population of 3,309 living in Great Coggeshall in Essex, 7·1 per cent. of the children between 0-15 years were attacked; between 0-3 years the attacks were at the rate of 4·0 per cent.; from 3-12 years they were 8·4 per cent.; and from 12-15 years 4·8 per cent.; the incidence of the disease upon children from 3 to 12 years being about 50 per cent. greater upon those known to have attended school than upon the

* In his Morton Lecture, delivered before the Royal College of Surgeons on Nov. 11, 1887, Sir James Paget, in referring to the resemblances between innocent and cancerous tumours, and to the examples of intermediate forms occupying the space between the two extremes, refers to this "gradation" as affecting "all groups of diseases". His remarks on this point deserve study in this connection.

remainder.* Much the same incidence of diphtheria on school-going children of the ages specified has been noted elsewhere; and at times school attendance has appeared to have constituted a very important factor in the recrudescence of the disease, and in imparting to it special characteristics. Mr. W. H. Power's report, in 1882, on an epidemic in Pirbright is very instructive from this point of view.†

"On four successive occasions while the village school was open, well-marked diphtheria occurred among the scholars . . . .; and this although the school premises were free from recognisable sanitary defect; and although the school was not, after its first closure, reopened until the disease had seemed extinct in the parish, and careful measures of disinfection had been used." But, beyond this, the reopening of the school appeared on each of four occasions to be responsible for giving a serious quality to the resulting diphtheria, and this although the assembling of the children only took place some weeks after the disease had apparently subsided, and when skilled investigation, subsequently carried out, could only detect the existence of certain neuroses amongst convalescents and a few attacks of so-called "colds". On each occasion, and "with startling suddenness", multiple cases of diphtheria, and fatal diphtheria, promptly occurred, and this without the intervention—except perhaps at one period—of cases of intermediate gravity. Indeed, there are grounds for believing that the aggregation together of cases of diphtheria and of allied throat-affections under circumstances such as those obtaining in elementary schools constitutes one of the conditions under which a form of disease of particular potency for spread and for death may be, so to speak, manufactured.

And, as far back as 1878, during an epidemic of diphtheria in North London, Mr. Power connected the incidence of the disease with the distribution of a certain milk-supply; and this under circumstances which raised the question whether the cow herself might not have been concerned in the infection of the milk.

In 1883, again, evidence that may be regarded as conclusive was forthcoming to show that diphtheria was capable of being brought about by the distribution of milk from a particular dairy; and in referring to two such outbreaks, investigated respectively by Mr. Power at Hendon and Dr.

* Dr. Downes's "Contributions to Diphtheria", in *The Practitioner*, vols. xxxi and xxxii, are of great interest in this connection.

† Report of the Medical Officer of the Local Government Board for 1882. [C.—3778—I], 1883.

Parsons at Devonport, in 1883, Dr. Buchanan says, in his report for that year, that "at Hendon it was difficult to refuse this explanation of the facts, since the milk at the very time it was operative for harm . . . exhibited a peculiar ropiness and unpleasant taste, which caused some of its habitual customers to return it to the dairy; and for these phenomena no condition about the dairy or its utensils could be regarded as responsible." Here it was again foreshadowed that diphtheria in man might have relation to some disease in the cow; and it is well at this stage to recall the fact that different observers had, even before this date, expressed suspicion that the contagium of the disease might be due to conditions affecting the brute creation, and that others had associated it with fungoid growths of a vegetable character.

Amongst the latest developments of these views I would call attention to several reports. First, there is one presented by Dr. Simpson to the Local Government Board in 1885, on diphtheria in and about Shaftesbury. In it he refers especially to an outbreak, in three successive Novembers, in an old, dilapidated house, standing in a water-logged soil, where the walls of the ground and upper floors were found to be wet and covered with moulds, and the woodwork rotten. "There could scarcely", he adds, "be a more favourable soil for the growth of lower forms of vegetable life."* Next, there is the contribution by Dr. George Turner on his experiences of diphtheria, especially in its relation to the lower animals, embodied in Dr. Buchanan's report for 1886. And, lastly, in the same volume, that by Mr. Power on diphtheria at York Town and Camberley. In this latter case the outbreak was ascertained to have been due to milk which had clearly received its infectiveness before it left the farm where it was produced; but the incidence of the disease on the better class of consumers, as compared with the cottagers and trades-folk, was as 29·3 to 6·2; and, it having been ascertained that this excess of disease was brought about apart from the use of cream, it became apparent that the *amount* of milk consumed, involving, as it did, *inter alia*, differences of *number of opportunities* for infection, had been all-important in bringing about this result. And, again, the better-class people stored their milk, and did not, like the other class, use it directly after purchase; and thus an opportunity was given in the former case for the development and multiplication of any infective

* See also a paper by Dr. Michael W. Taylor, in *Transactions of the Epidemiological Society*, Session 1886-87.

material present in it, and so of giving to the milk a superior ability to convey infection.

In this case there was a complete absence of evidence as to infection of the milk directly by human agency, or owing to unwholesome conditions at the farm; but, as regards disease in the cow, it could only be stated that the outbreak had been related in point of time to the process of calving and "cleansing" in two of the cows belonging to the dairy-farm, and to the existence in another cow of a "scab or crust", not unlike those which at a later stage of their malady had been observed to replace ulcers on the udders of certain Hendon cows referred to in connection with scarlatina.

The etiology of diphtheria is thus still involved in considerable obscurity. By some the disease is regarded as having distinct alliance with scarlatina, there being no clearly defined boundary-line dividing affections which, in their more marked forms, present obvious differentiating characteristics; and the views which were expressed by Dr. Franklin Parsons, in the paper which he read before the Epidemiological Society during the Session 1883-84, are worthy of careful consideration in this connection. It is also interesting to note that, whilst in the three quinquennial periods ending 1885 there was a diminution in the scarlatina death-rate for England and Wales, there was a coincident increase for that of diphtheria. Thus, for the periods 1871-75, 1876-80, and 1881-85, the scarlatina death-rate per 100,000 living in England and Wales was 75·8, 67·9, and 43·4 respectively, whereas the corresponding diphtheria rates were 12·1, 12·2, and 15·6.

How far this may be a question of nomenclature brought about as the result of improved diagnosis it is difficult to say; but there can be but little doubt that, quite apart from any such consideration, there is evidence to show that, for some cause or another, diphtheria has, during recent years, exhibited a tendency to increase, and to acquire a more permanent stability of type amongst us. Yet there is reason for some confidence that the study devoted to the etiology of the disease, especially during recent years, has tended towards the ultimate discovery of those measures by means of which a substantial check may before long be put upon the increasing fatality of diphtheria.

VI.—Phthisis.

I believe that the first comprehensive report relating to the prevention of pulmonary phthisis which was made during the present reign was that which Dr. Greenhow prepared, in

1858, and which was submitted by Mr. Simon to the President of the General Board of Health. In addressing the President, Mr. Simon stated that "pulmonary affections, including phthisis, cause very nearly a quarter of the annual mortality of England. Every 100,000 of our population yields on an average 552 annual victims to this deadly class of disorder." He also expressed the opinion that pulmonary phthisis then killed, on an average, in England more than 50,000 persons a year, in addition to some 8,000 others, whose deaths were set down to such causes as scrofula and tabes mesenterica; and he urged the need for further study of this disease and its causes, not alone because of its cruel mortality, but because of the tendency of tubercular disease to transmit itself from parent to child; and he added, that "whatever tends to increase tubercular disease among the adult members of a population must be regarded as assuredly tending to produce a progressive degeneration of race."

The investigation which was made at that time went to show, amongst other things, that "in proportion as the male and female populations are severally attracted to indoor branches of industry, in such proportion, other things being equal, their respective death-rates by phthisis are increased"; that imprisonment and barrack-life, under the then circumstances, exerted a considerable influence in inducing the disease; that inadequate ventilation, both in and about dwellings, was a factor in the disease; and that high phthisis death-rates were associated with certain branches of industry, in which sustained irritation of the air-passages was brought about, as, for example, by the diffusion in the atmosphere of the work-places of particles of materials used in the manufacturing processes, and in which the operatives were exposed to marked vicissitudes of temperature.

This inquiry was continued under Mr. Simon, after he was appointed medical officer to the Privy Council; and in his 3rd and 4th annual reports he published the details of an investigation, the result of which had for its object to elicit the influence of certain branches of industry on the production of phthisis. The summary of this inquiry, as set out in the 4th report, refers to the following as some of the more important influences at work in producing the excess of disease and death in question.

In the first place, and generally affecting most of the callings and pursuits, the unwholesomeness of work-rooms, through want of ventilation, stood out prominently—this evil obtaining "from the humblest cottage industry even up to

the highest development of our factory system". Adding to the mischievous results of this lack of ventilation were conditions such as the vitiated products of combustion following on the use of artificial light; the methods of heating, other than by open fire-places; and the failure to rid the air of injurious dust and other matters. Another cause of phthisis was found to be the sedentary nature of many employments, and often the constrained bodily posture. Numerous details in connection with various methods of manufacture were cited in support of these views, and a number of remedial suggestions were made, which have since been widely acted upon, with a result of substantially diminishing the risk of pulmonary phthisis in a large number of callings.

I have already referred, in connection with enteric fever, to an inquiry which was made by Dr. Buchanan in 1865-66, under the instructions of the Privy Council Office, and by which it was sought to ascertain what had been the results obtained by local authorities, who had, by means of such works as water-supply and sewerage, sought to improve the public health within their jurisdiction. One main purpose of the inquiry was, "that the then Central Public Health Authority should fulfil one of the principal duties expected of it by making new local experiences conducive to general enlightenment"; and this object was, in one important respect, attained in an altogether unexpected direction. That the prosecution of such works as have been referred to should have led to a diminution in the general death-rate, as well as in that for certain of the infectious fevers and allied diseases which are known to be intimately associated with pollution of air, soil, and water, was indeed to be anticipated; but as the inquiry progressed it became obvious that an influence had been exercised by such works on the amount of mortality from pulmonary phthisis; and in the end it became clear that "the drying of soil", which had, in most cases, accompanied the laying of main sewers in the improved towns, "had led to the diminution, more or less considerable, of phthisis mortality."*

Some brief summary of the results of Dr. Buchanan's inquiry, in so far as it relates to phthisis, will be of interest. Amongst the towns visited there were many in which a striking diminution had taken place in the phthisis mortality; that diminution being not only, at times, the largest amendment discovered, but it had taken place at a period

* Ninth Report of the Medical Officer of the Privy Council, with Appendix, 1866.

which must necessarily have preceded, by some years, the accomplishment of such objects as the construction of proper house-drains, the abolition of cesspools, etc. Indeed, it was quite evident that any mere improvement in the methods of dealing with excreta and refuse, both solid and liquid, the bringing in of new water-supplies, and a host of other conditions that were considered, bore no necessary relation to the improvement effected. But a distinct etiological connection was established between local dampness of soil and phthisis. Where drying of soil had resulted from works of sewerage, there the phthisis mortality had undergone diminution; where no such drying of soil had as yet been brought about, there no improvement as to phthisis had been effected; and not only so, but the amount of reduction in the phthisis mortality appeared largely dependent on the extent to which permanent drying of soil had been brought about. The results recorded, as regards Leicester and Chelmsford, afford crucial proof of these contentions. In the case of Leicester, we have a town "in the main situated on a saucer of loam, principally stiff clay", and formerly having an undrained subsoil and "a wet surface". In these circumstances it had, for a series of years before the execution of any sanitary works, a mortality from phthisis amounting to 43⅓ per 10,000 living; during six years, in which 30 miles of sewers were constructed, the rate fell to 25⅛; though, after the completion of the sanitary works, it rose again to 29¼; the comparatively small rise during the third period here referred to corresponding, doubtless, with a damper state of subsoil than that which obtained during the period when the works were in actual progress, and when the stiff clay was intersected with deep trenches for the reception of the sewers. Chelmsford, on the other hand, lies at the confluence of the rivers Chelmar and Cann; the town being situated on a gentle incline on a bed of gravel, some 15 to 30 feet in depth, overlying the London clay. In 1853 works of sewerage and water-supply were commenced. During the ten years preceding that date there had been a death-rate from phthisis of 32⅓ per 10,000; whilst the works were in execution—a time when certain wells were dried—the rate exhibited a slight fall to 30⅓; but the rate for a period of eleven years after their completion practically reverted to its previous state, and is recorded as amounting to 32⅔ per 10,000. In the case of Chelmsford the sewers are deep and the soil is porous; and it is under these circumstances that drying of soil is most likely to take place. But, half a mile below the town, on the river Chelmar, stands a mill having flood-gates, with

which the mill-owners alone have a right to deal; by means of these the subsoil water of Chelmsford is artificially kept up, and, at the date of the report, fogs prevailed in the meadows; and the town was, in winter, surrounded on more than one side by marshy lakes. Both Leicester and Chelmsford thus teach the same lesson, although the data are altogether different. At the close of his inquiry Dr. Buchanan learned that Dr. Bowditch, during the course of an inquiry as to the causes of consumption in Massachusetts, had come to conclusions similar to his own; and it is needless to add that the circumstance of two observers having, independently of each other, arrived at precisely similar results gives additional importance to the discovery made.*

The discovery was, indeed, of the utmost value. It afforded indications as to the prevention of phthisis which it soon became impossible to ignore; and we see some of their developments both in the efforts made, by works of drainage and otherwise, to reduce the level of the sub-soil water of towns and villages, and in those codes of bye-laws which require that the ground surface of every domestic building shall be covered with a layer of cement concrete, and that a damp course shall always be so placed in the walls of such buildings as to be beneath the level of the lowest timbers. The value attaching to residence at high altitudes, and in a dry atmosphere, also receives significant explanation as the result of Dr. Buchanan's inquiry. Indeed, it may be asserted that, on the lines indicated by the results of the several inquiries to which I have adverted, vast improvements have been going on, which have influenced health and life in town and village, factory, workshop, and home, to a degree that has already materially diminished the production of pulmonary consumption, both in this country and in many parts of the world.

* I am aware that, in his sixth report (1879) on the Combined Sanitary District of West Sussex, Dr. Charles Kelly, basing his views on certain experiences derived from that rural area, has expressed a doubt as to the correctness of these conclusions. He states that the phthisis death-rate had been "distinctly lowered" in that district "in recent years", "while very little, if any, change has taken place during the same period in the drainage of the soil." Without entering into any criticism of some of the statistical data brought forward by Dr. Kelly in support of his views, I would here merely note, 1st, that the large amount of agricultural drainage which had then already been effected throughout the kingdom would be expected to have produced a very similar result in rural districts to that brought about by sanitary drainage in towns; and, 2nd, that Dr. Kelly offers no explanation of the definite and striking relation shown by Dr. Buchanan to have existed between the amount of diminution of phthisis death and the extent and permanence of the lowering of sub-soil water.

Unfortunately, confusion between pulmonary phthisis and other diseases of the respiratory organs has not, even at this date, been eliminated from our mortality returns; and comparative statistics as to these several diseases are further vitiated by reason of changes in nomenclature which have followed on improved diagnosis. But, at the same time, some indication of the improvement I have referred to may reasonably be assumed to underlie the figures included in the following table, which shows the annual mortality per million from "phthisis" at all ages, and at certain typical ages in both sexes during the last three *decennia*.

PHTHISIS IN ENGLAND AND WALES.

	All Ages.	15—	20—	25—	35—
1857-60	2679	2961	4181	4317	4091
1861-70	2475	2651	3928	4243	4026
1871-80	2116	2036	3117	3619	3745

I cannot quit this subject without making some reference to the investigations which are being made in this country, in Germany, in the United States, and elsewhere, as to the etiological relationship of bovine and human tuberculosis; and to the allegations of some observers, that bovine tuberculosis may be communicated to man as the result of the use of milk of cows suffering from that disease, and even that human tuberculosis has a source in the use for food of the flesh of tuberculous animals. So far, the propriety of accepting the probability of such transmission of this infective disease appears to depend materially on the question of the identity of bovine and human tuberculosis, as to which different views are entertained. But the fact that the milk of tuberculous cows has, apart from certain complications, not yet been found to contain tubercle bacilli, alone forms a ground for suspending judgment on this important point.

VII.—CHOLERA.

The history of cholera in this country is very intimately associated with the story of progress in the department of public health and of sanitary medicine. As already stated, the first appearance of cholera in England was in 1831-32, before the commencement of the Victorian era. The disease reached this country by way of the Baltic, first attacking several of our eastern ports in the autumn of 1831, and becoming

widely diffused in the following year. Unfortunately, no accurate information is available as to the extent of its ravages. But that invasion led to an important movement in the direction of sanitary reform, and of legislation to effect that object; such legislation being largely based upon the skilled observations of those who had recorded the circumstances under which cholera had prevailed in this country.

The second great visitation of cholera occurred in 1849. On the occasion of this epidemic, cholera, after having extended its destructive course over the eastern part of Europe, penetrated into Prussia, Hamburg, and Holland, and, in September 1848, crossed the German Ocean, and broke out in several towns in Scotland and England. At first the progress of the disease was slow and irregular; the total deaths in England numbering 988 from the date of its first appearance, in 1848, to the end of March 1849. But between June and the middle of September of that year there was a rapid increase, after which the disease gradually declined, and finally ceased in December. In all, the fatal attacks during 1849 numbered 53,293, in addition to 18,887 diarrhœa deaths; and, from the literature in which the epidemic is recorded, it is evident that there was recognition at this date of the circumstance that its spread was largely facilitated by conditions of filth, affecting air, soil, and water. In the words of the Royal Sanitary Commission, 1869: "The reports then made by the medical officers who had been established . . . . clearly traced the most fatal ravages of the epidemic to the crowded alleys of large old towns, impure air and water, and foul streams." And whilst the diffusion of the disease was largely dependent on human intercommunication, direct or indirect, it was found to differ in its mode of spread from such affections as typhus and small-pox.

In August 1853 cholera appeared again in London; in September it was widely prevalent in Newcastle, Gateshead, and other places in Great Britain; and in the following year, 1854, England suffered from a third great epidemic of this disease. Cholera in this country had again followed on an epidemic spreading from Russia, through Norway, Sweden, and Denmark, and the Baltic ports of Germany, from whence it was communicated to this country.

In the metropolis the disease was maintained during September, and in the months of October, November, and December 1853 the fatal attacks were 335, 288, and 43 respectively. From January to June 1854 only occasional

deaths took place; but during the last three days of July no less than 138 fatal attacks occurred. From this latter date there was a rapid rise in the amount of cholera and diarrhœa fatality. The epidemic spread in various parts of the kingdom, and by the close of the year the total deaths from cholera in England and Wales had numbered 20,097.

Dr. Snow was amongst the earliest contributors to the etiology of cholera in this country. He had previously dealt with the epidemics of 1832 and 1849, in connection with the theory of water-pollution. As regards the 1832 epidemic, he had maintained that its exceptional incidence, in certain localities, was in large part due to the polluted water in use in London, whether derived from wells liable to receive cesspool soakage or from the sewage-contaminated Thames. Between 1832 and 1849 cesspools had been largely abolished, but water-closets, the contents of which passed into the Thames, had replaced them, with the result of bringing about a state of river impurity far exceeding that of 1832. And during the 1849 epidemic the parts of the metropolis most heavily visited were those within the area of the Lambeth and the Southwark and Vauxhall Water Companies on the south side of the river, their supply being then the worst of all those derived from the Thames. The "contamination of pump-water" was also ascertained to have been associated with the spread of the disease. And now again, in 1854, Dr. Snow pointed to the proof which the incidence of cholera afforded "of the powerful influence which the drinking-water containing the sewage of a town exerts over the spread of cholera". Between 1849 and 1854 important changes had taken place in the water-supply of the metropolis. The Lambeth Company had sought a supply higher up the river, and, to quote Mr. Simon,* that Company was now "furnishing as good a water as any distributed in London"; while its southern colleague, the Southwark and Vauxhall Company, "was purveying perhaps the filthiest stuff ever drunk by a civilised community". Here were materials for comparison, and the result was as follows:—On a population of 166,906 persons living in houses supplied by the Lambeth Company, the cholera deaths were at the rate of 37 to every 10,000 living; whereas, on 268,171 persons supplied by the Southwark and Vauxhall Company, they were at the rate of 130 per 10,000. "The population drinking dirty water accordingly appears to have suffered 3½ times as much mortality as the population drinking other water."

* Report addressed to the President of the General Board of Health in May 1856, by the Medical Officer of the Board.

It was during this epidemic that the circumstances occurred which have made the pump in Broad Street, Golden Square, historic in the annals of English cholera. According to Dr. Snow,* 89 cholera deaths were registered in three sub-districts in the week ending September 2nd, 1854; 79 of these occurring in the two last days of that week, and "nearly all" in persons residing within a short distance of that pump. Indeed, "except among the persons who had been in the habit of drinking of the . . . . pump-well", there had been no particular outbreak or increase of cholera in that part of London. It was a celebrated pump. "The water was used for mixing with spirits in all the public-houses around. It was used likewise at dining-rooms and coffee-shops . . . . it was also used at various little shops" in the making of sherbet. And amongst occasional consumers of it, some living at a distance from the pump, numerous fatal attacks occurred; whilst, on the other hand, the disease was markedly absent from several large groups of people living in the immediate neighbourhood, in institutions or other places having a different water-supply.

But it also became recognised that polluted water was by no means the sole cause of cholera spread. Dr. Greenhow, in reporting to the General Board of Health, pointed out that the mortality had also "generally borne a direct ratio to the amount of atmospheric contamination"; and although some of the channels by which the poison operated in its various wanderings still remained undiscovered, yet observers, such as Mr. Simon, detected the operation of "specific influences", with capability for propagation, such as is exhibited by "ferments", in the presence of conditions of filth affecting air, soil, and water.

It was as a sequel to this epidemic that the need for a medical adviser to the State was recognised by the appointment of Mr. Simon as Medical Officer to the General Board of Health.

The fourth cholera epidemic from which this country has suffered took place in 1866. As on the occasions of the former epidemics, the disease manifested itself in the year immediately preceding its wide diffusion. But it entered this country by an altogether different route. Early in 1865 cholera was causing an appalling mortality amongst the Mecca pilgrims, and from Mecca it was diffused into Egypt, and thence along the lines of steam-boat communication between Alexandria on the one hand, and the Levant and Southern Europe on the other; extension subsequently

* *Snow on Cholera*, 1855. London, John Churchill.

taking place in a more northward direction. Ultimately the disease manifested itself in Southampton, on more than one occasion, during the month of July 1865; this extension being probably connected with arrivals from Alexandria, Malta, and Gibraltar; and being the first occasion in which cholera had entered the kingdom by way of our southern coast. After these preliminary warnings, a definite outbreak, involving some sixty persons, took place in Southampton during September–November; and cases also occurred at other places, such as Weymouth, Portland, Dorchester, and at Theydon Bois in Essex.

Towards the close of April 1866, a trader arrived at Bristol from Rotterdam—sick of the disease; in May, German and Dutch emigrants had conveyed the infection to Liverpool. Birkenhead and Swansea were next attacked. In June the *Poonah* arrived at Southampton with the disease on board; and England was evidently developing cholera at various points. Then came the invasion of London, the first death being returned from Poplar on July 18th,* after which the disease spread rapidly. The story of this epidemic has been told in detail by the late Mr. Netten Radcliffe, who also, as the result of a laborious inquiry, came to the conclusion that the exceptional incidence of the disease in the eastern districts of London was the result of the distribution by the East London Water Company of a polluted water, which had "in it a power of direct infectiveness for those who drank it". During the course of 1866 the cholera deaths amounted to 14,378 in England as a whole; 5,548 of these occurring in London.

The circumstances of the 1866 epidemic acted as a great stimulus to epidemiologists in various parts of Europe to study afresh the conditions under which cholera prevailed, and to obtain a knowledge of the right principles of defence against it; the practical sanitary experiences gained in England being especially supplemented by the labours of distinguished German pathologists, who, at that date, sought for the essential cause in some fungoid growth. In this country it had been conclusively shown that the laws of cholera contagion bore a striking resemblance to those relating to enteric fever, and that, to quote the words of Mr. Simon, "excrement-sodden earth, excrement-reeking air, excrement-tainted water, these are for us the causes of cholera."

But though this lesson had been brought home more

* Reports of the Medical Officer of the Privy Council for 1865 and 1866.

convincingly than ever, it was not a new one; and for many years past a movement—often but too slow—had been on foot towards the removal of those conditions of filth which had been found to be essential to the diffusion of the cholera infection. And we have a right to assume that it was largely owing to these labours that the power of the disease for harm had become less and less during its last three visitations. This result is shown in the following table:—

CHOLERA MORTALITY.

Date.	England and Wales.		London.	
	Total Deaths.	Deaths per 10,000 living.	Total Deaths.	Deaths per 10,000 living.
1849	53,293	30	13,565	51
1854	20,097	11	10,684	43
1866	14,378	7	5,548	18

Since 1866, cholera, though more than once brought to our shores—as during the European epidemics of 1871-74 and 1884-87—has never succeeded in establishing itself in our midst, a result which is no doubt due to the steady removal from amongst the people of those insanitary conditions which are essential to its epidemic spread, and to the increasing security afforded by those measures of imperial and local sanitary administration by which it is sought to diminish sickness and mortality from all preventable diseases, including cholera.

The beneficial results which have ensued in regard to the saving of life from cholera have, in no small measure, been due to the fortunate abandonment by this country of all endeavours to keep cholera away from her shores by a system of quarantine. I recently brought under the notice of the Epidemiological Society the attitude we have finally adopted in this matter, in a record of the Proceedings of the International Sanitary Conference of Rome, 1885, which I had the honour of attending as one of the delegates of Her Majesty's Government.* It is one of distinct opposition to the entanglement of this country by means of a delusive system, offered anew under the plea that another reduction should be effected in the duration of a detention which, even under its most stringent forms, had been mainly characterised by ever-repeated failure; and I would now only refer to a summary of the grounds on which I justified this opposition when dealing with this subject at the meeting of the British Medical Association in 1887. I

* *Transactions of the Epidemiological Society*, vol. v, New Series. Session 1885-86.

concluded by observing that "the system of quarantine has again and again shown itself to be impotent for good, and, being so, its vexatious and inhuman characteristics stand out the more prominently. Above all, it has a blighting effect upon sanitary progress. So long as Governments tell their peoples that a line shall be drawn around them across which disease shall not pass, so long will those peoples be reluctant to spend their money on the promotion of true measures of prevention. The quarantining countries are essentially those which cholera invades. Taken as a group they are those where true sanitary progress is at its lowest ebb; and, with the experience we have before us, I would, in conclusion, ask, much in the words I used at the Rome Conference: Is it likely that this nation will sacrifice her well-tried system of prevention for a restriction of five days' quarantine?"

I cannot close this subject without referring to one noteworthy contribution to the etiology of cholera, which has recently been made by Dr. W. J. Simpson,* Health Officer for Calcutta. An outbreak of cholera having occurred on board the *Ardenclutha*, which was moored in the river, he succeeded by a process of elimination in at last tracing the disease to the use of a certain milk-supply. Fourteen of the crew who had not partaken of this milk remained altogether free from sickness; whereas, of ten who had drunk it, nine, or ninety per cent., sickened, four with fatal cholera, and five with "diarrhœa"; the one who escaped having only drunk a "thimbleful". The milk-supply being stopped, all sickness was stayed. In this case the milk—which was procured from a distance, namely, from Howrah, on the other side of the river Hooghly, and had been brought on board by a native—was admittedly and somewhat copiously diluted with the contents of a tank polluted with cholera excreta. Cases of cholera occurred amongst the natives using the tank water directly after the contamination took place, and this localised outbreak at Howrah was simultaneous with that on board the *Ardenclutha*. This is, I believe, the only case on record in which milk has been definitely ascertained to have served as a vehicle for cholera transmission.

I am deeply conscious of the shortcomings of this record. The time at my disposal has of necessity placed limits on its completeness, and has compelled me to omit all but the most cursory reference to labours and discoveries which are calculated to exercise a most important influence on public health

* Report on the Health of the town of Calcutta, for the first quarter of 1887.

in the future, and which are identified with the names of many with whom we have been and still are associated. One such omission will, I am glad to say, speedily be supplied by Dr. Klein, who will shortly bring before us the subject of "The Infectious Diseases common to Man and the Lower Animals". We have also in prospect a communication which will bring our information as to the relation of micro-organisms to preventable disease up to current date.

The complete story of the progress of sanitary medicine during the memorable period under discussion would carry us far beyond such bounds as I have had to place upon myself. It extends to the saving of human life, as the result of advancing knowledge, in every quarter of the globe; and those whom I address, having, during the best years of their lives, devoted singular gifts to epidemiological research in every portion of this great Empire, may well remember with satisfaction how our Society has been the repository of learning that has so largely tended to the lessening of death, which has been so distinguishing a characteristic of the past half-century.

If we need any fresh incentive to our labours we have but to compare the terrible death-roll of the past with that diminished mortality which has gone hand-in-hand with the scientific application of the principles of sanitary medicine to the circumstances under which we live. And when we look at the recent advances in our knowledge of the intimate nature of infection, and of the still hidden, but yet far-reaching, influences of disease, we are further stimulated to enter into friendly rivalry with those to whom the laurels of the past have so deservedly fallen.

It has, indeed, been contended that there are limits beyond which the saving of human life is not consistent with the public welfare, and that the present tendency is towards an undue multiplication of the human race, and to a corresponding increase of poverty and misery. But I venture to assert that, so long as the work we are engaged in goes, as it has gone hitherto, to the lessening of death, so long must it ensure the diminution of sickness, and a corresponding promotion of a higher vitality, and a greater capacity for remunerative work amongst the living.

And our determination to continue in a labour so full of promise will have the more binding force as we recall the progress which has marked a reign on which we, in common with the great British world, look back to-day with feelings of pride and loyal affection.

SOME OF THE INFECTIOUS DISEASES COMMON TO MAN AND THE LOWER ANIMALS.

By E. KLEIN, M.D., F.R.S.,
Lecturer on General Anatomy and Physiology at St. Bartholomew's Hospital Medical School.

(*Read*: 14*th December* 1887.)

Mr. President and Gentlemen,—At your invitation, Sir, I shall have the honour this evening to lay before you a summary of a good deal that has become known within the last ten or twelve years as to a group of communicable diseases which are common both to man and animals. That the study of these diseases must be of special interest to you, gentlemen, to whom the study of the origin, spread, and prevention of communicable diseases in the human race is essential, I need hardly emphasise; nor need I remind you that this study, at any rate as regards some of the diseases communicable to man and beast, is of very old date; *e.g.*, that glanders is communicable from the horse to man has been known long before this generation; and also that hydrophobia in man is caused by the bite of a dog affected with rabies; and here we must provisionally include small-pox of man transmissible to the cow, therein producing a mild disease, viz., cow-pox, the lymph of which being used to produce a local mild distemper in the human subject, protecting against the severer disorder, has been the great discovery, and is known wherever Jenner's name is known and held in reverence. But there are others amongst these maladies about which only within comparatively recent years it has become understood that they are common to, and interchangeable between, man and animals. Amongst these I may mention anthrax or malignant pustule, tuberculosis, foot-and-mouth diseases, actinomycosis, scarlet fever, and probably also diphtheria.

I should have greatly wished to give you as full an account of all that has been definitely and experimentally ascertained of these maladies by a host of workers, but the time at my disposal is far too limited to do so. The literature of this subject, as far as it must be of interest to epidemiologists and health officers, is so enormous, that to give a

fairly full account of our present knowledge on these matters would fill a big volume. I must ask you, therefore, to excuse the cursory manner in which I shall treat of some of the above disorders, and I will also ask you to follow me more fully into others, such as anthrax, tubercle, and scarlet fever.

Well, then, the diseases of which I will give you an outline are: small-pox and cow-pox, foot-and-mouth disease, hydrophobia, glanders, actinomycosis, and diphtheria; and in this account I shall limit myself to the etiological data, as far as they are of special interest to epidemiology.

1. *Cow-pox.*—Jenner's views as to the relation of human variola and cow-pox are so well known that it is unnecessary for me to enter into them, and likewise Ceely's classical papers in the *Transactions* of the Prov. Med. and Surg. Assoc., 1840, and Dr. Badcock's results, have more than once been discussed and commented upon. Ceely, by direct inoculation of human variola into heifers, has succeeded in two instances in producing vesicles identical with those of the natural cow-pox, and in transmitting the lymph from them into generations of human beings as vaccinia; he has thus proved that vaccinia is a modified human variola, having obtained this modification by its passage through the cow. Now it is known that the French Commission under Chauveau arrived at a different conclusion. They were unable to produce such a result as Ceely did by inoculating calves and heifers with human variola; in some instances they assert to have produced a papular eruption at the seat of inoculation; the matter of such papules when inoculated into human beings produced again variola. They concluded from these experiments that human variola is transmissible to the bovine species as variola, but does not produce vaccinia. That this conclusion does not necessarily follow from the experiments of the French Commission has been very ably argued by the late Dr. Seaton and others. All that can be said of the work of the French Commission is, that their attempts to produce vaccinia by means of human variola were negative.

I can fully appreciate these negative results, because, some years ago, I have myself had opportunity—under the late Dr. Seaton and under the late Mr. Ceely—to make such inoculations on a large scale. Over thirty milch cows and heifers had been inoculated with lymph of human variola; in fluid condition and dried on points, taken from cases of confluent small-pox in different stages; clear lymph, purulent lymph, and crusts. Every animal was inoculated in 6-12 places, each place being made up of a number of simple or crossed incisions about half an inch and more, and a large quantity

of lymph was rubbed in into each place of inoculation. Many of the animals were thus repeatedly inoculated. But in no instance was there produced a vesicle, or anything comparable to a cow-pock. We concluded from these experiments that, to obtain a positive result, an unknown additional condition is required which was absent in our experiments.

The objection that has been raised against Ceely's positive results are these: Ceely succeeded in two cases of heifers, where variolation was performed almost simultaneously with vaccination, several places of the skin at one side of the vulva receiving vaccinia, whereas several places of the other side receiving variola lymph. In each experiment one variola puncture developed into a typical vesicle several days after the first vaccinia vesicles of the other side had made their appearance. Now, the objection that has been, and no doubt can be fairly raised against these results, is the great proximity of the insertions of the vaccinia and variola lymph, for thereby an accidental inoculation of one of the variola punctures from a vaccine puncture is quite within the range of possibility. But, looking at Ceely's drawings and description, it is clear that these vesicles at the insertion of variola were not identical with those at the insertion of the vaccinia.

Dr. Voigt published a few years back the results of his experiments, which show that in several instances of calves he had succeeded in producing typical vaccine vesicles after inoculation with human variola. In order to meet the objection raised against Ceely's results, he vaccinated and variolated his calves at distant places of the body; vaccine was put into the skin of the perineum, variola into the skin of the ear, and on the latter typical vesicles made their appearance a few days after the vaccine vesicles on the perineum. So that he claims to have established the original proposition of Jenner and Ceely on a firm experimental basis. But I think the objections raised against Ceely's conclusions apply to a certain extent also to those of Voigt, since there is no guarantee that by the tip of the tail the animal may not have transferred the vaccinia lymph put into the insertions at the back into the insertions on the ear. It is, however, necessary to add that also in these experiments the vesicles that rose at the insertions of variola were not identical with the insertions of the vaccinia, neither as to size, progress, areola, and duration.

I am afraid that, as long as the present uncertainty as to the exact nature of the virus of vaccinia and variola remains, no clear experimental proof will be obtainable, unless it be

that a different method of experimentation with variola lymph is employed than has hitherto been followed. As regards the first, *i.e.*, the nature of the virus, various assertions have been made from time to time, without, however, bringing us much nearer to the exact understanding. F. Cohn has, many years ago, ascertained the presence of micrococcus of the character of streptococcus in the lymph of variola and vaccinia; Sanderson confirmed this; Weigert found micrococci in the lymph of the vesicles and lymphatics of the variola eruption; Pohl Pincus described them also in the tissue of the epidermis of vaccinia. Dr. Paul Guttmann isolated, by cultivation after the modern methods, different varieties of micrococci from the lymph of vaccinia and variola; so did also Dr. Buist. I show you here a streptococcus which is constantly present in human vaccinia, which corresponds to one of the streptococci isolated by Buist, and which he regards as the vaccinia streptococcus. Quite recently it has been, however, maintained that none of the species of micrococci hitherto isolated by the gelatine method can be the virus of vaccinia, but that it probably is some other organism not capable of growing in gelatine.

The fact of the matter is, no one has yet succeeded in making a successful inoculation experiment with any of the organisms isolated from either variola or vaccinia. In saying this I am quite aware of the assertions of Dr. Buist, who claims to have succeeded in this; but reading his statements, and comparing them with his experiments, I think you will agree with me that they do not carry a great amount of conviction.

When we have got hold of the organism of vaccinia and variola isolated and pure, then we shall be able with exactness to compare their action by experiments on the bovine species.

As to the second, *i.e.*, the method of experimentation, a great deal is left to be done. If, as Jenner, Ceely, and others maintain, true cow-pox naturally arising in cattle is a modified form of human variola, then I think inoculation of human variola lymph into the skin of cattle is not an exact imitation of the method in which infection is carried out by nature. Introducing the virus by the alimentary canal or the respiratory passages seems the more feasible way. Ceely has attempted this mode by wrapping blankets and sheets, that had been used by small-pox patients, round cows, but with no result. But I think this negative result does not justify our drawing any final conclusions, for repeated attempts will have to be made, and I think the small-pox

lymph and crusts in substance will have to be introduced directly and repeatedly into the alimentary canal and air-passages, before we are justified in drawing any conclusions. For we must not forget the fact that, in a disease like foot-and-mouth, notorious for its easy spread amongst cattle under natural conditions, artificial infection by means of saliva and fluid of the mouth is a matter of very great difficulty indeed, as all those have observed who have tried experimentally to reproduce the disease in cattle.

2. *Foot-and-Mouth Disease.*—That foot-and-mouth disease is communicable from cattle to the human subject, directly by contact and indirectly by milk, has been clearly established. I refer you to Dr. Thorne Thorne's paper, and also to the repeated observations of veterinary surgeons, who, by contact with diseased cattle, contracted the disease. In 1885 I isolated, by cultivation from the lymph of the vesicles of sheep affected with the disease, a streptococcus which, when administered by the mouth and nose to heal the sheep, produced the disease; from the eruption of these I isolated again the identical streptococcus; in the tissue of the vesicles of sheep naturally affected, and in those artificially infected, the same organism was observed, so that I have no hesitation in saying that this streptococcus is the essence of the disease. A curious fact demonstrated by these experiments was this: that while cultivations of this streptococcus when introduced by mouth and nose of sheep produced the disease, when inoculated into the skin failed to do so, though it seemed to protect them against subsequently contracting the disease when subjected to the first method of infection. The mode of growth of this streptococcus in artificial cultivation in broth, milk, gelatine, in gelatine-plates, in Agar-Agar, and on blood-serum, are characteristic, and by these it can be distinguished from other streptococci. In some respects it presents the same or similar characters with the streptococcus isolable from vaccinia, from erysipelas, from puerperal septicæmia, from pus (the *streptococcus pyogenes*), and from scarlet fever; but, on carefully comparing these different streptococci in the different culture-media, definite differences will be noticed in many details; and so, also, as regards their action on animals. (See below.)

3. *Hydrophobia.*—The subject of hydrophobia has, by the researches of M. Pasteur, received important additions. His long and arduous labours, carried on for several years, and confirmed by many independent workers, have established, beyond doubt, the following facts:—

(*a*) The fresh brain and cord of an animal or man affected

with the disease contain the virus of rabies in an active form.

(*b*) The intracranial injection of such virus is followed by the disease with certainty after an incubation varying in duration in the different species.

(*c*) Drying the cord or brain diminishes its virulence; this shows itself by a delayed incubation, and by a modification of the character of the disease, inasmuch as it gives place to "paralytic" or "dumb rabies". If the process of drying is carried sufficiently far, but not too far, no rabic disease, though an immunity is produced against rabies by inoculation with the fresh material.

(*d*) The inoculation with virulent matter (natural or experimental) simultaneously with, or followed by, that of attenuated virus of the proper degree, is not followed by the outbreak of the disease, hence it is possible to overcome the action of the virulent poison.

(*e*) The disease is also, but unreliably, produced by subcutaneous inoculation of cord and brain matter; as also, but still more unreliably, by the saliva.

From these facts Pasteur has drawn certain conclusions, which, if established, would give a means, not only to stamp out rabies amongst dogs, but also prevent rabies in man: for it must be obvious that, by subjecting all dogs to inoculation with attenuated virus, no rabies dangerous to man could develop in them; and further, by inoculating human beings bitten by a rabid animal with attenuated virus, the disease would be prevented. These conclusions, though fully justified, cannot, however, be considered as fully established. Their applications to the human subject are far from understood.

In the first place, the method of inoculation, as practised by M. Pasteur on the human subject, *i.e.*, subcutaneous inoculation—intracranial injection not having been, and is, for obvious reasons, not likely to be employed—is not sure of success, if as a basis for such a proceeding the knowledge gained by animal experiments is to be relied upon. For in the series of experiments made by Von Frisch in Vienna, and by Mr. Dowdeswell at the Brown Institution, it is clear that the results in the rabbit and the dog are altogether dissimilar; for whereas in the latter species this method did lead to protective inoculation in some animals, in the first species —*i.e.*, the rabbit—it produced true rabies. M. Pasteur's protective inoculation in man also led, in a certain percentage of cases, to fatal results.

Secondly, the question of degree of attenuation the cord

undergoes by drying, although established for the rabbit's cord, is not established as far as its application to the human subject is concerned. Pasteur's and others' method of using the cord of rabid rabbits in successive degrees of attenuation —starting with the use of a higher, and proceeding to that of a lesser degree of attenuation, either slowly or rapidly (intensive treatment)—is tentative; and for this method no firm and clear basis is as yet available, since no one knows what is the minimum and what is the maximum degree of attenuation of rabbits' cord *quoad hominem.*

Another uncertain factor in the application to man of the protective inoculation is the fact that the incubation-period of rabies in man exhibits such a conspicuous difference in the different cases. As is well known, persons bitten by rabid dogs and seized with hydrophobia show a wide range of incubation-period. If, as Pasteur assumes from his animal experiments, a difference in incubation-period indicates a different degree of virulence of the rabic poison, then no assumed degree of attenuated virus could rationally be recommended for protective inoculation, since the duration of the incubation-period in a given human case can obviously be only a matter of conjecture.

But, above all, the intimate nature of the rabic virus being as yet unknown, no definite and conclusive insight into its *modus operandi* is available. Various observers have described organisms as being the cause of rabies, such as those by Fol; but there is really no definite proof forthcoming. Mr. Dowdeswell has seen masses of micrococci in and around the central canal of the cord in a rabid dog, but has missed them in other cases. Quite recently a Russian observer described bacilli as the cause of rabies; but from his experiments it is quite clear that the microbe of rabies remains to be discovered. When this is done, and not until then, definite and clear proof will no doubt be obtained on some of the most essential points of the problem. When artificial cultivations of this microbe are available, then a clearer knowledge and understanding will be forthcoming as to the best methods of its attenuation, as to the most useful manner of its inoculation for protective inoculation, and as to a variety of other matters intimately connected with the subject. I need only refer to the large amount of useful knowledge that has been possible in the case of protective inoculation against anthrax, against charbon symptomatique, and against erysipelas in swine.

4. *Glanders or Farcy.*—Thanks to the exact and thorough researches of Löffler and Schütz, Franck and Weichselbaum,

and the detailed monograph by Löffler in the *Arbeiten d. k. Gesundheitsamtes*, i, 3-5, we have now a full and complete account of the etiology of this disease. It is due to a bacillus (*bacillus mallei*), which is present in all the morbid products—nodules, ulcers, and abscesses—in farcy or glanders. All nodules, ulcerations, or abscesses in the skin or mucous membranes (nose, larynx, and lungs) contain the bacilli. They are slender rods of about the length of the well-known tubercle bacilli, but a little thicker, often slightly curved; they are present singly or in groups in the tissue of the nodules, are more numerously found in the young nodules both of man and animals than later, when the nodules have become ulcerated and purulent. They grow well on boiled potato, forming in two to three days a brown slimy scum; on blood-serum (solidified) they form after three days, at 37 deg. C., minute translucent droplets; also on Agar-Agar mixture they grow at 37 deg. C. in the form of greyish or white droplets; in gelatine they only grow at 37 deg. C., *i.e.*, in liquefied gelatine. By inoculation of cultures into field-mice, guinea-pigs, horses, donkeys, and young dogs, glanders in all its symptoms has been produced, with the characteristic bacilli in the nodules.

House-mice are insusceptible; rabbits get only occasionally a few local ulcers, which, however, soon heal up. In guinea-pigs, on the third or fourth day, an ulcer appears at the seat of inoculation, and a subsequent enlargement of the nearest lymph-glands, which is followed by nodules in the ovaries or testes, feet, and nasal cavities. When small quantities are used for inoculation, the local ulcerations and the swelling of the nearest lymph-gland remains *in statu quo* for several weeks. Now this condition in the rabbit and in the guinea-pig (in the latter when small quantities are used for inoculation) seems to show that the body of the rabbit, and, *mutatis mutandis*, of the guinea-pig, are not the most suitable soil for the manifestation of the virulence of the bacilli, and it is quite possible that when taken from this new soil and transferred to horses and donkeys—*i.e.*, animals which represent the best natural soil for the bacilli—they would not exhibit the same virulence as those bacilli originally derived from a case of natural glanders in the horse. That is to say, by using the nodules of a rabbit or a guinea-pig (in which only a local effect has been produced by the bacilli) for inoculation of horses, it is quite possible that a mild transitory disorder would be produced, protecting against the virulent form of the disease. This line of research is, at any rate, well worth trying, bearing in mind that this disease, glanders,

is a widespread one, and is easily communicable, and when gaining entrance into large stables—say those, for instance, of the omnibus, tramcar, cab, and other companies—is capable of causing great loss to the owners, and is at the same time of great danger to the stablemen.

5. *Diphtheria.*—There are various statements as to the identity of diphtheria in man with a disease in pigeons, fowls, and other domestic animals (horses, cats). But examining the evidence carefully, it is found that there is no definite proof for these statements, and, besides, what evidence there exists is very contradictory.

Emmerich states that what is known as diphtheria in pigeons is caused by a certain bacillus, which bacillus he also found in the diphtheritic membrane of human cases. This bacillus is a thick rod, twice the length of its breadth, which on nutrient gelatine grows in the form of whitish colonies of the size of a pin's head; in pigeons, rabbits, and white mice, the introduction of the cultures on to the mucous membrane of the trachea produces greyish, croupous membranes; death of the animal follows in a few days, the bacilli being found in the blood and internal organs. This result is produced equally with the bacilli obtained from the diphtheritic membrane of human cases, as also from those of pigeons in the natural disease. However, Emmerich's conclusions are not accepted by the Berlin School and its followers, on account of his results clashing with those of Löffler. This last observer found, in the great majority of human cases, in the diphtheritic membranes, a bacillus which is altogether different from that of Emmerich. Löffler's bacillus is a non-mobile bacillus, slightly curved, with one or both ends slightly enlarged. It grows only very slowly and imperfectly on nutrient gelatine, but grows well on blood-serum at 37° C., as whitish translucent droplets. It also grows fairly well on Agar-Agar mixture. On this latter it forms round or oval brownish granular discs. Cultures introduced into the mucous membranes of the trachea of rabbits, fowls, and pigeons produce herein extensive pseudo-membranes, followed by hæmorrhages in the lymphatic glands and pleural cavity. Young animals are more susceptible than adult ones. Subcutaneous inoculation into guinea-pigs and small birds produces extensive œdema and hæmorrhage, starting from the seat of inoculation. But Löffler did not succeed in finding these bacilli in all diphtheritic membranes of human cases; and in some cases of children and adults, not affected with diphtheria, he has succeeded in isolating the same bacilli from the fluid of the

mouth. By culture, staining, and experiment, its identity with that isolated from diphtheritic membranes was established. Besides, quite recently, Rosenbach isolated the same bacillus from the normal mucus of the human tonsils and pharynx.

The disease known as diphtheritic ulcerations in the calf, and investigated by Messrs. A. Lingard and E. Batt as ulcerative stomatitis, and by Löffler as diphtheria of calves, is different from human diphtheria, and is due to an altogether different bacillus, longer and thicker, and growing in an altogether different manner. Mr. Lingard has succeeded in cultivating these bacilli, and successfully to inoculate with them the mucous membrane of the tongue of calves and the skin of the ear of rabbits.

Löffler investigated also the disease known as diphtheria in pigeons, which resembles to a certain extent that of man. The back of the tongue and the pharynx of pigeons, the tongue, palate, nose, and conjunctiva and larynx in the fowl, show an initial hyperæmia leading to the formation at these places of a thick, yellowish deposit. The disease lasts for weeks and months, and in a certain percentage leads to death. Löffler isolated from the deceased parts in pigeons a special bacillus, the cultures of which produce, by inoculation into the mucous membrane of the mouth, the same disease as observed naturally.

Since this bacillus does not affect fowls, the disease in the fowl must be of a different nature; its contagium has not been isolated. The opinion that human diphtheria stands in relation to the disease of pigeons and fowls has been repeatedly maintained. Thus, Gerhardt and Stumpf maintain a direct connection between human diphtheria and the disease in the pigeon.

The observations of Dr. Turner (Report by the M.O., L.G.B.) as to a relation between membranes in the trachea of fowls, cats, and horses (spoken of as diphtheritic membranes) and human diphtheria, although very interesting and highly suggestive, do not claim to furnish any definite scientific proof.

6. *Actinomycosis.*—This is a chronic disease not unfrequently found in cattle. It shows itself in the appearance of hardish tumours of the jaw, in the tongue, pharynx, and larynx, sometimes in the lungs, and then resembling tubercle: the tumours under the microscope appear in the young state as round-cell growth, in the centre of which there is situated a yellowish granule of the size of a millet-seed; this, under the microscope, is composed of a central solid mass, from

which extend in a radiating manner, and densely placed side by side, longer or shorter club-shaped filamentous glistening elements, their broadest part at the periphery. These elements are sometimes branched at or near the peripheral end, wavy or twisted. In the central mass, occasionally a felted mass of fine threads is to be made out. This fungus is called, owing to its arrangement, actinomyces. It is, no doubt, the cause of the new growth, since, when freed of the surrounding tissue and transplanted into the subcutaneous tissue of cattle, it is capable of producing the same disease. These actinomyces have been found in the crypts of the tonsils of pigs in the normal state, and more recently by Virchow in the flesh of pigs, where to the naked eye they might be mistaken for the chalky capsules of trichinæ. Israel and Pomfick have shown that this disease, actinomycosis, affects also the human subject. Actinomyces have been found by them in abscesses of the jaw and lachrymal canals, and, spreading from here to the viscera of the chest and abdomen, cause abscesses in the pharynx, pleura, peritoneum, and lungs; further, they have been found in the wall of the alimentary canal, and by further spread cause peritonitis. In all these lesions the actinomyces have been discovered.

Until quite recently, the position of these actinomyces amongst parasites was not well understood, but Böstrom has shown by artificial cultivation that they probably belong to the group of algæ known as *cladothrix*.

7. *Anthrax, charbon, malignant pustule, splenic apoplexy, or Milzbrand.*—This disease is known to occur naturally amongst sheep, goats, cattle, and horses, and to be communicable to human beings. Its etiology, owing to the researches of Davaine, Brauell and Pollender, Bollinger, and above all of Koch and Pasteur, is now well understood. It is due to a special bacillus, the *bacillus anthracis*, capable of living in all vegetable and animal infusions, and, when well exposed to air, of forming spores or permanent seeds. When finding entrance into the animal or human system, it sets up a general blood disease, ending in many instances fatally. The blood is the most suitable habitat for these bacilli; in it they multiply with great rapidity, and cause hæmorrhages and extravasations in the different parenchymatous organs, leading to death. When animals like sheep or cattle become naturally affected with the disease, and they die, there are always more or less copious sanguineous discharges from the mouth, nose, and bladder, which abound with the bacilli; these bacilli are capable of multiplying on and in the soil receiving the discharges, and being here well exposed to the air, are capable

of forming spores, which, in the same season or later, or even after years, can, when taken up by a healthy animal, either by food or breath, set up the disease. One animal can thus become the cause of an extensive area becoming richly supplied with spores of bacillus anthracis. Add to this the fact that, by the spring and summer rains, and owing to the temperature being favourable for the germination of the spores and the multiplication of the bacilli, a meadow may become well stocked with an abundant crop of bacilli and spores of anthrax. If an animal that had died of anthrax in a given field were there and then buried, and the soil all round it were turned up and well disinfected, a limit to the further development of the bacilli and the formation of their spores would no doubt thereby be established, but as a rule this simple mode of precaution is not followed; in some cases the animal or animals are dragged or carted away to a distant point of the field, where they are buried, or in other cases, worse still, they are skinned before being buried; and since every drop of blood and exudation contains numerous bacilli, these become scattered over a wide area, and represent then so many foci where new colonies and crops of bacilli and spores become established. There is every reason for saying that whenever in a field anthrax attacks the animals grazing and sojourning in this field, the infection is produced by spores in the manner above indicated, *i.e.*, either by old spores, viz., spores formed in a former season, or by spores recently formed; since it is well known that bacillus anthracis, growing on a soil containing animal and vegetable matter, and well exposed to air—*e.g.*, the surface-layers of a field—is capable of forming spores within a few hours. There is experimental evidence that infection by spores by way of the alimentary or respiratory organs does easily take place, and there is likewise experimental evidence that with sporeless bacilli—*e.g.*, the bacilli of the blood—this does not succeed; for the bacilli are destroyed by the gastric juice, and do not penetrate easily by the respiratory mucous membrane, provided there be no lesion of the tissues; in the latter case infection would undoubtedly ensue as much as by subcutaneous inoculation. Pasteur maintained that an animal dead of anthrax and buried, could, nevertheless, become a source of infection, inasmuch as the bacilli in the blood and tissues would form spores, which, when brought to the surface and deposited there by earthworms, become available for infection. But there is no reliable evidence whatever that the bacilli are capable of forming spores in the blood and tissues, *i.e.*, away

from a free access of air; on the contrary, it can be shown that an animal dead of anthrax, and whose tissues and spleen teem with the bacilli, if left unopened or buried unopened, loses in a few days all power of infection as far as its blood and tissues are concerned. By putrefaction the bacilli in the blood and tissues become destroyed. The time in which this process of destruction of the bacilli in the blood-vessels and tissues of an animal unopened and buried is completed would, of course, vary with the size of the animal. In small animals—*e.g.*, guinea-pigs, rabbits, and mice—I have found that all infectivity has disappeared before or at the end of a week.

In all precautions against the spread of anthrax in a given locality, and in all methods of stamping the disease out, it must therefore be remembered that the effusions from mouth, nose, and bladder and rectum, as also the blood of an animal dead of anthrax, contain the bacilli anthracis; that these are capable of multiplying with great rapidity in soil that contains vegetable or animal substances, and that under free access of air the bacilli are capable of forming spores; further, that the usual mode of natural infection in a field is by spores taken in by the respiratory or alimentary organs; that the bacilli of the blood and tissues are destroyed by the gastric juice and by drying, but not their spores; and that if the bacilli are not supplied with free air they do not form spores; and, lastly, that the bacilli in the blood-vessels and in the tissues do not form spores, but gradually are destroyed by the natural putrefactive processes occurring in a dead animal.

The manner in which human beings become infected is twofold:—(*a*) either by inoculation with the fresh bacilli, or (*b*) by spores. In both instances it is generally infection with anthrax of sheep, goats, or cattle. In the first mode infection is produced if a cutaneous injury on the hands or fingers is brought in contact with the blood or effusions of an animal (sheep or cattle) dead of anthrax. Such occurs amongst butchers or others who handle the fresh skin or fresh carcases. On the River Plate Company's estate such cases of infection, I was informed by Mr. Derbishire, the manager, are not rare when there are many cases of anthrax amongst the sheep and cattle. Quite recently, two cases (butchers) occurred in Chelmsford in connection with anthrax in cattle, no doubt known to you from the medical journals. The curious fact about these instances is that infection seems, as a rule, to remain more or less localised to and around the place of inoculation. Malignant pustule

or carbuncle makes its appearance with all the clinical and anatomical characters of localised anthrax with the typical bacilli in the carbuncle. And though in the severer cases there may be and generally are present severe constitutional symptoms, such as high temperature and fever, it rarely comes to a general infection with anthrax and a fatal termination. Destruction of the local tumour and its surroundings generally leads to complete recovery. It is different in the second mode of infection, viz., when spores of anthrax (of goats, sheep, or cattle) find entrance into the system by way of the alimentary canal or respiratory organs. In mycosis intestinalis, infection with spores of anthrax starts from the alimentary canal, and the bacilli germinating from these spores pass into the wall of the alimentary canal, further into the mesenteric glands, and then into the general circulation, with generally fatal result. Such cases have been several times observed and carefully described; or, as is the case in wool-sorters' disease, a general anthrax infection, not seldom with fatal result, is brought about by the spores of anthrax adhering to the wool of sheep or goats that had died of anthrax, and where the wool during the process of skinning the animal would become smeared with the anthrax blood, and the anthrax bacilli having had time to form spores; or in the general anthrax infection observed in workers of hides. These cases of infection have been admirably described by Dr. Spear, whose valuable Reports are no doubt known to you. In both these latter instances —*i.e.*, wool-sorters' disease and the anthrax in workers with hides—the general infection could be obviously only brought about by spores, since the anthrax virus present here, *i.e.*, in the wool or on the hides, can be only in the form of spores, for the bacilli themselves, which originally adhered to the wool or to the hides (either naturally, as blood-bacilli on the inner surface of the skin of an anthrax animal, or accidentally, by having been smeared with blood of an anthrax animal), are not able to exist as bacilli for any length of time, drying and putrefaction being inimical to them as bacilli, whereas they are able to form spores in the course of a few hours, under favourable conditions of temperature, if they are exposed to access of air, as is the case in the processes of preservation of these materials.

What I wish to point out particularly is, that there seems to exist a difference between infection with fresh anthrax bacilli and that with spores—in the former a local, in the latter a general infection being produced. This is in harmony with various facts which have been ascertained in

experimental investigations of anthrax. It has been ascertained that blood of mouse, dead of anthrax, and containing the anthrax bacilli in the sporeless state, when inoculated into sheep, produces, as a rule, only a local effect, which sometimes may be severe, but with no general anthrax infection and no fatal result; whereas, if such bacilli are allowed to form spores—*e.g.*, in artificial cultures—and these spores are inoculated into sheep, fatal general anthrax infection is the result. Similarly, fresh blood of a guinea-pig, dead of anthrax, inoculated into cattle, does not, as a rule, produce general infection with fatal results, but only a local swelling, sometimes very extensive, whereas, if such bacilli are allowed to form spores, these, when inoculated, do produce, as a rule, general infection, with fatal result. But these considerations must not make us lose sight of another set of facts, viz., that in the first place the anthrax virus of the blood of the different species of animals differs in degree of virulence; and, secondly, that not all anthrax virus, even when derived from the same last source, possesses the same degree of virulence. I will refer to the following ascertained facts. Anthrax of mouse is less virulent than anthrax of guinea-pig, the virus of this is weaker than that of sheep, and this, again, weaker than that of cattle; the anthrax bacilli derived from the blood of a given animal that had become infected with bacilli of a gelatine culture are less powerful than the anthrax bacilli of the blood of an animal of the same species that had been infected with spores of a broth-culture.

8. *Tuberculosis.* — As is well known, tuberculosis is a disease to which many of our domestic animals are subject, though in varying degrees; thus, while cattle (young and adult), guinea-pigs, and rabbits are perhaps the most susceptible; others, like sheep, pigs, fowls, dogs and cats, and mice, are so in a lesser degree. That in all these instances the tubercular deposits contain the tubercle bacilli of Koch, that these bacilli possess definite morphological, chemical, and cultural characters, and that, whether tubercular material or pure cultures of these bacilli are introduced into a suitable animal, either by subcutaneous inoculation or by way of the respiratory or alimentary canals, the typical disease with the tubercle bacilli in the tubercular deposits is again reproduced, may be considered as fully established. These facts, first demonstrated by Koch, have been confirmed in all details by a host of workers. But there are various important points which have not, as yet, received a full and satisfactory explanation. In the first place, as to the tubercle bacilli them-

selves. It is well known that in order to demonstrate the tubercle bacilli, say, for the sake of diagnosis, the materials are stained in fuchsin, then treated with nitric acid, because it has been shown by Ehrlich that amongst a host of bacteria, which may be, and often are, present in the expectoration of the lungs, the tubercle bacilli stained with fuchsin and then subjected to nitric acid are capable of retaining the dye, whereas the other bacteria accidentally present become decolorised. Though this method is not the only one used for the staining of tubercle baccilli, it is, no doubt, the best and readiest for purposes of differentiation. Now the tubercle baccilli have, besides this chemical character, also certain morphological characters,—*e.g.*, size, shape, and aspect,—and for the purposes of diagnosis it is necessary to pay attention to them, as well as to the chemical character just referred to. For within the last few years it has been shown that the tubercle bacilli are not the only ones capable of withstanding the action of nitric acid. The bacilli of leprosy, the so-called syphilis bacilli, have been known to possess also this power; more than that, it has been fully established that others—*e.g.*, certain septic bacilli, which under ordinary conditions of growth have not this power—are capable of acquiring this same power of withstanding the action of nitric acid when growing in a fatty medium; and it is not at all impossible, or even improbable, that the tubercle bacilli of Koch possess this chemical character, on account of the nature of the soil in which they grow, since it is known that they are numerously found in tubercles that have commenced to undergo, or have advanced in process of, caseous degeneration.

It is therefore of importance to bear this in mind; and therefore all other characters of these bacilli, as well as the anatomical and clinical evidence, must be taken into account. But there is an experimental means of fully ascertaining the nature of the material in question, viz., whether tubercular or not. Guinea-pigs are extremely susceptible to tubercle; inoculated under the skin of the groin, they show after some days (eight to fourteen) hard swelling of the inguinal lymph-glands nearest the seat of inoculation, which gradually increases and leads to the formation of an abscess. This sooner or later bursts, and leads to the formation of an open sore involving all the tissues around, the swelling and caseation of the lymph-glands spreading wider and wider, the bone-marrow also becomes involved; then the viscera (lungs, spleen, liver, peritoneum, intestine, and even kidneys), with their lymph-glands, become the seat of tubercles, at first small and grey, then larger and caseating in the centre, and,

when numerous, becoming confluent. There are slight deviations from this picture, which represent an extreme case of general tuberculosis, since the extent of tubercular deposit in the organs of the chest and abdomen may be in some instances slight, in others very pronounced; as also the progress and duration of the disease is subject to great variations, from several weeks to several months. The tubercles taken from such experimental animals introduced into fresh guinea-pigs always set up the same chain of morbid processes. Another, and even more characteristic experimental test, is that of Cohnheim. It is this. Introducing into the anterior chamber of the eye of rabbits small bits of tubercular material, it is always noticed that, after the lapse of a few weeks, it produces a crop of grey, characteristic tubercles in the tissue of the iris, which gradually undergo the usual process of caseation, and which are followed by the production of the general disease, *i.e.*, disseminated tubercles in the internal viscera.

A second, and as you will readily see, a most important point is the relation of human to bovine tubercle. It must be obvious that if bovine tuberculosis is identical with human tuberculosis, the consumption of meat, milk, etc., derived from tubercular cattle may become a serious source of human tuberculosis, considering that tuberculosis of calves and adult cattle is not by any means rare. In connection with this the following points must be taken into consideration:—(1) Tuberculosis can be induced experimentally in guinea-pigs, fowls, and pigs by feeding these animals with tubercular matter; and since the human subject is susceptible to tuberculosis, there is *à priori* no reason why human beings should not also be capable of contracting the disease by the way of the alimentary canal. In the experimental tuberculosis produced by feeding animals with tubercular matter, deposits in the wall of the digestive tract in the form of tubercles in the lymphatic follicles (Peyer's and solitary follicles), at first grey and then caseous, followed by tubercular deposits in the mesenteric glands and peritoneum, liver and spleen, clearly indicate the course of the passage of the tubercular virus. And as regards tuberculosis of man, the well-known cases of intestinal tuberculosis are very instructive, and indicate that most probably also here the tubercular infection was produced by the passage of the virus directly into the wall of the alimentary canal. (2) The tubercle bacilli found in bovine tubercles, and the distribution of the bacilli in the lungs of cattle, present some well-marked differences from those found in the human

subject. The bacilli of the former are smaller than in the latter, are invariably at first lodged within the well-known multinuclear cells, and when caseation sets in and the cells disintegrate, the bacilli become free; whereas in the human subject the bacilli are, as a rule, found between the cells constituting the tubercle. But these differences may be explained by the difference of the soil, the tissues of cattle presenting slightly different conditions from those of the human subject. A more important difference between the two viruses is, however, to be found in their different action on animals. While the tubercular matter derived from a bovine source acts very virulently on both guinea-pigs and rabbits, that from a human source acts considerably less virulently on rabbits than on guinea-pigs; and, in the case of guinea-pigs, the process of tuberculosis produced by bovine tubercular matter is considerably more rapid and general than that induced by human tubercular matter. This difference, which undoubtedly exists, may be, however, explained by the assumption that the tubercular virus in its passage through the cow gains in virulence, in the same way as the anthrax poison, passing from the mouse through the guinea-pig or sheep, increases in its virulence. (3) The anatomy of tuberculosis in the cow differs from that of the human subject, though particular cases of human tuberculosis have been described (Creighton) which bear a striking resemblance to that in cattle; but whether this is owing to the fact that these particular cases of human tuberculosis are causally more related to bovine tuberculosis than the ordinary typical human pulmonary tuberculosis, is a point not yet cleared up. (4) But there is one point about which there is as yet no evidence forthcoming, though this is sadly wanted. It is this: if the virus of bovine tuberculosis is the same as that of human tuberculosis, and as bovine tuberculosis is comparatively a widespread disease in cattle, there ought to have come to light cases where human tuberculosis has followed the consumption of bovine tubercular matter. True, the milk of tubercular cows does not contain the tubercle bacilli, unless there be present tubercles in the udder itself, as is sometimes the case in advanced stages of the disease; and feeding experiments carried out on calves and other animals with milk of tubercular cows, in which the udder was free of tubercles, and consequently the milk was free of tubercle bacilli, yielded negative results. It is likewise true that by exposure to the heat of boiling water the life and action of the tubercular virus become destroyed, provided this be carried on for five minutes. And further:

though the marrow of bones contains tubercles and tubercle bacilli in guinea-pigs, experimentally infected, already at a very early stage of the general disease, and when this has made but slight progress as yet, the muscular tissue is free of tubercles and tubercle bacilli, unless, owing to the close vicinity of tubercular deposits, these spread into, and involve the muscle; notwithstanding all these facts, there remains a wide margin within which infection of the human subject with tubercular materials of cattle (calves and adults) would still be possible, considering the extensive use for human consumption we make of the organs and tissues of calves and adult cattle. The assumption that such infection of the human subject has occurred, and does occur, frequently expressed, is not based on actual evidence—either direct or circumstantial—and though it may be extremely difficult to produce such evidence, it seems to me premature to accept at present such mode of infection; all we can say and do is, that it is safer to avoid such possible risk, by rejecting the use for food of tubercular cattle.

There is one more consideration which I have to add: it refers to tuberculosis of poultry. Various observers have drawn attention to the possible connection existing between the tuberculosis of fowls and human phthisis, this disease having been noticed to prevail amongst poultry in farms where human tubercular sputum became available, and where it was picked up by fowls. There can be no question about the possibility of producing tuberculosis in fowls by mixing with their food human tubercular sputum; and though the disease thus produced in the fowl is exceedingly slow, and limited chiefly to the abdominal viscera, occasionally only in the form of a few discreet tubercles, the assumption that the body of such an animal may, in its turn, again become the source of infection of human beings is not quite without basis, considering that fowls artificially tuberculised with human sputum in the majority of instances present an extremely well-nourished body, and would therefore be considered as well fit for the market.

Before concluding this part of my paper I wish to remind you also of the evidence brought forward since Koch's work on tuberculosis, as to the etiology of scrofula and lupus. In both these affections bacilli have been found, which in morphological and chemical characters bear the greatest resemblance to the tubercle bacilli, and cultivations of them have been produced which are identical with those of the tubercle bacilli; and lastly, experiments made on guinea-pigs with material both of scrofulous lymph-glands and of lupus-

tissue, or with artificial cultures of the bacilli, have produced a disease which cannot be distinguished from that produced by true tubercular matter. The literature on the subject, on the identity of scrofula and lupus with tuberculosis, is enormous, and has been very ably put together by E. Finger, in the *Centralblatt für Bacteriologie*, ii, 12, 13, 14. The prevailing opinion at present is unquestionably this, that scrofula as well as lupus are localised tubercular processes, and that lupus is probably due to an accidental cutaneous infection with tubercular virus. But whether these localised tubercular processes owe their difference from general tuberculosis to the nature of the locality into which the tubercular virus is brought, or whether it is due to a difference in the general disposition of the person, or is caused by an initial modification of the tubercular virus itself, are questions waiting for an answer.

10. *Scarlet Fever.*—It will be no doubt in the recollection of this Society that at the end of 1885 and the beginning of 1886, an extensive outbreak of scarlet-fever occurred in Marylebone, which Mr. Winter Blyth has traced to be due to consumption of milk coming from a particular farm at Hendon.* Mr. Power supplemented this by finding that cases of scarlet-fever occurring also in other districts of the metropolis were likewise to be referred to the milk of the same farm.

With Dr. Cameron, Medical Officer of Health of the Hendon District, Mr. Power was able to exclude all and every accidental contamination of the milk of this farm from human source. Mr. Power has further shown "that only certain sections of the milk-supplies within the farm, and eventually certain cows, had to do with the infectivity of the milk." The Report of Mr. Power is known to you, and I think you will agree with me that in reading it, and following from step to step the clear and precise observations of Mr. Power, we cannot refuse admitting the correctness of his conclusions. It is not my object or my place to defend Mr. Power's conclusions, he is quite capable of doing so himself; but I will undertake to say this, that to an unbiassed reader the methods by which he arrived at those conclusions are unimpeachable, and the proof amounts almost to a mathematical demonstration. You have, no doubt, during the last

* How imperfectly the nature of such inquiries and the methods employed are understood and appreciated abroad, is best shown by the manner of scepticism in which some foreign medical papers receive the accounts of milk-epidemics, be it of scarlet-fever, diphtheria, or typhoid fever, so thoroughly understood in this country for the last fifteen or eighteen years.

year, heard and read a good deal about Mr. Power's and my own "theories", and you have also been told in so many words by some of the daily and medical papers that these investigations have been disproved by others; but I should like to say that when all the evidence is known, which at present it is not, Mr. Power and myself will be able to show that our critics are not quite so infallible as they try to make out.

Now some of the opposition raised against Mr. Power's and my own conclusions is of a kind which would be raised against anyone who, directly or indirectly, interferes with monetary interests; with this opposition I obviously cannot deal; another form of the opposition has been offered in a more or less scientific manner, and it is this with which I will now trouble you.

Dr. Thin, in his address to the British Medical Association, August 1887, said this: "Two men, I am assured, who were engaged as milkers at Panter's farm, lived in 'the Mead', walking to and from their work, a distance not over half-a-mile. There is not the slightest proof that these men conveyed the scarlatinal infection from the 'Mead' to Panter's farm, but it is quite conceivable that they may have done so; the very fact that 'the Mead' contains so many laundries, that scarlet-fever existed at Child's Hill, and that there was daily communication between the 'Mead' and the milking-sheds, justifies a suspicion, or at all events affords a possible explanation of how the milk became infected."

From these words you see that Dr. Thin has neither shown nor succeeded in ascertaining himself that two milkers passed daily between the milking-sheds and "the Mead", but he merely says that he is assured that such was the case. Now, in the first place one would very much like to know by whom Dr. Thin was assured of this, and when was he assured of it. This Dr. Thin, I presume, was assured several months after the farm had become closed, when everyone there had come to look upon Dr. Cameron and Mr. Power as instrumental in bringing about this unwelcome result. In order that there should be no uncertainty about these two men, who, although free from scarlet-fever themselves, might, nevertheless, infect the milk with scarlatinal virus, Dr. Thin suggests that these two men might have done what he was informed from another source to be a habit in the milk trade, viz., adding something to the milk known as "colour", and "this colour" is mixed by the dairyman plunging his naked hand into the milking-can and stirring round the "colour" in the milk. So you have two men coming to the farm every day from an infected locality, "the Mead", about half-a-mile

distant, and for weeks, day after day,—*i.e.*, as long as the milk of the farm has been shown to be possessed of infection,—contaminating the milk by plunging their hands, to which the scarlatinal contagium adhered, into the milk. Now against these statements of Dr. Thin, based on hearsay, we will place the statements of Mr. Power referring to the conditions at the farm during the time the epidemic was being caused and kept up by the infected milk.* This is what Mr. Power says:

"The dairy farmer, though willing to afford, and though indeed desirous of affording us every assistance, was utterly incredulous of the presumptive evidence tending to connect disease with the milk supplied from his farm, and till a late period of the inquiry he remained so. His cowmen were perfectly incredulous also. And truly, having regard to the facts that we first elicited, as to freedom from illness of those at the farm, and as to the peculiar care given to the sanitary affairs of the farm and its dairy, the farmer's incredulity could not but be regarded as justifiable. He had certainly done his best to avoid known conditions of danger, and had not suspected that any such condition, known or unknown, had been present on his farm. The farm was found to have had especial pains taken to render it, as the phrase is, sanitarily perfect. At the instance of one of the London retailers with whom the farmer had dealings, the place had for several years been the subject of special supervision by the Medical Officer of Health of the district, my coadjutor in this inquiry, Dr. Cameron. He had seen that the West Middlesex Company's water was laid on to the farmhouse, to the dairy, and each of the several cowsheds; he had seen specially to the wholesomeness, as regards drainage, cleanliness, ventilation, and the like, of the house, the farmyard, the cowsheds, and the dairy, securing for the last all needful appliances for effectual cleansing of dairy utensils by hot water or steam; and, month by month, he had inspected the farm premises with reference to these and similar details, for the express purpose of safeguarding the milk against contamination of any detectable kind. Further, under the same arrangement, Dr. Cameron had specially attended to the health-conditions of those employed about the farm and their children, with a view to early detection of any malady among them that might by chance injuriously affect the milk with which they had to do. He had even undertaken to observe and to report to the London retailer, by whom his services were retained, on any occurrences of infectious illness in the

* Fifteenth Annual Report of the Local Government Board, Supplement containing the Report of the Medical Officer, page 75.

neighbourhood of the farm, even though it did not directly affect the families of people employed there. The farmer, too, who had consented to the exercise of this supervision over his doings, had attended to every suggestion made to him, and had taken every precaution to secure his farm and his milk against any known sanitary fault or misadventure. He had a separate shed for any sick animal, and a separate shed for the observation of newly arrived animals.

"Thus, with Dr. Cameron's aid, the point was speedily reached at which it could provisionally be affirmed of the Hendon farm milk that, if indeed it had caused scarlatina among its consumers, it had not acquired the ability to do so in any commonly accepted way, such as through unwholesome conditions of water or drainage, or through careless handling of milk or milk utensils by persons carrying scarlatina infection. Nor, during the long subsequent acquaintance with the farm gained in the course of this inquiry, did any reason appear for modifying this conclusion."

The assumption, therefore, of Dr. Thin stands in as marked a contrast to the statements of Mr. Power, as any hearsay and superficial assumption made by an irresponsible person stands to the direct, careful, detailed, and precise statements made by two responsible experts.

The next point that our opponents have brought forward refers to a disease in the cow which, it is alleged, is the same, and has the same origin as the Hendon disease, but has not produced infection of human beings with scarlatina. Now let me first remind you what the conditions of the Hendon disease were. I will quote the concise summary given by the Medical Officer of the Local Government Board, in his Report of 1885-86, pp. vi and vii: "In the end", says the Medical Officer, "he (Mr. Power) has demonstrated beyond reasonable doubt the dependence of the milk-scarlatina of December on a diseased condition of certain milch cows at the farm; a condition first introduced there in the previous month by some animals newly arrived from Derbyshire; and he finds strong circumstantial evidence for believing that the later phenomena of this dependence were brought about through the extension of the diseased condition of one set of animals to another set, after the fashion of an infection."

This disease in the Hendon cows manifested itself in the following symptoms*:—ulcerations covered with scabs on

* The description given by Dr. Cameron in a paper read before the Epidemiological Society, is admittedly a description based partly only on his own observations of the Hendon disease, and is to a certain extent collected from various persons who allege to have seen the disease at

the teats and udder, ulceration, scabs, scurfiness, and loss of hair in patches in different parts of the skin; the animals were thin, and showed slight cough, but showed no rise of temperature. The viscera of two animals killed showed: slight pleuritis, with recent fibrinous deposits and adhesions; congestion and extravasations of blood in many lobules of the lungs; discoloured softened patches in the liver-tissue; ecchymoses and petechiæ in the tissue and in the capsule of the spleen; swelling of, and extravasations of blood in, the lymphatic glands; congestion and glomerulo-nephritis of the cortex of the kidney; in some also interstitial new growth.

With the material of the ulcers the disease was reproduced in the skin of calves; from the tissue of the ulcers of the Hendon cow an organism was isolated by cultivation, of which the characters in the depth and on the surface of gelatine, on Agar-Agar mixture in broth, milk, and on blood-serum, were minutely studied, and hereby its identification became possible.

This streptococcus, when inoculated into calves, produced a visceral and sometimes cutaneous disease, mild in character, which very much resembled that of the Hendon cows. With this organism, by feeding or inoculation, a disease ending fatally in a large percentage was produced in grey house-mice; from the heart's blood of these the same micrococcus was recovered by cultivation.

Professor Axe has presented a Report to the Agricultural Department of the Privy Council Office, in which he states that in two different localities cows had been affected with ulceration of the teats and udder, and that the disease had been introduced at the same time into these localities by infected cows coming from the same dealer in Derbyshire who had supplied those incriminated first cows to the Hendon farm, but in those two localities no scarlet-fever had been produced amongst the consumers of the milk. Now let me here say that this dealer in cows in Derbyshire had, at first, stoutly refused any and every information to Mr. Power; but some months afterwards, when this investigation at the Hendon farm had to a great extent implicated the stock of this Derbyshire dealer, he had altered his mind, and offered the above information. It would seem, in such a case, that some corroboration of the statements and observations of the Derbyshire cow-dealer should have been forth-

various places in former years. That a milker got infected from the cows, mentioned by Dr. Cameron, refers to an informant of previous years, but such infection, as far as could be ascertained, *did not occur* in any of the milkers at the Hendon farm in connection with the Hendon cow disease.

coming. I do not question for a moment that Professor Axe had correctly reported what he heard and saw as to the ulceration on the teats and udder in the two herds, and that he had been told and had observed that the disease spread in each herd, and that it was possibly introduced there by cows coming from the dealer in Derbyshire above-mentioned. But what I should like to be permitted to question is the reliance that ought to be placed on the information offered by the above Derbyshire dealer, *i.e.*, whether about the same time and from the same stock that he supplied cows to the Hendon farm, he also supplied those two other localities. That the disease observed in these two localities is the same disease as that at the Hendon farm there is no evidence whatever; sores and ulcers on the teats and udder of cows of an infectious character are known in veterinary pathology, and they are known to be of various kinds. In connection with this I will mention one striking coincidence that refers to this Derbyshire dealer. It will be no doubt in your recollection that an extensive outbreak of scarlet-fever occurred at and about Wimbledon at the end of the last and the beginning of this year. This epidemic was traced distinctly to milk coming from a particular farm. Now the curious thing about this farm is that it belongs to a dairy farmer of the name of Keevil, who happens to be the brother of our Derbyshire dealer, and who had received some of his cows from this same dealer, shortly before the outbreak of scarlatina in Wimbledon.

The way that this Wimbledon milk had received the contagium has not been ascertained by Mr. Power, but the fact that the Wimbledon dairy farmer Keevil had got cows from the Derbyshire dealer Keevil, who had supplied the Hendon farm, is, I think, extremely suggestive.

I have, in my last year's inquiries, isolated from acute cases of human scarlet-fever, both from the heart's blood as also from the blood of the living, a micrococcus which in all respects is identical with that of the Hendon cows. Experiments made last year on calves and on grey house-mice with cultivations produced the same visceral disease as did those of the Hendon cows. I have, in the course of the spring and summer of this year, inoculated six milch cows, recently calved (about two to three weeks), and have produced in two of them ulceration on the teats; in all of them scabs, scurfiness, and loss of hair in different parts of the skin, and in all when killed there were the same visceral lesions as were observed in the Hendon cows, *i.e.*, in the lung, pleura, and lymph-gland, liver, spleen, and kidney. There

was no distinct rise of temperature, and the animals, just like those at Hendon, fed well, gave plenty of milk, but got thin, and some showed slight cough. From the blood and pericardial fluid of the calves inoculated last year with cultures of the micrococcus of human scarlet-fever, and from the milch-cows experimented upon this year with similar cultures, the same organism was recovered by culture. Let me add that I have found in the kidney, liver, and lung, and spleen particularly, and in the ulcers of the skin of the Hendon cows; of the calves and cows experimented upon with the organism of human scarlet-fever; further, in the kidney, skin, and liver of human scarlet-fever, the same kind of organism, in the form of diplococci and short chains.

This is, briefly, the evidence which I have to offer as to the bacteriological and anatomical part of the inquiry. Part of this work (last year's) has just been published in the Reports of the Medical Officer of the Local Government Board; another part of the work (this year's) will not appear, I am afraid, before next year's Report comes out.

Now Dr. Thin has told us in his address that the organism which I have isolated and described, of the Hendon cows and of human scarlet-fever growing on gelatine, coincides with that of the common streptococcus pyogenes found often in pus. Dr. Woodhead has expressed himself in a similar sense. This suggestion is perfectly unwarranted. The characters in cultivations in the different media of the organism which I have called the micrococcus scarlatinæ bear only a slight resemblance to the streptococcus pyogenes, no greater than they do to six other species of streptococci. I show you here (1) streptococcus of opaque vaccinia, (2) streptococcus of foot-and-mouth disease, (3) streptococcus of human puerperal septicæmia, isolated by Dr. W. R. Smith, (4) the streptococcus of scarlet-fever, and (5) the streptococcus of an infectious disease of the cow, showing itself in ulceration of the teats. When you look at these cultivations on the surface of gelatine and in the depth of gelatine, you will not easily detect any difference, and the same applies to the streptococcus pyogenes and the streptococcus of erysipelas. To all these a description such as quoted by Dr. Thin from Flügge's book on micro-organisms would fully cover the appearances of all these different species; for in all of them you have the same kind of greyish, whitish, round, discreet dots on the surface of the gelatine, the same small spherical spots, more or less brownish, and closely placed side by side in the track of the needle used for the inoculation of the depth of the gelatine.

And yet only one imperfectly informed of the different characters presented by these different species when growing in different culture-media could offer such an assertion as that offered by Dr. Thin, viz., that because the growths appear so similar on the surface and depth of the gelatine they are the same. Very little trouble would show that all these seven species are totally distinct species. The varying rapidity with which the growth appears, the manner of progress, and the extent and aspect of the colonies of the growth as time goes on, the manner in which the organism grows in broth, in milk, on Agar-Agar mixture, are different in all these different species.* Above all, their mode of action on suitable animals is altogether different.

The streptococcus of foot-and-mouth disease is inactive on rabbits and mice. The streptococcus pyogenes produces in mice and rabbits local suppurations which may lead to metastatic pyæmic processes of the viscera; the streptococcus of erysipelas produces, on subcutaneous inoculation into the ear of rabbits, a true and progressive erysipelatous rash; the streptococcus vacciniæ has no effect on mice and rabbits; the streptococcus of human puerperal septicæmia produces on subcutaneous inoculation of white mice septicæmia and death in a few days, and on the ear of a rabbit has an effect similar to, but slighter than, that of the streptococcus of erysipelas; the streptococcus of scarlet-fever produces in a small percentage of white and in a large percentage of house-mice, in the course of one, two, or more weeks, a distinct visceral disease leading to death; on rabbits it has no effect.

There can then be no manner of doubt that cultures in various media and the animal experiment prove that they are distinct and different species of micrococci, though they all belong to one group of micrococci, having certain characters in common, as to form chains in fluid media, and to grow on the surface and in the depth of gelatine, in a manner which, for the different species of the group, presents certain similar characters.

Owing to the kindness of Professor Brown, I have been enabled quite recently to observe two cows which belonged to a herd in which a contagious ulcerative disease of the teats and udder prevailed, which was, and is supposed to be, the same as that in the Hendon cows. No cases of scarlet-fever have been known in connection with these cows. The

* In a paper read by Dr. Crookshank at the Pathological Society on December 15th, he also expresses the belief that all these different streptococci are the same; Dr. Crookshank is evidently but imperfectly acquainted with the distinctive characters of streptococci in their biological and cultural relations.

ulcers on the teats were very extensive, superficial, irregular, and covered with brown crusts, the teats themselves much swollen.

Taking off the crusts and scraping the superficial part of the ulcers, and then taking material from the deeper part of the ulcer and inoculating gelatine, I succeeded in isolating four different species of micrococci—(*a*) micrococcus citreus; (*b*) micrococcus aurantiacus; (*c*) staphylococcus pyogenes; (*d*) a micrococcus, which in plate cultivation, on the surface and in the depth of gelatine, presented characters resembling the micrococcus scarlatinæ obtained from the Hendon cows and from human scarlet-fever. But it soon became apparent that it is an altogether different species; it grew much more slowly, and its colonies (minute circular dots) are much more transparent than those of the Hendon cow organism; further, while the micrococcus of the Hendon cow and of human scarlet-fever grows well in particular broth, producing after two days, at 35 deg. C., a distinct turbidity, and also well on the surface of particular Agar-Agar mixture, as isolated whitish dots; the micrococcus in question, isolated from the ulcer of Professor Brown's cows, does not show any growth on this Agar-Agar mixture, or in the broth, even after a fortnight's sojourn in the incubator at 35 deg. C., but grows well in these media at 20 deg. C.

One of the animals in question was killed after about a fortnight, when the ulceration and swelling of the teats was still considerable. The left hind-quarter of the udder was found very tense, and greatly swollen; on incision, a large quantity of thick, grumous pus was found in this portion. The lungs were normal, the spleen was normal, the liver showed a few whitish irregular spots, and the kidney presented a few fatty patches on the surface of the cortex; none of the lymphatic glands showed the hæmorrhage into their substance.

During life the animal had, all the time while under observation, high temperature, varying between 105 and 104 degs. Fahr.,—and no wonder, seeing the extensive suppuration that was going on in the udder.

There can then be no question that this cow did not show the same disease that was observed in the Hendon cows, nor was the same organism obtained. The second cow showed on *post-mortem* examination the same appearances as the first. By cultivation the same organism was obtained as from the first cow.

It has been urged that the disease which was observed in the Hendon cows, and the visceral and cutaneous disease that is produced with cultures of the micrococcus from human scarlatina in calves, and during the present year in

the milch-cows, is not the same disease as human scarlet-fever. If proof is expected that fever, ulceration of the tonsils, and scarlatinal rash should be produced in the calf and in the cow, by the virus of human scarlet-fever, I am afraid such proof will not be forthcoming; but that the micrococcus of human scarlet-fever produces in calves and milch-cows a cutaneous and visceral disease which, though mild, is nevertheless distinct, and is the same as produced by the micrococcus of the Hendon cows, of this I have had ample evidence. True, there is no definite rise of temperature, and the animals feed well, and appear otherwise not affected, though many of them get thin, and show a slight cough; in some no distinct cutaneous disorder is produced, in others this is distinct (*e.g.*, the scabs, scurfiness, and loss of hair in different parts, and in milch-cows even a few ulcers on the teats and udder), but it is necessary to make a *post-mortem* of the animals in order to show the presence of the visceral lesions above referred to.

Professor Axe and also Dr. Thin lay it down that unless a disease is produced in animals which is identical in every respect with human scarlet-fever, no proof has been given. I must altogether demur to this proposition; there is, as far as I know, no theoretical reason why this should be essential. Who can deny to consider cow-pox and human small-pox as closely related diseases? Who can deny that a localised carbuncle, produced by infection of the skin with anthrax blood of a sheep or cow, is the same disease as the general anthrax infection of the sheep or cow from which the virus is derived? Or who would deny that wool-sorters' disease is due to the same bacillus anthracis as the above localised carbuncle? And further, is it not maintained by the majority of observers—pathologists, surgeons, and physicians—that scrofula and lupus are the same disease as tuberculosis; and yet can there be a more striking difference between lupus in a human being, and general tuberculosis in man, or perlsucht in the bovine species? I think, if we can show that one and the same species of organism is present in a disease in man, and is also present in an animal affected naturally with a certain disease, and that this species of organism grown in artificial cultivations produces in experimental animals the same disease, no matter whether derived from the human or the animal body, we are justified in saying that these two diseases, the one in man and the other in the animal, are the same. But in our case of human scarlet-fever, and the Hendon disease in the cow, there is

unquestionably a resemblance between the two diseases, though they are certainly not identical in symptoms, any more than a general anthrax-infection is with a localised carbuncle, or perlsucht in cattle is with lupus.

The last point of opposition urged is that of the so-called bacillus scarlatinæ of Edinburgh. Dr. Edington has isolated from the epidermis during the stage of desquamation of scarlet-fever cases a bacillus which he considered to be the bacillus of scarlatina. He maintained that this bacillus is present in all cases of scarlet-fever in the blood before the third day; it then disappears from the blood in order to appear in the skin, whence it is discharged into the epidermis, and here forms spores during the stage of desquamation. In the first place let me remind you that various species of bacilli and micrococci have been hitherto isolated from the epidermis of the normal skin. In the second place, this bacillus of Edington has not been found in all early cases of scarlet-fever by a Committee of the Medico-Chirurgical Society of Edinburgh, appointed to test Dr. Edington's assertion, only in the blood taken from a prick in the finger of two out of ten cases of scarlet-fever on or before the third day. Anyone acquainted with the modern methods of bacteriological inquiries will agree with me when I say that there is no difficulty whatever for anyone who devotes the necessary time to it to isolate by the modern exact methods new species of bacilli or bacteria in any material he chooses to select, be it epidermis of normal or pathological skin, fluid of the mouth, mucous of the throat or lungs, or any other material, because the systematic branch of bacteriology is only in its infancy; owing to the exact methods of cultivation now employed, the morphological characters of bacteria can be studied in a more satisfactory and perfect manner than was possible in former years, before Koch introduced the method of cultivation in solid media.

The point, therefore, in the present state of bacteriological researches is not whether in any morbid material of the body this or that new species is present, but whether in its distribution and its action it bears any definite relation to that morbid process. Now everyone acquainted with these fundamental conditions will understand that one of the first and essential conditions would be that a microbe which is to bear any relation to scarlet-fever must be found in the blood or tissues in a definite distribution. Examining the desquamating epidermis and finding a new species of bacteria may mean nothing more than this: besides those several species already imperfectly known to exist in the

epidermis, another species has been added; which, in itself, is no doubt an interesting discovery, as much as, for instance, a new species of diatome or a new beetle is in a locality where no one has seen it before. But this new addition to the list of bacilli in the epidermis may have nothing whatever to do with the scarlet-fever. True, Dr. Edington tells us that he has not met with this bacillus in the epidermis during the stage preceding desquamation, but it is quite permitted to doubt whether sufficient time and care has been devoted by him to this point, or to the examination of other normal and morbid epidermis, seeing that in the first place he is "young and enthusiastic" (for so he is described by Dr. Jamieson); and, secondly, it was a postulate by Dr. Jamieson, that in order to confirm his (Dr. Jamieson's) view as to the nature of the scarlatinal virus (as described in *The British Medical Journal* for June 1887), this must be discovered as spores in the epidermis of scarlet-fever cases during the stage of desquamation.

As to the presence of the same bacillus in the blood taken from a prick in the skin of scarlet-fever cases, surely no one can say that the bacilli were not derived from the epidermis. In order to show that this bacillus is present and circulates in the blood of scarlet-fever patients on or before the third day, it is essential to find it in the blood, but without any danger of contamination with epidermis; *e.g.*, taking the blood directly from the heart of a case that died on or before the third day in scarlet-fever—taking, of course, care that the chest and pericardium are opened with the proper precautions; and similarly, in order to be enabled to say that this bacillus passes out during the stages of desquamation into the epidermis, where, being exposed to air it forms spores —it is essential to show the presence of these bacilli in the tissue of the cutis itself previous to desquamation.

It is therefore clear from this that the preliminary conditions that have to be fulfilled, are: to find this bacillus in the blood of the circulation—*e.g.*, the heart—before the third day, and to find it in the cutis before desquamation begins. Before anyone can make any assertion as to the relation of this bacillus to scarlet-fever, those two essential conditions must be first complied with. Now neither Dr. Edington nor the Committee appointed by the Medico-Chirurgical Society of Edinburgh have even attempted to do so. No *post-mortem* was made; the blood of the circulation, or of the heart, was not tested for the presence of this bacillus; no sections of the skin or organs were made to discover this bacillus. And such a research as that of Dr. Edington

was called by Dr. Crookshank, at the British Medical Association, a "fairly complete" bacteriological research. I can say this, that I have had the opportunity of examining the blood of the heart at the *post-mortem* in cases of scarlet-fever on and before the third day, as, also, others have done in previous years, and I have failed to discover this bacillus by microscopic examination or by cultivation. Nay, in a number of cases in which I took the blood from the finger, I have not been able to come across this bacillus,—probably my methods were different from those of Edington. I have examined a large number of sections, as also others have done, through the skin and organs, kidney, liver, lymphatic glands, and spleen, of cases of scarlet-fever, and have not found this bacillus.

The organisms which I found in the heart's blood and in the skin and kidney were diplococci and short chains of micrococci, except the ulcerated tonsils and the swollen and purulent lymphatic glands of the neck, where a variety of different bacilli and micrococci were present.

I have had an opportunity of testing this so-called bacillus of scarlatina (sent to me kindly by Dr. Edington) on animals. Masses of bacilli and spores were injected under the skin, but no result of any kind was hereby produced, either locally or generally. The bacillus forms readily, after a day or two, a thick coherent pellicle on the surface of broth-cultures; such pellicle is easily lifted out, washed in salt solution to free it from chemical products that may have been produced in the culture-fluid, and such pellicle, consisting of masses and felt-works of the bacilli, with copious spores, can then be easily injected into animals.

This so-called bacillus of scarlatina is a bacillus which belongs to a well-defined group of species which in former years would have been considered as bacillus subtilis, viz., rods of a certain length and thickness, mobile; they have a tendency to form long chains or leptothrix; they grow rapidly in broth and form here on the surface a scum, which is already well developed after twenty-four hours; the scum thickens during the next days, and becomes corrugated, the fluid itself remaining clear, but gradually becoming brownish, the more brown the longer time elapses; they liquefy gelatine rapidly, and after some days form also here a scum on the surface; they form in a few days a corrugated scum on the surface of solid Agar-Agar mixture; they grow rapidly in milk which is curdled, and by which a white precipitate is formed, while the fluid, at first turbid, gradually becomes clear; on its surface there is a thick pellicle.

In plate-cultivations in gelatine, the bacilli form minute

whitish colonies, at first—*i.e.*, during the first day—angular, after thirty hours or more, gradually liquefying the gelatine, and forming circular colonies of liquefied gelatine with an opaque or slightly granular outline, and a central, whitish spot due to precipitate of masses of bacilli. On potato they grow rapidly, and form a more or less corrugated layer, gradually turning brownish.

Now, I know of five different species, all notoriously septic bacilli, which show these characters besides the Edinburgh bacillus; they show, nevertheless, slight differences, and may be considered as different species, but as to those general characters just mentioned, there is no doubt they belong to the same group of organisms.

To sum up, then: the Edinburgh bacillus, judging by its mode of growth, clearly belongs to a group of species of bacilli which are notoriously septic bacilli; it has been found in a small percentage of cases of scarlet-fever in blood taken from a prick of the finger; its spores occur in the epidermis of cases of scarlet-fever, amongst other species of bacteria; it has no action when the bacilli or spores are injected subcutaneously, and in a pure state, into animals. You will, I think, agree with me that, after such evidence, it would be quite unjustifiable and premature to call this bacillus the bacillus of scarlet-fever. This whole question of the etiology of scarlet-fever is no doubt an extremely difficult one; and only by combined work on an extensive scale, and by careful and accurate methods, can we hope to get at the truth of the matter; and in these efforts no criticism should be shunned or objected to, but, on the other hand, it must be criticism made in an unprejudiced and scientific spirit.

ON THE MICROBIAN DOCTRINE OF YELLOW FEVER.

By JUSTIN F. DONOVAN, M.D., Surgeon R.N.

(ABSTRACT).

(*Read: February 8th*, 1888.)

YELLOW FEVER seems to have practically disappeared from the Navy of late years; except an occasional outbreak among the naval forces of Port Royal, Jamaica, this pestilence has ceased to be the scourge it was when Lind wrote: "It is reputed that in the early part of the eighteenth century this island (Jamaica) buried to the amount of the whole number of its white inhabitants once in five years."

The writer had an opportunity of studying the disease clinically in 1882, during an outbreak of yellow fever at Port Royal among the naval community. Since then he has devoted some time to the study of its literature, and, in submitting to this learned Society the following *résumé* of Professor Freire's researches on the "Doctrine microbienne de la fièvre-jaune et ses inoculations préventives", he must claim its indulgence for the many imperfections which the paper contains. Whatever of merit or of novelty it may be found to possess is entirely due to the learned Brazilian Professor; and, in submitting a condensed translation of this monograph, his sole object has been to bring to the notice of this Society a work of sterling merit and originality on a subject whose literature, though voluminous, has, up to the present time, formulated no scientific doctrine on the genesis of this dire pestilence.

It was reserved for Professor Domingos Freire, of Rio de Janeiro, to discover, describe, and classify a vegetable, acrobic organism, to which he gave the name of *Cryptococcus-xanthogenicus*, as present in the blood, urine, ejecta, etc., of patients suffering from the disease. At page 244 of his monograph the following passage occurs bearing on this question: "My microscopical observations clearly demonstrated the co-existence of the micro-organism (*Cryptococcus-xanthogenicus*) with the evolution of the disease; subsequently it will be proved that it is dependent on these same organic elements, and,

further, that the symptoms of yellow fever are the outcome of this physiological development. Also, that clinical experience, too much depreciated at the present day in the elucidation of such questions, furnishes us with the most vital test for establishing this conclusion."

In investigating the relation of micro-organisms to disease the following conditions require to be fulfilled, according to Koch, before it can be established that a particular micro-organism is probably the cause of a particular disease. 1. The presence of the micro-organism in question in the blood or the diseased tissues of man, or of an animal suffering from the disease. 2. To cultivate artificially these micro-organisms in suitable media, excluding the accidental introduction of other micro-organisms, and to cultivate them from one culture to another for several successive generations. 3. When thus cultivated for several successive generations it is necessary to introduce them into the body of a healthy animal susceptible to the disease, and to show that this animal becomes affected with the same disease. 4. It is necessary that in this animal so affected the same micro-organisms should again be found.

As Professor Freire has fulfilled these conditions in his experimental researches on yellow fever, he has established a strong *primâ facie* case in favour of the doctrine he maintains. We will examine in detail each of these requirements which the Professor has effected in his experimental researches. The following extracts from his writings will illustrate those points sufficiently:—

"The era inaugurated by Pasteur", writes Dr. Freire, "has happily brought a truce to the rival combatants (contagionists and non-contagionists) who expended their intellectual powers in pursuing a conquest which they failed to accomplish. They were like mariners without a compass or a rudder to steer by. Following the precepts of this eminent master, I discovered, in 1880, the microbe of yellow fever. I sought to demonstrate the close relations which exist between it and the disorders which constitute this morbid condition; and so successful was I, that in less than three years I found that my work was being discussed in Europe and America."

"Corroboration of the constant presence of the xanthogenic micro-organism has been made by various physicians in Mexico, Peru, Havanna, and in Central America. . . . ."

"The means to which I resorted for establishing these conclusions were: experimental physiology and comparative pathology; clinical facts, the study of the course of epidemics,

the vague influence of telluric and meteorological agents. Such ideas have had their day; it was a field rendered barren by the illusions of observers. . . . ."

"I have succeeded by my experiments in giving a body and a form to the supposed *miasma*, to the disputed virus of yellow fever. I have brought it to light under a material form; pointing it out as endowed, on the one hand, with a microscopic organism of a phyto-microbe, with a fixed evolutionary period; and, on the other hand, with toxic substances of the alkali-ptomaïne class, products of elaboration of the secretion or abnormal assimilation effected in the tissues by these very microbes."

"Already in a great many diseases the determination of the actual cause has been arrived at. Splenic fever, fowl-cholera, typhoid fever, septicemia, are among the number. Yellow fever may now be placed in the same category. This disease has its well-known cause, its specific agent, its materialised virus (*virus materialisé*), viz., the *cryptococcus xanthogenicus*. . . . ."

"When we diligently and carefully follow the course of development of the germs which produce yellow fever we find little specks like minute grains of sand, at first almost imperceptible, but gradually increasing in diameter, from 0.001 mm. or 0.002 mm. to 0.007 mm. or 0.008 mm., and even larger. When they arrive at maturity these cells burst and discharge their contents, composed of spores mixed with a glutinous substance of a yellow colour, made up of a pigmentary substance and of the liquid of the parent cells. Such being a *résumé* of the evolution of the microbe outside the organism, in the earth, in water, in alimentary substances, it follows that the microbe of yellow fever enters the human organism in various stages of development, seeing that in the air which we breathe, and in the food and water which we ingest, the spores which have left the parent cell are accompanied by fully developed organisms, and even detritus, resulting from the disintegration of cellules. . . . ."

"The minute dimensions of the xanthogenic organisms, even when fully developed, may be conjectured from the fact that a power below 450 diameters is insufficient to distinguish the microbes. It may be imagined how easily they find an entrance through the epithelial cells, and thus become absorbed, either by the chyliferous vessels and the venous capillaries of the intestines, or by the bronchial or pulmonary mucous membrane through which they enter the circulation, and in a short time induce a febrile condition. Hence, in the first stage of yellow fever, as soon as the

primary symptoms manifest themselves, it is not unusual to meet with fully developed organisms by the side of others more numerous, which have only passed through the first phase of their existence.

"This fact is in accordance with the clinical observation that there is no fixed time for the first stage of the disease. If none but the spore-germs could enter the organism the duration of this stage (save some modifications dependent on the constitution, the temperament, or the idiosyncrasies) would be pretty accurately determined. However, from the moment that the fully developed microbes and others in a less less mature condition enter the economy, it is evident that the duration of the first stage will depend more or less on the relative quantity of microbes which have arrived at an advanced state of development. If the adult microbes have gained an entrance in greater proportion than the less mature ones, the transition to the second stage will be much more rapid, consequently the duration of the first stage will be shorter. On the other hand, if spores and only a few adult microbes have penetrated, the former (spores) will require a longer period to reach the age of proliferation which marks the limit of the second stage, and it is evident that the initial stage of the disease will be much longer. Moreover, it is not difficult to account for the presence of a greater number of the latter than of the more mature microbes. Spores and granules can more easily insinuate themselves through the interstices of tissues, not only on account of their very minute size and of their more or less spherical form, but also on account of the rapid movements which they execute, movements of which the adult microbes are deprived."

"It may naturally be asked, What time is necessary for the absorption of microbes, and of their destructive consequences when introduced? We may answer, that generally the time is very short. I had an opportunity this year (1884) of observing cases among individuals who had been only one or two days at Rio de Janeiro, and in whom well-marked yellow fever developed within that short time. The period of incubation, consequently, may be of very short duration. A few days suffice for the spores, which find an entrance through the respiration or alimentation, to proliferate and to set up symptoms more and more grave. . . . ."

"This rapidity of action of the xanthogenic microbe is analogous to that observed in splenic fever, which, according to Chamberland, is not always preceded by premonitory symptoms, the onset is frequently quick (*brusque*) and the termination rapid. The same writer mentions that, having

inoculated a guinea-pig with a few drops of urine containing bacteria, which was characteristic of splenic fever, in thirty hours after its blood swarmed with bacteria. . . . ."

"The experiments which I performed on animals with liquids containing the microbes of yellow fever furnished similar results; cultures of the microbes introduced subcutaneously into guinea-pigs were followed by the appearance of similar microbes in different parts of the animal's body."

"In the early stage of their development these products are endowed with very rapid movements of rotation as well as progression. In the mature state these cellules are much larger, becoming sometimes ovoid in shape. Masses of pigment, yellow, greenish, or violet colour, are plainly visible in the interior of the cellules with a 740 diameter. Spores are enclosed in these. At first I was of opinion that these cellules multiplied by congregation—by the union of sporanges and antherides—but subsequent observation, carefully made and repeated a great many times, demonstrated that such was not the case, that the tendency of union among considerable masses of these organisms is entirely fortuitous. Reproduction, which I have frequently observed, is carried out by rupture of the parent envelope. Each mature cellule, having been transformed into a little sac, full of spores and pigment, bursts and scatters its contents in the liquid which surrounds it. The spores thus discharged increase in volume, and proliferate in the same manner as the cellules. We may observe that the cellules which are about to rupture, or are close to this phase, are motionless; and the spores which are adherent to the masses of yellow pigment, and which are glutinous in consequence of being covered with liquid ptomaïne, whose consistency is more or less oleaginous, are likewise motionless. Frequently the spores which are fixed to masses of yellow pigment become grouped in a regular or more or less symmetrical form, sometimes pear-shaped, at other times like a pineapple, etc. The membranes of the cellules, after their rupture, form irregular *débris*, ash-colour, black, and sometimes violet or greenish, and are carried along the circulatory current, inducing functional changes which have an important bearing on the pathogeny of the fever, which we shall see later on. The yellow pigment dissolves in the blood, and in this way infiltrates through all the tissues."

Résumé of the Evolution of the Cryptococcus-Xanthogenicus.

They (cryptococci) flourish in the blood, in the vomit, in the brain, in the muscles, in parenchymatous organs, and generally in all the tissues and fluids of the economy, and *in the fluids of* albuminous cultures.

They commence as small black dots, which pass to the stage of round cellules, with grey or black borders, highly transparent, sometimes variegated, with granular interior (when they become mature), and containing yellow and greenish pigment.

The mature cellules split up.—1. At different points at the same time: this is of most frequent occurrence. 2. At one spot, the spores escaping by one orifice (rare). 3. By a circular section, the cellule taking the form of a pyxide (rare).

Sometimes the spores are scattered without order, sometimes they adhere to the pigment without any regular form; sometimes they stick together, assuming different shapes, as that of a pear, pine-apple, etc. The *débris* occurring or resulting from the disintegration of the cellules form different amorphous heaps, black, violet, or greenish colour.

"The presence of the xanthogenic microbes in the earth which cover the graves of yellow-fever corpses is unfortunately a fact which can be easily verified by those who wish to make the necessary microscopic examinations. Having gone to visit the Jurujuba Cemetery, wherein those dying in the Maritime Hospital of Santa Isabel are interred, I gathered, from a foot below the surface, some of the earth from the grave of a person who had died of yellow fever about a year ago. This earth showed nothing remarkable in appearance, smell, or other exterior characters; but, on examining a small quantity microscopically (740 diameters), I found myriads of microbes exactly identical with those found in the vomitings, urine, blood, and other organic fluids of persons with yellow fever, *i.e.*, cells of the cryptococcus-xanthogenicus in various stages of development. Many of these organisms were endowed with spontaneous movements. Yellow masses, due to the pigment of the cells, were also seen, as well as vibriones with rapid movements. These observations, which were verified in all their details by my assistants, show that the germs of yellow fever perpetuate themselves in the cemeteries, which are like so many nurseries for the perpetuation of new generations, destined to devastate our city. These germs spread into the atmosphere through the pores of the earth, others are disseminated by the rains to the squares and streets.

"The presence of the microbes of yellow fever in cemeteries is completely in accordance with Pasteur's observations in relation to anthrax. We recommend this fact to the attention of hygienists. It appears to me that cemeteries ought to be removed to places as far as possible from the city, where the prevailing winds cannot carry with them the spores of the microbe.

"A guinea-pig, whose blood was previously examined and found to be normal, was enclosed in an atmosphere in which some earth from the infected cemetery was placed: in the space of five days the animal died, and the autopsy revealed all the lesions which are ordinarily met with in those who die from yellow fever; its blood simply swarmed with cryptococci in different phases of their evolution. The urine, boiled and treated with nitric acid, gave the reaction of albumen. A guinea-pig placed in an atmosphere saturated with spores of organisms died in seven days with all the symptoms of typhus icterodes; the blood of this animal was injected into another, which died in six days; not content with these facts, we mixed some of the earth of the Jurujuba Cemetery with pure water, and having taken a few drops of this mixture, diluted with a gramme of distilled water, and inoculated a guinea-pig with it, the next day the animal died; this animal's blood was inoculated into another, which died three days after. . . . "

"Yellow fever, being at first contagious, does not delay long in becoming infecto-contagious—our experiments on animals have furnished us with direct proof of this. The air of our laboratory was infected in such a manner that latterly all the animals coming direct from the market for our experiments contracted the disease and died in a few days. In the face of these facts it is necessary to have recourse to special hygienic measures, such as preventive inoculation, or cremation. If we extol cremation it is not from caprice, but influenced by the force of circumstances as an energetic measure of public hygiene. As to the preventive inoculations, the specific and contagious nature of the disease, its transmissibility, and its property of attacking the same individual but once, have induced us to think that the question was not insoluble. The entire difficulty consists in finding the best means of attenuating the virus. We shall see that this difficulty has been surmounted, and the results which have been obtained have proved that vaccination applied to yellow fever is a reality. One fact which vividly strikes the observer who studies yellow fever is the periodical rhythm manifested by the epidemics, which in our climate (Brazil) occur only at a certain and fixed time of the year, between February and June. The influence of season on the evolution of the microbe of yellow fever is consequently very powerful. A number of guinea-pigs were inoculated with gelatine cultures fertile in characteristic organisms, their energy having been proved by the inoculations causing the death of several of these animals; however, similar inoculations performed in

July and August, furnished negative results; the animals had but a slight rise of temperature, but survived the effects of the inoculation. . . . ."

"Blood found in cases which have died of yellow fever is black and glutinous. When hematemesis supervenes, the ejected blood assumes a dark colour; hence the confusion of ideas entertained by some physicians as to the cause of the black vomit. They do not differentiate between the two causes—black vomit without a trace of blood, and black vomit accompanied with hematemesis or hæmoptysis. In the first case, the colour of the vomit is due entirely to the pigment elaborated by the microbes during their evolution; in the second case the colour of the vomit, besides this cause, is due to the deoxygenation of the red blood-corpuscles induced by these organisms.

"In addition to deoxygenation there is the production of ptomaïnes, which are a thick, oleaginous material, the work of the microbes. We can easily recognise the cause of the glutinous consistence, pitch-like, of which we have previously spoken, and which is equally found in the bodies of animals dead of splenic fever. Different manifestations of yellow fever are to be attributed to this viscidity; the arrest of *stasis* in the capillary circulation, as well as *hæmatosis*, are amongst its effects. The elaboration of new elements by the microbe can scarcely be questioned; indeed, all living beings perform some work, which consists, amongst others, in disintegrating the materials which serve nutrition, assimilating certain elements which result from this decomposition, and rejecting others as useless and unassimilable. To destroy for the purpose of creating appears to be the great law which not only the minutest, but all animated beings obey. During its existence the microbe of yellow fever not only elaborates liquid ptomaïnes, as well as different gaseous products, but it also absorbs oxygen, disengages carbonic acid, nitrogen, as well as a gas which is rapidly absorbed by sulphuric acid. The functions of the *cryptococcus-xanthogenicus* are therefore sufficiently complex: its mission is to manufacture a yellow colouring matter, a black colouring matter (liquid ptomaïnes), nitrogen, and carbonic acid gases. It lives at the expense of the red blood-corpuscles and other albuminoids, and induces disintegration in different tissues whose functions it perturbs.

"The question of ptomaïnes opens up an important field for discussion, whose aim it is to establish if certain parasitic diseases are due to the production of ptomaïnes during the course of the morbid process, or to the destructive effects

of the micro-organism. Ptomaïnes are found both in the blood and in the urine of patients; in the ejected matters they are in the form of butyrite of ptomaïne, verified by decomposing a small quantity of the material by a certain degree of heat and sulphuric acid, which sets free butyric acid. My experiments show that several ptomaïnes are found in yellow-fever patients. We have demonstrated that these products are due to the elaboration of microbes.

"A ptomaïne of yellow fever may be defined as a liquid alkaloid, of a pungent aromatic smell, volatile, of a slightly yellowish colour, oleaginous, forming an emulsion with water, soluble in alcohol and ether, turns red litmus paper blue, and contains a large proportion of nitrogen. It is inflammable; heated on a thin plate of platinum, it burns with a yellow flame, leaving a residue of carbon—carbon, 20.976; hydrogen, 15.098; nitrogen, 63.926: 100.000.

"A litre of 'black vomit' gives about five grammes of ptomaïne. This alkaloid is precipitated by iodide of potash as well as by tannin; it gives, with ferro-cyanide of potassium, perchloride of iron, and a few drops of hydrochloric acid, a beautiful emerald-green colour, like that produced by quinine and chlorinate of ammonia. It gives another curious reaction, which may serve to characterise it, viz., it sets free iodide of starch.

"*Typical black vomit.*—I took a small quantity of this vomit and found in it an enormous number of small granules and vibriones, endowed with very rapid movements. I also saw amorphous masses of a yellowish colour, and a great number of large cells of a smoke colour, motionless, isolated, or grouped together two and two, three and three, or more. These cells are the cryptococci of which we have been speaking. On the following day the same vomit, when examined, was found to contain a great number of granules (recognised the previous day), increased in size, and becoming more opaque. Some mycelia were also noticed, as well as the yellowish masses strewn with granules. On putting a drop of distilled water on the slide, some vibriones and granules commenced to execute a tumbling motion. On the third day the cells appeared still darker, their opaque margins increased in density; granules, vibriones, and bacteria were still visible, which became more apparent by the addition of a drop of water. On the fourth day there were a considerable number of large-sized cryptococci. . . . . On the sixth day many cryptococci of various dimensions were to be seen. Their great number indicates their proliferation by means of conjunction. A period of four or five days is there-

fore necessary for their fecundation. The innumerable granules, which surround the generative cryptococci, follow the same evolution as the primitive granules, and in this way perpetuate a series of generations, which continues as long as the germs find material for their nutrition. The foregoing microscopic observations show us that the corpuscles of yellow fever are soft *algæ*, which we have designated *cryptococcus-xanthogenicus*.

"*Yellowish brown vomit.*—Having taken a particle of this vomit, which was ejected from a moribund patient, and placed it under the microscope, an enormous number of molecular granules, scattered or adhering to the organic detritus, was found in it. The scattered ones were endowed with rapid movements. On the following day the same granules and filaments of bacteria were observed, which, as well as we know, are none other than metamorphosed vibriones. Cryptococci of variable size were also observed, some very small, others larger, and some giant-cells. They all presented a clear nucleus. Having moistened the slide, some granules began to move. I remarked in the same vomit large mycelia, crooked and thick (*leptothrix*); they represent a more advanced form of bacteria. On the fifth day the same cryptococci were seen to be on the increase, with movements of a vibrating character. . . . . On the seventh day a prodigious number of granules, some of them having a diameter of 0.002 mm., presenting a dark border, were observed. . . .

"In blood and in pus, products analogous to those which we described as existing in vomited matter were found. . . .

"We stated before that the black colour of the vomit was due to the *débris* of the cells of the cryptococci after their rupture. We performed experiments to prove that hæmoglobin is not the cause of the black colour of the vomit, for typical 'black vomit' does not contain a single globule of blood, except there may be some concomitant hæmatemesis or epistaxis. It is easy to corroborate this fact by means of the microscope, which fails to demonstrate a trace of blood in typical 'black vomit'. If there is not a single drop of blood, whence comes the hæmoglobin to communicate to the vomit this colour of burnt paper which characterises it? To place the matter beyond doubt, we had recourse to a method even more decisive, viz., the spectrum test.

"We did not notice the slightest alteration in the position of the spectrum which characterises hæmoglobin, which we know shows two absorption-bands of unequal size, situated between D and E, and that the hematine is known by the presence of the absorption-band which appears between C

and D. Consequently, the microscopical examination, filtration, the insolubility of the black elements, and the spectrum analysis demonstrate that it is not the hæmogobin or its products on decomposition which communicates the black colour to the vomit in yellow fever, as pathologists have heretofore believed. This colouring matter is due to the disintegration of cryptococci, which ensues after the act of fecundation and the dispersion of germs. In like manner the yellow colour of the vomit is not always due to the presence of bile pigment. We examined yellow vomit of the first and second stages, and not a trace of bile has been revealed by chemical analysis. Biliary acids and their derivations are easily detected by Pettenkoffer's test. The colour of the vomit is due entirely to the pigment elaborated by the microbes during their evolution.

"I made cultures with complete success in beef-broth, in milk, and in gelatine solution; in these, proliferation of the germs is very profuse.

"On the 25th of March 1883 I placed some blood of a yellow-fever patient in gelatine culture contained in a sterilised flask (Pasteur). Five days after, on examining the flask by reflected light, a slight discoloration on the surface of the liquid was noticed. Various cultures in broth, milk, and gelatine were made with a few drops of black vomit and urine, and even with a particle of earth taken from a cemetery (Jurujuba), in which only yellow-fever bodies are buried. A like result ensued in each case, namely, the appearance of a material, more or less dark-coloured or even entirely black. This material is due to pigments elaborated by the micrococci. The black deposit is exactly the same as the black vomit of the advanced stage of yellow fever. Previous to my microscopical studies on yellow fever, pathologists believed that the black colour of the ejected matter was due to blood more or less altered. In my work, published in 1880, I placed beyond doubt that the black colour which was produced by the cryptococci was due to the black pigment peculiar to these micro-organisms.

"Whether the little organisms are observed in the blood, in the bile, or other fluids of the economy of man or animals; or whether in the artificial cultures, or even in the earth taken from a yellow-fever cemetery. Always the same little black specks (*points noirs*), and afterwards little cells, somewhat larger, surrounded with a dark border, and later on adult cells, proliferating, and containing a large quantity of black and yellow pigment. The evolution in the culture does not differ from that which takes place in man or

in animals. For this reason, we say with certainty that the microbe, such as we describe the *cryptococcus-xanthogenicus* to be, is never found in organisms affected with other diseases; it is specific of this affection. The presence of this microbe in the blood and in the urine of a patient constitutes the surest diagnostic sign of yellow fever. . . . ."

Professor Freire has not, in his experimental researches, strictly fulfilled Koch's third condition, viz.: 3. When thus cultivated for several generations, it is necessary to introduce them into the body of a healthy animal susceptible to the disease, and to show that that animal becomes affected with the same disease. He (Freire) transmitted the disease from one animal to another by inoculating them with blood, in the first case from a person who had died of yellow fever, and with this animal's blood inoculating a second, and so on to a ninth. The blood and urine in all these cases revealed cryptococci, identical to those discovered in the blood and fluids of yellow-fever patients; each animal died from the effects of the disease induced by the inoculation, and, on autopsy, revealed *post-mortem* lesions identical with those observed in man.

A small portion of muscle taken from a subject dead of yellow fever was triturated in a mortar, the paste obtained was mixed with a small quantity of distilled water and filtered through fine muslin. A drop of the fluid, examined under the microscope, revealed a multitude of microbes in the field. Subsequently a gramme of the liquid was dried, by subjecting it to a temperature of 280 deg. C.; mixing this with a little water, it was injected subcutaneously into a guinea-pig, who experienced no ill-effects from the operation. Another guinea-pig injected on the same day with the same liquid, non-sterilised by heat, sank under the disease in two days. The logical inference to be drawn from this experiment may, we think, be assumed to be that the cause of yellow fever resides in the microbe, which infects both the blood, the urine, and the tissues of patients affected with the disease.

The origin of the germ has been variously assigned to malarial or miasmatic effluvia, to animal or vegetable decomposition. The belief in the malarial origin of yellow fever is no longer entertained, the general tenor of medical opinion being opposed to it. Acclimatisation (?); immunity after one attack; resistance to the sulphate of quinine; portability by means of ships, individuals, clothes, etc.; the exemption of some highly malarious localities; the fact that it is an urban disease (like typhus), in contradistinction to malarial fever,

which is generally associated with rural districts; the epidemic occurrence of the fever on ship-board; and, finally, the marked and persistent difference at all times, and in all places between the mortality of the two diseases, establishes that the *materies morbi* of paroxysmal fever and yellow fever are quite distinct.

It is highly probable that the evolution of the germ is intimately associated with the process of decomposition in organic matters; but, whether the products of decomposition and the effluvia arising therefrom constitute the morbific agent, and represent the *causa intima* of the disease, or whether the production of the pestilence is due to the introduction into the system of specific virus, for whose evolution and reproduction these products afford merely suitable soil, is still *sub judice*, and will remain unsolved as long as we are ignorant of the nature of the poison itself. From the experiments and investigations of Professor Freire, which are strongly supported by analogous researches in other diseases, both in man and animals, there is strong presumptive evidence that the microbe of this pestilence has been isolated, and that the soil is its home and serves as a nursery for its evolution.

"Let us suppose that the virus and the vaccine of yellow fever has been discovered, as a Brazilian physician has flattered himself: what are we to do? We French people, who stay at home, need do little, or nothing; but merchants, sailors, and soldiers, who repair to yellow-fever regions, such as South America, Senegal, or the West Indies, have every object in getting vaccinated."

Yellow fever, with very rare exceptions, does not attack the same individual twice. Accepting the doctrine of microorganism in this, if we could succeed in attenuating the culture of its virus, and with it vaccinate individuals, and by this means produce a change in the composition of the blood and in the tissues, we might obtain for yellow fever that which has been already gained for small-pox in man and anthrax in cattle.

Professor Freire has described, in his report to the Brazilian Government, what he terms "des expériences concluantes faites sur des animaux dans le but de résoudre un problème si ardu."

He says: "It is singular that not only the cultures made from gelatine solution, milk, etc., become weak out of the epidemic season, but that likewise the cryptococci, which are found in the blood and fluids of the organism, become equally so. On the 30th of July, when the epidemic was plainly on the wane, I injected into a guinea-pig one gramme of blood from the corpse of a yellow-fever case, which was

extracted at the height of the epidemic, and at that time caused the death of several animals; this blood, which was preserved in a bottle under a layer of ether, was found to contain masses of cryptococci on the above date (30th July). The animal inoculated with this blood, though experiencing slight rise of temperature for a few days, sustained no ill-effects.

"The question may be asked, Can the inoculation of attenuated cultures be made on man without risk, and with an entire confidence in its success? Having demonstrated by numberless experiments that rabbits and guinea-pigs are (as well as man) susceptible of yellow fever, we may assume that man ought to resist the effects of these cultures as well as those animals. This opens up another question—What is the most propitious season for vaccination? It is clear that the propitious season for employing vaccination is the interval during the epidemic season, as the blood-cultures become inert then—a fact which has been clearly demonstrated by repeated experiments on animals.

"The various viruses may be divided into two classes: those with an intermittent and those with a continued action. The virus of rabies, glanders, anthrax, syphilis, etc., is of the latter class; for, no matter what the season, climate, or altitude may be, a little blood of a patient suffering from one of those diseases inoculated on a person susceptible of the virus will produce the same disease. But the virus of yellow fever, and probably of other parasitic affections, has an intermittent action, being periodic, and having a season during which it recovers energy; it has its climatic and its altitudinal limit.

"The next question to consider is the manner of verifying the preventive action of the cultures. The method adopted by Pasteur, Chamberland, Roux, and others, in dealing with anthrax (*charbon*) was the one employed in the demonstrations, with this difference, that a smaller number of animals were experimented on. At first, five guinea-pigs, marked A, B, C, D (the fifth had no distinguishing mark), were employed. On the 25th May the temperature of the five animals was as follows: 38.8°; 38.7°; 38.9°; 39.1°; 39° (centigrade). A and C were vaccinated by hypodermic injection with nearly a gramme of prepared liquid culture. The following is a record of the temperatures of the above animals:

	May.						June.			
	26th	27th	28th	29th	30th	31st	1st	2nd	3rd	4th
A—deg.	38.5	38.8	38.2	38.3	38.5	38.4	38.2	38.4	37.9	38.5
C—deg.	39.3	38.7	38.	38.6	38.9	38.7	38.9	38.9	37.4	38.6

"At the expiration of twelve days I inoculated these two animals A and C with a virulent culture of blood, and employed the same culture and a like quantity in inoculating B and D, which had not been previously vaccinated. All the animals were placed in the same cage.

"The following is a record of their temperatures:

Animal B.				Animal D.			
June	5th	6th	7th	June	5th	6th	7th 8th
Deg.	38.6	38.	37. (died)	Deg.	39.2	38.	37. (died)

The autopsy on these two animals revealed lesions peculiar to yellow fever. Microscopic examinations disclosed a very considerable number of cryptococci, some spirilla, in addition to masses of yellow and black pigment. The same may be said of the animal D, in whose urine, in addition, albumen was found.

"Let us now see the modifications of temperature presented by A and C.

	June.														
	5	6	7	8	9	10	11	12	13	14	16	17	18	19	20
A—	38.	39.	38.4	39.2	39.	38.2	39.2	38.8	39.	39.	39.2	39.2	38.3	39.4	39.4
C—	38.1	39.2	39.2	38.5	38.4	38.7	38.2	38.8	38.4	38.	38.3	39.	39.2	39.	39.3

Both animals (A and B are still alive, 11th November) having experienced no ill-affects from the experiments to which they were submitted. The other guinea-pig, not labelled, who remained as a witness, underwent no change; his temperature being 38 deg. on the 20th June. In a second experiment ten animals were vaccinated with a culture made in gelatine; twelve days afterwards the same procedure was employed as in the former experiment. Four animals, unvaccinated, were inoculated with blood taken fresh from a yellow-fever corpse; the ten vaccinated were treated likewise. The result was as in the former experiment, viz., the four non-vaccinated died and the ten vaccinated lived. These results induced me to continue the same experiments during the month of July, and I was surprised to find the cultures, which up to then had been most virulent, had lost or had become diminished in strength. The favourable results already obtained, and the proof of the attenuation of the cultures experienced by the number of animals experimented on, induced me to try inoculation on the human species.

"In the face of these encouraging results, I addressed myself to the Imperial Government, soliciting authority to invite all persons who wished to resort to this prophylactic measure, vaccination.

"Up to the 7th May 1884 some 418 persons were vac-

cinated; their names, ages, sex, nationalities, residences, and the duration of their stay in Brazil will be found in the Appendix. All these individuals were exposed to the consequences of a sufficiently severe epidemic. The results of the experience are favourable, and clearly demonstrate the parasitic doctrine of the fever."

Résumé of a Report on the preventive inoculation of yellow fever during the epidemic of 1883-4 *at Rio de Janeiro, presented to H.E. the Minister and Secretary of State of the Empire by Dr. Domingos José Freire, President of the Central Board of Health, etc., etc.*

"The epidemic cycle of yellow fever, commencing last October 1883, having just terminated, I deem it my duty, having been commissioned by the Imperial Government to study the disease, to convey to your Excellency succinctly the practical results of the researches which I have made since March 1883.

"It was only after long and patient experiments on the virulent power of the microbe of yellow fever that I was emboldened to inoculate the human species. In the first place I applied myself to the verification of the contagious nature of the malady; this I effected by inoculating susceptible animals with blood of yellow-fever patients, and in this manner conveyed the disease from animal to animal in the same way that in its natural evolution it passes from man to man. Having demonstrated the contagiousness of the disease, I applied myself to find a process of attenuating the virus, which I found, after a certain number of cultures had been made, had lost a great part of its virulence, and might, without causing death, be inoculated into susceptible animals, inducing only slight elevation of temperature for a few days. This result urged me to experiment further, and, accordingly, twelve animals which had been vaccinated some days previously with attenuated cultures were inoculated with virulent blood extracted from a yellow-fever patient: none of these animals died, whereas other animals (of the same species), non-vaccinated, and on whom a like inoculation was performed, all died.

"These twelve cases had, however, much significance for me, and as I had demonstrated in upwards of fifty experiments on animals the innocuousness of attenuated cultures, I had a sufficiently secure basis on which to support my opinion as to the safety of attempting preventive inoculations on the human species.

"On no occasion have I ever inoculated in man with the

blood or other organic fluid extracted directly from a corpse, or a patient ill of the fever, as it has been insidiously attempted to impute to me. I proceed in the following manner. The blood of a yellow-fever patient is passed through an extended series of animals which have been successively inoculated, and their blood it is which is subject to cultivation, and only on having transplanted the culture more than four times is the inoculation commenced on man, and even then not until it has been verified, by inoculations on animals, that the cultures do not produce in them any serious consequences. . . ."

APPENDIX.

Extracts from the Report on the preventive inoculation of yellow fever during the epidemic which obtained at Rio de Janeiro in 1883-4, *presented to H.E. the Minister of State of the Empire by Professor Domingos José Freire.*

"I performed 418 vaccinations in the human subject, not only during the month preceding the epidemic, but also at the time of its gravest intensity. I endeavoured to vaccinate only persons placed under the most usual morbid conditions, and the 418 individuals vaccinated may be classified as follows:

"1st. *Age.*—From 16 to 25 years, 241; from 31 to 45, 94; from 1 to 15, 64; from 45 to 60, 13; no age mentioned, 6.

"2nd. *Nationality.*—Portuguese, 158; Italian, 122; French, 11; Spanish, 9; English, 1; Swiss, 1; Polish, 1; German, 1; Dutch, 1; North American, 1; Argentine, 1; Brazilian, 111. The 111 Brazilians came, with rare exceptions, from the high lands of the interior of the province of Rio de Janeiro, or the southern province; few came from the northern province; they were therefore all in the most favourable conditions of morbid receptivity.

"3rd. *Period of sojourn in Brazil.* A few days, 39; from a month to 12 months, 166; from 2 to 5 years, 114; more than 5 years, 26. There were 73 persons whose stay was not known, but of the number were a great many children of a few months to a few years (residence). The greater number of the vaccinated dwelt in houses called *estalagens*, situated in those quarters where the pestilence is wont to break out with most violence, as at Cidade Nova, etc., in the centre of commerce, and on the sea-board from the flat of Santa Lucia to Botofogo.

"During the epidemic season a great number of those

vaccinated were seized with yellow fever, but of a benign form. The number of persons non-vaccinated who died of the fever during the past epidemic numbered 650. On the other hand, seven of those vaccinated figured among the deceased, and of those there is grave doubt as to the true diagnosis in some cases. But, even admitting that the seven deaths were from the malady, the ratio per cent. would be very favourable, since it would be only 1.6 per cent. In some of the *estalagens*, where I vaccinated nearly all the occupants, all of them escaped during the epidemic; those only who refused to be vaccinated succumbed to the fatal malady. Likewise, in a great number of houses inhabited by vaccinated as well as by non-vaccinated persons, the latter only were fatally attacked, whereas the vaccinated were attacked very mildly, or were completely spared. To recapitulate:—From October 1883 to the present time (June 1884) 650 non-vaccinated persons died, and, during the same period, *at most* seven vaccinated succumbed to the fever. These 650 deaths refer to individuals who were placed in identical conditions with the 418 vaccinated. Ages of non-vaccinated who died of yellow fever during the epidemic, 1883-4:—From 16 to 30 years, 353; from 45 to 60 years, 36; from 30 to 45 years, 158; over 60 years, 5; from 1 to 15 years, 88; no age given, 10.

"The nationality of deceased non-vaccinated was as follows:

Portuguese	361	Argentine	2
Italians	126	African	1
Spaniards	30	Belgian	1
English	18	Dutch	1
German	13	Russian	1
French	12		
Norwegian	5	Strangers	578
Swedes	3	Brazilian	72
North American	2		
Austrian	2	Total	650

"238 persons between 16 and 30 years of age were vaccinated; 64 persons between 1 month and 15 years of age were vaccinated; 353 persons between 16 and 30 years of age, non-vaccinated, died; 88 persons between 1 month and 15 years of age, non-vaccinated, died. These figures show that between those two limits of age I vaccinated nearly as many persons as died of the disease non-vaccinated. I vaccinated 307 strangers, and 575 strangers died who were not vaccinated. It may be laid down that in very severe epi-

demics the mortality ranges from 50 to 80 per cent., and in less intense ones from 20 to 35 per cent. All authorities are unanimous on that point.

"In order to determine the proportion of attacks among receptive subjects, from whom alone we choose our vaccinated, we must be guided by the experience obtained in other countries. For example, according to the dates furnished with care in 1881 by Dr. Jemble, who published an epidemiological report on Senegal, it was established that during the epidemic of that year, among strangers who were from one to three years in that locality, 3 out of 4 were attacked, and 2 out of 3 died, or at the rate of 75 per cent. for the former and 66.6 per cent. for the latter. . . . . If we calculate the number of receptive individuals living in Rio de Janeiro during the epidemic, and under similar circumstances to be vaccinated, we will find the number to be approximately 4,737. Admitting the mortality per 100 of the receptive as 13.5, and of the vaccinated as 1.6 per 100, it evidently follows that the mortality of the vaccinated has been 8½ times smaller than the non-vaccinated."*

* This proportion comes close to that obtained by Pasteur in his vaccinations against splenic fever.

ON SOME SPECIFIC FEBRILE DISEASES OF MALARIOUS ORIGIN,

AND ON THE NECESSITY OF THE EXISTENCE OF GERMS FOR THEIR PRODUCTION.

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(ABSTRACT.)

(*Read: Feb. 8th,* 1888.)

THE subject chosen in this paper has for object the causes in action which originate certain specific febrile diseases, but refers more especially to those which have a malarious origin.

In all alluvial soils where heat, moisture, and rank vegetation combine—whether at the mouths of the great rivers of Europe, Asia, Africa, or America; whether along the shores of tropical lands; in inland swampy districts; or where a soil, untouched for ages, has been newly turned up —remittent and intermittent fevers may be found. But besides the remittent and intermittent, other fevers are met with in these same localities, under circumstances similar to those which favour the development of remittent and intermittent fevers. I refer more particularly to such specific malarious disorders as plague, cholera, yellow, remittent, and intermittent fevers, and dysentery, but still more especially to the three first mentioned. These three—viz., plague, cholera, and yellow fever—are bound by certain exactions for their generation and development, in addition to those which are demanded of the fevers of a remittent or intermittent type. Limited to definite parallels of latitude and meridians of longitude, they are never met with away from their circumscribed zones, unless carried to climes and soils suitable to their growth. These malarious specific diseases have chosen the deltas of large rivers as their especial habitat. The plague (*pestis bubonica*) is found to generate in the delta of the Nile; cholera (*cholera Asiatica*) has its birth and its habitat in the Sunderbunds of the Ganges; yellow fever (*febris flava*) in the deltas of the Mississippi and Orinoco;—and in the choice thus made—although the cosmic and meteorological circumstances are to all seeming alike—we never find yellow fever generating in

the delta of the Ganges, cholera in the delta of the Nile, nor plague in that of the Mississippi.

It may be asked what are, or what can be, the influences —in the deltas of these great rivers—which operate to constitute in one locality a specific form of virulent disease, whilst in another—under circumstances equally similar to all appearances—to bring into existence a specific disease of equal virulence, though differing in its symptoms.

When we consider the huge amount of matter brought down to the deltas by the waters of the great rivers—when we have grasped the idea of the vast extent of country through which these waters pass, the variety and nature of the soil, and the multitudinous mass of life that abounds everywhere—we can readily bring ourselves to admit that with these waters innumerable living organisms are con veyed, which, developing by change of locality and circumstance, have become vested with a potentiality they had not before.

But here, again, a doubt arises, for if this element or germ be possessed of virulent properties when in the delta of these great rivers, how does it happen that this virulence does not manifest itself at its source, and in the locality from whence the germ was forcibly carried away; and what can be the especial agencies which, by a simple change of place, have effected so marked a change in its nature? Where in one instance heat, moisture, and an aptness of soil are essential conditions, cold, dryness, and their concomitant conditions are absolute in another. The germ of yellow fever will be found to luxuriate under the heats of a tropical sun on the low shores of Mexico and Louisiana; whilst that of the Ureda Nivalis can only find development in an Arctic climate, or an elevation above the snow level.

As an instance of germs of a specific nature having a special habitat, we may quote the fact that yellow fever was not known to Europe before the discovery of America by Christopher Columbus, for no record nor description of this disease is known prior to that period; nor can we satisfy ourselves of the occurrence of any well-authenticated invasion of this disorder in Europe before the epidemic which devastated Lisbon in 1723.

The Great Plague of Athens (433 B.C.), described by Thucydides, and the pestilence known as Budhe connail, which, during the reign of the Saxon King Oswy (A.D. 644), devastated the islands now known as the United Kingdom of Great Britain and Ireland, are now declared by competent judges to have been epidemics of Oriental plague. In spite

of the name given by some to the Budhe connail of "Pestis flava" and "Infirmitas icteritia", those well acquainted with the symptoms of yellow fever know that yellowness of skin does occur, as a symptom, in other diseases, whilst it is not always a concomitant symptom of yellow fever. The causes which gave rise to the epidemic of yellow fever in Lisbon in 1723 had their origin in the marshy shores of America, and the evidence afforded of their importation into that city has sufficient and satisfactory proof. The circumstances in existence in this city at the time of the invasion of this fever may be accused of having predisposed to and assisted in the development of the germ, but they certainly had nothing towards generating a specific febrile disorder such as yellow fever.

If we seek for further proof in favour of the individuality of germs, we shall find it in the established fact that Oriental plague (*pestis bubonica*) has never yet been known in any part of the New World. It has no habitat there, nor, fortunately, has the germ ever been imported.

Though there would seem to be many differences and much divergence in the agency of the specific germs which originate plague, cholera, yellow fever, and remittent, and though in the practical observation of each we meet with many deviations from the accepted standard, there will be found nevertheless, on the whole, broad features of resemblance which give to each a unity whereby each may be recognised and each distinguished.

A germ demands a multiplicity of conditions for development; in the absence of these conditions it remains torpid, as in the instance of the mummy wheat and the germs in granitic formation. Unless these conditions are fulfilled the germ can effect nothing of itself. If seed be sown in a sluggish soil and an ungenial season, little hopes can be entertained of its fruitful issue; but where circumstance, place, season, and apt soil are present, the result will, and must be, favourable.

What is termed a poison is not so, in its natural state, neither to the plant which generates it nor to the venomous creature which secretes it. The poison in the fang of a venomous serpent; opium in the juices of the poppy; strychnia in the nux vomica, effect no evil until placed in a soil whereupon they can work, and where, by a concurrence of circumstances, their development is favoured and their destructive powers brought into action. Even the virus of rabies, implanted in the system, may remain in a state of latency for indefinite periods. Sir Thomas Watson, some

fifty years ago, published certain views of his own, wherein he imagined " that the virus implanted by the rabid animal may remain lodged in the bitten spot, shut up perhaps in a nodule of lymph, or detained somehow in temporary and precarious union with some one of the animal tissues, without entering the blood itself for a longer or shorter time", only declaring itself in symptoms of hydrophobia when occasion presents favourable for its development.

Some individuals are, from hereditary proclivity, more liable than others to contract disease. Some again possess a power of resistance unaccountable and eccentric. Though habited in clothes impregnated with the germs of specific febrile diseases, men have been known to communicate and propagate disease, whilst they themselves escaped unharmed.

The *trousse galant* of France, named from the speedy death it inflicted on young and robust men, followed fast upon the disasters of the French army before Naples in 1528, and was the contemporary of a long and grievous famine. It was carried about from place to place by the starving people in search of food. These people, it is said, were so saturated with the poison, that they communicated it to persons in health, without being themselves affected; in this respect reminding us of our own gaol-fever, and of the fever which followed the Irish famine.

I have heard related by old inhabitants of Malta of the devotion of a daughter to her parents when stricken with the terrible plague of 1813 (which carried off 4,668 of the inhabitants of that island, out of a population numbering 100,000). Unassisted, she attended both her plague-stricken parents during their illness, nursed them, and at their death, when her friends and neighbours, panic-struck, fled from and abandoned her, she gave to them the sacred rites of burial with her own and only hands. Yet through this heroic filial devotion she lived unhurt and untainted.

In allusion to the facility of contracting disease, due to some exceptional proclivity or eccentric predisposition, I may mention here the susceptibility of one race to succumb to influences when coming in contact with another and a different race.

In our first visit to the Samoan group of islands in the South Seas, in H.M.S. *Calypso*, in the year 1848, I remarked, shortly after communication had been established between the ship and shore (the sanitary state of the ship being good at the time), that the natives became affected with symptoms of influenza, some exhibiting these symptoms in a more severe form than others. When I

expressed my astonishment at this sudden appearance of disease—for which sufficient reason could not be given, either in the meteorological or other influences which then obtained—I was answered that the man-o'-war had brought the disease and had given it to the natives; and I was further told that the arrival of foreign ships, howsoever healthy their crew (provided a certain given interval had elapsed between their visits), was attended with similar evil effects.*

It would seem from this that the exhalations emanating from the bodies of a race foreign to the clime become, when in the presence of another race, possessed of a virulence hitherto unknown. Innoxious to the race from whence they emanate, these exudations may be said to be somewhat analogous in action to the poison of venomous serpents, which, harmless when implanted within the body of a snake as venomous as itself, proves most deadly to those snakes not endowed with similar venomous properties.

It has been observed, where a race of high intelligence and of high nervous energy comes in contact with one less marked for these qualities, that the weaker race gives way to the stronger, a noted decrease of population follows, until at length it gradually disappears. The Red Indian, once counted by millions, is now only to be numbered by thousands. The Australian is fast disappearing before the determined advance of the Anglo-Saxon race.

Though soil, climate, and habits have a mighty action in their effects in both the improvement and the deterioration of a race, the influences which the contact of different races engenders should not be excluded nor set aside without a searching inquiry. The vigorous youth now emigrating in numbers from Europe into Australia, New Zealand, and our establishments in the Pacific, carry with them fresh, robust, and healthy seed. The energy of their nature throws a new life into those who have preceded them; but at the same time it assists in intensifying the evil influences caused by intercourse; casts a species of glamour over the darker race,

* The people of St. Kilda and those of the Faroe Islands say "that the first stranger of the year brings with him the 'Krujm' or cold fit, which runs its race through the islands." (*Some Faroe Notes.*)

Martin, who visited the island of St. Kilda in the seventeenth century, says of the inhabitants: "They are not subject to many diseases; they contract a cough as often as any strangers land or stay for any time among them, and it continues for some eight or ten days. They say the very infants at the breast are affected by it."

The natives call the affection "Conatan-na-gall", or strangers' cold. (C. R. Macdonald, M.D., *St. Kilda, its Inhabitants, and the Diseases peculiar to them.*)

forcing it to recede before it, as forests before population, as wild beasts before man.

I am one of those who believe, with Professor Knox, that unless this infusion of new blood is maintained by a continuous stream of immigration, unless an imported current of white blood flows constantly into them, a degeneration must necessarily follow of the white race itself.

It has been known from time immemorial that individuals suffering from phthisis pulmonalis enjoy comparatively a greater degree of ease and comfort when breathing the soft, balmy air of malarious districts.* It is supposed that the germ of intermittent fever, when inhaled, has the power of checking the growth or even of killing the bacillus which is believed to be the cause of tubercle. Some give it greater power, and attribute to it an antagonistic action against the progress of other diseases. Sir Thomas Watson (*Principles and Practice of Physic*) tells a story he had heard Dr. Graham of Edinburgh relate, how a patient, afflicted with epilepsy, was sent to pass the night in a marshy place, where the malaria was known to be so abundant and so powerful that few thus exposed escaped ague. The twofold design succeeded admirably. The patient gained an ague and lost his epilepsy.

Specific fevers are believed to be the result of a progressive change in the blood, induced by the introduction into this fluid of some element, principle, or germ which, constituting a *modus operandi* antagonistic to what is observed in a state of heath, reveals itself in symptoms which characterise a specific febrile disease of the same nature as that of the parent offspring from whence the germ had birth. The many shades and distinctions which are observed in this class of fevers are touched upon but slightly, for justice could not be rendered them in so short and sketchy a paper as this. These fevers, however, depend upon the morbid action which the blood has taken upon itself by the contact with the virulent germ; the blood thus vitiated is the chief cause at fault. The odours which arise from the bodies of persons affected with fever are characteristic of the fever itself. They reveal the state and changes through which the blood has passed and is passing. This smell resembles in no ways that which emanates from the same body in health. Even in health this smell differs under different conditions. It is characteristic of the phases of the body at the time. It is

* In the days of Celsus and Pliny the Romans thus suffering were recommended to seek, for ease and comfort, the dense atmosphere of the Delta of the Nile.

even said that the smell which emanates from a person after a fit of passion is specially significant of the passion itself. This smell in healthy states of the constitution differs in man, in woman, and in child. It likewise differs in individuals in accordance with the colour of the hair.*

In the special febrile diseases of which we are treating we are able to discriminate between the smell that emanates from a subject affected with yellow fever and that which exhales from another affected with small-pox, dysentery, or typhus. Each of these affections is characterised by its especial odour, denoting the state of the blood at the time, and helping materially towards a surer diagnosis.

The *eucalyptus globulus* planted in marshy districts has effected the destruction, or at least has diminished the virulence, of the malarious germ by the absorption of the moisture in which the germ luxuriated or by the disintegration of the germ itself. The sunflower is said to possess the virtue of preserving a neighbourhood from intermittent fever, and is planted for this purpose in several marshy districts of the South of France.

Although this paper does not refer exclusively to that form of malarious fever known as yellow fever, it may not be considered irrelevant to say a few words regarding it, its habit, its individuality, its infectious properties. It is now, I believe, generally admitted that a special and well-defined geographical position on the globe is assigned to it as a habitat; that this habitat is confined to the shores of North and South America—north of the equator, commencing in the north at or about George Town in South Carolina, lat. 33° 25′ N., sweeping round the southern shores of North America and the eastern shores of Mexico and Central America, coasting the northern lands of South America, and terminating in the south at or about George Town (Demerara), in lat. 6° 49′ N. Though yellow fever is likewise observed on the western shores of Mexico and Central America, the division of these shores, by the range of mountains which runs north and south through the continents of both Americas from Alaska to Patagonia, has a remarkable and characteristic influence; for, whilst the eastern shores are possessed of a climate hotter, moister, and less bearable by the human constitution, where serpents and insects are more venomous, and where fevers are endowed with greater virulence, and are of more frequent occurrence, the western differ in as much as they possess,

* Dealers in hair will tell you that they are able to ascertain by the smell the sex and age of the person to whom the hair once belonged.

comparatively, a milder climate, serpents and insects less venomous, and fevers less virulent in their nature.

That this fever may be, and is, conveyed away to other localities—not its natural one—and does thrive in them, we have many examples. The qualifications which are required for this development are: the importation of the germ, a temperature above 70 deg. Fahr., an apt soil, and appropriate pabulum. Importation is accomplished through ships coming from an infected district, their crews, their cargoes, their passengers and effects. A lower temperature than 70 deg. Fahr. will arrest the development of the germ, either by stamping out its vitality or by reducing it to a state of dormancy and seeming effacement. It is on this known antagonistic power which a lower temperature possesses that the Admiralty have enforced the necessity of the immediate removal from the West Indies of all ships of war, upon which fever has broken out, to a northern and colder climate, such as Halifax in Nova Scotia.

Yellow fever is possessed of an individuality *sui generis*. It differs, in this essential, from remittent, from plague, from cholera, as each differs one from the other; for though the symptoms which declare themselves in each may in their first aspect assume a similarity, they diverge widely and characteristically as the fever pursues its course; each takes its determined line, each exhibits symptoms essentially its own. It prefers the white to the dark race, youth to age, the new comer to the old resident. Though in some sweeping epidemics it works its ravages irrespective of age, of race, of colour, of vigour of constitution, as it did in that epidemic which devastated Vicksberg, Memphis, and other towns on the Mississippi in 1878, it is, nevertheless, as a rule, observant and choice in the selection of its victims.

It was during the period of my administration as Deputy-Inspector-General of the Royal Naval Hospital at Jamaica in 1869 that this predilection of yellow fever for young and vigorous subjects fell markedly under notice. It was at a time when the disease was in existence in an epidemic form that a number of supernumerary boys, whose ages ranged from sixteen to twenty, arrived in the West Indies for distribution among the ships on that station. Every means to shield these youths from the reigning contagion, dictated by prudence and experience, was at once resorted to. Yet, notwithstanding the excellence of the arrangements made by the medical officers of the squadron; in despite of the attention paid to the isolation and seclusion of the fever-stricken men, and of the adoption of the general sanitary and hygienic

precautions, the poison did its cruel work on these untainted constitutions, and many fell victims to the scourge.

It has been remarked that a constitution weakened by abuse is overlooked by this germ. In several instances, when men were allowed on leave in Kingston (Jamaica), where, though the sanitary state was considered and reported good, germs of this fever were in existence in a state of latency, the temperate fell victims whilst the drunkard escaped untouched. The physician of former days, knowing this predilection of yellow fever for vigorous subjects, recommended those about to visit the tropics to adopt a system and regimen whereby they might ensure a general lowering of their system.

The infectious properties of this specific fever are denied to it by some. Having lived in lands in which epidemics of yellow fever frequently prevailed, and having, with a few others, escaped infection, they framed their belief upon, and drew their inferences from, the isolated facts which fell directly under their immediate observation. They did not ensure to themselves the safer belief that theories only gather strength by the interpretation of accumulated facts well and duly considered; they set aside the combined experience of others, and evidently were not cognisant of the circumstance—though they had passed the ordeal safely and soundly—that they had notwithstanding absorbed into their system an amount of the virus, and that they were at the same time possessed of a power of resistance which enabled them to combat the evil effects of the poisonous element. This circumstance of resistance was fully appreciated by the College of Physicians of Philadelphia as far back as the year 1793, when it was remarked by them that "the contagium of the disease might be taken into the body and pass out of it without producing the fever, unless it be rendered active by some active cause." The instances which can be adduced as proofs of the infectious properties inherent to this fever are now many, and so well observed, that a doubt cannot, with any show of justice, be entertained about this question.

As yellow fever and remittent fever are observed to be in existence at one and the same time, and as they have frequently been mistaken one for the other, it may not be inopportune to show at a glance the differences which obtain between them, and to note the chief points that characterise each. These distinctions are summarised in the following table:

Characteristic distinctions between Yellow Fever and Remittent Fever.

Yellow Fever.	Remittent Fever.
Is essentially of an infectious nature.	Is not of an infectious nature.
Young, vigorous, and strong constitutions, as a rule, fall victims to it. Coloured population *less* liable than white.	All ages and constitutions are liable, the weakest most so. Coloured population as liable as the white.
Restricted to the yellow-fever zone, viz.: the deltas of the Mississippi and Orinoco rivers, and the coasts and islands adjoining these deltas.	Is to be found in all the parts of the world where marshy soils prevail.
Is of a continued type; no remissions.	Remissions observed in the morning.
Rachialgia more pronounced in this fever than in any other, except small-pox.	Rachialgia slight.
Abstraction of blood not tolerated.	Abstraction of blood tolerated.
Albuminous urine the rule.	Albuminous urine the exception.
Liver affected.	Liver not affected.
Spleen not affected.	Spleen invariably affected.
Partial or total suppression of urine the rule.	Suppression of urine the exception.
Hæmorrhages from stomach in bad cases the rule.	Hæmorrhages from stomach in bad cases the exception.
Quinine useless as a therapeutic agent.	Antagonistic power of quinine beyond the reach of question.
Death occurs on the fifth day.	Death seldom occurs before the seventh day.
Yellow fever never merges into intermittent.	Remittent merges into intermittent.
One attack affords an immunity from future ones.	One attack affords no immunity from future ones.
Immunity almost perfect after a three years' residence in the lowlands.	No immunity obtained by any length of residence.
Convalescence less protracted than in remittent fever.	Convalescence protracted.
Peculiar smell in yellow-fever cases.	Though a smell is observable in remittent fever, it is different and less pronounced than in yellow fever.

From the above sketch we may conclude: 1. That the various specific febrile diseases of malarious origin are possessed each of a distinct and separate individuality, and are dependent upon some principle, element, or germ which —implanted within the system, and developing—marks each with characteristic pathognomonic symptoms. 2. That the germs producing these specific febrile diseases are many and diverse, each limited to a habitat, each exhibiting a special *modus operandi* producing its like, and in its following marked by a precise and determined conformity, never

undergoing transmutation, but persisting in a unity of action, and perpetuating its specific qualities to the end of time. 3. That epidemics are simply an intensified state, an aggravation of the natural conditions, arising either from a multiplication of germs, or from a greater degree of virulence of these germs.

EPIDEMIC CEREBRO-SPINAL FEVER IN THE FIJI ISLANDS, 1885.

By BOLTON G. CORNEY, Colonial Surgeon.

(*Read: March 14th*, 1888.)

THE year 1885 was productive in the islands of Fiji of two notable epidemic outbreaks of disease. The earlier in the order of their incidence was dengue, which had never occurred before in the Archipelago. This, it was pretty conclusively proved, became developed in Fiji through the medium of a white woman travelling from Sydney, N.S.W., who received the infection at Nouméa, in New Caledonia, which is a port of call *en route*. The latter place obtained it by means of the line of mail steamers belonging to the Compagnie des Messageries Maritimes, by which communication is maintained at monthly intervals between that colony and the islands of Réunion (Bourbon), where dengue has been long domiciled. Before this occurrence in New Caledonia I am not aware that dengue had ever visited any of the islands in the Pacific Ocean south of the Equator.

The other epidemic was cerebro-spinal fever, and a truly appalling visitation it was. Whatever may have been the nature of the "black death" in England during the fourteenth century, I am greatly tempted to adopt that simple designation for this fever, not merely as metaphorically applicable, but also as descriptively truthful in more than one of its elements.

There is reason to suppose that cerebro-spinal fever in Fiji was of extrinsic origin from the fact that its ravages were confined to the persons of certain immigrant labourers introduced from other islands in the Western Pacific. But on the other hand these people had been too long in the colony to allow one to believe that they had brought the disease with them in the way in which ordinary zymotic diseases can be transplanted. They were natives of the New Hebrides, the Solomon Islands, and the New Britain group, collectively an extensive tract of the world's surface, spreading from Tana, in lat. 19° 30′ S., long. 164° 20′ E., down to New Hanover, in lat. 2° 30′ S., long 150° E. They emigrate yearly to Fiji in varying numbers, and maintain in that colony an

average aggregate muster-roll of about 5,000 individuals, of whom, however, only six or seven per cent. are females.

It is a somewhat notable fact that out of this multitude of different islands the epidemic showed marked preference for natives of four particular localities, viz., New Britain, New Ireland, Buka, and Malakula. The first three lie about a thousand miles away from the last-named, and their respective peoples differ widely from each other. The distribution of them in Fiji will not account for this preference, as they were living and working side by side with batches of natives from almost every island in the Melanesian tract just mentioned; nor can it be justly attributed to numerical predominance, because that condition cannot be said to have applied to them; nor did others suffer to even a proportionate extent in relation to their respective populations.

The Fijians themselves, of whom there are 110,000, as well as all the pure members of the Malago-Polynesian race in the colony, escaped altogether. These included Samoans, Tongans, Rotumans, natives of the Gilbert or Tokalan group, and a few others. The Indian immigrants from Bengal, to the number of about 5,200, also escaped altogether. Three white children were attacked and died. They had all been born in Fiji. One of them was nine years old, the other two were each less than one year. The total white population was, excluding half-castes, under 3,000.

The peculiarity of the distribution of the cases of cerebro-spinal fever in this epidemic early suggested to me the probability that the disease was infectious from man to man, but that very close association was necessary for its propagation. Subsequent observation failed, in my opinion, to disprove this theory, although I am still unable to assert that any very salient facts support it.

The total number of cases constituting the epidemic was 128, of which 90 terminated in death. The duration of it did not exceed five consecutive months in all, and it consisted of three nearly synchronous portions, each lasting rather more than four months. Strange to say, two of these portions appear to have come into activity independently of each other, and although nearly concurrent in date, were remote in locality. If indeed they possessed a common origin, it must have dated back no less than three years; and if this be true of two of the outbreaks, it is probably also applicable to certain cases in the third.

The two separate centres, however, contributed during the epidemic numerous healthy persons to the third or common centre, where many of these arrivals developed the disease,

and where other persons previously and continuously resident there were also attacked. One of the first two centres being a small island with infrequent communication, had a circumscribed epidemic of its own, with the exception of its contributions (if they may be assumed to have originated within it) to the third centre. This was the island of Mañgo.

The other of the first two centres was less circumscribed, being an alluvial district in the large island of Viti Levu, called Rewa. In it cases were scattered in a linear direction to a distance as the crow flies of ten miles from the point of outbreak, equal to nineteen miles by the river, which is the ordinary highway of communication, or fourteen miles by pathways.

The third centre was the town of Suva, where the Melanesian immigrants' depot is, to which the indentured labourers from Rewa and Mañgo were returned, according as their various periods of service expired. A few cases occurred at Suva, however, as may be noticed by referring to the schedule of cases appended, in immigrants newly arrived from other and non-infected districts—cases which argue strongly in favour of the infectiousness of cerebro-spinal fever.

The two primary centres, Mañgo and Rewa, are situated 150 miles apart, of which 130 are over sea; and there had been no communication between the immigrants at these two places for many months previously. The third or common centre, Suva, is in daily communication with Rewa both by water and by land, being only fifteen miles distant; but it too is 150 miles from Mañgo, communication being by means of a local schooner or steamer.

The period at which the epidemic prevailed was the cool season of the year in 1885, an unusually cool one for Fiji, although the temperature did not fall in the littoral districts below 60° Fahr. There are no immigrants in the mountains, but only Fijian natives, who, as has been said, did not suffer.

Text-books tells us that regarding cerebro-spinal fever very little information is to hand; but they concur in recording its apparent preference for winter seasons. It is a noteworthy circumstance that this condition not only obtains in countries where ice and snow are common, but is now imitated in this epidemic within the tropics; and the fact deserves consideration in thinking over the microbe question, if it be sought to apply that theory to this disease.

By the merest accident I have been afforded an opportunity of glancing over a report on cerebro-spinal fever by

Dr. Collins of Dublin, in 1868.* In it there is given the most complete geographical and chronological history of the disease that can be desired. But the terrible *gêne*, especially as to literary privation, which medical officers necessarily suffer when living and working in outlandish corners of the world's surface, has debarred me from consulting any contemporary writings on the subject. I therefore crave the indulgence of my more fortunate and better informed brethren of this distinguished Society who live in London, and are privileged to breathe there an atmosphere of learning, of contentment, and of smoke.

The History of Cerebro-Spinal Fever in Fiji.

The Fijians, an intelligent and observant race, do not recognise cerebro-spinal fever as a specific disease, profess themselves unacquainted with its existence prior to the 1885 epidemic, and have no name for it. The earliest recollection of it in the islands by white men dates from 1876, when a few cases are said to have been observed in immigrant labourers in the depot at Levuka. No written record is known to exist of the occurrence. From time to time, during the succeeding eight years, a case of obscure, nervous, or febrile disease, with delirium or stupor, would occur on plantations amongst the same classes as those already mentioned; and would be reported by employers as "tetanus", "fits", "brain-fever", "sun-stroke", or "madness"; or death would be attributed to "moping", and a determination to seek the shades of Burotu—the Polynesian's Paradise—rather than longer endure the regularity and thraldom of plantation labour under white men's *régime*.

I can call to mind some eight or nine such cases, but the widespread nature of the population, and the hindrances to speedy or comfortable locomotion by medical officers, coupled with the necessity in this climate for early burial, have been the means of thwarting the performance of any properly conducted *post-mortem* examinations in such cases until last year.

It is probable, therefore, that death was not infrequently registered as due to idiopathic tetanus, sunstroke, or meningitis merely, when cerebro-spinal meningitis or fever should have been quoted as the true cause, even though not epidemic at the time.

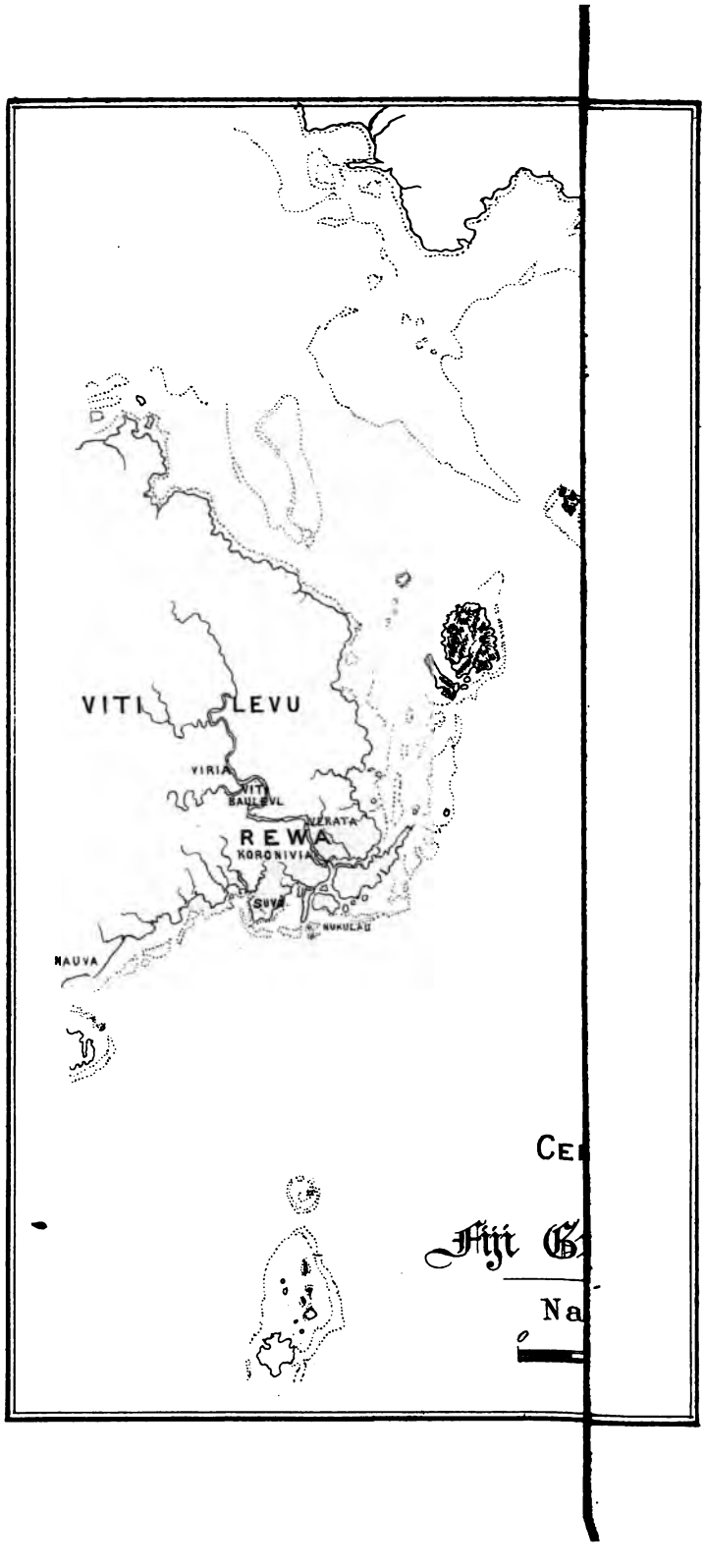
One series of cases, however, was, I am convinced, a genuine

* *Dublin Quarterly Journal of Medical Science*, August 1868.

epidemic of this disease; and, taken in connection with the visitation of 1885, is of the greatest interest. It occurred at sea, in the persons of men who had never been in Fiji, but who at the time were on their way thither as emigrants. The *Falcon* was a three-masted schooner engaged by a company of sugar planters and mill-owners in Fiji to recruit, under Government regulation, immigrant labourers. The vessel went in search of them as far as New Ireland, and in June 1883, with seventy-four recruits on board, turned her bows homewards, on the return voyage to Fiji. Sixty-eight of these people were New Ireland natives, collected at their homes between the 30th of May and the 9th of June. On the last-named day one of them named Lauhut, No. 20, was found to be suffering from a strange illness. He had been seven days on board. The Government agent of the *Falcon*, to whose official journals I am indebted for the particulars of the event, was not sufficiently familiar with medical distinctions to record the symptoms with scientific precision, but he had a general knowledge of minor surgery and "domestic" medicine. An examination of his records, however, which I have summarised in the table, when made in conjunction with the observations afforded by the epidemic of 1885, is sufficient, in my mind, to prove that the nature of the disease on board the *Falcon* was no other than that which two years later broke out among people of the same and neighbouring nationalities in the colony.

The survivors of those who embarked in the *Falcon* have recently completed their period of three years' indentured service and have returned home. On the eve of their departure I had a long conversation with them relative to the events of the outbreak, and their general condition when at home. Their testimony, now that they have acquired a fluency in the Fijian language, was very clearly given, and entirely corroborates the opinion I had formed that the epidemic on board the *Falcon* was cerebro-spinal fever.

What is of great interest, moreover, is the assurance, volunteered to me by the survivors of the *Falcon* people, that their countrymen who followed to Fiji by the brigantine *Marie*, three months after them, brought word that the *Falcon's* visit to the neighbourhood of their village had been immediately followed by the outbreak of a severe epidemic such as they had never witnessed before, of a very fatal character. They assert also that this sickness was the same as that subsequently witnessed by them in 1885 in Fiji, and that it was different from anything they or their oldest men had ever experienced before, save then.


VITI LEVU
VIRIA
VITI BAULEVU
NAUVA
REWA
KORONIVIA
SUVA
NUKULAU
NAUVA

It is rather surprising that they did not credit the *Falcon* with the introduction of the disease to their country; but it is also difficult to understand how her visit could have occasioned it, even if they had.

I am much more inclined to regard the occurrences as indicating a spontaneous origin of a cerebro-spinal fever epidemic among a tribe of New Ireland natives, some of whom happened to embark in the vessel during their incubative period, as no one on board, except the people from this place, was attacked by the disease, either before or after their embarkation. It must be noted, however, that while the natives of New Ireland generally, in Fiji, suffered more severely than most other immigrants, those who came thither in the two ships just now alluded to, although located on the same plantations, furnished only one case in the 1885 epidemic.

In 1882 I observed two well-marked cases of cerebro-spinal fever in children of European parents. One was a strong and lusty boy of 4¼ years of age, born in Fiji. The other was a stout little girl of eight years, who came thither from New Zealand. She had been eight or nine months in Fiji at the time of her illness, and lived in a house adjoining the other case. The boy died on the twenty-first day, while the girl recovered. Both cases occurred at the same time, in the month of June.

Shortly after the arrival of the *Falcon* I proceeded to England on leave of absence; but from the time of my return to the Colony, in September 1884, up to the end of May in the succeeding year, when the epidemic commenced, some six more cases of meningitis, non-tubercular, came under my observation in native and Melanesian subjects. There was, however, no connection between them, evident or suspected.

This brings us down to the epidemic now under review, to understand the points of which some local geographical knowledge is necessary. A map is appended for the assistance of the reader.

I wish it to be understood that when using the term Melanesia I desire only to include the islands peopled by the dark-skinned, woolly-haired Papuan or hybrid Papuan tribes, viz., the New Hebrides, Solomon Islands, and, speaking roughly, the New Britain and New Ireland groups. When it is wished to indicate the light copper-coloured, straight, or wavy-haired native of Melago-Polynesian origin, the term Polynesian is employed.

Navua.—The unfortunate collapse of a large firm of

sugar-growers and mill-owners during the early months of the year became the means of leaving about five hundred indentured immigrant labourers unprovided with food or necessaries. Of this number 298 were Melanesians, and the remainder Indian coolies. They were left in a condition which, but for the succour afforded them by the Government, would have speedily resulted in starvation or mutiny; and the Melanesians did, in fact, subsist on what wild yams they could dig in the bush, and what bananas they could appropriate about the deserted plantations. The hot season of the year was over, when exceptionally cool weather, with strong trade winds, set in, and continued during the whole of the temperate months in 1885; and at the time of this impending starvation the cold weather had already begun to make itself felt.

Navua, where these people had worked, is quite the coldest of the coast districts of the Fijian islands, being on the southern side of the largest one, and closely approached by high mountains extending from the interior. The thermometer sometimes, though rarely, falls to 55° at night; and may average 70° for days together as the maximum shade temperature. In 1885 the season was exceptionally cool and prolonged; and as the great majority of the Melanesian immigrants already mentioned were from countries lying within six degrees of the Equator, and had never been away from home before, the climatic conditions which they met with in Fiji may be said to have been in a considerable degree foreign to them.

The practice followed by most large plantation managers of giving their coloured labourers wooden and corrugated iron barracks to live in greatly contributes to their discomfort, as they are generally precluded from having fires in them at night—a custom inseparable from all South Sea Islanders in their own homes, and one of great hygienic value to them in many ways.

Means having been found by the Government to provide the Indians at Navua with fresh employment, it was speedily decided to remove the Melanesians to the immigrant depot at Suva, that they might be properly cared for while arrangements were being concluded for transferring their services to new employers also. A few hours after arrival there two of their number were taken suddenly and alarmingly ill; and one of these died, while the other eventually made a complete recovery. Both were admitted to the public hospital, and the cases appear in the register there as

"meningitis" and "stiff neck" respectively. Judged by the light of subsequent events, there is little doubt, I think, that both were cases of cerebro-spinal fever; and they seem, if one may be permitted to draw a comparison between the two diseases, to occupy a position parallel, as it were, to those so-called sporadic instances of cholera which, happening mysteriously in unconnected localities, so usually herald in India the advent of an epidemic outbreak of that disease.

Two other adventitious cases of cerebro-spinal fever also occurred at Suva in the month of July. Both died; one of them being also the subject of advanced phthisis. These did not come from Navua, but one of them had arrived only a week before from Taveuni, an island 112 miles distant to windward, which was not affected by the epidemic at any period. In the case of this man a tumour was unexpectedly found in the cerebellum. These four cases may perhaps fairly be called sporadic at Suva.

I now pass on to the consideration of the distinctly epidemic cases at the other centres, but shall revert to those of that nature which occurred at Suva.

Mañgo.—A week later than these events a local manifestation of the epidemic began its course in the island of Mañgo. The nature of the disease was not primarily recognised, and its ravages were attributed to the ingestion of poisonous fish. To this part of the subject also I shall again turn, as it did not come to my knowledge before the middle of July—so seldom was there communication; and in the meantime the first serious alarm came from Viria, an inland sugar plantation in the valley of the Rewa river, situated twenty-five miles above its mouth. This happened during the second week in July, and the earliest victims were natives of New Britain—young, adult men of vigorous physique. Seven of them presented febrile symptoms, with prostration and cerebro-spinal complications, between the 9th and the 14th of the month; and the district medical officer's attendance was procured. Three of the patients speedily died, and *post-mortem* examinations confirmed the diagnosis and testified to the absence of tubercle or other means of accounting for the occurrence. The Government authorities were at once communicated with, and it was resolved to remove all the New Britain people from the Viria plantation and to receive them in an immigrant depot (then vacant) on a small island situated a mile outside the mouth of the Rewa river, on the sea-reef. Here the sick could be isolated in the infirmary building and the healthy

drafted into one of the empty barracks, with the purest of sea-breezes surrounding them all. The islet is called Nukulan, and is twenty-three acres in extent, consisting only of sand, triturated coral, and shells, fertilised by the decaying vegetation which continuously falls from the cocoanut-trees, harringtonias, lemon-bushes, guavas, and many other varieties of brushwood which grow there. To this spot the New Britain people, thirty-one in number, were conveyed from Viria on the 21st July; but three more cases, making ten in all, had in the meantime declared themselves.

Eighty-four other Melanesian immigrants and 117 Indian coolies were residing on the Viria plantation when the disease broke out there, but, although they worked side by side with the New Britain men, no cases followed after the removal of the latter. Not so fortunate, however, were these themselves, for during their stay at Nukulan, which extended over six weeks, no less than eight more cases occurred amongst them, and seven found their graves in the sand. The duration of these fatal cases varied from twelve hours to twenty-six days. Thus, out of forty-one New Britain natives originally at Viria, eighteen in all developed cerebro-spinal fever within a space of thirty-four consecutive days, viz., between the 9th July and the 12th August. Of this number twelve died, being a proportion of 66.66 of the cases, or 29.26 of the whole batch, per centum. From the first seizure to the last death seven weeks elapsed. A circumstance which deserves mention is that thirteen of the eighteen cases are described as occurring in strong muscular men, while out of the five who overcame the disease two were noted as weakly subjects. The ages varied from fourteen or fifteen years to twenty-eight. All were males, and the employment upon which they had been engaged was the ordinary field-work of a sugar plantation. Their diet consisted of yams, sweet potatoes, and rice with sugar, with an allowance of meat or salt fish twice a week. They never used flour or bread, and their only drink was water. They emigrated from their own country in October 1883, and arrived in Fiji on December 2nd of the same year. They were at once indentured to the Viria plantation, and had thus worked there rather more than two years and a half prior to the outbreak of meningitis among them.

Rewa.—From the centre of outbreak at Viria the epidemic spread by degrees to several other plantations on the banks of the Rewa river. These were Koronivia, Navutoka, Bauleva, Viti, and Muainaweni, situated at various distances below

Viria, but all within ten miles. The most notable of these was Koronivia, a large sugar estate employing fifty-six Melanesians and 382 Indian coolies. The first case there happened on the 5th September, and it was speedily followed by ten others within three days. One of these cases was so sudden, that a man who had embarked with others in a canoe to cross the river, in apparent health, was semi-conscious and unable to stand or articulate when he arrived at the other side, yet the river is not wider than the Thames at Kew. The first five of the Koronivia cases were fatal, but five others, although precisely similar at the onset, terminated favourably within thirty hours of seizure. The Melanesians from this plantation were therefore removed to Nukulan, which the Viria people had vacated a week before, and they remained there exactly a month, during which only three new cases occurred.

Fifteen other cases occurred in the Rewa district; the particulars of their distribution may be gathered by a reference to the list of cases appended. One of them, however, was distinctly traceable to Sura, where the disease was then rife. The subject of it was a native of Malakula, who, having left the depot at Suva in health on the 28th August, arrived at Rewa the same day, and died of cerebro-spinal fever on the next. He had entered the depot on 17th July, after three years' service at Kanathea.

Mañgo.—Mañgo is a nearly circular island, three miles in diameter, situated in lat. 17° 27′ S., long. 179° 9′ W., forming one of the Fiji groups, and lies, as has been already mentioned, about 150 miles to the eastward, or windward, of Suva, Rewa, and Viria. It is owned by an Australian company, and produces sugar-cane, cocoanuts, cotton, coffee, maize, ground-nuts, and various food-crops, such as yams, bananas, taro, sweet potatoes, and cassara. For the cultivation of these products and the manufacture of sugar about 560 immigrant Melanesians are employed and domiciled on the island; with thirty-five Indian coolies. There are also about a dozen white men, mostly without families, but no Fijian natives resident on the island; 120 miles of the distance from Suva to Mañgo are over sea.

On the 29th May the first recorded case of cerebro-spinal fever at Mañgo occurred. Its subject was a native of Malakula, about twenty years of age, male, who had been two years in Fiji, at Mañgo. On the succeeding day a New Britain native was similarly attacked, and both died on the 2nd June.

From June 1st to the 9th nine more cases occurred, three only of them fatal; one being a native of Malakula, three of New Britain, and the remainder of New Ireland. During July six more cases were recorded, in various natives, all fatal except one. In August there were two only, but in September a recrudescence took place, and seven new cases occurred, six of them eventually fatal. In October there were three, all fatal, but no more occurred after the 14th of that month.

The majority of the victims were natives of New Britain (10), New Ireland (8), and Malakula (5); the remaining 7 belonging to Malata (3), Buka, Malo, Amhrym, and Epi.

A curious feature in the Mañgo epidemic was that its nature was not at first recognised. There being no medical man on the island, the manager, who had had long experience of Melanesians both at Mañgo and elsewhere, felt himself at no loss to diagnose the complaint, and promptly set it down as fish-poisoning. As this is a somewhat common accident at Mañgo and elsewhere in Fiji, he did not immediately report the earlier cases to me, and it was not until the 17th July that I received intelligence of what was going on. The description of the symptoms given by the manager in his letter to me was, however, so clear and so vivid that, profiting by my newly acquired experience of cerebro-spinal fever at Suva and at Nukulan, I at once disregarded his diagnosis, and was able with full confidence to ascribe the deaths to the disease under review. I subsequently visited the island and some of the patients, and satisfied myself on this point, although, unfortunately, I was not afforded an opportunity of making a *post-mortem* examination while there. The only female attacked during the whole of the epidemic was at Mañgo, a strong, healthy-looking adult woman from New Ireland, who died.

The nature of the employment, food, and general hygienic conditions at Mañgo was similar to that at Viria and other places, but the climate was somewhat drier and the nights less cool. The drinking water was from springs, instead of from the river, or from iron roofs, as at Viria.

Suva.—I now pass to the consideration of that portion of the epidemic which occurred at Suva, the third or common centre. During the earlier period of outbreak at Mañgo and at Rewa, several of the immigrants in those places terminated their indentured service, and were sent into the depot at Suva to await re-engagement or a passage to their homes. The majority of those from the Rewa plantations were natives of Buka, and a portion of them were people who had

been transferred a few months previously from the plantation already mentioned as having been abandoned at Navua. Of the Mañgo contingent a large number were natives of Malakula, while some came originally from New Britain and from New Ireland.

There had, up to the third week in August, been only four cases of cerebro-spinal fever at Suva which could be regarded as having any possible relation to the epidemic. A large batch of Buka natives arrived in the depot on the 8th and others on the 15th August. I inspected them all closely on the latter date. I held a long conversation with some of them on several topics, including the nature and names of various diseases occurring in their own country. Only three were detected as requiring treatment: one with boils, one an ulcer, and one with (monocular) conjunctivitis. At morning muster, two days later, one of these men failed to appear, and on being inquired for was found in the barrack, unconscious. His friends stated that he had complained of fever, headache, giddiness, and nausea, on the previous afternoon, but they had not then reported it. He was transferred to the hospital, but died on the 20th from cerebro-spinal fever.* Eight days later, on the superintendent being summoned at 5 P.M., another Buka man was found dead in the ward. He had been apparently quite well at morning muster, and his immediate friends stated that he had continued so up to noon. He then complained of pain in the head and neck, seemed giddy, ceased to speak or to swallow, became very restless, and had a hot, dry skin. In an hour's time he was unconscious and had convulsions, and in three more he was dead. On inspection, another man was then found ailing, a native of Malakula, and he owned to having been ill at least eighteen hours. He was sent to hospital, and went through all the stages of a typical case, but ultimately recovered in about six weeks.

The outlook was now sufficiently alarming, there being no fewer than 285 immigrants in the depot at this time. They were therefore mustered three times every day for close inspection by the superintendent, and were frequently medically examined by myself. It was impossible, through force of circumstances, to practise a high degree of segregation, but the cases, as soon as suspected, were isolated and sent to a special ward at the public hospital. The Buka people in depot were domiciled in a barrack by themselves, where carbolic powder was freely used, cleanliness enforced, and

* Notes of the *post-mortem* will be found appended.

the most ample and constant aeration employed—an easy matter in the tropics. The nights being chilly, a large open fire was kept burning in a trough of sand placed in the centre of the ward. The men were encouraged to live out of doors as much as possible in the day time, but a sort of panic seemed to take hold upon them, and all their movements indicated an irresolute, scared, and hopeless frame of mind, and they herded impotently together as if wondering, but scarcely caring, who was to be the next victim. Their position, indeed, was alarming enough to induce this feeling had they been other than semi-barbarous people.

The natives of Buka, which is a smallish island contiguous to the north end of Bougainville, in lat. 5° 5′ S., long. 154° 34′ E., are the blackest of all the Pelagian Negritos, or Papuan race, and possess many distinctive qualities altogether apart from the Malago-Polynesian stock, with whom ethnologically they appear to have no affinity whatever. There exists among them, however, a considerable degree of common-sense, and their perception is perhaps more acute than that of many of the mixed races in which the Papuan and Polynesian blood is blended. Although, about 1867, several of them were kidnapped by lawless traders and labour-seekers, it is only within the last half-dozen years that they have voluntarily ventured into white men's settlements, and as yet but little is accurately known about the Buka people's civilisation at home. For its size their island is immensely populous; but the establishment of German stations in New Britain and New Ireland promises to speedily drain Buka of its native race, as the white settlers there employ them, not only for agricultural labour, but also for purposes of defence, and in some cases offence, against the natives.

At this stage the epidemic claimed daily from one to three victims out of the people in the depot, and a special ward had to be set apart at the hospital to receive them. Up to the end of August only Buka and Malakula natives were attacked, although they formed together only a third of the total number of inmates. During the week ensuing after the occurrence of the first case (August 17), in the Buka men who came from Rewa no other cases declared themselves; but during the second week no less than seventeen cases were recorded, and several others suspected of being cerebro-spinal fever, though the symptoms and rapid recovery left them doubtful as to diagnosis. I seldom entered the depot without detecting one or two in an early stage who a few hours before had been in perfect health. Unfortunately, Melanesians are often backward in reporting their ailments, and

many lives are lost through the want of prompt remedial measures at a time when they may do the most good. This is especially true with regard to dysentery, their chief disease.

Of the seventeen above-mentioned all died, seventeen days being the duration of the longest case and four hours that of the shortest.

On the 1st September the Buka people were embarked in the ss. *Southern Cross* for their home. At this time there were four of them, all youths about fifteen years of age, convalescent from the disease, and it was difficult to know whether the sick, or indeed any of the batch, should be allowed to proceed home before the epidemic was thoroughly extinct amongst them. On the whole, I considered that it would be checked rather than fostered by the circumstances of a sea voyage; and as the ship was specially fitted for carrying immigrants, and they were to be accompanied by a gentleman as Government agent, possessing fair medical attainments, I determined to give the men the benefit of the doubt, and to risk the possibility, so remote as it was, of conveying infection into their own homes. The end proved satisfactory, as the four convalescents all made a good recovery, and only three fresh cases occurred during the voyage. These happened on the 1st, 5th, and 7th September respectively, and were all Buka men. The first two, after a severe struggle, were landed at their village on the 15th in a fair way to recovery; the other died within twenty-four hours of seizure. All the Buka passengers (eighty-five) were landed on the 15th September. There were thirty-six other immigrants on board for various islands in the Solomon group, but, after careful inquiries, I have failed to hear of any outbreak of cerebro-spinal fever in any of them, or in Buka either.

During this time the epidemic in the depot at Suva also waned. From September 1st—the date of the departure of the Buka people in the *Southern Cross*—to the 13th nine cases occurred. Three were natives of Malakula, one New Britain, two Malata, one Guadalcanar, one Aoba, and one Pentecost Island. Three of them eventually survived, and the fatal cases varied from one to twenty-eight days in duration. After this, only three more cases occurred in Suva, but they originated in the town, and could not be directly traced to the depot, which is a mile outside it. They all three terminated in the death of the patient—one after a few hours, one three days, and the other eight days.

These remarks conclude the narrative of the epidemic as it occurred actually at Suva. But as it had been ushered in

by four sentinel cases, so to call them, so its decline was marked by a few isolated instances in ships at sea conveying time-expired immigrants from the depot to their homes in the New Hebrides and Solomon Islands.

Those on board the *Southern Cross* can scarcely be classed among these, as one may safely ascribe a direct connection between them and the outbreak in the depot by means of the four convalescents on board, and the case of Sipiku (No. 122), which occurred on the day of sailing. But other cases presented no such link, and occurred in the schooner *Glencairn*. I must, however, refer to some cases that occurred on board the brigatine *Meg Merrilies* and the schooner *Albatross*.

The *Glencairn* sailed from Suva on the 1st August with ninety-nine natives, returning to the New Hebrides and Solomon Islands. Eighty of them were natives of Bougainville Island, and had been employed at the plantations at Navua, already mentioned. At the time this vessel quitted Suva the Buka people from Rewa had not come in to depot, and only three preliminary cases had occurred there when the passengers by the *Glencairn* embarked on July 29.

On the 15th August, however, an undoubted case of cerebro-spinal fever occurred on board in a native of Bougainville; and was followed by two more on the 16th, one on the 20th, and another on the 21st of the same month. In this instance, also, death was attributed by the Government agent of the vessel to fish-poisoning; but, although four of the five died, it could only be proved that one had eaten any fish at all. These people, then, were attacked with the disease during their third week at sea, at a period coeval with the outbreak in an epidemic form at Suva. The *Glencairn* was at this time at Espiritu Santo, in the New Hebrides, and did not finish landing the passengers before the 16th October; but no fresh cases occurred.

The cases in the ss. *Southern Cross* have been already alluded to; and the next cases in chronological order were those on board the *Meg Merrilies*. This vessel offers a curious instance in which it would appear that a man became infected with cerebro-spinal fever through association with others who, although they came on board from an infected place, did not themselves suffer from it. In her case a number of passengers were to embark who had been working in Taveuni, an island in the Fiji group, but 200 miles to the N.E. of Suva, where no cases of the disease occurred from first to last. That they might have the best possible chance of escaping, I did not permit these Taveuni people, on arrival

at Suva, to go to the depot, but ordered the coaster alongside the *Meg*, then lying at anchor in the harbour, and drafted them straight on board. They were ashore for half a day only, receiving their pay (in goods) at the contractor's warehouse. To make up the complement, however, it became necessary to send away in the *Meg* some of the immigrants who were in the depot awaiting a passage home, during the height of the epidemic there. Strange to say, although none of these became ill on board, one of the others from Taveuni did; being attacked on the seventeenth day out with unmistakable symptoms, and dying four days later. Unless his illness was independent of the epidemic altogether, this case lends some colour to the theory of the infectiousness of cerebro-spinal fever. But no other case of it has ever been recorded in a native of his island—Espiritu Santo—and there are many of them in Fiji. There were no Buka or Bougainville men in the *Meg Merrilies*, but only New Hebrideans.

The case which occurred in the schooner *Albatross* was that of Turia, one of the boat's crew. He was a native of Guadalcanar, in the Solomon Islands, had been nearly seven years in Fiji, and was taken ill on the 17th November, after being six weeks away from Suva. He died on the 23rd with well-marked symptoms. From that time to the end of 1886 a few more isolated cases of cerebro-spinal fever have occurred at sea in immigrant vessels—perhaps five in all; but the *rationale* of this singular and erratic disease remains, I regret to say, as obscure as ever. About an equal number of cases have occurred also since the termination of the epidemic in Fiji on shore, but they too have shown no particular connection with places or with one another.

Symptoms and Post-Mortem Appearances.

The symptoms and *post-mortem* appearances observed in the victims of this epidemic were sufficiently terrible. To detail them in all the cases would occupy too much space, and would be wearisome. As, however, full notes were taken of nearly every case, I may with advantage describe a typical one, and it will be sufficient to give a mere general outline of the others.

I was visiting the island of Nukulan on the 11th August, when the New Britain men from the Viria plantation were there located. I had turned towards the beach, with the intention of starting homewards, when I noticed a man, who, I learned, was named Taki, walking along the beach towards me. He was a big, stalwart, well-nourished native of New

Britain. I observed him become suddenly wild-looking, his body swaying from side to side, his gait staggering. His facial expression alternated between astonishment and fear, and his head was frequently bowed forward and thrown back. He came on a few paces, and then tottered to the ground, first on to one knee and then to a sitting posture, with his hands behind him on the ground so as to support his body erect. He did not speak, but was picked up by myself and three or four of his comrades, my boat's crew (natives of Malata, in the Solomon Islands) being afraid to approach him, and declaring him to be bewitched. On our way to the infirmary, about 100 yards distant, he struggled a good deal, and reminded me of a case of epilepsy. When deposited on a stretcher-bed Taki wriggled off it on to the floor; squatting sideways, and supported by myself and an assistant. He resisted attempts to control his movements or position; and spat out everything that was put between his lips. His teeth were not clenched; his tongue was white and furred, and his skin burning hot. His eyes were suffused, and presented a dazed, staring appearance. From his actions I judged that photophobia was present; but he could not speak. The head continued to be alternately cast forwards and thrown back, generally with a sudden, almost convulsive, movement; and the whole body and limbs were violently agitated and irritable on any attempt being made to alter his posture. The greater part of the surface appeared hyperæsthetic, more especially the trunk and thighs. The pulse was normal in force and frequency, though the elevation of temperature must have been very considerable. The patient's extreme restlessness prevented my using a thermometer.

I had examined this man three-quarters of an hour previously, in common with the rest of the people; and he then appeared to be quite well, and said he was so. About twenty minutes after the beginning of the illness I got him on to the bed again, and succeeded in placing a blanket over his body and applying a gentle cold douche to the head. He remained quiet, but soon afterwards lost consciousness.

On the following day Taki indicated that he was suffering severe headache, with pain and stiffness in the neck, and his head was retracted. He was almost unconscious, but when spoken sharply to in Fijian could answer questions. He could swallow but sparingly, and always rejected fluids or semi-solid nourishment. He occasionally uttered an abrupt, sharp shout—a feature which was frequently observed with cases in the other divisions of the epidemic, and which was

very characteristic. It resembled in some degree the short, single bark or yelp which a dog sometimes gives. Towards the close of the day Taki ceased to notice questions, and the eyes assumed a fixed gaze, like those of a person under chloroform, and he gradually became quite quiet, except his stertorous breathing and unconsciousness. At this time, the bowels being constipated, a drop of croton-oil was given, but was inoperative. An enema of castor-oil and turpentine, however, produced the desired effect. The urine was never retained too long, neither did it dribble; on the contrary, the act of micturition was performed with proper regularity, although unconsciously, and this was so in every case. A semi-purulent mucous discharge came from one nostril—another very frequent symptom in this epidemic; and on the third day there was conjunctivitis of one eye, while the other was dry, shrivelled, and deathlike. The pulse varied from full and slow to rapid and thready, and Taki gradually sank, dying on the 14th August, after three-and-a-half days' illness.

In addition to the symptoms I have detailed in narrating Taki's case, it was a common thing to meet with vomiting and diarrhœa at the onset, premonitory lassitude or prostration, severe and persistent headache, with pains in the back of the neck, along the cervical and lumbar (but not often dorsal) regions of the spine, confusion of ideas, fixed pain in one knee, or in a wrist or elbow, subsultus tendinum, and jactitation. The herpetic rashes, said to be so common a characteristic of this fever, were occasionally present about the corners of the mouth, and in one or two cases also over the pectoral region and the sacrum. But the purpuric spots alluded to by most observers were altogether absent, or else were undistinguishable in a native's skin. The Buka natives are extremely black in hue; those of New Britain and New Ireland are less so; and most of the other subjects of the disease were of a rich coffee-colour. But even the three white children failed to exhibit any approach to purpura.

In the later stages, after pus had been formed on the pia mater, paralysis of one or more limbs or sides was a common occurrence, but it often shifted its ground, one limb recovering and another being involved. These cases also suffered from great marasmus, and developed bed-sores, but generally died before the latter had existed long enough to become extensive. The whitey-brown or greenish muco-purulent discharge from one or both nostrils was an almost invariable accompaniment, and stuff of a somewhat similar appearance and nastiness was occasionally vomited. In some cases the

subsultus tendinum was very constant and wonderful to witness for a day or two together, and it was in these that I chiefly noticed the characteristic shout I mentioned in Taki's case, and the hyperæsthesia of the skin and joints. Busy, muttering delirium was present in some, while others reached the stage of profound stupor, without being noisy or chattering. I noticed that the patients invariably chattered, when delirious, in their own native tongue; while, if called by name and not too far gone to answer at all, they always replied in Fijian, which is the common medium of conversation in the colony. In most other instances of delirium occurring in Melanesians in Fiji I have found them employ the Fijian language throughout.

The Suva cases all began with diarrhœa and emesis. The Nukulan cases were all constipated at the onset, and remained so, while none of them vomited. In some the pulse was frequent and thready; in others full and slow. But it would vary surprisingly in an individual case, and seem to bear no relation to the outward signs of the patient's condition. The duration of fatal cases varied from four hours to a month. A great many died on the fourth day.

In a considerable proportion of the cases the onset of the disease was slow and insidious; in another set of cases it was of the type known as *foudroyante*, or *fulminante*—expressive and rather apt terms. Conjunctivitis, generally monocular, was not an uncommon event; and in one case deafness ensued.

Of the *post-mortem* appearances it is not possible to give a single general description to suit all the stages of the malady. Those in the cases which died early were generally as follows: On turning back the scalp an extensive blotch of black blood was found at the vertex, as if all the venous blood in the scalp had gravitated after death to a patch about three inches and a half in width. On lifting the skull-cap the dura mater was found strongly adherent in the middle line; and the first object which struck the observer was the gorged condition of all its veins and of the sinuses. Removing the dura mater, the arachnoid membrane was seen to be dull, and, where crossing the sulci, appeared semi-opaque, owing to the presence beneath it of a lymphoid or semi-purulent deposit of a milky, yellowish, or greenish colour, varying, according to the duration of the case, in hue, consistence, and amount. In some places this was enough to fill the sulcus, and occasionally was present in large quantity over various portions of the brain, but more often at the base than at the vertex, although the longitudinal fissure had its share.

Bright, green creamy pus was found in one or two instances in the lateral ventricles, and with frequency in the fourth. The medulla, indeed, and the pons, were sometimes bathed in pus; but this was only observed in cases of from three to five days' duration or longer. The pia mater was evidently deeply involved in the inflammatory process, and presented quite a pink aspect, with numerous diminutive bright red arteries standing half in relief from its surface, especially the spinal portion. Pus was generally present in the spinal canal, and was most plentiful in the lumbar portion. It varied, according to the duration of the case, from a mere "blink" of it investing the pia mater of the cord, to a couple of drachms of pure pus in the subarachnoid space. The entire brain was abnormally hyperæmic, if the expression be permissible—a condition shared alike by its membranes and by its substance. On section the white matter was seen to be plentifully flecked with hæmorrhagic puncta, here and there amounting to a couple of lines in diameter, but in general scarcely so large as a pin's head. Its substance throughout was, from this cause, darker than usual, especially the grey cortical portion and the bodies of the convolutions.

In sudden and short-lived cases, of the *foudroyante* variety, fully formed pus was generally undistinguishable; though the products of inflammation could be seen even with the naked eye, tending towards its development, especially in the sulci. Portions of these matters, when examined microscopically, revealed the presence of moderate-sized granular round cells, with nuclei scattered round their margins, looking as if young cells were forming, five or six at a time, by self-division from the parent cell. To all intents and purposes they resembled ordinary pus cells, and there was nothing else of note in the field. For the rest, the *post-mortem* appearances consisted chiefly in a preternaturally dark and fluid state of the blood. The muscles participated in this appearance, and were of a singularly livid hue throughout. The liver and spleen were usually intensely congested, and full of dark unoxygenised blood. The kidneys were less so. The lungs would present infarctions at the base, with patches of interstitial inflammation at the apices or middle. They were usually extremely friable. A good deal of frothy mucus was in them, and occasionally some of the bronchial tubes, a line or so in calibre, were occluded with viscid pus.

Recent pericarditis was met with in three cases. Two of these were like ordinary pericarditis; in the third the sac contained six ounces of a nasty-looking compound, which, in my notes, I find likened to a "mixture of pea-soup with beef-

tea". The membrane itself was "thickly inflamed, and coated with lumpy, blotchy, sticky pus". Most bodies were those of particularly athletic, well-developed men; but in cases where the disease had been long continued, such as a month, it was usual to meet with extreme emaciation. Nearly all the *post-mortem* examinations were made at Suva, a few only at Rewa, one at Mañgo, and one at Nukulan.

Treatment.

With reference to the treatment of cerebro-spinal fever, I will only say that no specific line of treatment appears to be of avail. I usually began by giving a minim of croton-oil if I saw the case early, and in following it up with quinine and arsenic in liberal doses. Cold irrigation to the head may be applied with good effect in some cases, but it is advisable to keep the trunk and limbs warm, and even to apply sinapisms. Most patients evince great difficulty and unwillingness in taking food or medicine by the mouth; and as there is no time to be lost in this disease, the latter should be administered hypodermically whenever possible, and stimulant or nutrient enemata be given. Bromide of potassium was used in several cases without apparent benefit. In fact, speaking generally, the progress of the disease may be said in nearly all cases to have been influenced by remedial measures. Local symptoms may sometimes be treated with advantage, as they arise, on general principles.

Conclusions.

The conclusions to which the facts of this epidemic lead one are, I think, mainly of a negative order. Their tendency is to impeach the correctness of many of the various theories hitherto enunciated as to the causation of this singular disease; whilst, it must be owned, they have not made clear the way to any new insight to its mysteries.

So far as I know, it is the first recorded instance in which an epidemic of cerebro-spinal fever has run its course within the tropics. It is probably, however, not the only one, as I have heard of eight cases—or eight deaths: I am not certain which—occurring amongst Indian immigrants at sea, while on the voyage from the West Indies to Calcutta, during the same year. This fact was communicated to me by the surgeon of the ship in question, Dr. F. Hasard; but, unfortunately, I kept no note of it, and cannot say whether the epidemic occurred while the ship was within the tropics or not.

The etiology of the disease is still shrouded in mystery.

It appeared in the cool season, it is true, but the cool season of the tropics. It attacked 127 males and only one female. But the female population in Fiji among the classes that were affected is less than five per cent. of the total. It attacked chiefly persons who were engaged in the cultivation of sugar-cane, or who had recently been so occupied. But it also attacked some who were not, and it spared many hundreds who were. It happened in a dry district, but also in a wet one; in a small sea-girt plantation on an island formed by coral upheaval (Mañgo); and along the alluvial banks of a river in an island, ninety miles by sixty, formed of volcanic products, and not merely by volcanic force. The water-supply in one case was from springs; in another from the rain caught in iron tanks, and from the river. The food was of varying quality, and derived from sources thousands of miles apart. The ages of the victims were various.

The occurrence in 1885 appears to have been an epidemic outbreak of an endemic disease, yet it began simultaneously in two different places, separated from each other by 120 miles of sea. Nevertheless, it showed a preference for natives of particular islands, 1,500 miles away, in both these centres, although there were large numbers of other people, living and working with them under precisely similar conditions, who were not attacked by it. The general hygienic conditions of all were good, and the size and ventilation of their dwellings were regulated by Government rules.

For a few weeks, however, some of those who afterwards were attacked suffered some degree of privation at Navua through the collapse of their employers. But yet it is not possible to trace a connection from any of these cases to those at Viria or at Mañgo, although the Buka men who died in the depot may have had the disease communicated *to* them by the New Britain men at Viria, occurring, as they did, a month later than the latter.

This leads me to the subject of communicability. On the whole, my impression is that cerebro-spinal fever may be communicated from one person to another. For instance, 329 immigrants, during the first half of the year were working at an island called Kanathea, about ten miles from Mañgo, and belonging to the same owners. A batch of eighty-seven of these was transferred in June to Mañgo. Three of them soon after became infected and died there, although none of those who remained at Kanathea suffered throughout the epidemic. Another, whose indenture expired, left Kanathea and returned to the depot at Suva, in May, before any cases had occurred even at Mañgo. This man

was seized on the 24th August with the disease, and went through all the most typical symptoms, although he ultimately recovered. He was then in the depot, and it was at the time that the principal outbreak occurred there.

In addition to this case, fifty-five others, whose indentures at Kanathea expired, left that island and arrived in the depot during the month of July. Of these, eight more were stricken by the disease while in the depot, and seven of them died there; while none of the immigrants still left at Kanathea took it, although they numbered 187. On the other hand, of those who left Mañgo itself, where the epidemic was in full vigour about this time, and returned to the depot, only two were attacked after their arrival there.

The most potent argument in favour of infectiousness lies, I think, in the cases of certain immigrants, who, when attacked, had only been a few weeks in Fiji, being new arrivals from the Solomon Islands in the months of June and July. They were quartered in the depot, at a time when the epidemic there had not exceeded the four sentinel cases, but, upon its developing as it afterwards did amongst the more recent arrivals from plantations, these four new immigrants became infected, and two of them died. It must be added that they were only four out of 183 arrivals in the months just mentioned, the remaining 179 being not affected by the disease. Only one other ship came in during the continuance of the epidemic, and her passengers were not allowed to communicate with the depot, but were received at the Quarantine Island for safety, where none of them fell ill.

One peculiar feature of cerebro-spinal fever which has been noticed in former epidemics was remarkably borne out in this one. This was its preference for attacking people in batches or gangs.

Thus, among the cases which occurred in the depot at Suva were some natives of one island, Malakula, who had worked together at Kanathea for the preceding three years, having emigrated in the same vessel. Kanathea was not an infected place during the whole of the epidemic, yet other Malakula natives in the depot at this time from other plantations did not suffer, with one exception. In the same manner the Buka men afford an example of the disease running in batches, as do also the New Britain men at Viria, the New Ireland people at Mañgo, and the Malata men and others at Koronivia.

I confess the circumstances attending this tendency of

the disease are so wanting in congruity that I am entirely at a loss to explain it. The mortality in this epidemic will be seen, on reference to the following table, to have been 70 per cent., within a small fraction.

CEREBRO-SPINAL FEVER IN THE FIJI ISLANDS, 1885.

Summary of Cases, Results, and Dates.

Locality.	Date of First Case.	Date of Last Case.	Number of Recoveries.	Number of Deaths.	Total Cases.
Suva... ...	May 17	Oct. 2	9	30	39
Rewa ...	July 9	Nov. 16	17	19	36
Nukulan ...	July 22	Sept. 11	1	9	10
Mañgo ...	May 29	Oct. 14	10	20	30
At Sea ...	Aug. 15	Nov. 17	3	7	10
Europeans ...	...	...	nil	3	3
Total ...	May 17	Nov. 17	40	88	128

Mortality, 88 out of 128 = 69.5 per centum.

THE DANGERS OF WATER-CONTAMINATION DURING DISTRIBUTION IN MAINS AND SERVICE-PIPES.

By JOHN SPEAR, Esq.

(*Read: April 11th*, 1888.)

The possibility of the entry of morbific material into water-pipes during intermissions of water-service is self-evident. It is not so evident, nor is it, I have found, generally known, that strong insuction may likewise occur during the continuance of general water-pressure. This phenomenon may, however, be brought about in various ways (I will show afterwards by experiment how readily it occurs); and there is ground for believing that street-mains, and especially service-pipes, may not unfrequently present all the conditions necessary for its production. Let me endeavour to explain the *modus operandi*. The "energy" of a column of water under conditions existing in a main or service-pipe is expended (*a*) in pressure or (*b*) in velocity; when a descending or even horizontal main or pipe is opened full diameter, so that water may escape in a full stream, all pressure (*a*) (above the normal pressure of the atmosphere, that is to say) disappears near the opening (as may be verified by the application on the wall of the pipe of a water-pressure gauge), the total "energy" being converted into velocity (*b*). But velocity is at certain parts of the water-stream greater, of course, than at others; in a straight pipe this part will be at the centre, in a curved one probably at the larger curvature. Here velocity (*b*) will be a *plus* quantity, and pressure (*a*), correspondingly reduced below the normal atmospheric pressure, a *minus* quantity. In other words, at that part of the stream where velocity is greatest, there will be a partial vacuum; and if it happen to be "tapped"—placed in communication with the external air or what not, by an accidental or other opening in the wall of the pipe—strong insuction, all the stronger for a greater "head" of water, will there occur. Again, an excessive and sudden "draw" upon a water-service at the lower levels of a system may lead to the formation of

partial and momentary vacua above; or, in the lower levels, the same result may follow interference above (as, for example, by the formation of "air cushions") to the flow of water.

Dr. Buchanan, investigating this subject in connection with his inquiry into typhoid-fever prevalence in Croydon, summarises as follows the conditions under which he found by experiment that indraught occurred through apertures in the sides of pipes running full of water:—"(1) The lateral incurrent is freely produced when the water-pipe is descending, and when the pipe beyond the hole is unobstructed; (2) if the force of water-flow in a descending pipe be moderate, a moderate degree of obstruction beyond the hole does not prevent the incurrent; (3) in horizontal pipes of uniform calibre, when the flow is strong, or the pipe beyond the hole is long, or when the end of the pipe is at all turned upwards, the incurrent does not take place; but (4) momentary interference with flow, a *tergo*, or momentary reduction of obstruction, *a fronte*, allows a momentary incurrent through the hole; (5) incurrent through a lateral hole takes place with incomparably greater ease when the hole is made at a point of constriction of the water-pipe."

Notwithstanding these various chances of contamination of water during its distribution—a chance exceedingly imminent in cases of intermittent water-service, and by no means remote even with constant supplies—the fact remains that mischief has very seldom been traced to this cause. Can this be supposed to be due to real immunity, or is it attributable only to shortcomings in our means and methods of investigation? In almost every town of any size localised outbreaks of enteric fever—"household epidemics" as they have been called—from time to time occur, and are never properly explained.

It has been my business recently to investigate an epidemic of enteric fever in a Glamorganshire town, and I propose to bring a summary of the evidence obtained before you. I believe you will agree with me that it demonstrates the cause of the epidemic to have been the contamination of drinking-water as it passed through one particular main. But this is not all. A study of the earlier history of the locality showed that ever since this water-main was laid, in 1885, recurring and hitherto wholly unexplained outbreaks of enteric fever have occurred amongst the houses supplied from it; and this under circumstances which, now that they can be collated and viewed as a whole, clearly point to the new water-service as the cause. The water at its source was pure, and the method of its distribution did not differ from

what no doubt may be found in hundreds of other places; or, if small house services be taken into account, in many thousands of other places. If from risks so general a remarkable series of outbreaks can be established in one place, it is reasonable to infer that many outbreaks, isolated and inconspicuous, have occurred from the same cause elsewhere.

The direct evidence of this, however, has been hitherto, as I have said, exceedingly meagre. The following well-authenticated instances only can be quoted:—An epidemic of enteric fever in 1873 at Sherborne, Dorsetshire, investigated by Dr. Blaxall; the well-known outbreak of enteric fever at Caius College, Cambridge, in the same year, reported upon by Dr. Buchanan; an extensive epidemic of the same disease at Lewis, Sussex, in 1874, reported upon by Dr. Thorne. Dr. Buchanan, again, in his "Report on Enteric Fever in Croydon in 1875", attributes a considerable share of the fever prevalence to "multiple local contaminations of the water in various parts of the Croydon water-system".

From Continental and American literature I have not been able, after, I confess, a very inadequate search, to obtain any clear evidence in this connection. Several outbreaks of enteric fever, attributed to the infection of streams, and more or less open aqueducts, are reported, but that is a different matter. At Winterthür, Canton Zürich, in 1872, an epidemic (reported on in the *Zürich Dissertations*), occurred along the line of one particular water-main, the rest of the town remaining comparatively free, and those who investigated the circumstances were unanimous in considering the water to have been the source of mischief. Because, however, the supply was constant—because, as Dr. Banson says, the pipes were always filled with constantly flowing water—it was held to have been impossible that contamination within the pipes could have occurred; and, accordingly, although the actual source of contamination was not discovered, a certain water-chamber at the head of the main was suspected.

The above instances exhaust my own stock of information upon this point, except for the striking occurrences recently the subject of my own investigation, to which I will now ask the Society's attention.

In the autumn-quarter of last year a severe epidemic of enteric fever occurred in the Mountain Ash urban sanitary district, Glamorganshire. The district consists of several detached mining villages, but the four central ones only, two on either side of the river Cynon, were affected by this outbreak. There was no doubt whatever as to the nature of the disease: clinically and pathologically the

typical appearances of enteric fever were presented. The epidemic was sudden in its onset. In the four weeks of July the attacks numbered, respectively, 4, 12, 52, and 95. Epidemic spread continued during August and September; in October the weekly number of cases again fell to single figures. In all, during the epidemic, 518 cases in 265 houses occurred.

The central villages contain a population of 12,700; but in them there was very marked localisation of the disease. On the right bank of the river, where the villages of Mountain Ash and Miskin are situated, there was a well-defined area in which the disease, in popular phraseology, "raged". Above, in the Mountain Ash village, the boundary of this area appears at first sight arbitrary or artificial, for it includes the lower part only of Oxford Street and the short streets *immediately* branching therefrom; below it includes practically the whole line of district, comprising the village of Miskin and Penrhiwceiber Road, until it terminates abruptly at the entrance of the Penrhiwceiber village. Within this strip of ground, about a mile and a quarter in length, 52 per cent. of the 225 houses were invaded; whilst in the adjacent localities beyond it only scattered cases occurred; the corresponding percentage being 2.4.

The history of family invasions was significant: the proportion of multiple attacks in households to total number of households implicated was 55 per cent. in what I may call the epidemic area; in the area beyond, 30 per cent. The proportion of multiple attacks *within the week succeeding that of first invasion,* in the one area was 35 per cent., in the other 5 per cent. Inquiry, moreover, showed that, as to the exceptional family invasions (*i.e.*, those outside the epidemic area), the first or only sufferer had been exposed to opportunities of infection within that area in 29 of the 40 cases—eight of these sufferers having, indeed, lived within its limits up to the time of seizure, and others having been frequent visitors (taking food, drink, etc.) at infected houses there situated.

Evidently the cause of the outbreak was to be looked for in some condition, acting with enormously greater force, probably exclusively, on what I have designated the epidemic area. Further, neither age nor sex was spared, so that this condition must have been independent of occupation; and, still further, the rapid occurrence, as already stated, of multiple attacks in so large a proportion of families living within the epidemic area, excluded any theory of the spread of the disease as an epidemic by personal contagion.

Very little inquiry (as to food supplies, beverages, places of popular resort, diseases of domestic animals, etc.) served to limit the influences that might be regarded as possibly fulfilling the requirements of the now restricted problem to three, viz., that of sewage contaminated air, of infected milk supply, and of polluted water.

The sewers had, in local estimation, been regarded as the source of mischief. Especially, there was a defect in the Miskin main district sewer, resulting in the diversion of the sewage from its proper outfall channel and its discharge, near the entrance of the village, into the river Cynon, which was thought to have had some causative relation to the outbreak. Concerning this it is to be remarked that the whole of the infected district is well elevated above the river, a line of railway separating the two throughout; that the nearest infected house is thirty-five yards from the river; that the streets in Miskin which suffered most are the furthest removed, 110 to 270 yards away; and that another deeply infected district, the lower part of Mountain Ash village, is situated more that 500 yards *above* the point where the sewage found entrance to the stream. Then, as to the possibility of infection from the sewers themselves, three several portions of the "epidemic area"—the Mountain Ash portion, Miskin, and Penrhiwceiber Road—draining each to its own sewer system, have nothing in common in the matter of sewerage except the main outfall which is common to the whole valley. Further, on the one hand, a small part only of the localities draining respectively to the Mountain Ash local sewer system and the Penrhiwceiber local system are included in the "epidemic area"; and, on the other hand, several collections of dwellings within the "epidemic area", but situated intermediately between the local sewer systems, so as to be beyond the reach of any sewers, suffered quite as severely as those for which sewers had been provided.

The milk supply came early under suspicion, for a very large number (afterwards ascertained to be nearly 70 per cent.) of the infected families in the epidemic area obtained milk from one, not very extensive, purveyor. It was found, however, that Miskin, the centre of this area, was practically his "preserve"; so that the small quantity of milk which the families here obtained (rarely more than half-a-pint a day and often only half-a-pint on Sunday), was mainly supplied by him. On the outskirts of the epidemic area the coincidence vanished, the milk-walk extending in certain directions beyond that area, and *vice versâ*. Thus of the thirty-

one infected families in the upper or Mountain Ash portion of the area only three were supplied by the dealer in question, while the Penrhiwceiber village below, within the range of his milk distribution, was beyond the area of the epidemic disease.

The water for the supply of these central villages is collected from springs and surface drainage of the steep hill-sides, away from cultivated land, and at its source it appears to be fairly exempt from risks of contamination. On the right bank of the river it is collected into a reservoir above Mountain Ash, from which all the Mountain Ash division, Miskin and Penrhiwceiber Road, with the exception of a few individual houses, were, for some time after June 30th, 1887, supplied. Starting from the reservoir, an 8-inch iron main passes down Pryce Street in the Mountain Ash village towards the bridge over the river, giving off branches to streets that together constitute rather more than seven-eighths of Mountain Ash village; and all this part, except for a few scattered cases, several of them with distinct histories of exposure to infection elsewhere, was free from fever. At the foot of Pryce Street, near the bridge over the river, the 8-inch main bifurcates; one 6-inch pipe passing across the bridge to unite with a water-system on the other side, the other bending to the right down Oxford Street. For the first 160 yards this Oxford Street main supplies only service-pipes to twenty of the houses in Oxford Street, but thenceforward serves both sides of the street, and gives off at short intervals branches to Henry Street, Bruce Street, the lower part of Daren Road, and Ifor Street. Continuing as a 3-inch pipe it supplies, with a few individual exceptions, the whole of Miskin, and is then carried along Penrhiwceiber Road, supplying most of the scattered blocks of cottoges on either side, until it terminates at the entrance of Penrhiwceiber village. The "epidemic area", and the one supplied by the various ramifications of this particular water-main are thus shown, so far as their marginal limits are concerned, to be exactly conterminous.

The coincidence is likewise carried within the area. There certain individual houses or groups of houses are not supplied with water from the common source, but depend upon local springs, etc., from the rising ground behind. This is the case with a collection of seventeen houses known as the "Bush" at Miskin, and with smaller groups and a few isolated dwellings on the Penrhiwceiber Road and elsewhere. In all, thirty-five houses within this area are unprovided with the supply from the public main, and whilst in the remaining

396, 224, or 57 per cent., were invaded by fever, in these thirty-five only one case occurred, the sufferer being a young woman who had been employed for some time up to the date of her attack in helping at infected houses, washing soiled linen, etc.

The water-service I have described was the one in existence from June 30th to August 19th. Before the 30th June the upper streets of Miskin had been supplied from the "Bush" spring, an independent source. On August 19th a further change was made, by which the Mountain Ash supply was cut off from the *lower* streets of Miskin and from the Penrhiwceiber Road; so that if the water from the Oxford Street main had been alone instrumental in distributing the fever poison it might be expected that some fourteen days after the 19th (taking 14 days as the limit of the period of incubation), or about September 3rd, fresh attacks in these particular streets would practically cease.* As a matter of fact the disease by this time was showing signs of abatement generally; still, there is marked difference in its decline in these streets and in the others of the area. Of the 1,634 inhabitants who continued to receive water from the Oxford Street main, fifty-four, or 3.3 per cent., suffered from fever after September 3rd; of the 712 who ceased to be so supplied on that date five only, or ¾ per cent., thereafter developed symptoms of infection.

There were other variations in the incidence of the disease within the epidemic area, as I will presently show. In no case, however, did they weaken, but on the contrary they served to strengthen, the conclusion deducible from the foregoing facts. They may be more conveniently discussed in considering the origin of the water-contamination.

The earlier history of enteric fever in the district is found to have an important bearing upon this inquiry. Taking the year 1886 and the months of 1887 (January to June) that have not already come under consideration, it is found that forty-four household invasions were reported by the Medical Officer of Health to the Sanitary Authority as having occurred during that time in the villages on the right bank of the Cynon. Of these household invasions twenty-five occurred in August and September; the outbreak being reported at the time as obscure in its origin. It now appears that all these twenty-five, and all but four of the forty-four infected houses, are situated in the epidemic area of 1887; so that for the whole

* Unless, indeed, it could be supposed that the street or house service-pipes (which, of course, were the same), having been infected, might still retain the contagium.

period the proportion of infected houses in the "epidemic area" is equal to 9.3 per cent., in the other to less than ½ per cent. Moreover, in one of the four exceptional cases, the subject of attack had been engaged for some time before her seizure in washing, nursing, etc., at an infected house in the "epidemic area"; and, in another, the sufferer attended a school within that area.

It may be remembered that several streets in Miskin were not supplied from the Oxford Street main previous to July 1887. Up to June 30th of that year the upper streets received water from the Bush spring. The houses known as the "Bush" also, as well as several scattered ones elsewhere, always obtained water from independent sources, as I have already explained. Again, then, an opportunity is afforded of comparing the incidence of the fever upon two sets of people living in this area, but receiving water from different sources. Of the 231 houses that received water from the Oxford Street main during the period, January 1886-May 1887, thirty-six, or 15.6 per cent., were invaded by fever; of the remaining 200, four, or 2 per cent., suffered.*

Of the 200 houses last spoken of, 165 were, on June 30th, 1887, brought within the range of the service from the Oxford Street main; and as it is interesting to see how they fared under the altered circumstances, the epidemic of 1887 may be again cited. So far from continuing to enjoy immunity, 70.7 per cent. were then invaded by fever; whereas, of the remaining 231 houses so supplied, only 46.6 per cent. suffered. The cause of the lighter infection of the latter at this period appears to have been threefold. The removal (as already explained) of 124 of their number from the area of the infected water-service before the entire cessation of the epidemic, accounts for 5 per cent. of the 24 per cent. difference; the prevalence of the disease amongst their inhabitants in the previous year, leading to some diminution in the number of susceptible individuals, accounts, it may be estimated, for an equal share. The difference remaining (equal to 14 per cent.) is attributable to conditions that I shall have again to remark upon, and is associated with the unequal incidence of the disease upon houses supplied directly from the trunk water-main, and upon those supplied from terminal and "dead-end" branches.

As to still earlier experience, a search of the death-registers

* In three of these four exceptional cases the attacks were single ones. In one the sufferer had been employed in a house where the water of the Oxford Street main was used; in two others the sufferers had, within a week of their seizure, removed from infected houses.

shows no undue prevalence of enteric fever during the years 1880-84 in the area subsequently involved in the epidemic disease. In that area, during the five years in question, two deaths only were ascribed to this cause, while in the remaining part of the district there were eleven. That is to say, the proportion of such deaths to total population was 0.2 per 1,000 per annum in the smaller area, and 0.3 per 1,000 in the other.

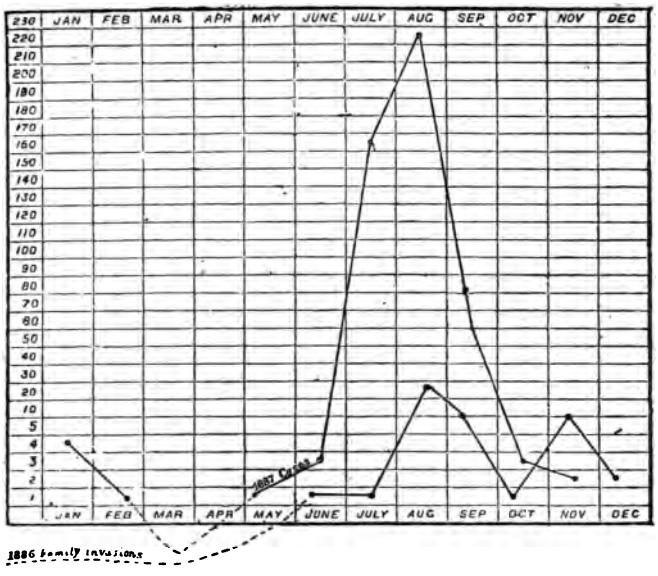
Up to the early part of 1885, Miskin village was wholly supplied from the "Bush" spring. Then, owing to increased demands, a supply from the Oxford Street main was laid on to the lower part of the village. In August 1885 a new and larger (6-inch) main was laid in Oxford Street, under circumstances which I shall presently have to explain.

In the autumn of that year a small outbreak occurred, that in the light of after events cannot but be regarded as of importance in the inquiry. Several cases occurred in Miskin in the streets newly supplied from the Oxford Street main, while, according to the testimony of medical men and inhabitants, the upper streets of Miskin, supplied from an independent source, remained entirely free. Two deaths were registered, one in September and one in October. The mortality for the year in the area (comprising 200 houses) now supplied by the Oxford Street water-main was equal to 1.7 per 1,000 of the population; in the remainder of the district the corresponding proportion was 0.4. The provision of the new water-supply was coincident with the commencement of the endemic prevalence of the fever.

I have given now a summary of the facts and figures by which, it seems to me, it is shown that the water of one particular main has been the vehicle of contagion, not in one epidemic only, but during a prolonged prevalence of enteric fever.

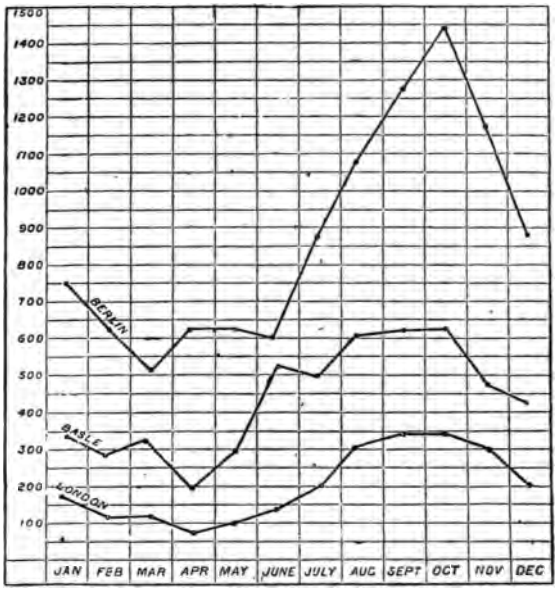
The inquiry, however, as to how the water became infected is not materially forwarded. The immediate and complete extension of the disease in 1887 to the streets newly supplied from the Oxford Street main, and, afterwards, its immediate disappearance from other streets so soon as that water was cut off, go to show that the sufficient cause of the mischief was in the water of the trunk main itself. Still, it is quite possible that the sources of that infection were accidental and ephemeral—that the epidemic outbreaks of 1886 and 1887 (to confine attention to the later and more fully investigated years), and the single household invasions of the intermediate period, were, each one, due to independent infections of the water; or it may be that throughout there

FIG. 1.



Epidemic area (*i.e.*, area of Oxford Street water-service) (1887), population 2,346.
" " " " " (1886), " 1,356.

FIG. 2.
COMPARATIVE MONTHLY FREQUENCY of TYPHOID FEVER in BERLIN, BASLE, and LONDON (after Liebermeister).



NOTE.—The Berlin curve represents the time of death of the patients; those of Basle and London the time of their reception into hospital.

has been one continuing focus—that at one particular point in the neighbourhood of the main, from which access to the water was possible, the elaboration of the fever poison has been in progress.*

Evidence bearing upon this point (one, it will be seen, of great etiological interest) is found in the study (keeping still to the later years) (1) of the locality of initial contamination of the water, as indicated by the earliest appearance of the disease in its various manifestations; and (2) of the general behaviour of the disease itself in its endemic prevalence.

As these general considerations naturally assist in the comprehension of the more particular phenomena, they may be first discussed.

The diagram (Fig. 1) illustrates the monthly incidence of the disease in the epidemic area during the two years 1886 and 1887. In comparing the curves it has to be remembered that the one for 1886 represents *household invasions*, that for 1887 *individual cases;* that in the earlier year a smaller area containing a population less by some 40 per cent. was involved; and that, as to period of maximum intensity, the curve of 1886 represents the time at which the fever was reported, that of 1887 the time of individual seizures. Bearing the qualifications in mind, it will be seen that the outbreak of 1886 was almost an exact counterpart, on a smaller scale, of that of the following year. (Fig. 1.)

Comparing now the above chart with the one that follows illustrating the seasonal prevalence of enteric fever in places where the disease is endemic, or places in which the study is of such broad proportions that *natural* characteristics attaching to the disease are necessarily displayed, it will be found that its curves are, with slight exceptions presently to be noticed, those natural to the disease itself. (Fig 2.)

In this small area, then, at Mountain Ash the all but continued presence of the disease for a period of some twenty months is demonstrated; its large development and wide spread take place in orderly sequence and due season. The contagium follows, in short, as though operating from its proper home, undisturbed by accidental external conditions, the normal course of its existence; and in so doing affords presumptive evidence that independent and fugitive foci of infection have played at most only a subordinate part in its local manifestations.

* I exclude, on *à priori* grounds, the possibility of continued existence in the main itself of the contagium. Running water may frequently act as the vehicle of its distribution, but it would be contrary to current scientific opinion to suppose that in such a medium it could complete the various stages of its life history.

The view which holds the soil to be the natural habitat and place of germination of the typhoid contagium has a mass of widely collected and varied evidence for its support; and it has been argued with much plausibility that the later incidence of the disease in certain endemic localities, notably in Munich, is due to the deeper situation in the subsoil of the infecting germs. The influence of the summer heat is apparent, and we know that the temperature of the soil to a considerable depth follows slowly the temperature of the air at the surface. In Mountain Ash, it may be remarked, the elaboration and activity of the fever poison, as indicated by the curves above, are slightly earlier in point of time than in other plans selected as standards for comparison. The fever curves follow with somewhat unusual promptitude the variations of the thermometer at the surface; and we might suspect, from the situation of the water-mains and the continued infection of the water, that the fever poison there lies in the superficial soil.

To return to the more exclusively local phenomena: 160 yards from the commencement of the Oxford Street main, the first street branch, that to Henry Street, is given off, and it is at this point that evidence of water-contamination arises. In the next 160 yards of the main's course short branches are given off to Bruce Street, the bottom of Daren Road, and Ifor Street. In all, from this second 160 yards of main, ninety-nine houses, together with the Ifor Street School, are supplied. In the outbreak of 1886, houses in Henry Street were amongst the first infected (of the first nine reported as infected five were situated in that street and one in Ifor Street). In the epidemic of 1887 Henry Street was again amongst the first to suffer, and the six houses infected before July 9th were all amongst the ninety-nine above referred to. In the interval between these epidemic periods, from the end of September 1886 to the beginning of June 1887, of the fourteen infected families four lived in Oxford Street itself (within the 160 yards below Henry Street), one in Henry Street, three in the lower part of Daren Road, and one in Ifor Street; in other words, nine of the fourteen were amongst the ninety-nine supplied from the second 160 yards of the Oxford Street main. In the first 160 yards (above the Henry Street bifurcation) there was no case of fever at any time throughout this period, nor, indeed, in 1885.

Here then is a focus of infective activity permanent in duration, primary in point of time and place. The question of its sole sufficiency has now to be examined.

The evidence already adduced as to the natural and cha-

racteristic behaviour of the disease, showing as it does little room for the disturbing influence of fresh accidental infections, is not to be forgotten; but, apart from independent sources, such as that evidence tends to exclude, there is the question of secondary foci of water-contamination. Given a fever poison circulating in a water-system, it is quite possible that in the dead ends of pipes, or at other places were stagnation occurs, or, again, at any point where escape may take place into the soil, further development of the poison, leading to secondary infection of the water, so to speak, may ensue. This question is not, as will be seen, touched by evidence as to the general behaviour of the disease, but requires examination at the closest quarters.

In such a scrutiny this important fact is at once apparent, that whereas the upper (Oxford Street) part of the infected area suffered to a greater degree than the lower (Miskin) part, as I have shown, at the commencement of each epidemic period, and in the endemic interval, so soon as each epidemic was established the proportions were reversed. In the first-named periods the proportion, making due allowance for the difference of population, was approximately as five to one; in the latter as one to two; so that while the fever prevalence was more continuous in the upper locality, in the lower it was more severe.

This reversal of experience is, it seems to me, explicable by one of two hypotheses only: either it was due to some difference in the physical circumstances of water-service during the two periods, or at the epidemic times there was increase and development of the fever poison at the lower levels of the system; or possibly these two conditions may have contributed their influence.

Examining this question, it is found that the comparative immunity during epidemic periods was not shared in by the whole of the upper locality. Henry Street and Ifor Street, situated at opposite extremities of the second 160 yards of the Oxford Street main, contain together thirty-seven of the ninety-nine houses. In the epidemic of 1887, 70 per cent. of those in Ifor Street, and 56 per cent. of those in Henry Street, were invaded; and in the outbreak of the previous year the houses in Henry Street suffered to the extent of 22 per cent. In the lower (Miskin) locality the corresponding proportion in the two epidemics was, respectively, sixty-two and twelve.

The immunity spoken of was enjoyed then by the remaining sixty-two houses only, so far as the epidemic of 1887 was concerned, and in 1886 was not shared by Henry

Street. Of the sixty-two houses, forty-five are situated in Oxford Street itself, thirteen in Daren Road, and four in Bruce Street. Amongst these nine only (seven in Oxford Street, one in Daren Road, and one in Bruce Street), or 19 per cent. of the total number, were invaded during the epidemic period of 1887, and in the smaller outbreak of 1886 only one. On the other hand, this was the very locality that suffered most during the endemic interval. At that time Oxford Street and the half-dozen houses immediately abutting upon it in the Daren Road contributed the majority of the fever cases.

There was this difference in the circumstances of water-supply during the two periods. In the summer, when a nightly discontinuance of the service has to be enforced, it has been the custom to close a valve at the top of the Oxford Street main, thus cutting off the supply of water to the whole of the district served by it, but preserving the unity of the system; in the spring and winter there have been frequent intermissions necessitated for the purposes of repairs, etc., but these have been quite temporary, and, when made in Oxford Street, a second valve at the lower part of the street, just below the Daren Road turning, has been closed in addition to the first, in order, as it is stated, to prevent the water from beyond that point from "coming back upon the workpeople"; for the descent from Oxford Street is not continuous throughout the system, the mains having again to rise at Miskin. The short section thus isolated is then quickly emptied of water. The difference would result in this, that after the nightly intermissions of the summer months the morning inflow of water would find (except perhaps for some dip in the pipes here and there where stagnation of the previous day's water might have occurred) a free course open for it throughout the system; after the irregular intermissions I have spoken of the inflow would meet, possibly the closed second valve, or if that had been opened, unless the intermission of supply had been unusually prolonged, a body of back flowing water. In either of the latter cases the tendency would be to limit the first flow of water, and with it the impurities that had reached the pipes during the intermission, to the first section of the main and its house-services. This will be better understood when I explain the exact circumstances of the latter.

The houses in Oxford Street are supplied direct from the 6-inch main, the house service-pipes leaving the main through a ferrule inserted in the crown of the latter, those of Daren Road and Bruce Street from sharply rising 3-inch branches.

It might be supposed that impurities reaching the Oxford Street main during intermissions of supply would be swept onward past these channels towards the lower levels, by the first flow of incoming water, *providing that the course was clear.* The steep gradients of Bruce Street and Daren Road would, moreover, effectually prevent any lodgment or stagnation during the intermissions of supply.

The circumstances of the Henry Street main are exactly the reverse of all this. It is a descending terminal (dead-end) branch. It would take its full share of the first rush of water down Oxford Street, and all impurities would have to be withdrawn through the house-taps.

The Ifor Street service-pipe is a terminal (dead-end) branch from Daren Road, and on the theory of a sole sufficient focus of infection on the line of the Oxford Street main it is difficult to understand why this street should have suffered more than its immediate neighbours, Oxford Street and Daren Road. In the outbreak of 1886, indeed, only one house of the ten was invaded, but in that of 1887 there were seven, and 30 per cent. of the inhabitants suffered.

The pipe (a 3-inch) is about fifty-three yards long, possesses a gradual ascent from Daren Road, and when laid bare was found in good condition. On the other hand, at the top or distal end it supplies by service-branches, without the intervention of any cistern or flushing box, two school closets, the pipes passing straight from the main into the closet-pans. When the water in the main was turned off, and the tap of one of these closets opened for a moment, suction of air up the pipe was immediately heard. The school was broken up from July 13th to August 16th for the summer holidays. Two of the children were attacked by the fever before that date and others subsequently, so that the closets would be almost certain to receive the specific poison of typhoid. Evidently this particular main was liable to secondary local contamination.

In the lower division of the infected area (Miskin and Penrhiwceiber Road) no such diversity in the distribution of the fever as that experienced in the upper locality was exhibited. In the epidemic of 1887, Glyn Gwyn Street showed the lowest proportion of infected dwellings, viz., 54.5 per cent., and Mount Pleasant Terrace the highest, 76 per cent.; and this divergence is, as I have already shown, to an extent accounted for by the fact that the water of the Oxford Street main was discontinued on August 19th to certain of the streets in this locality (Glyn Gwyn Street amongst the number), and by the previous incidence of the disease.

Still, the actual divergence has not yet been wholly explained, and the experience of the upper locality affords a clue to its meaning. Comparison of the relative incidence of the disease upon houses supplied direct from the main water-pipes, and those supplied from branch and terminal street services, gives the following results. The comparison has to be confined to the epidemic of 1887, for before that year there was only one "dead-end" branch in Miskin; and, in order to avoid the disturbing element of the change on August 19th in the water-supply, to the period antecedent to September 3rd.

	Epidemic Area.	Percentage of Houses invaded during period in question.
Upper Locality.	Houses supplied from 6-in. main ...	11.1
	From Branches	37.0
Lower Locality (Miskin).	Houses supplied from 3-in. main ...	56.0
	From Branches of ditto	65.4
	Terminal extremity of 3-in. main (Penrhiwceiber Road) ...	66.7

Comparison may be carried still closer with apparently the same results. One large 3-inch branch from the Miskin main passes along Victoria Street and thence up Mount Pleasant Terrace, where it terminates in a dead end. These streets were supplied throughout the epidemic period with Mountain Ash water. The 95 houses of Victoria Street suffered to the extent of 70.4 per cent. during that period; the 50 of Mount Pleasant Terrace to the extent of 76 per cent.*

Chemical analysis of the water and, still more, microscopical examination afford results confirmatory of much that has been advanced. Dr. Dupré, to whom samples were sent for analysis, reports that water taken from a house-tap in Henry Street, before the nightly intermission of supply, was pure; while that from the same tap after intermission gave evidence of animal contamination and of the appearance of living organisms. Water from a tap in Victoria Street, Miskin, showed similar deterioration (the presence of

* The evidence supplied by the statements of the water consumers is not without significance in the question under consideration. Throughout Miskin the complaint of the water being, in the summer, on first drawing in the morning, peculiar to taste and smell, was general, and in Ifor Street and Henry Street similar evidence came from, I think I may say, most of the thirty-seven houses; but in Oxford Street such complaints seem to have been far less common. The point was not the subject of any special inquiry, but statements to the above effect were made to me in only two of the houses of Oxford Street and Daren Road.

fungoid growths and of large and small animalculæ, etc.), both in the morning and evening supplies.

Reviewing the evidence, it seems clear that, under the circumstances of intermittent water-supply such as existed at the epidemic period, some special danger attached to the consumption of water from branch mains and "dead-end" pipes. Whether this arose from the simple physical circumstances of water-flow and distribution I have described, or from the development and multiplication of the fever poison in pipes permitting more readily stagnation of contents, the evidence cannot satisfactorily decide. The main fact of its being so, and the intensification from the upper to the lower levels of the system of the infective capacity of the water during the epidemic period, admit of either interpretation or of both combined. The fact that the disease promptly disappeared from those streets from which the Oxford Street supply was discontinued on August 19th, points to the infection of the Oxford Street main as the sole sufficient cause; only this evidence is somewhat discounted by the circumstance that, with one exception, these very streets were never supplied from terminal or dead-end branches. Minor points tend some to one and some to the other conclusion; *e.g.*, there is the experience of Ifor Street on the one hand, and, on the other, the very uniform infection, not materially disturbed by some partial though definite escape on the line of main pipes, of the several streets of Miskin village.

Whatever may be the true answer to this particular question, the facts all tend to bring into more and more prominent relief the point of initial contamination of the water; and, in view of the now clearly established premises of my argument, the exact condition of the Oxford Street main and its relation to immediate surroundings become matters of exceeding interest and importance. I have shown that the water as it passed into the main, and for the first 160 yards of its course there, produced no ill-effects in its consumers; and I have identified that point, just at the Henry Street bifurcation, as the one where continued infection of the water occurred. Was the main at that point carried through a breeding-ground of the typhoid contagium receiving habitually infection therefrom?

It was almost permissible to hope from the evidence that one could have foretold the very spot at which to find a defect in the Oxford Street main permitting the entry of specific contamination; but a little further examination showed difficulties in the way of this precise identification of

the spot. The Oxford Street and Henry Street mains are so related that during intermissions of water-supply any air-borne matter gaining access to either of them would probably be carried back some distance, by the aspiratory action of the other, to the point of their bifurcation. Indeed, for some yards below that point, water will flow back from the Oxford Street into the Henry Street pipe, a fact that is made use of when it is desired to empty the former quickly.

The Henry Street pipe was laid some twenty-five years ago; that of Oxford Street was relaid, as I have said, in 1885. There is no doubt that the relaying was very carelessly, not to say recklessly, done. Owing to the exigencies of water-supply, it was done mostly at night, and the works, being in private hands, were subject to no supervision on the part of any official or authority having concern with the public health. Accordingly, the main was carried, without any special precaution, immediately above, alongside, and even through old rubble drains; and when, in the course of the trenching, drains were cut through, no trouble was taken to replace them, as is shown from the number of stopped drains that were soon afterwards discovered. It may be said that, as one result of the careless laying of the main, the latter was at different points, from time to time, bathed in refuse matters, and habitually, at certain points, in sewage-contaminated air.

The two pipes (those of Oxford Street and Henry Street) were laid bare for some distance below the point indicated as that of initial contamination of the water. At ten different places leakage was discovered, in three places from the main itself. At two points water was found to be escaping into drains.

In addition to the instance of direct communication between water-mains and closet-pans at the Ifor Street School, already mentioned, two other such cases were found in connection with the Oxford Street main, and these, I believe, were the only others in the district.

The water-service was intermittent in the summer months, as I have said, pressure being shut off at night; in the winter it was on the constant-service system, although frequent temporary intermissions for purposes of repairs, etc., appear to have been necessary in the Oxford Street main. During such times insuction of air or liquid matter, from the soil or from the drains, as the case might be, must have occurred at the points where fracture or other defect of the pipes leading to leakage was found. I had myself opportunities of seeing how great the suction into the mains was during discon-

tinuance of water pressure. From taps in Ifor Street I allowed three or four pails full of strong solution of burnt sugar to be drawn into the main; on resumption of supply, complaints of discoloured water were found to be most numerous at the distal end of the system, particularly from the "dead-end" main of Mount Pleasant Terrace, where, it may be remarked, the fever prevalence had been most severe.

The circumstances of the Oxford Street main are, moreover, such as would appear to well fulfil the conditions set forth at the commencement of this paper as productive of lateral incurrents during the continuance of general water-flow. It is a descending pipe from its commencement to some 120 yards below Oxford Street; there it is carried in, in full diameter, to large colliery workings, where, for the purpose of supplying boilers, locomotives, etc., etc., it has been the custom to take, from time to time, very large draughts of water. Here, then, is the "momentary reduction of obstruction, *a fronte*", spoken of by Dr. Buchanan. Again, at the commencement of the main, a twin branch (6-inch), from Price Street, is given off to join the system on the other side of the river. Usually, I am assured, no flow of water occurs through this pipe, but, under circumstances leading to diminished supply, or larger consumption, on the opposite side, it would be, perhaps suddenly, fully drawn upon. Hence there might arise in the Oxford Street main the "momentary interference with flow *a tergo*".

It results from the foregoing observations that, during intermissions of the service, large contamination of the water of the Oxford Street and Henry Street mains must have occurred; and that contamination, although on a comparatively minute scale, during continuance of supply, is probable.

NOTE.

The results of this inquiry recalled to mind a sudden and very remarkable outbreak of diarrhœa which occurred in the Mountain Ash district in March 1885. From the clinical histories the affection seems to have exactly resembled that occasionally met with after the ingestion of animal matter (pork, veal, etc.) in a state of peculiar incipient decomposition. Caegarrow, on the opposite bank of the river to Mountain Ash village, was chiefly affected. Some 800 to 900 cases occurred within two or three days, and two of them terminated in death. The Medical Officer of Health at first suspected the water, but afterwards, the water being found pure on analysis, inclined to the view that emanations from manure, with which some neighbouring fields had been spread,

were the cause. Dr. Davis, who investigated the circumstances for the Local Government Board, regarded the early and more numerous cases at Caegarrow as due to "a specific infection either water or air-borne" (food as a medium was excluded), and subsequent cases as resulting from "a limited infective capacity possessed by the disease".

The circumstances of water-supply and service on the Caegarrow side of the river are very similar to those of Mountain Ash opposite; and, indeed, as I have explained, the two systems are connected. There is reason to believe also that similar defects are to be found there. Not long ago a leak of apparently some standing, and communicating directly with one of the old rubble culverts, was discovered in the trunk main of the locality. The possibility of passing contamination could not of course be negatived by chemical analysis.

AGE, SEX, AND SEASON IN RELATION TO SCARLET-FEVER.

By ARTHUR WHITELEGGE, M.D., B.Sc., Medical Officer of Health, Nottingham.

(*Read: May 9th*, 1888.)

THERE are many points in the natural history of scarlet-fever upon which the text-books are almost silent, but which are not without practical importance. The influence of age upon liability to the disease, for instance, has not, so far as I know, been worked out thoroughly; and indeed it could scarcely be measured with any approach to accuracy by hospital statistics, dealing as they necessarily do with a more or less picked population. The unrecorded cases, treated at home, must materially affect the result.

In certain towns, including Nottingham, the system of compulsory notification now affords facilities for studying scarlet-fever as it affects a whole community, and I have long thought that an attempt to clear up some of the points of obscurity by this means might be of interest to the Society.

It has often occurred to me that the very certainty of the exact origin of many cases of scarlet-fever, by infection traceable to other persons attacked, has impeded the study of its etiology. Comparatively little attention has been given to the marked influence of season, locality, age, and sex in modifying the facilities for acquiring infection.

Scarlet-fever and diphtheria present many points of close resemblance, and among them ready transmissibility from person to person. Still the majority of persons apparently exposed to risk of infection of either disease escape scot-free, while the majority of attacks cannot be traced with any degree of certainty to a definite source. Attacks of diphtheria being comparatively rare, the occasional difficulty of finding even a plausible theory of connection with a previous case has led to the well-founded suspicion that direct, or even indirect relation with previous human diphtheria is far from being the whole truth in the etiology of the disease. Besides milk-diphtheria and aerial convection of germs, we have now to bear in mind the possibility of allied diseases in lower

animals, defects of drainage or scavenging, dampness of houses, and telluric conditions, even if we exclude origin *de novo* as being only a confession of ignorance.

Scarlet-fever, on the contrary, is practically always present in every large community, and it is still customary with medical practitioners and the public to limit their etiological research to seeking for some direct or indirect contact with a previous case; and, knowing that previous cases always abound, to content themselves, if need be, with any such clue, however remote or unpromising.

A scarlet-fever patient gives off infective matter by the breath and desquamation; this infective matter may lodge in crevices, or be carried in clothing or in food (especially milk), and retain for months or years its power of producing scarlet-fever when grafted upon a susceptible person. Probably most observers will admit, also, that it may be carried in a current of air, say across the sick-room, without losing any of its virulence; but a wider diffusion by this means is not generally recognised. As a matter of common experience, the majority of persons in whom an attack would be readily accounted for do not acquire the disease, with the exception, perhaps, of those in intimate contact with the patient during the infectious period.* Still there can be no doubt that the contagium must become very widely diffused, if only by convection by clothing, etc., making all due allowance for destruction by washing and by the ordinary perfunctory attempts at disinfection. It is scarcely possible to exclude such sources of infection from consideration in any given case.

The Hendon and Wimbledon outbreaks investigated by Mr. Power and Dr. Klein give strong proof of the existence of at least one possible alternative mode of infection, viz., infection carried by the milk of cows suffering from a specific disease. Here the relation with a previous case becomes very remote, unless we regard the cow in that light. But this discovery, important as it is, does not materially help us to account for the numbers of cases in which we cannot clearly trace any exposure to infection. Out of some fifteen hundred cases of scarlet-fever, I have found no special incidence of the disease upon any particular dairy, such as I should expect

* A few months ago my suspicions were aroused by the notification by different practitioners of ten cases of scarlet-fever which, upon inquiry, I found to have attended the same class at a school. I examined the seventy children who remained in the class, and found one whose hands were peeling. He had had an illness which was not recognised as scarlet-fever, and had returned to school four days before the first of the ten children was taken ill. I could trace no special contact or proximity with the ten sufferers beyond that with the rest of the scholars.

to characterise outbreaks of this nature. There is some evidence that dogs and monkeys may be attacked by scarlet-fever, and Thomas gives a list of other animals suspected of susceptibility.

The existence of one exception to the general rule suggests the possibility of others, even apart from the analogy with diphtheria, and it is at all events clear that infection is directed and controlled, not only by the accident of contact, but by other conditions which have not perhaps received sufficient attention.

The Registrar-General, in his Annual Report for 1886, issued in January last, has anticipated some of the conclusions which I have drawn from the statistics of Nottingham and other notification towns. It is reassuring to find that, starting from entirely different data—viz., the official record of 5,000 cases in Christiania—he obtains results which harmonise with mine, so far as the two are comparable.

The points which I have selected for present consideration are—

1. *Age-incidence*, that is, the proportion of attack at each age-period, or the liability to attacks at any given age.
2. *Age-mortality*, or the proportion of scarlet-fever deaths at each age-period; in other words, the liability of the population at any age to death from scarlet-fever.
3. *Case-mortality*, or the proportion of deaths to attacks at different ages; that is, the severity, as measured by the danger to life, of attacks at any given age.
4. *The Influence of Sex* upon the number and severity of attacks occurring at each age.
5. *The Influence of Season* upon scarlet-fever; using the word in a broad sense to include monthly, weekly, or even daily differences.

Age-Incidence.

Thomas, in Ziemssen's *Cyclopædia*, says that the predisposition to scarlet-fever is very limited in the youngest children, increases during the second six months, is strongest from the second to the fifth or seventh year, and rapidly diminishes after the tenth year.

This diminution of susceptibility with age is mentioned by almost all writers, but is, I think, scarcely recognised by the majority of the medical profession, by whom the decrease in

the number of attacks is attributed to the increasing protection of the population by previous attacks.

The Registrar-General (*Annual Report for* 1886) has dealt with a part of this question, on the basis of 5,000 cases in Christiania, supplemented by 18,000 cases recorded in London hospital reports. He gives annual details for the first five years of life, corresponding pretty closely with those which I have worked out in Table A, and extended to the fifteenth year. His conclusions are summarised as follows:—

1. The mortality from this disease is at its maximum in the third year of life, and after this diminishes with age, at first slowly, and afterwards rapidly.

2. This diminution is due to three principal causes:—*a*. The increased proportion in the population at each successive age-period protected by a previous attack. *β*. The diminution of liability to infection in successive age-periods of those who are as yet unprotected. *γ*. The diminishing risk in successive age-periods of an attack, should it occur, proving fatal.

3. The liability of the unprotected to infection is small in the first year of life, increases to a maximum in the fifth year, or soon after, and then becomes rapidly smaller and smaller with the advance of years. (I have found the maximum to occur in the fourth or fifth year.)

4. The chance that an attack will terminate fatally is highest in infancy, and diminishes rapidly with years to the end of the twenty-fifth year; after which an attack is again somewhat more dangerous. (My figures seem to indicate a rather higher lethality in the second year than in the first, but otherwise are in accord.)

5. The female sex throughout life, the first year possibly excepted, is more liable to scarlet-fever than is the male sex. (I find the same curious exception in the first year.)

6. But the attacks in males, though fewer, are more likely to terminate fatally. (The Registrar-General's figures show an exception in the first year to this rule also.)

The five-yearly age-periods given by the Registrar-General are short enough for all practical purposes at ages above fifteen years; but it is important to carry on the yearly analysis from the fifth year, where the Registrar-General leaves it, to the fifteenth. By this means we shall determine the exact age of greatest liability to scarlet-fever, a point which the Registrar-General leaves in some doubt.

To the 1,340 cases of scarlet-fever which came under my notice in Nottingham in 1885, 1886, and 1887, I have been able to add 4,948 others, sufficient particulars of which are

given in the Reports of the Medical Officers of Health for Salford and Leicester. The total is thus raised to 6,288, and represents practically the whole of the cases occurring in three large English towns, having a population of considerably over half-a-million. The number of attacks at each year of age is given as a percentage of the whole number; and as the Leicester figures are not given in detail for each year of age beyond the tenth, the later percentages are calculated upon the Nottingham and Salford data only. (Table A.)

This curve rises sharply from infancy to the fourth year of age, after which it declines gradually and without interruption. The fifth year is little below the fourth, and exceeded it not only at Christiania, but at Salford, and at Nottingham in 1885. Beyond this, there seems to be no material discrepancy between the towns in question. We may assume that the true summit of the curve occurs somewhere about the end of the fourth year, so far as the mere number of recorded attacks is concerned.

I have found it convenient to use percentages of total cases rather than percentages of persons living at each age. We are now concerned with the *relative* frequency of attack at different ages; the number of attacks per hundred or thousand persons living at each age will vary with every year, in every town. To show that the correction for the age-distribution of the population does not materially alter the curve, I have applied the test to Table A, with the result shown in Table A A. The only effect of the correction is to make the curve more abrupt. The maximum is still in the fourth year.

I shall presently have to refer to two other corrections—for the accumulation of protected persons among the population, and for the "secondary" cases. The result of these three corrections is to transfer the point of maximum susceptibility from the fourth to the fifth year.

Deferring for the moment the consideration of several points suggested by this curve, I will pass to the next section, viz.:

Curve of Age-Mortality.

The Registrar-General's Annual Reports, together with the decennial Supplements, give us abundant material for this part of the question, which, however, I will dismiss as briefly as possible. A curve, which is practically constant for all years and all localities (providing only that the number of deaths be sufficiently large), is obtained by taking as ordinates the percentage of the total scarlet-fever deaths which happen

TABLE A.

Known Cases of Scarlet-Fever.

Years of Age.	0+.	1+.	2+.	3+.	4+.	5+.	6+.	7+.	8+.	9+.	10+.	11+.	12+.	13+.	14+.	0-5.	5-10.	10-15.	15-20.	20-30.	30-40.	+.	All Ages.
Salford, 1883-4-5 ...	59	200	232	318	331	301	243	188	160	126	89	69	52	40	35	1140	1018	285	57	56	26	9	2591
Leicester, 1885-6 ...	59	132	291	374	307	305	227	235	122	99	...	...	...	...	...	1163	988	...	...	...	...	...	2357
Nottingham, 1885-6-7	19	77	123	171	168	148	116	103	79	53	42	46	31	25	21	558	499	165	43	44	22	9	1340
Total	137	409	646	863	806	754	586	526	361	278	131*	115*	83*	65*	56*	2861	2505	450*	100*	100*	48*	18*	6288
Per cent. of cases at all ages.	2.2	6.5	10.3	13.7	12.8	12.0	9.3	8.3	5.7	4.4	3.4*	3.0*	2.1*	1.7*	1.5*	45.5	39.8	11.5*	2.6*	2.6*	1.2*	0.5*	

* Salford and Nottingham only.

TABLE A A.

	0+.	1+.	2+.	3+.	4+.	0-5.	5-10.	10-15.	15-20.	20-30.	30-40.	40+.
A. Percentage of cases at each age. (Table A.)	2.2	6.5	10.3	13.7	12.8	45.5	39.8	11.5	2.6	2.6	1.2	0.5
B. Percentage of population at each age. (Census, 1881.)	2.9	2.6	2.7	2.7	2.6	13.6	12.1	10.8	9.8	16.8	12.6	24.2
C. Comparative age-incidence corrected for age-distribution of population.	7.6x	25.0x	38.1x	50.7x	49.2x	33.5x	32.9x	10.6x	2.7x	1.5x	1.0x	0.2x

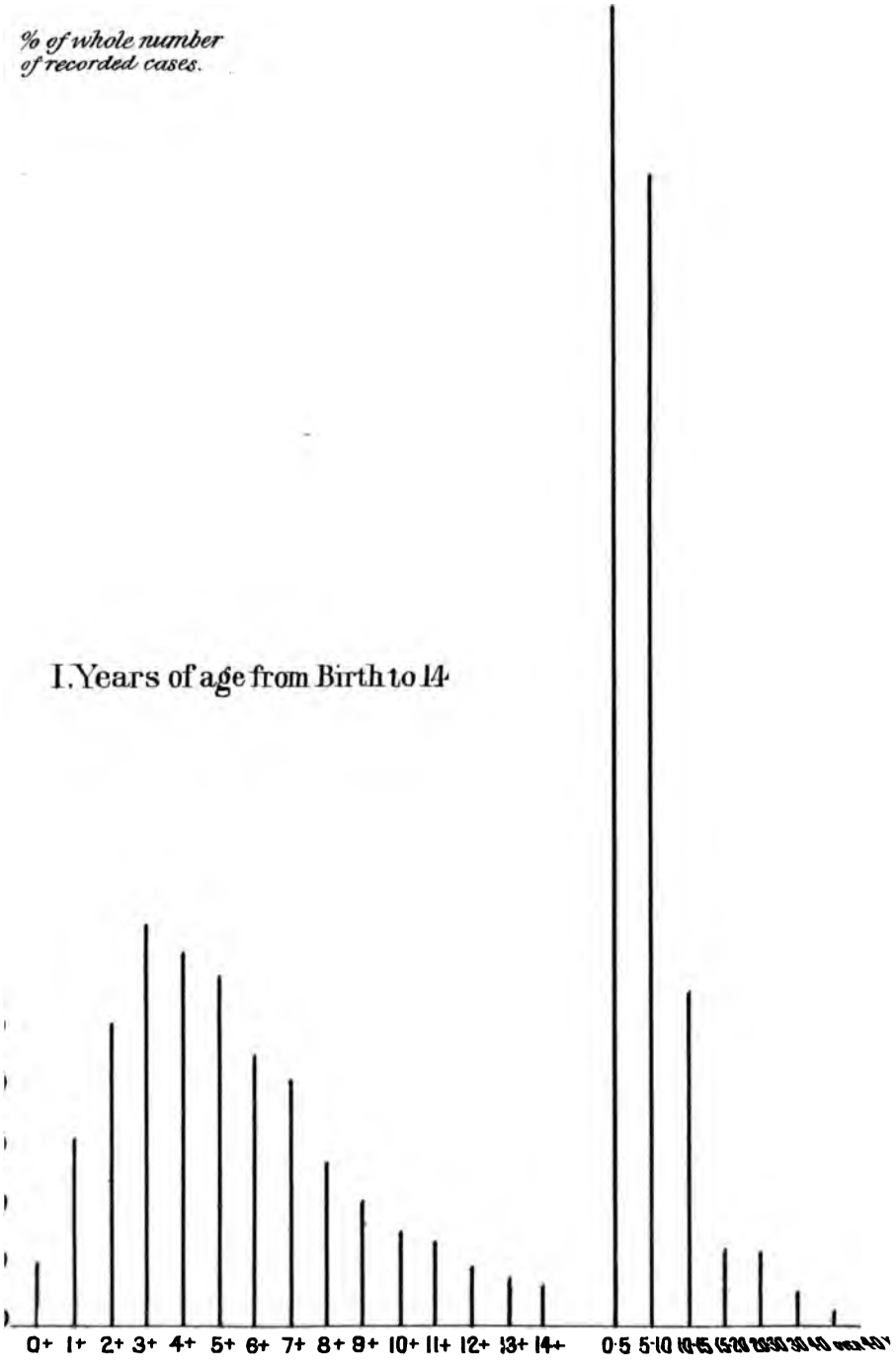
entage of Cases of
RLET FEVER occurring
tain ages & groups of ages.

To accompany Table A.

II. Groups of ages.

% of whole number of recorded cases.

I. Years of age from Birth to 14



at each age. It is substantially unaffected by correction for age-distribution, and rises rapidly from infancy to the third year of age, after which it declines throughout the rest of life, but more gradually than it rose.

This curve is shown in Table B, based upon the 382,000 deaths from scarlet-fever in England and Wales during the twenty years 1861-80. For the sake of uniformity, quinquennial age-periods are retained throughout, but the figures for ages above twenty-five years are merely the halves of the decennial totals. Here again I have preferred to take the percentages of total scarlet-fever deaths at each age, instead of the more strictly scientific mode of estimating the deaths in proportion to population at each age. Comparison with Table C shows that the former method is sufficiently exact for the present purpose, the resulting curves being closely similar at all points.

The Registrar-General only gives the figures for single years of age under five, but this instalment is sufficient to enable us to localise accurately the actual maximum, viz., at 2+.

Now whether we take comparatively small populations and obtain sufficiently large numbers by using decennial statistics, or take larger districts and only annual figures, we get practically this same curve. The only noteworthy exceptions to the rule which I have met with are, first, that in certain districts in Durham and Lancashire there has, from time to time, been an apparent excess at 3+ over 2+, which, however, was not constant even in those districts; secondly, that, in districts with low scarlet-fever mortality, the proportion of deaths at ages under five years was rather lower than in districts with high scarlet-fever mortality, and conversely with the higher ages. (Table D.)

The counties with low scarlet-fever mortality contain few large urban centres, and perhaps the difference between the conditions of urban and rural life may have some influence in the matter. The accumulation of protected survivors among the population at higher ages would produce some little effect in reducing the number of attacks, and, therefore, the mortality. This effect, slight as it may be, would, no doubt, be more felt in districts in which scarlet-fever was often prevalent, and which had therefore a high scarlet-fever death-rate.

These variations have no very wide range, however, and the curve shown in Table D may be taken as characteristic of scarlet-fever, irrespective of locality, time, or degree of prevalence. There must necessarily be corresponding uni-

TABLE B.

Scarlet-Fever Age-Mortality. England and Wales. (Deaths at each age-period per 100 scarlet-fever deaths at all ages.)

	0+.	1+.	2+.	3+.	4+.	0-5.	5-10.	10-15.	15-20.	20-25.	25-30.	30-35.	35-40.	40.45.	45-50.	50-55.	55-60.	60-65.	Deaths.
1861-70 ... M. ...	3.4	7.4	8.3	7.7	6.2	33.0	13.5	2.5	0.73	0.35	0.17	0.17	0.07	0.07	0.03	0.03	0.01	0.01	105368
F. ...	2.8	6.8	8.1	7.5	6.0	31.2	13.0	2.9	0.79	0.51	0.29	0.29	0.10	0.10	0.03	0.03	0.015	0.015	102499
	6.2	14.2	16.4	15.2	12.2	64.2	26.5	5.4	1.5	0.86	0.46	0.46	0.17	0.17	0.06	0.06	0.025	0.025	207867
1871-80 ... M. ...	3.2	7.5	8.8	8.1	6.4	34.0	13.0	2.4	0.72	0.32	0.17	0.17	0.08	0.08	0.03	0.03	0.01	0.01	88874
F. ...	2.6	7.0	8.4	7.8	6.2	32.1	12.4	2.5	0.69	0.45	0.30	0.30	0.11	0.11	0.023	0.023	0,01	0.01	85358
	5.8	14.5	17.2	15.9	12.6	66.1	25.4	4.9	1.4	0.77	0.47	0.47	0.19	0.19	0.05	0.05	0.02	0.02	174232

TABLE C.

*Scarlet-Fever Deaths, per million living at each Age.**

		All Ages.	0-5.	5-10.	10-15.	15-20.	20-25.	25-35.	35-45.	45-55.	55 65.	65-75.	75+.
1851-60	M. ...	909	4311	1984	461	146	67	39	30	20	15	10	7
	F. ...	845	4071	1999	528	154	79	48	31	19	14	11	7
	Persons	876	4191	1992	494	150	73	44	31	19	15	10	7
1861-70	M. ...	1011	4745	2220	468	149	80	47	23	15	8	5	8
	F. ...	934	4503	2144	534	157	105	73	33	12	10	6	5
	Persons	972	4624	2182	501	153	93	61	28	14	9	6	6
1871-80	M. ...	750	3591	1554	318	107	55	35	21	10	5	3	1
	F. ...	683	3387	1475	330	101	70	56	28	7	5	3	2
	Persons	716	3489	1515	325	104	63	46	24	9	5	3	2

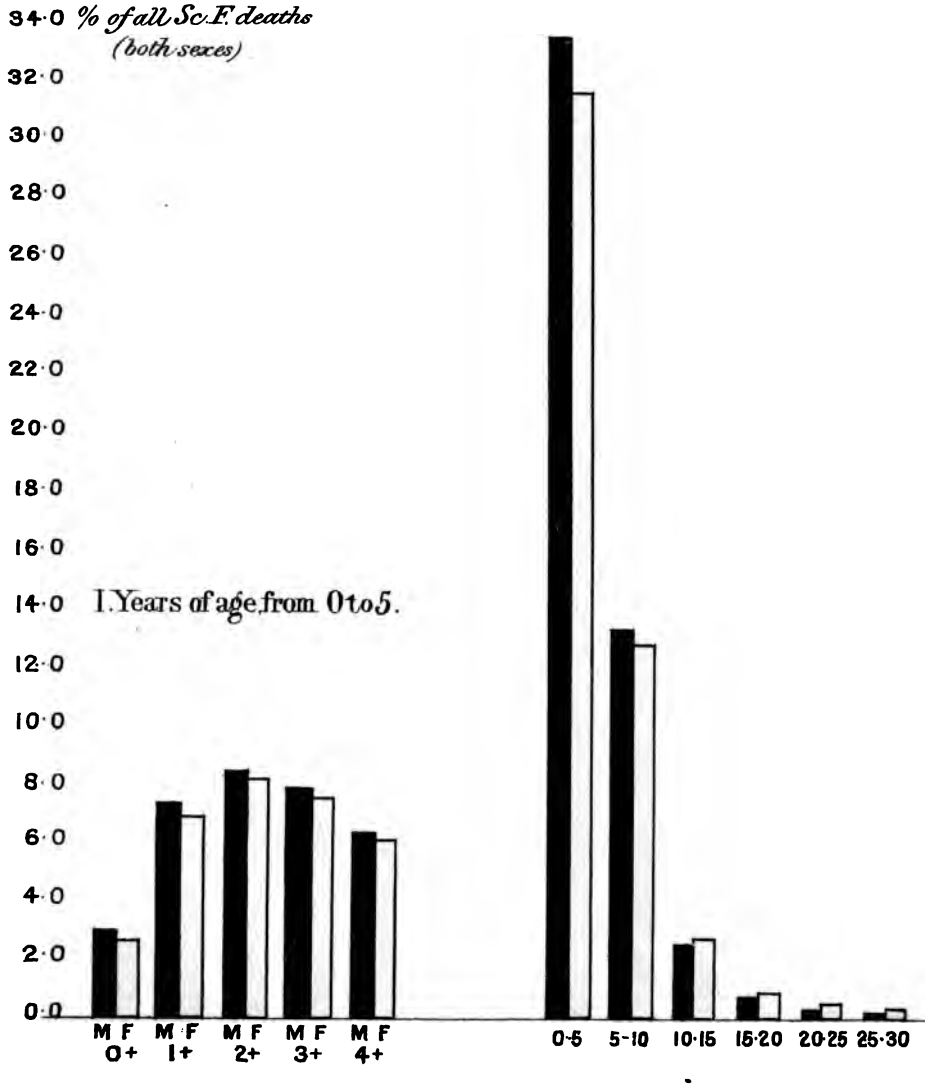
* Ogle *Supplement to the Forty-Fifth Annual Report of the Registrar-General*, p. cxii.

SCARLET FEVER MORTALITY.

ENGLAND AND WALES, 1861-80.

To accompany Table B.

Per centage of total Scarlet Fever deaths (all ages and both sexes) occuring in each sex at certain ages and groups of ages.



formity in the proportion of cases which occur at each age, and their respective fatality. I have already pointed out that the number of attacks of scarlet-fever in a population reaches its maximum at the fourth or fifth year of age, and shall presently have to show that the ratio of deaths to attacks is highest in the first two years, diminishing thereafter year by year. Up to the fourth year, therefore, the number of attacks increases year by year, while the severity of these attacks as steadily decreases, so that the

TABLE D.

Showing Influence of Total Scarlet-Fever Death-Rate upon Relative Age-Mortality from Scarlet-Fever in England and Wales, 1871-80, *and Percentages, at certain Ages, of Total Scarlet-Fever Deaths in groups of Registration Counties.*

	0+.	1+.	2+.	3+.	4+.	0-5.	5-10.	10-15.	Over 15.	Total Deaths from Scarlet-Fever.
14 counties* with scarlet-fever death-rate less than 0.40 per 1,000 living.	5.0	12.4	15.2	14.4	11.8	58.8	28.0	7.1	6.1	14,922
21 counties† with scarlet-fever death-rate from 0.40 to 0.80.	5.8	13.7	16.3	15.1	12.5	63.4	26.0	6.0	4.6	36,229
9 counties‡ with scarlet-fever death-rate of 0.80 and upwards.	6.0	15.2	17.7	16.2	12.6	67.9	24.9	4.3	2.9	101,834
England and Wales: Scarlet-fever death-rate, 0.72.	5.8	14.5	17.2	15.9	12.6	66.1	25.4	4.9	3.6	174,232
London: Scarlet-fever death-rate, 0.60.	5.7	14.0	17.3	16.2	13.2	66.5	25.4	4.0	4.1	21,247

position of the apex of the age-mortality curve at the third year is readily explained; after the fifth year both the number of cases and their lethality diminish, and the age-mortality curve consequently declines rapidly.

The age-mortality curve is therefore immediately influenced, and indeed produced, by the other two dissimilar curves; and

* Sussex, Kent, Hants, Bucks, Herts, Berks, Cambs, Suffolk, Devon, Hereford, Dorset, Oxon, Surrey, Middlesex.

† Northants, Hunts, Beds, Essex, Norfolk, Wilts, Cornwall, Somerset, Gloucester, Salop, Worcester, Leicester, Rutland, Lincoln, Notts, Derby, E. Riding, N. Riding, Westmoreland, Monmouth, N. Wales.

‡ Stafford, Warwick, Cheshire, Lancashire, W. Riding, Durham, Northumberland, Cumberland, S. Wales.

since it is uniform in all times and places, they, too, must each be practically unchangeable. There must always be more attacks in the fourth or fifth year than in any other, and the lethality must always be highest in infancy, whether we are dealing with severe epidemics, mild epidemics, or a mere accumulation of sporadic cases. Not only this, but the ordinates for each age in both curves must be liable only to very slight relative variation; otherwise it would be difficult to account for the steadiness of the resultant curve of age-mortality.

It seems reasonable to conclude that the curves of age-incidence and case-mortality which are found to be true for Nottingham, Salford, Leicester, and Christiania, hold good likewise for other towns and districts, since their resultant curve is the same in all.

Case-Mortality.

The proportion of fatal cases to attacks of scarlet-fever varies greatly. Ten per cent. seems to be generally accepted as a fair average, but it may be as low as three or as high as thirty per cent. The Salford returns indicate a total case-mortality of about eleven per cent., as compared with six per cent. in Nottingham and Leicester; recently in Nottingham we have had several hundred cases with a mortality of less than four per cent. The available returns from these three towns are, in themselves, too few for any satisfactory detailed analysis of the case-mortality according to age. Fortunately we have large hospital statistics to fall back upon, and the fact of their representing a picked population does not interfere very materially with their value in this respect, although it makes them unreliable as regards age-incidence. So far as they go, the notification-figures confirm the hospital statistics upon this point in all respects, so that the results may be accepted as trustworthy. Handford,* reasoning from hospital statistics, finds that the case-mortality is highest in the first five years, and diminishes with age, but rises again slightly after twenty-five. The Registrar-General draws the same conclusion from hospital returns, and finds that the case-mortality is highest in infancy, diminishing not only quinquennially but annually, up to the age of twenty-five. These hospital returns deal with nearly 18,000 cases.

The Nottingham, Salford, and Leicester figures show the same continuous fall from infancy to the twenty-fifth year, and subsequent rise with slight irregularities fairly attributable to the smallness of the data. In all three towns, how-

* See Table G.

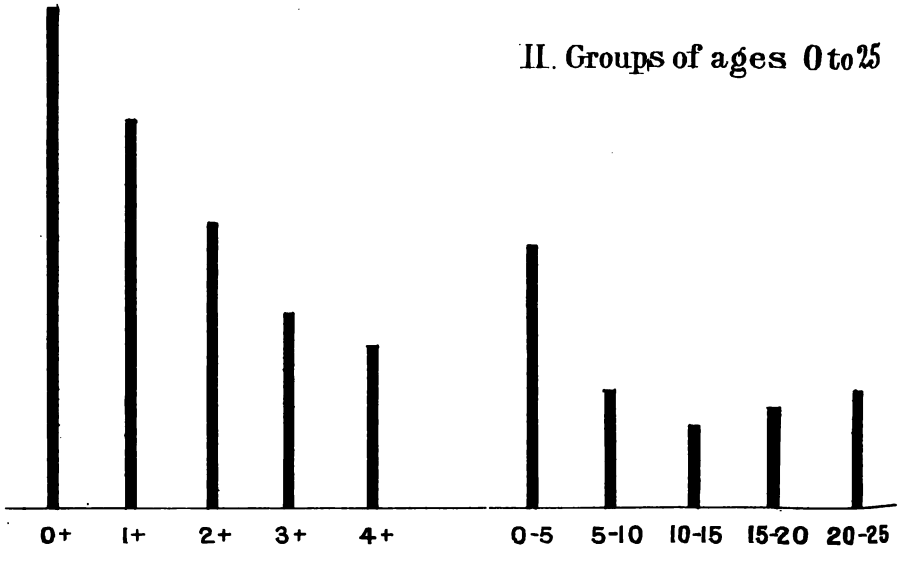
SCARLET FEVER

Relative Case-Mortality at certain ages & groups of ages.

To accompany Table E.

I. Years of age, 0 to 5.

II. Groups of ages 0 to 25



ever, the case-mortality was rather lower in the first than in the second year, this being the only point of divergence from the Registrar-General's conclusions.

Although the cases are numerous enough to give a tolerably reliable curve, the deaths are comparatively few, and it is preferable to utilise the far larger and more general mortality figures given in Table B. The case-mortality varies directly as the age-mortality (Table B), and inversely as the age-incidence (Table A), so that, taking a suitable constant x, the case-mortality at each age will be

$$x \frac{\text{age-mortality}}{\text{age-incidence}}.$$

Thus, the case-mortality at ages from 0 to 25 years is as follows:—

TABLE E.

	0+.	1+.	2+.	3+.	4+.	0-5.	5-10.	10-15.	15-20.	20-25.
$x \frac{\text{age-mortality}}{\text{age-incidence}} =$	$\frac{6.2}{2.2}x$	$\frac{14.2}{6.5}x$	$\frac{16.4}{10.3}x$	$\frac{15.2}{13.7}x$	$\frac{12.2}{12.8}x$	$\frac{64.2}{45.5}x$	$\frac{26.5}{39.8}x$	$\frac{5.4}{11.5}x$	$\frac{1.5}{2.6}x$	$\frac{0.86}{1.3}x$
Case-mortality =	$2.82x$	$2.18x$	$1.60x$	$1.10x$	$0.95x$	$1.37x$	$0.67x$	$0.47x$	$0.58x$	$0.66x$

That is, the case-mortality is highest in the first year of life and decreases year by year to the fifteenth or thereabouts, after which a slight increase occurs.

At adult ages, however, the notification-figures quoted in Table A are too few to constitute a safe basis for calculation. Probably the increase (real or otherwise) in the case-mortality belongs rather to the 25-30 age-period than to the 15-20.

It is possible that the somewhat increased danger to life in case of attack after twenty-five is only apparent. Among adults scarlet-fever is frequently unaccompanied by rash, and a slight sore-throat may be the only prominent symptom. If the unrecorded and often undiagnosed scarlatinal sore-throats could be added to the list, the case-mortality might be much lowered. We hear of all the deaths, but many of the attacks are so slight as to escape record; at all events, the case-mortality among adults of any age is far less than it is in childhood. The most important general conclusion to be drawn from the evidence is that the proportionate risk to life in case of attack at different ages remains pretty constant. Whether the prevailing case-mortality be high or low, the chance of recovery from an attack is least in infancy, and increases every year up to the twenty-fifth, if not further.

TABLE F.

Age-Mortality.

AGE.	NOTTINGHAM, 1885, 1886, 1887.			SALFORD, 1883, 1884, AND 1885.			LEICESTER, 1885 AND 1886.			TOTAL.		
	Cases.	Deaths.	Case-mortality per cent.	Cases.	Deaths.	Case-mortality per cent.	Cases.	Deaths.	Case-mortality per cent.	Cases.	Deaths.	Case-mortality per cent.
Under 1 year	19	4	21	59	15	25½	59	11	19	137	30	21.9
1+	77	17	22	200	52	26	132	36	27	409	105	25.6
2+	123	9	7⅓	232	39	17	291	34	12	646	82	12.7
3+	171	16	9⅓	318	35	11	374	21	5½	863	72	8.3
4+	168	3	2	331	50	13⅓	307	18	6	806	71	8.8
5	148	8	5½	301	35	11⅔	305	12	4	752	55	7.0
6	116	0	0	243	18	7⅓	227	7	3	586	25	4.3
7	103	8	8	188	10	5⅓	235	6	2½	526	24	4.5
8	79	9	11	160	7	4½	122	5	4	361	21	5.8
9	53	0	0	126	7	5½	99	2	2	278	9	3.3
10	42	0	...	89	2	...	...	...	...	131*	2*	...
11	46	1	...	69	4	...	...	...	...	115*	5*	...
12	31	0	...	52	2	...	...	...	...	83*	2*	...
13	25	0	...	40	0	...	...	..	...	65*	0*	...
14+	21	1	...	35	1	...	...	...	...	56*	1*	...
0 to 5	558	49	8.8	1140	191	16.8	1163	120	10.3	2961	360	14.1
5 to 10	499	25	5.0	1018	77	7.6	988	32	3.2	2505	134	6.7
10 to 15	165	2	1.2	285	9	3.2	...	...	...	450*	11*	2.4
15 to 20	43	0	0.0	57	2	3.5	...	...	...	100*	2*	2.0
20 to 30	44	0	0.0	56	4	7.1	206	5	2.5	100*	4*	4.0
30 to 40	22	1	4.5	26	1	3.8	...	...	...	48*	2*	4.2
Over 40	9	0	0.0	9	0	0.0	...	...	...	18*	0*	0.0
All ages	1340	77	5.7	2591	282	10.9	2357	157	6.2	6288	516	8.2

* Nottingham and Salford only.

TABLE G.

Case-Mortality.

Ages.	Hospital Statistics (Handford, *Practitioner*, xxxviii, p. 313). Percentage of Total Cases.*	Number of Cases.	Deaths.	Case-Mortality.	Hospital Statistics (Reg.-Gen. *Ann. Rep. for* 1886) Case-Mortality. M.	F.	Nottingham and Salford Notification Returns. Case-Mortality.
0-5	21.6	3864	929	24.0	24.1	21.7	14.1
5-10	34.0	6084	664	10.9	10.7	9.7	6.7
10-15	17.2	3084	168	5.4	5.6	5.3	2.4
15-20	11.5	2054	84	4.0	4.0	3.4	2.0
20-25	8.3	1498	57	3.8	3.9	3.2	4.0
25-35	5.7	1020	58	5.6	7.5	4.3	4.2
35-45	1.1	199	15	7.5	8.5	6.5	
45-55	.2	39	3	7.7			
55+	...	2	1	[50.0]			
All ages	...	17,844	1979	11.1			

Several of the following tables introduce a distinction, which I think is of considerable importance, between a first case in a household and the secondary cases which may follow it. The latter, or the great majority of them, are doubtless due to direct infection from the first case, and their etiology is sufficiently simple. The first cases include all of doubtful origin, and are the more significant for present purposes.

TABLE H.

Primary and Secondary Cases, Nottingham, 1886-7.

Age ..	0+.	1+.	2+.	3+.	4+.	5+.	6+.	7+.	8+.	9+.	10+.	11+.	12+.	13+.	14+.
Primary ...	10	35	60	94	105	75	58	58	38	27	17	20	13	13	8
Secondary...	4	16	33	31	25	27	15	17	20	10	10	10	8	7	9
Secondary, per cent. of total	29	31	36	24	20	26	21	23	34	27	37	33	38	35	53
Ditto (Bloxam)	...	34	30	26	23	22	24	25	28	33	32	36	35	41	...

* (*Cf.* Table A.

TABLE H—*continued.*

Age	0-5.	5-10.	10-15	15-20.	20-25.	25-30.	30-35.	35-40.	40+.
Primary	304	256	71	28	11	4	3	4	2
Secondary	109	89	44	6	7	7	9	5	4
Secondary, per cent. of total.	26	26	38	18	39	64	75	56	67
Ditto (Bloxam) ...	...	28	28	34	32	56	66	67	...

The classification in Table H suggests that the difference between towns in regard to the position of the summit of the age-incidence curve may perhaps be partly due to the absence of discrimination between primary and secondary cases. Unfortunately, the only data available in respect to secondary cases are those of two years' records in Nottingham; but the subtraction of the secondary cases from the Nottingham totals leaves a curve with a distinct maximum at the fifth year, like Salford and (probably) Christiania, whereas the secondary cases obscure this considerably.

I cannot say what importance, if any, should be attached to the indication which the small number of secondary cases give of a maximum at the third year, two years earlier than that of the primary.

Bloxam's method enables us to obtain from these scanty materials a tolerably smooth curve, which goes to show that the proportion of secondary cases is about one-third of the total in the first two or three years; descends to one-quarter or one-fifth about the sixth year; after which it rises without much interruption, reaching one-third again about the twelfth year, one-half about thirtieth, and two-thirds about the thirty-fifth. The proportion of secondary cases among the whole number recorded is about 30 per cent.*

Influence of Sex.

Thomas concludes that "there is no evidence to prove that sex influences the predisposition to scarlet-fever . . . . young boys are more frequently affected because their number is greater." Judging from the English census returns of 1871 and 1881, however, the preponderance of males only occurs in the first year. Gee (Reynolds' *System of Medicine*) says that both sexes are equally liable to scarlet-fever, but women

* The Report of the Medical Officer of Health for Newcastle, which has just reached me, gives certain data respecting the number of cases of scarlet-fever in each household attacked. The proportion of secondary cases is only 21 per cent.

are more exposed after puberty. Squire (Quain's *Dict. of Medicine*) expresses similar views.

The Registrar-General points out that sex has a bearing upon the prognosis of an attack of scarlet-fever, since the case-mortality among males is greater than that among females at every age except perhaps infancy. He shows also that females are more frequently attacked than males at all ages (except infancy), and that in both sexes the mortality in relation to population is greatest in the third year.

Commencing with the age-mortality, we find that the net result of the greater severity and smaller number of male attacks is an excess of male deaths over female; but there are noteworthy exceptions to this rule.

From an examination of the returns for England and Wales, for sixteen years (Table I), I find that the female

TABLE I.

England and Wales, 1871 *to* 1886. *Scarlet-Fever Deaths at certain Ages per cent. of Scarlet-Fever Deaths at all Ages.*

		0–1.	1+.	2+.	3+.	4+.	0–5.	5–10.	10–15.
1871 ... M.	9407	3.6	7.5	8.4	7.3	5.8	32.7	13.3	2.8
F.	9160	2.9	7.3	8.0	7.2	5.8	31.2	12.3	3.1
	18,567	6.5	14.8	16.4	14.5	11.6	63.9	25.6	5.9
1872 ... M.	6052	3.6	8.0	8.8	7.7	5.7	33.9	11.9	2.8
F.	5870	3.0	7.3	8.3	7.6	5.8	32.0	12.1	2.6
	11,922	6.6	15.3	17.1	15.3	11.5	65.9	24.0	5.4
1873 ... M.	6660	3.7	8.1	9.2	8.0	5.7	34.7.	12.3	2.3
F.	6484	2.9	7.3	8.8	8.1	6.0	33.1	11.9	2.6
	13,144	6.6	15.4	18.0	16.1	11.7	67.8	24.2	4.9
1874 ... M.	12,651	3.1	7.7	9.0	8.4	6.2	34.4	12.2	2.5
F.	12,271	2.6	7.1	8.8	7.9	6.1	32.5	12.0	2.6
	24,922	5.7	14.8	17.8	16.3	12.3	66.9	24.3	5.1
1875 ... M.	10,502	3.1	7.4	8.6	8.4	6.7	34.3	12.7	2.4
F.	9967	2.7	7.2	8.0	7.4	6.4	32.1	11.8	2.6
	20,469	5.8	14.6	16.6	15.8	13.1	66.4	24.5	5.0
1876 ... M.	8616	3.0	7.3	8.4	8.2	6.7	33.6	13.2	2.4
F.	8277	2.7	6.9	8.5	7.0	6.0	31.8	12.7	2.3
	16,893	5.7	14.2	16.9	15.9	12.7	65.4	25.9	4.7

TABLE I—*continued.*

		0-1.	1+.	2+.	3+.	4+.	0-5.	5-10.	10-15.
1877 ... M.	7405	3.3	7.7	8.8	7.7	6.1	33.5	13.8	2.4
F.	7051	2.6	7.1	8.1	7.5	6.1	31.4	12.9	2.4
	14,456	5.9	14.8	16.9	15.2	12.2	64.9	26.7	4.8
1878 ... M.	9592	3.0	7.6	9.5	8.0	6.7	34.6	13.1	2.1
F.	9250	2.6	7.1	8.7	8.0	6.5	33.0	12.4	2.1
	18,842	5.6	14.7	18.2	16.0	13.2	67.6	25.5	4.2
1879 ... M.	9148	3.1	7.2	9.0	8.5	6.7	34.7	13.7	2.1
F.	8465	2.2	6.5	8.5	8.1	6.4	31.7	12.6	2.0
	17,613	5.3	13.7	17.5	16.6	13.1	66.4	26.3	4.1
1880 ... M.	8841	2.8	6.9	8.2	7.9	7.1	32.8	14.3	2.3
F.	8563	2.4	6.4	7.9	8.0	6.9	31.5	13.5	2.1
	17,404	5.2	13.3	16.1	15.9	14.0	64.3	27.8	4.4
1881 ... M.	7145	3.0	6.7	8.0	7.9	6.8	32.4	13.8	2.3
F.	7130	2.3	6.5	7.8	8.2	6.5	31.3	13.9	2.4
	14,275	5.3	13.2	15.8	16.1	13.3	64.7	27.7	4.7
1882 ... M.	6904	2.9	7.0	8.2	7.8	6.4	32.2	14.3	2.3
F.	6828	2.7	6.6	7.7	7.7	6.3	31.1	14.2	2.6
	13,732	5.6	13.6	15.9	15.5	12.7	63.3	28.5	4.9
1883 ... M.	6449	2.9	7.1	8.2	7.9	6.8	33.0	14.0	2.6
F.	6200	2.2	6.4	7.5	7.2	6.5	29.9	14.5	2.5
	12,649	5.1	13.5	15.7	15.1	13.3	62.9	28.5	5.2
1884 ... M.	5504	3.1	7.1	8.2	7.5	6.4	32.3	14.1	2.8
F.	5359	2.3	6.9	7.7	8.1	6.2	31.3	13.5	2.7
	10,863	5.4	14.0	15.9	15.6	12.6	63.6	27.6	5.5
1885 ... M.	3210	3.3	7.8	8.5	7.6	6.6	33.8	12.6	2.3
F.	3145	2.9	7.4	7.7	7.0	5.9	30.8	13.4	2.8
	6355	6.2	15.2	16.2	14.6	12.5	64.6	26.0	5.1
1886 ... M.	2999	3.7	8.5	9.3	7.1	6.0	34.6	12.3	1.9
F.	2987	2.9	7.8	8.5	7.8	6.6	33.4	12.4	2.1
	5986	6.6	16.3	17.8	14.9	12.6	68.0	24.7	4.0

deaths in the first two years of life were always below the male deaths; in the third year they once exceeded them; in the fourth year they once equalled and five times exceeded them; in the fifth year they twice equalled and three times

exceeded them. Looking now to the mortality returns for the ten years 1871-80 (Table B), we see that while in both sexes the deaths in the fourth year are fewer than in the third, the decline is greater among males than among females. These differences may be in part accidental, but seem to indicate some constant deviation between the sexes, either in regard to age-distribution of cases or case-mortality, about the fourth year, which, as we already know, is the turning point of the age-incidence curve. Here I may note, as an instance of the marked parallelism between scarlet-fever, diphtheria, and enteric fever, that the two latter have an age-mortality curve closely resembling that of scarlet-fever during the first five years of life, but with this difference, that the female mortality from enteric fever and diphtheria outstrips the male in the third, fourth, and fifth years.

TABLE B B.

Percentage of Total Deaths from Diphtheria and Enteric Fever (all ages and both sexes) occurring at each Year of Age from 0 to 5 Years.

		Diphtheria.					Enteric Fever				
		0+.	1+.	2+.	3+.	4+.	0+.	1+.	2+.	3+.	4+.
1861-70 ...	Male...	6.6	9.3	7.6	7.9	6.6	1.5	2.0	2.2	2.0	1.7
	Female	5.0	8.9	8.4	8.7	8.2	1.3	2.0	2.3	2.1	2.0
	Total	11.6	18.2	16.0	16.6	14.8	2.8	4.0	4.5	4.1	3.7
1871-80 ...	Male...	3.2	5.2	4.7	5.4	5.2	2.6	4.3	4.8	4.2	4.1
	Female	2.5	4.5	4.9	6.2	5.7	2.3	4.2	4.7	4.7	4.5
	Total	5.7	9.7	9.6	11.6	10.9	4.9	8.5	9.5	8.9	8.6

With these occasional exceptions, the mortality from scarlet-fever is greater among males than females during each of the first five years, and is always so in this period as a whole.

From five to ten years of age there is still a slightly higher mortality among males, but from ten to forty-five years the female death-rate exceeds the male, especially at those ages (twenty to thirty-five) at which the charge of children might be expected to bring with it special exposure to infection. After forty-five the proportions are about equal.

The following table is taken from the Registrar-General's Annual Report for 1886, and indicates that the case-mortality, in both sexes alike, diminishes from infancy to about the twenty-fifth year, and then increases somewhat. It is always

higher among males than females, except in infancy. The scanty data at my disposal are in harmony with these conclusions. I have already called attention to the possibility that the increased case-mortality at ages over twenty-five may be fallacious.

TABLE J.

Male and Female Case-Mortality per 1,000 *attacks* (*Reg.-Gen., loc. cit.*).

Age ...	0+.	1+.	2+.	3+.	4+.	0-5.	5-10.	10-15.	15-20.	20-25.	25-30.	35+.
Male ...	395	384	255	184	130	241	106	56	40	39	75	85
Female ...	442	346	226	174	112	217	97	53	34	32	43	65

Turning now to the inquiry whether there is any characteristic difference between the curves of age-incidence in the two sexes, it is important to observe the distinction between primary and secondary cases. The primary cases show a distinct though not very great excess among females for the first ten or eleven years of life, after which the numbers are too small to draw sound conclusions from; so far as they go they indicate some excess among males. Among secondary cases, however, the conditions are reversed. Up to the fifteenth year males and females are almost equally affected, the males being in slight excess; but beyond the fifteenth year the female secondary attacks greatly exceed the male, owing to exposure to infection in nursing children suffering from scarlet-fever, and, according to many authorities, increased receptivity during the puerperal state.*

Table K shows the same excess of male attacks over female in infancy which the Registrar-General notes in regard to the Christiania returns. With this exception, the female numbers exceed the male slightly up to the sixth year, and more markedly from the sixth to the twelfth year, after which the figures are small, but still indicate some excess among females.

Females therefore are more liable to take scarlet-fever than males are, but less liable to die from it.

It does not seem probable that there can be any difference in the degree of exposure to risk of infection between boys and girls in the first two or three years of life, whatever may be the case later on. In the absence of any other explanation, we may assume that the susceptibility varies for

* It is an old theory that menstruation predisposes to infection. The greater prevalence among females from ten years of age is not inconsistent with this view.

physiological reasons; and the inequality of case-mortality in the two sexes to some extent supports this view.

TABLE K.

Sex and Age of Primary and Secondary Cases, Nottingham:

Years of Age.	All cases, 1885-6-7.				Primary Cases, 1886-7.				Secondary Cases, 1886-7.			
	M.	F.	F. Per cent. of total.	F. Per cent. of total (Bloxam).	M.	F.	F. Per cent. of total.	F. Per cent. of total (Bloxam).	M.	F.	F. Per cent. of total.	F. Per cent. of total (Bloxam).
0 +	11	8	42	...	6	4	40	...	3	1	25	...
1 +	38	39	51	51	14	21	60	56	11	5	31	45
2 +	57	66	54	51	26	34	57	56	15	18	55	47
3 +	85	86	50	52	44	50	53	53	16	15	48	54
4 +	82	86	51	51	53	52	49	54	10	15	60	49
5 +	74	74	50	52	31	44	59	54	16	11	41	49
6 +	53	63	54	55	26	32	55	58	8	7	47	46
7 +	39	64	62	57	23	35	60	55	8	9	53	50
8 +	36	43	54	58	20	18	47	58	10	10	50	49
9 +	24	29	55	55	8	19	[70]	56	6	4	40	48
10 +	19	23	55	57	8	9	[53]	62	5	5	50	47
11 +	17	29	63	56	8	12	[60]	50	5	5	50	50
12 +	16	15	[48]	56	9	4	[31]	52	4	4	50	48
13 +	12	13	[52]	53	5	8	[61]	44	4	3	43	46
14 +	13	8	[38]	...	5	3	[58]	...	5	4	44	...
0–5	273	285	51	...	143	161	53	...	55	54	49	...
5–10	226	273	55	53	108	148	58	55	48	41	46	48
10–15	77	88	53	54	35	36	51	56	23	21	48	48
15–20	23	20	47	54	15	13	[47]	50	1	5	[83]	58
20–25	11	20	[65]	54	5	6	[56]	51	0	7	[100]	85
25–30	5	8	[61]	58	1	3	[73]	56	2	5	[71]	70
30–35	8	5	[39]	49	2	1	[33]	45	5	4	[44]	57
35–40	5	4	[44]	51	3	1	[25]	33	2	3	[60]	59
40 upwards	2	7	[78]	...	1	1	[50]	...	...	3	[100]	...

Why should children become more and more liable to take scarlet-fever every year of their life up to the fourth or fifth, and less and less liable every year afterwards? The second half of the question appears at first sight to present less difficulty, since with increasing age we have a smaller population, and an increasing number of persons protected by a previous attack; but a simple calculation based upon the figures already given will show that these facts afford no adequate explanation.

Let us trace the gradual acquisition of protection by a population of 10,000. For the sake of simplicity, let us assume a scarlet-fever death-rate of 1.0 per thousand per annum, with an average case-mortality of ten per cent. We

have then 100 attacks yearly among 10,000 people, and the age-incidence table, together with the census returns, enable us to allot the cases and persons living as shown in Table L.

Given the attacks and the numbers living at each age, we can ascertain (*c*) the proportion of protected persons at the end of each period, and from this again the number of attacks at each age per 1,000 *unprotected* persons.

TABLE L.

	All Ages.	0+.	1+.	2+.	3+.	4+.	0-5.	5-10.	10-15.	15-20.
a. Census population, 1871-81.	10,000	305	270	278	271	267	1391	1232	1107	993
b. Scarlet-fever cases (taking Sc. F. death-rate of 1.0 per 1000, with case-mortality of 10 per cent.).	100	2.2	6.5	10.3	13.7	12.8	227.5	199	57.5	13
c. Attacks at each age per 1000 living.	10.0	7	24	37	51	48	164	162	52	13
d. Protection at end of each age-period, per 1000 living.*	...	7	31	68	119	167	[164]	326	378	391
e. Attacks per 1000 *unprotected* persons living at each age.	...	7	24	38	54	54	[164]	193	78	21

Hence the survivors at the age of twenty years, by which time the prospect of future attack is slight, have become protected to the extent of less than two-fifths of their number. After correcting for age-distribution, and eliminating also the protected part of the population, we find that the only material effect upon the age-incidence curve has been to give a slight excess in the fifth year over the fourth in liability to attack.

Further confirmation of the fact that the majority of persons escape scarlet-fever throughout life is afforded by the result of inquiries which I have made respecting 2,585 persons living in houses in which cases of scarlet-fever occurred; 1,409 of these were over fifteen years of age, and only 414, or 30 per cent., were stated to have had scarlet-fever in earlier life.

Murchison expressed the opinion that a large number of persons must remain exempt, and attain middle life without being protected by a previous attack. Thomas states that observations of epidemics in isolated regions, where scarlet-fever seldom prevails, give the same result. Since neither

* No deduction is made for fatal attacks.

age-distribution of population, nor increased protection due to previous attack, is sufficient to account for the lessened prevalence at ages after five years, we are driven to the conclusion that for some unexplained reason the average liability of the individual diminishes from that point, quite apart from protection as ordinarily understood.

The difficulty is even greater in regard to ages under five years. With a diminishing population and an increasing proportion of protected persons, we have a rapid increase in the liability to scarlet-fever.

There is no very obvious coincidence with the recognised stages of development in childhood, as, for instance, the first and second dentition. The comparative immunity of infants suggests some connection between weaning and exposure to scarlet-fever, but I have not met with confirmatory evidence of this among the few cases on my list. *A priori*, one would have expected to find the excessive case-mortality among infants to have coincided with a high proportionate liability to attack, as is so clearly shown in statistics of small-pox in vaccinated and unvaccinated persons. It is not easy to conceive that a child begins life with a certain degree of physiological susceptibility to scarlet-fever, which increases rapidly for five years, and then declines during the rest of life. On the other hand, one is almost equally at a loss to imagine in what way, apart from increasing susceptibility, the facilities for access of *materies morbi* can be so much greater in the third year than in the second, and in the fourth year than in the third. There are considerable differences, as regards diet and contact with other persons, between infants and older children; but even if we accept these conditions as sufficient to account for the increase year by year up to the fourth, it is strange that the increase ceases and gives place to a decrease just at the age when contact with other children at school and elsewhere is becoming most free.

There is evidently a real and progressive diminution of susceptibility after the fifth year, and it may be that the true susceptibility is highest at birth, like the case-mortality, and that it is only masked in the earlier years by a rapid increase in the unknown facilities for infection.

The slight variation between different towns in the age of maximum liability may perhaps be regarded as suggestive rather of varying local conditions affecting the facilities for infection, rather than of varying physiological susceptibility.

The statistics of small-pox and vaccination show that the case-mortality varies as the susceptibility; that is, that unvaccinated persons, being more liable to take small-pox, also

incur greater danger to life in case of attack. This relation seems a natural one, and it is a little surprising to find so many exceptions to it in regard to scarlet-fever, according to the figures before us. It is true that from five to twenty-five years of age the case-mortality and susceptibility to attack seem to decline together; but there the parallelism ends. From birth up to the fifth year susceptibility increases, while case-mortality decreases, and *vice versâ* at ages over twenty-five years. The male sex, with a higher case-mortality, has a smaller number of attacks in proportion to population than the female sex; and, curiously enough, in infancy, at which age alone the attacks are more numerous among males than females, the relative case-mortality also is reversed, and the danger to life is greater among females.

It is clear that, with each year of age after the fifth, a condition of immunity from scarlet-fever is gradually acquired, apart from the protection of individuals by non-fatal attacks. The immunity thus acquired stands to scarlet-fever much in the same relation that the protection due to vaccination does to small-pox, but with the difference that the one increases while the other diminishes with lapse of time. Scarlet-fever without rash, which is not an uncommon form among adults, ought perhaps to be regarded as a modified attack, occurring in an individual partially protected by age (or, it may be, by a previous attack).

I have never met with detailed statements as to *ages* in reports of milk-epidemics of scarlet-fever. These involuntary experiments are sometimes made upon a large scale, and might throw considerable light upon the susceptibility at different ages.

In shielding a child against infection during the first few years of his life there is a double gain; every year of escape from scarlet-fever renders him less and less susceptible, until finally he becomes almost insusceptible; and, secondly, even if he should ultimately take the disease, every year that the attack is deferred reduces the danger to life which it brings. In other words, attacks of scarlet-fever become both less severe and less frequent with every year of age after the fifth. Up to the fifth year the liability is less, but the risk to life in case of attack is very great.

In the *British Medical Journal* of November 5th, 1887, Mr. R. W. Parker raises a question which to me seems to admit of only one answer. He says: "The question has frequently passed through my mind whether it is really a wise thing to try and escape an attack of measles or scarlet-fever, seeing how often escape merely means postponement—

postponement from childhood, perhaps, when the fever may be gone through with comparative ease and comfort, to adult life, with all the inconveniences, anxieties, and additional dangers which advancing age entails." The obvious reply is, that it *is* wise to avoid an attack of scarlet-fever whenever possible; during the first few years of life, because of the great risk to life at that age in case of attack; and at all ages after the fifth year, because every year of delay brings a double security. Even under the old *régime* of carelessness in all matters relating to infectious disease, two-thirds of the adults seem to have escaped; and the liability to take scarlet-fever, or to die of it, is small even among those adults who are not protected by a previous attack.

If the suggested exposure does not lead to infection, the risk has been incurred uselessly; if it does, we increase the risk to life by every year that we anticipate the attack—an attack which, in at least two out of three persons, would never have occurred at all, had ordinary precautions been observed.

Seasonal Curve.

The increased prevalence of scarlet-fever in autumn is noticed by almost every writer upon the subject.

Hirsch* states that out of 435 epidemics in various parts of the world, 22 per cent. occurred in spring, 24 per cent. in summer, 29 per cent. in autumn, and 25 per cent. in winter.

Longstaff, in a paper read before this Society in April 1880, has a diagram based upon the deaths from scarlet-fever in London during thirty years, showing that the mortality increases throughout the summer, reaching its maximum at the end of October, and falls continuously throughout the winter to a minimum in April. The Registrar-General, in his Annual Summary for 1880, gives a similar diagram, based upon the death-returns for forty years. Swedish statistics (Hirsch) also show an autumn maximum.

Matthews Duncan† has traced the weekly curve of deaths from scarlet-fever in London for twenty-eight years (1848 to 1875), the autumnal rise being evident in each of these twenty-eight years without exception.

Hirsch points out that while most epidemics coincide with cold and humidity, several have occurred in hot and dry seasons, and have even increased with heat, and commenced to decline only when colder weather set in.

Longstaff, in the paper already referred to, shows that

* *Geographical and Historical Pathology*, vol. i, New Sydenham Soc. 1883.

† *On the Alleged Occasional Epidemic Prevalence of Puerperal Pyœmia or Puerperal Fever and of Erysipelas.* 1876.

the epidemic maximum occurs for the most part in years of scanty rainfall. It would seem, however, from a chart furnished by Barnes,* that the maximum average mortality (at the end of October) coincides pretty closely with the maximum average rainfall in London.

Squire (Quain's *Dictionary of Medicine*) says that heat favours the diffusion of the disease, but lessens the severity of the attack. Epidemics often extend in dry seasons, and subside after wet ones.

Hirsch states that the autumnal increase in prevalence is attended by no special change in case-mortality, so that the cases, like the deaths, rise from a minimum in April to a maximum in October.

The seasonal curves given by Dr. Longstaff and the Registrar-General are calculated upon the returns of deaths only. I have constructed a similar seasonal curve based upon the 23,000 cases of scarlet-fever notified in nine large English towns† during the years 1885-6-7. The necessary details were obtained from the columns of the *Sanitary Record*; and from the same source I have obtained corresponding information regarding 13,000 cases notified in Aberdeen, Edinburgh, and Dundee. For the purpose of comparison with other seasonal curves, I have treated the figures according to Buchan and Mitchell's method, that is, each monthly total is stated as a percentage of the monthly average.

TABLE M.

Seasonal Curve of Cases and Deaths stated as percentage of monthly average.

	Jan.	Feb.	Mar.	Apr.	May.	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Cases.
	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	
Cases: 9 English Towns.†	81	71	67	59	62	73	83	104	140	176	156	128	22,882
Cases: 3 Scotch Towns.	70	89	80	45	50	60	82	118	162	185	155	101	12,923
Deaths: London, 30 yrs.‡	95	77	67	67	72	76	83	98	127	158	156	125	

* On the Causes, Internal and External, of Puerperal Fever, *Brit. Med. Journal*, Nov. 12th, 1887.

† Birkenhead, Bolton, Bradford, Leicester, Manchester, Nottingham, Oldham, Preston, Salford. The total population of these towns is about 1,700,000.

‡ Longstaff, *loc. cit.* The figures are only approximate, being taken by inspection from a diagram.

The curve of attacks differs little in outline from the mortality curve. The maximum of cases is in April, the minimum in October, both in England and Scotland, although the Scotch curve exhibits considerably greater deviations from the mean line. The irregularity in the February and March numbers for Scotland is due to an exceptional outbreak in Edinburgh in 1887.

If the seasonal curves of attacks and deaths were exactly parallel, it would indicate that the case-mortality is independent of season. At almost every point the deviation from the mean line is less in the mortality-curve than in the case-curve now before us; in other words, the mortality rises and falls proportionately less than the cases do. This, if it should prove to be a constant relation, would be important as showing that the autumnal prevalence of scarlet-fever is attended by a low case mortality, and conversely with the spring minimum.

I suspect, however, that another explanation is to be found in the short period for which the returns were obtainable. There was a very general increase in the number of attacks during the last few months of the record (*i.e.*, in the fourth quarter of 1887), which has not improbably led to an abnormal elevation of the seasonal curve of cases in autumn, and a corresponding depression in spring.

Why does the average unprotected individual become more and more liable to take scarlet-fever throughout the summer months, and most liable of all in October? And why should the facility for infection diminish month by month throughout the winter? There is evidently some varying influence at work, over and above the accident of contact with other victims of scarlet-fever—some influence which, as Hirsch puts it, is not any "particular kind of weather". If the maximum were at midsummer, or midwinter, or even if the hotter months exhibited a uniform excess, or deficiency, compared with the colder months, it would be much easier to devise plausible theories connecting the average climatic conditions with degrees of exposure to infection, by means of differences in habits of life in summer and winter. Enteric fever, a disease which, like diphtheria, has curious points of analogy with scarlet-fever, reaches its seasonal maximum at the same time, viz., the end of October, although its minimum occurs at midsummer; and diphtheria follows a very similar but rather later course.*

In enteric fever, however, infection by direct contact with an infected person is recognised to be exceptional, and a

* *Reg.-Gen. Ann. Rep.*, 1880; Longstaff, *loc. cit.*; Barnes, *loc. cit.*

totally different formula of explanation is assumed in cases of obscure origin. Hence, the seasonal variations of scarlet-fever are almost parallel with those of a disease which is etiologically very different from it; and they have presumably a further meaning than mere variation in the facilities for infection by contact in a way which admittedly would be inadequate to impart enteric fever. In contrast to the close similarity of the seasonal curves of scarlet-fever, enteric fever, and diphtheria, two of the mostt ypical infectious diseases—viz., small-pox and whooping-cough—have a characteristic curve which is exactly the reverse of the former, with a maximum in spring and a minimum in autumn; while measles has a peculiar curve of its own, very different from that of scarlet-fever.

I have already pointed out that scarlet-fever also resembles diphtheria and enteric fever in its curve of age-mortality in childhood, in both sexes. The age-mortality curve of small-pox is happily obliterated by infantile vaccination; that of measles presents an enormous peak in the second year, and that of whooping-cough in the first year, both being entirely devoid of any resemblance to the curve of scarlet-fever. These marked contrasts to the purely infectious diseases, and affinities with enteric and diphtheria, are very suggestive.

But although there is a distinct maximum both of epidemics and of sporadic cases in autumn, attacks of scarlet-fever occur at all seasons in every large community. I have a chart showing the daily number of attacks recorded in Nottingham for the last three years, and a close study of it brings out some interesting points. We have, not unfrequently, considerable periods of time with very few cases indeed; then a week or two with almost epidemic intensity, followed by a decline without further spread. The average mortality being only about three per cent., these minor waves would inevitably escape recognition if death-records, or even the experience of individual practitioners, were alone available. Every one of the cases on the chart has been investigated, and, with rare exceptions, there has been no such wholesale causation as infection at school, or by milk, or even grouping of cases in any one locality, to account for the temporary prevalence. Somewhat to my surprise, I have not hitherto found any clear indication of difference between holidays and school-time; nor any proof of greater prevalence after the great fairs which take place every few months. The most obvious suggestion is, that some concurrence of meteorological conditions occurs from time to time, which increases the facilities for infection.

Some days stand out conspicuously above the rest in the number of attacks credited to them, and it is probable that this is not altogether accidental. If we knew the incubation period of scarlet-fever as accurately as that of small-pox it would be a simple matter to determine the date of infection, and thus ascertain at once if there were any exceptional atmospheric conditions. As it is, the duration of latency is so uncertain, and the possible combinations of meteorological conditions so infinite, that any investigation is a matter of much labour and uncertainty.

I have been at considerable pains to investigate the relation between meteorological conditions and the daily, weekly, and monthly number of attacks of scarlet-fever, but the only point about which I feel any degree of confidence at present is that absence of rain is favourable to the spread of scarlet-fever. I mention this merely as an illustration of a line of inquiry which I hope to carry further.

The daily charts afford materials for one more calculation which might be expected to throw light upon the mode of origin of some obscure cases. Among all classes, one day in the week stands out from the rest, bringing with it considerable departure from the routine habits and surroundings of almost every individual. It seems reasonable to anticipate that the average Sunday would be marked by a notable deviation from the average week-day in respect to the facilities for infection, whatever those facilities may be. If the incubation period were constant, we should upon this hypothesis have a corresponding excess or deficiency in the number of onsets upon some one day in the course of the week, marking the appearance of the cases contracted on Sunday. The incubation period is, however, probably variable; but we might still hope to find some indication of the average latency. It is true that other exceptional conditions prevail on Saturday afternoons, and in Nottingham also on Thursday afternoons, but these are of minor importance.

The first attempt which I made at a classification of 418 primary cases in 1887 seemed to support this view in an unexpectedly complete manner, but the addition of the other cases recorded in 1886 and 1888 tends to show that the result was partly accidental. (Table N.)

The chief indication of difference between the weekly curves of primary and secondary cases is the reversal of the last two days of the week.

The apparent inference from the 1887 data was, of course, that the facilities for infection on Sunday were less than on other days; and that the usual period of latency up to the

TABLE N.

Comparative Frequency of Appearance of Rash on each Day of the Week.

	Sunday.	Monday.	Tuesday.	Wednesday.	Thursday.	Friday.	Saturday.	Total.
First Cases, 1887 ...	60	61	61	42	63	70	61	418
First Cases, 1886-7-8 ...	116	126	108	93	123	133	108	807
Secondary Cases, 1886-7-8	49	53	37	34	48	42	57	320
Total Cases	165	179	145	127	171	175	165	1127

appearance of the rash was three days. This interpretation, however, implies a shorter incubation than is usually assigned to scarlet-fever. The Wednesday minimum is still conspicuous in the complete records, but other inequalities become apparent. I think we are justified in regarding the Wednesday minimum as having some significance in Nottingham in the years 1886-7-8; but whether the determining conditions are local or general, temporary or permanent, remains to be seen. At all events the point is interesting, and may possibly prove to be important. I am not aware that it has been suggested before.

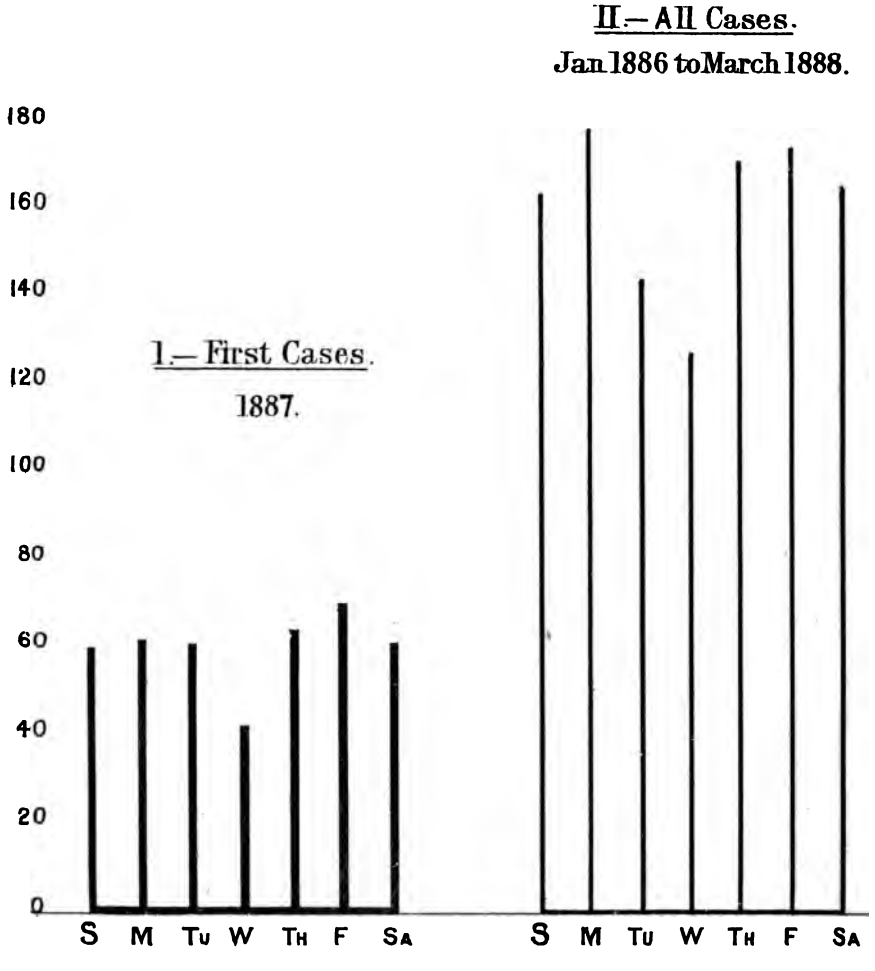
The rise and fall of epidemics of scarlet-fever may be regarded as due either to varying susceptibility of the population, or to varying facilities for infection. Similarly, the greater or less severity of type, as shown by case-mortality, may conceivably be determined either by the virulence of the contagium, or by the physiological resistance of the patient. But whatever the type of disease, and whatever its degree of prevalence, we can predict the relative numbers of its victims of each sex, and at every age, and also the relative mortality among all these groups. This relative constancy of result among all sub-divisions of the population seems to suggest that the variations in prevalence and severity depend rather upon the character of the contagium and the facilities for infection than upon any varying susceptibility of the whole population.

The occurrence of surgical scarlet-fever and puerperal scarlet-fever is too infrequent to affect the general prevalence of the disease. Dr. Alfred Carpenter has suggested that the decomposition of blood or other animal tissues may sometimes give rise to the disease. The possible existence of an analogous disease among lower animals has already been referred to.

WEEKLY CURVE.

Nottingham.

To accompany Table N.



There are two other possible factors in scarlet-fever infection which it may be worth while to examine briefly in reference to the seasonal variations in prevalence.

Several authorities, including Drs. Squire and Alfred Carpenter, hold that drainage defects play an important part in its etiology. If established, this would be one more link of association between scarlet-fever, enteric fever, and diphtheria; but I must confess that in my own limited experience I have found little confirmatory evidence.

The possibility of aerial convection offers a wide field for speculation. *Contagium vivum* in abundance is given off probably by every sufferer from scarlet-fever, both by the breath and skin. It is capable of retaining its vitality for years—under favourable conditions, at all events. There is no reason to doubt that it is portable, like the small-pox virus which we now know to be carried by the wind, or the omnipresent bacteria of putrefaction, or Lister's *bacterium lactis*, which is always forthcoming if milk is sufficiently exposed to air; and far more portable than the comparatively heavy and bulky particles constantly found in air. Whatever value we may attach to the "disinfection" sometimes attempted in the sick-room, we can scarcely suppose that it destroys the organisms which would otherwise be carried, by means of ordinary ventilation, out of the room into the open air.

This hypothesis lends itself pretty readily to the explanation of seasonal variation in scarlet-fever prevalence, but there are difficulties in the way of its acceptance. We have very little evidence of prevalence of scarlet-fever around hospitals, comparable to that of small-pox. Furthermore, the theory seems to explain too much, unless modifying influences are admitted—an objection which it shares with the ordinarily accepted view. If active germs of scarlet-fever can float about in the air for long periods, it becomes difficult to account for the temporary escape for years of persons whose susceptibility is afterwards proved by an actual attack. Dr. Ransom suggests that this objection may be met by the simile of a battle, where countless bullets are flying about, but few of the combatants are hit. It might also be argued that, for anything that is known to the contrary, a certain minimum charge of the contagium of scarlet-fever may be necessary for infection, so that dilution beyond a certain point would render it practically inert. Thomas* says: "It is an undoubted fact that a dilution of the contagious principle, by attentive ventilation of the sick-room,

* Ziemssen's *Cyclopædia*, art. "Scarlatina".

very much diminishes, or entirely removes, the opportunities for infection."

In conclusion, I may revert to what I said at the commencement of this paper. Infection from a previous case may be the explanation of all, as it is the undoubted cause of many, attacks of scarlet-fever. If so, we have still to discover, among other points, why it should happen more readily in the female sex, on certain days and weeks, in the autumn, and in certain years of early childhood, and less readily in spring, in the male sex, and in infancy, and at ages beyond the sixth or seventh year. Its associations in regard to age, sex, and season are rather with diseases in which infection from person to person is believed to play a minor part. It is possible that a complete explanation may ultimately be found in varying degrees of physiological susceptibility on the one hand, and in varying facilities for access of the virus on the other, without invoking the aid of any other agencies than those with which we are familiar; but it is also possible that in time to come modes of infection will be brought to light which are now as little suspected as bovine scarlet-fever was three years ago.

NOTE.—At the conclusion of Dr. Whitelegge's paper, the President brought before the Society the following paper upon the same subject, written by Dr. Ballard twenty years previously, but never published.

ON THE

PREVALENCE AND FATALITY OF SCARLATINA AS INFLUENCED BY SEX, AGE, AND SEASON.

By EDWARD BALLARD, M.D., F.R.C.P.

(*Read: May 9th*, 1888.*)

During the twelve years 1857-68, I have found recorded in the books of the Poor-law Medical Staff and of various medical institutions in Islington to which I have access, 3,850 cases of scarlatina. These twelve years include three epidemics, namely, those of 1859, 1863, and 1867-8; three years intervening between 1859 and 1863, and three years again between 1863 and 1867.

Sex and Age in their Relation to Scarlatina.—The following table gives the numbers of each sex and at each age of importance attacked with the disease during the twelve years.

The statements of authors upon this subject are neither precise nor accurate, as I shall point out as I proceed. In the first place, my tables show that there is scarcely any age, except, perhaps, the very earliest infancy, which is absolutely exempt from the invasion of scarlet-fever. The youngest patient whose age I have found in my records stated in weeks was a female infant aged 5 weeks; the oldest was a woman aged 74 years. Bouchut remarks that the disease is "scarcely observed until towards the end of the first year". Out of my 2,375 cases, 45, or about 2 per cent., were in children under 9 months. My observations, when the ages of attack are taken in quinquenniads, also bear out the statement of Bouchut, that the maximum of cases occur between the ages of 5 and 10 years; and that of Dr. Clark (as quoted by Willan), together with the opinion currently held in the profession, that children under 10 years of age are the most liable to attack. Dr. Copland gives a very broad range when he states that the susceptibility is greatest between the period of weaning and fully adult age. No doubt it is; but, inasmuch as comparatively

* See note, page 182.

TABLE I.

Total.	Under 3 Mos.	3 Mo.	6 Mo.	9 Mo.	Under 1 Yr.	1.	2.	3.	4.	Under 5 Yrs.	5.	6.	7.	8.	9.	5 Yrs. and under 10 Yrs.	10.	15.	20.	40.	60 Yrs. and upwards.
Males, 1112.	4	9	15	7	35	49	94	129	138	445	131	110	88	61	47	437	133	42	47	8	...
Females, 1260.	1	3	13	11	28	56	84	119	138	425	124	119	120	83	44	490	186	66	81	11	1
Sex not named, 3.	...	...	...	...	...	...	...	2	...	2	1	...	...	...	...	1	...	...	...	...	...
Both sexes, 2375.	5	12	28	18	63	105	178	250	276	872	256	229	208	144	91	928	319	108	128	19	1

It relates to 2,375 *pauper* cases in which the age and sex are specially mentioned. The proportionate liability of each sex and age to suffer from scarlatina may be deduced from the next table, in which the numbers are stated per 10,000 of the population at each age in each sex.

TABLE II.

	Under 1 Yr.	1.	2.	3.	4.	Under 5 Yrs.	5.	6.	7.	8.	9.	5 Yrs. and under 10 Yrs.	10.	15.	20.	40.	60 Yrs. and upwards.
Males ... 159	144	235	445	629	723	421	713	624	521	377	305	517	195	68	20	6	...
Females ... 148	118	275	413	625	717	414	668	665	697	502	278	570	251	75	27	8	2
Both sexes, 132	132	255	430	627	720	418	693*	645	610	441	291	544	224	72	24	7	1

*** The liability at separate ages from 5 to 10 is calculated on a basis of population obtained by assuming a regular arithmetical progression in the numbers living from 10 years to 5 years, as given in the Census Tables.**

very few cases indeed occur after adult age, such a statement is equivalent to giving no information at all. The greatest age at which this writer has observed it was between 50 and 60 years. According to my tables, the age at which the largest number of cases occurred was between 4 and 5 years; that is to say, in the course of the fifth year of life. This is also, according to Table II, the age of greatest liability to attack. The age of greatest proclivity to scarlet-fever, then, is a year later than that of greatest proclivity to measles (according to my observations). There is a gradual, but not absolutely regular, progression in the liability to scarlet-fever from birth to the fifth year of life. On the whole, this increase of liability is much more rapid than the decrease of liability year by year after the fifth year of life is completed. Hence it is that a larger proportion of cases is met with from 5 to 10 years than from 0 to 5 years of age. At 6 years of age, the liability is much the same as at 3 years, and at 8 years not very different from what it is at 2 years of age. After 8 years of age has been attained, the liability lessens rapidly to 10 years. From 10 to 15 years the decline of liability is again rapid, and continues so to extreme age. Dr. Tweedie gives a table of the ages of 200 persons of the two sexes admitted with scarlatina into the London Fever Hospital. Of these, 110 were between 15 and 25 years of age, and hence he says it "disproves the assertion of Sir Gilbert Blane, and others, that the majority of those who are seized with scarlatina are under puberty". But nothing can be more fallacious than the statistics of hospitals in respect of such a question as this, for not only are young children almost invariably excluded, but the tendency of mothers among the lower classes is distinctly to nurse their offspring at home, however infectious the disease, until they are overruled by the independent will of the latter. Among the pauper population of my district, so far from even half the cases being between 15 and 25, only 10.7 per cent. of all the cases happened at any age over 15 years. I may mention that the greatest age at attack mentioned by this writer was 57 years. As I remarked in speaking of measles,* my tables give no indication at all that the period either of first dentition or of puberty exerts any influence in predisposing to an attack. Nor is it clear that the period of second dentition has any remarkable predisposing

* The mention of observations as to measles here and in other parts of this paper has reference to a previous paper, similar to this, which was communicated to the Metropolitan Association of Medical Officers of Health, but still remained unpublished.—E. B.

influence; for although the age 5 to 10 corresponds with the eruption of the first molar incisor and bicuspid teeth, and during this period the liability to scarlet-fever is high, yet the greatest liability to scarlet-fever is observed between 5 and 8 years, when the influence of second dentition on the general health is, I believe, if noticeable at all, less than at a more advanced period. Comparing now scarlet-fever with measles, it is observable that, in the first quinquenniad of age, the latter is nearly four times more commonly met with than the former; in the second quinquenniad, scarlet-fever is a somewhat more common disease than measles; in the third quinquenniad, scarlet-fever is three times as common as measles; in the fourth quinquenniad, about five times as common as measles; from 20 years of age onwards, although the difference of liability is not so great, still it is very greatly on the side of scarlet-fever. As regards the first year of life, it is to be noticed in Table I that the liability to the disease increases in the several successive trimensual periods (allowing for the correction* necessary for the fourth of these periods). Taking this fact in connection with the regular increase of liability afterwards, we can have no difficulty in setting aside the far-fetched and not very scientific explanation of the immunity of sucking-children from scarlet-fever put forth by Dr. Copland, viz., that the infant is "then nourished by a secretion directly from the secreting organ of the mother, and thus possessing some measure of an invigorating vital emanation, thereby enabling the infant to resist the infection."

The general law of age thus enunciated for the two sexes taken together applies to each separately, although there are differences in the liability of the same ages in the two sexes, which I shall allude to presently. It is to be noticed that while there are 1,112 males included in Table I, the females attacked with scarlatina amounted to 1,260. Taking these numbers without proper allowance for the numbers of each sex living, the inference might be drawn that the female sex was more predisposed to scarlatina than the male. And this, indeed, is the statement made by some writers, and among them by Dr. George Burrows. Dr. Tweedie, basing his opinion upon the unreliable table constructed from the Fever Hospital cases, also states it as his experience that females are more liable than males. Among his 200 cases there were 138 females to 62 males, the females being in excess at all ages. I cannot explain this, except by some peculiarity attaching to the hospital itself, such,

* This correction is for the tendency of mothers in stating ages *about* twelve months to call them *one year*.

possibly, as the accommodation for female patients being greater than that for males, or the greater proportion of males being received into the hospital on account of other febrile diseases. Anyhow, it is utterly at variance with the facts as they come out in my more extended tables. Withering and Bouchut state that the number of boys and girls attacked are equal, and Dr. Clark that they are almost equal under twenty years of age. If Table II, however, be consulted, the fact becomes apparent that, taking the numbers of each sex living into account, males are, on the whole, rather more liable to scarlet-fever than females. A similar difference, but more strongly marked, was noticed in the case of measles. But while this is the fact as respects all ages taken together, it is not the fact when distinct periods of life are compared in the two sexes. Up to about six years of age, males are more liable to be attacked by scarlet-fever than female children, but beyond this age the greater liability is decidedly on the side of the females. A similar fact was noticed when I was considering the subject of measles, but, in this latter disease, the turning point of liability was the fourth, and not the seventh year of life. Both Clark and Withering noticed that adult females are more liable to attack than adult males. Hence, it results that, under five years of age, the preponderance of liability to suffer from the disease is slightly on the side of the male sex, and between five and ten years, and in each succeeding quinquenniad, very much on the side of the female sex. There are, however, two ages at which this rule does not seem to apply. One is the second year of life, the period of active first dentition, when it is clear that female children are more liable to scarlatina than male children. The other, my table indicates, is the tenth year of life; but, from the mode in which this part of the table is calculated, the indication may not be true.

Season, etc.—Authors are pretty well agreed that autumn is the season at which, as a rule, scarlatina is most prevalent, and my observations confirm their opinion. Stating its prevalence during the twelve years, as per 1,000 cases in the whole public practice, I find that they were distributed thus

1st or Winter Quarter.	2nd or Spring Quarter.	3rd or Summer Quarter.	4th or Autumn Quarter.
Jan. ... 65 cases	April ... 57 cases	July ... 91 cases	Oct. ... 145 cases
Feb. ... 68 ,,	May ... 56 ,,	Aug. ... 86 ,,	Nov. ... 114 ,,
March... 44 ,,	June . 60 ,,	Sept. ...122 ,,	Dec. ... 92 ,,
177 ,,	173 ,,	299 ,,	351 ,,

As a rule, then, this disease forms one wave of prevalence in the course of the year, the smallest number of cases occurring in the month of March, the largest in the month of October. Spring and winter are the seasons when it prevails least, and summer and especially autumn those in which it prevails most. Erasmus Wilson states that "the atmospheric conditions favourable to scarlatina are cold and moisture combined, and the existence of this state of the weather for any time gives rise to a medical constitution in which scarlatina is apt to be developed." There could scarcely ever occur a year in which this combination of conditions would be more marked than the memorable year 1860, yet in this year scarlatina, as an epidemic, was in abeyance throughout. It had prevailed as a severe epidemic in the summer and autumn of 1859; and in the first quarter of 1860, which, though cold in February, was not wet, there was an amount of scarlet-fever above the average for the season; but, throughout the rest of the year, which was very wet, cold, moist, and miserable, scarlet-fever was below the average prevalence. And, if it be said in reply that the susceptibility of the population had probably been exhausted, I may refer to the epidemic of 1867, which failed to exhaust the susceptibility of the population so far as to prevent the disease raging through the hot and dry year 1868. Indeed, it has appeared to me pretty clearly from my own comparisons of the meteorological conditions of the several forty-eight seasons with the prevalence of scarlet-fever in each, that its extension is favoured by quite an opposite state of weather to that mentioned by Mr. Wilson, namely, by a temperature above the average for the season, and that a dry state of the atmosphere with little rain favours its spread more than the reverse conditions. In the autumn, however, it was most prevalent in the years when the seasons on the whole were warm and rainy. My observations have led me to infer that the weather most favourable to an outbreak of scarlet-fever is one in which the mean temperature is somewhere between 56 and 60 deg., but that a temperature higher than this is not absolutely unfavourable. When the temperature falls below 53 deg., we may, when scarlet-fever is prevalent, look for the commencement of its decline. There is, as I have before mentioned, something like an indication of cyclical recurrence of an epidemic of this disease to be observed in my records, having a period of recession extending over three years. That it did not recede after the epidemic year 1867, may be due to the peculiarly favourable (to scarlatina) meteorological conditions of 1868. Dr. Tweedie says (and this observation has been copied by systematic

writers) that "epidemic visitations are most observed after a warm summer, especially when the heat has been accompanied by continual rains." Our epidemic of 1859 commenced in the third week of September, the mean temp. being about 58 deg. the week before. The summer throughout, from the beginning of June to the end of August, had been hot and dry, with the exception of two weeks of heavy rainfall. The epidemic of 1863 may be regarded as commencing in the third week of September 1862; but the preceding summer was remarkable for being cold and damp from the very beginning of June. The worst of the 1863 epidemic began about the first week in July, the previous winter and spring months having been comparatively warm, with very little rain. The epidemic of 1867 commenced with severity in the first week of October, although the disease had been prevailing unusually all the year since the autumn of 1866. There had been about five weeks' warm weather in August and September, with a good deal of rainfall—indeed, the quantity of rain that fell that summer was enormous (11.40 inches); but, with the exception of these five weeks, the weather had been comparatively cold, almost uniformly so, from the third week in May to the third week in August. So that thus far Dr. Tweedie's observation does not correspond with mine.

The Fatality of Scarlatina in Relation to Age.—This is represented in the following table*:—

TABLE III.

AGES.	CASES (among Paupers; *vide* Table I).	DEATHS (in entire Parish).	Proportion of Deaths in whole Parish, to 10 *Pauper* Cases.
Under 1 year ...	63	139	22.1
1 & under 2 yrs.	105	265	25.2
2 „ 3 „	178	365	20.5
3 „ 4 „	250	300	12.0
4 „ 5 „	276	232	8.4
Under 5 years...	872	1301	14.9
5 & under 10 yrs.	928	495	5.3
10 „ 15 „	319	106	3.3
15 „ 20 „	108	38	3.5
20 „ 40 „	128	54	4.2
40 „ 60 „	19	15	7.9
60 years and upwards.	1	1	10.0

* These old results of mine may be usefully compared with the results of a similar comparison of cases and deaths more recently made by the Registrar-General. (*Registrar-General's Forty-Ninth Annual Report*, pp. xiv-xviii.)—E. B.

Hence, taking both sexes together, we observe that *the fatality lessens from infancy, when it is greatest, up to about fifteen years, after which it gradually increases until advanced age.* But at no period of life is the fatality of the disease so great as it is at the ages below four years. This result corresponds with the observation of Mason Good, that scarlatina is not only rare, but, also for the most part, less violent in adults, and is opposed to the opinion expressed by Dr. Tweedie, that the disease is more severe in adults than in children; an opinion which Dr. Maunsell also adopts. Equally are my statistics at variance with the results of Dr. Copland's observation, who says that, according to his experience, the younger the child the milder is the attack.

The second year of life is only a little more disposed to impart a fatal tendency to scarlet-fever than the first; and although the period of puberty is the turning-point at which the disease begins to become again more fatal, there is no reason to believe that this period in itself is especially dangerous.

The Influence of Sex on the Fatality of Scarlatina.—In order to discover this I have tabulated the ages of 466 fatal cases in males and 414 fatal cases in females, which were all that occurred in Islington during the six years, 1857-1862: the following statement represents the result, together with the proportion borne by the deaths at each age in the entire parish to the cases in public practice at corresponding ages during the same period.

TABLE IV.

AGES.	CASES (among Paupers, Table I).		DEATHS (in entire Parish), 1857 to 1862.		Proportion of Deaths in entire Parish, to 10 *Pauper* Cases.	
	Male.	Female.	Male.	Female.	Male.	Female.
Under 1 year ...	35	28	34	20	9.7	7.1
1 & under 2 yrs.	49	56	56	54	11.4	9.6
2 „ 3 „	94	84	85	72	9.0	8.6
3 „ 4 „	129	119	79	64	6.1	5.4
4 „ 5 „	138	138	53	49	3.8	3.7
Under 5 years...	445	425	307	259	6.9	6.1
5 & under 10 yrs.	437	490	130	99	3.0	2.0
10 „ 15 „	133	186	20	29	1.5	1.6
15 „ 20 „	42	66	5	13	1.2	2.0
20 „ 40 „	47	81	4	11	0.9	1.4
40 „ 60 „	8	11	0	2	0.0	1.8
60 years and upwards.	0	1	0	1	?	10.0

The inference to be drawn is, that to a slight extent scarlet-fever is a more fatal disease with males than with females up to about the period of puberty, when the state of things is reversed, and the disease presses more fatally upon females than upon males. My experience quite confirms the opinion commonly held of the special fatality of scarlet-fever when it attacks women recently confined. As respects males, it is observable that the table indicates a steady decrease of fatality from infancy onwards. During the six years there were six cases of scarlet-fever in the public practice among males above 40 years of age, but no deaths; among females there were five cases above 40 years, and three deaths. But neither among adult males nor adult females is the fatality of scarlet-fever such as it is in infancy.

Fatality of Scarlet Fever in its Relation to Season.—During the twelve years, 1857-68, there were recorded 3,850 cases in public practice, and 1,993 deaths from scarlet-fever occurred in the entire parish. Distributing both into the seasons in which they happened, and calculating the proportion borne by the deaths in the entire parish to the cases in the whole public practice, we arrive at the following result.

TABLE V.

	CASES.	DEATHS.	Proportion of Public Cases, to 10 Deaths in entire Parish.	Proportion of Deaths in entire Parish, to 10 Cases in the Public Practice.
1st or Winter Quarter	670	459	15	6.9
2nd „ Spring „	671	362	19	5.4
3rd „ Summer „	1174	456	26	3.9
4th „ Autumn „	1335	716	19	5.4

The inference deducible from these numbers is that scarlet-fever has proved with us a less fatal and serious disease on the whole in summer months, and that it has been most fatal in the months of winter. In spring and autumn the fatality of the malady has been intermediate, but approaching in its severity nearer to the character of the disease in winter than to its character in summer. It is curious, therefore, to find Hebra stating that his observations do not lead him to attribute to the season of the year any perceptible influence on the issue of this exanthem. It is right, however, that I should state that the mortality given in my table includes deaths from such sequelæ as dropsy. In the summer-time, exposure during desquamation is less dangerous than at

other periods of the year, while the danger is greatest in the winter, and in those months of the spring and autumn quarters which approach nearest to winter in their weather characteristics. Dr. Tweedie states that the disease is generally of a milder character in the spring and summer than in the autumn and winter months.

[I cannot but feel that some apology is needed on my part for permitting the publication of this antiquated paper, which was originally (about the year 1869) communicated to the then "Metropolitan Association of Medical Officers of Health". But since the Society have considered that the facts recorded in it are even now worthy of preservation, in connexion with Dr. Whitelegge's more recent observations, I can only submit to their decision, leaving it to the discretion of the President to publish the paper in full (notwithstanding its criticisms of writers now more or less superseded) or to elide such portions as might be regarded superfluous.—E. B.]

NOTES ON DIPHTHERIA.

By ARTHUR DOWNES, M.D.

(*Read: June 13th*, 1888.)

Mr. President and Gentlemen,—In a recent report,* the Registrar-General defined two distinct and notable fields of diphtheria-prevalence in this country. The one extends from the south-eastern counties into Essex, Hertfordshire, Bedfordshire, and Cambridgeshire; the other has its nucleus in Shropshire and North Wales.

It has been my lot during the past twelve years to have, in both of these fields, an almost continuous experience of epidemic diphtheria; and I propose to lay before you to-night some few gleanings from that experience, more especially in regard to Essex. But, before doing so, I should like to say a few words on—

I.—*Country and Town.*

It has long been observed that diphtheria is chiefly a disease of healthy 'rural districts.† Dr. Farr recorded‡ that during the decade 1861-1870, the diphtheria-mortality in such districts exceeded, in the proportion of 2.3 : 1., the corresponding death-rate of unhealthy Liverpool. A like comparison of the metropolis and its surrounding country, or of the sparsely populated hills of Wales and our crowded towns, then told a similar tale. And for a yet later period the same rule held good, as the following table§ from the *Transactions* of this Society serves to show:—

Mean Annual Death-Rates from Diphtheria, 1871-84 (12 *years*), *per* 100,000 *of Population.*

Liverpool	9.8	London	14.4
Great towns	11.8	Standard Rural Districts	18.1
England and Wales ...	12.6		

I recall these familiar facts to your recoilection because,

* *Fourteenth Annual Report*, p. xii.
† *E.g.*, Greenhow, *On Diphtheria*. London, 1860, p. 195.
‡ *Thirty-seventh Annual Report of Registrar-General*, pp. 219, 220.
§ Parsons, *Trans. Epidem. Soc.*, New Series, iii, 128.

during the current decade, the old order has, seemingly, undergone a change.

Taking the statistics of the past six years (1882-1888), I find the incidence of diphtheria falling with chief severity on the great towns of this country; though, curiously enough, it passes lightly over the towns of second-class importance.

Mean of Annual Death-Rates from Diphtheria, 1882-7 *inclusive*, *per* 100,000 *of Population.**

28 great towns	16.5
50 next largest towns	9.1
England and Wales, exclusive of 78 towns ...	16.1
Whole of England and Wales	16.0

I have partially analysed the incidence on the great towns by a comparison of their mean death-rates in the fourth quarters—in this country the season of greatest mortality from epidemic diphtheria—of the six years in question. In the following list they are arranged in ascending order:—

Mean of Annual Death-Rates per 100,000 *of Population from Diphtheria, in Fourth Quarters of Six Years*, 1882-7.*

Blackburn	1.	Birkenhead	13.5
Derby	4.	Birmingham	14.
Sheffield	6.	Halifax	15.5
Bradford	6.	Oldham	15.5
Wolverhampton ...	7.5	Plymouth	16.
Bolton	8.	Brighton	17.
Hull	10.	Sunderland	17.
Bristol	10.	Newcastle	17.
Leeds	11.	Huddersfield ...	18.
Preston	12.	Cardiff	18.
Salford	12.5	Liverpool	19.
Manchester	13.	Norwich	21.
Nottingham	13.	London	27.
Leicester	13.	Portsmouth	30.†

Whole of England and Wales ...	19.
Dublin	10.
Edinburgh	42.
Glasgow	55.

The relatively large mortality of the Scotch towns is noteworthy.

Of the twenty-eight great towns, nine are seen to have for the periods above mentioned a diphtheria-rate above the average of the country generally, while eighteen have a mortality below that average.

Diphtheria statistics, to be strictly comparable, should take

* From *Quarterly Returns of Registrar-General.*

† Portsmouth had the highest scarlet-fever rate,—another example of the inverse ratio which is so often seen in the prevalence of these two diseases.

into account the age-distribution of the several populations, inasmuch as children suffer in far the greatest proportion. At the same time, I think that we may regard the above-given figures as fairly, though provisionally, approximate. It would seem, therefore, that during recent years diphtheria has invaded the great towns in a larger degree than heretofore, proportionately to the rest of the country. At the same time, this increase, so far, has been of a partial character.

I do not propose to pursue this branch of my subject on the present occasion, but, before I leave it, I may mention that the Medical Officers of Health of Glasgow, Edinburgh, Portsmouth, Norwich, and Liverpool have very courteously favoured me with some particulars of recent diphtheria-prevalence in their respective towns. From these it appears that, with the sole exception of Norwich, where no difference had been observed, the newer portions of the towns are those which have suffered most; and that the better class—or, in Portsmouth, "artizans", as opposed to the "very poor"—had borne the greatest share. London experience, by the way, generally confirms this. During the twenty-five years 1856-1881, Shoreditch, Bethnal Green, and St. Giles had the lowest diphtheria-mortality of all the metropolitan districts. Hampstead had the highest.

Nature of subsoil and configuration of surface seemed to have had little influence; nor in any case had the chief incidence of the disease in the towns in question been particularly associated with pail or privy system.

II.—*The Nosology of Diphtheria.*

Unfortunately, there still exists the utmost confusion as to what is, and what is not, diphtheria. The word, as I have said elsewhere, though current coin of medical language, has no mint standard, and may represent to different minds as many values.

From the epidemic and statistical points of view, the identity of diphtheria and croup would appear a matter of highest probability, their close association a certainty.* Yet the discussion which took place at the Royal Medico-Chirurgical Society's meeting, some few years ago, was little more than a faithful reflex of a general fog; and in our nomenclatures the word "croup", like Muhammad's coffin, still hovers doubtfully between two destinies.

The Registrar-General repeatedly notes and exemplifies the confusion which renders the diphtheria-returns "very

* Cf. Longstaff, *Trans. Epidem. Soc.* iv, 421.

untrustworthy", save on the broadest scale and with the greatest care.* Unfortunately, this chaos involves a very considerable amount of danger to the public health, as some examples taken quite at random from my journals may suffice to show.

Kate B——, a servant, sickened with diphtheria, then locally prevalent in a part of my district, was sent to her home, and died in a village fifteen miles away, also in my district. No diphtheria was then known for many miles around. Within a week, her child, at the same house—an inn—sickened and died also. The mother's death-certificate was "diphtheritic croup", the child's "cynanche trachealis". I was aware of the whole matter at the onset, and warned the school authorities of the village. In reply the rector wrote:

". . . . As to the disease, we are not very clear. Kate's was said first of all to be diphtheria, then croup, now diphtheritic croup. You evidently consider it infectious. The medical attendant told the mother that the child's complaint is not infectious, that she was not to be afraid. . . . I hope language is not used in any technical sense calculated to mislead the public mind."

Now, we had no more "diphtheria" in this village, but I will tell you what we did have—some "sore-throats"; and in the autumn came a fatal outbreak in the next village, followed early in the next year, in the parish beyond, by the following succession of cases, the record of which I take from the Medical Relief and Death-Returns:—

February	Eliz. B.,†	aged 5	...	"Laryngitis"	... *Fatal.*
May ...	Edith B.,†	„ 5	...	„	... *Fatal.*
April ...	Francis S.,‡	„ 2	...	"Glandular swelling."	
May ...	Ernest S.,‡	„ 4	...	"Laryngitis"	... *Fatal.*
June ...	Arthur R.,	„ 5	...	"Sore-throat."	
July ...	Emily W.,	„ 8	...	"Diphtheria."	
Aug. ...	John W.,‡	„	...	"Tonsillitis."	
„ ...	Mary W.,‡	„	...	„	
October .	Wm. M.,	„ 1	...	"Laryngitis"	... *Fatal.*

Yet I was assured on medical authority that, with the exception of Emily W——, there had been no diphtheria in this parish.

Again, one of the most fatal outbreaks I have ever witnessed was initiated by the school attendance of two diph-

* *E.g.*, see his *Forty-fourth Annual Rep.*, p. xxiii; *Forty-fifth Annual Rep.*, p. xviii; also *Quarterly Return*, No. 156, p. vii. Of twenty-seven foreign cities furnishing Table IX of the last-named *Return* (p. xvi), no less than eleven include croup with diphtheria.

† Twins. ‡ Same family.

theric children. They had recently lost a little brother, but "only of croup", and their own swelled necks were merely "mumps", though mumps of a kind that left behind it nasal voice and impaired vision. And let me say by the way that very often indeed do I find diphtheria masquerading in this guise.

My able predecessor in office drew a distinction between diphtheria and what he described as "spreading-quinsy"; but the medical attendant of cases regarded by Dr. Fox as spreading-quinsy tells me that paralysis subsequently followed in some of them. Again, the term "diphtheritic sore-throat" is becoming to the public as comfortable a euphemism as was, and to a great extent still is, scarlatina—a convenient excuse for shutting one's eyes to unpleasant responsibilities. Finally, a new candidate for popular favour has sprung up with an attractive title, which should become both popular and fashionable. I refer to the so-called "Sandringham sore-throat".

Now, I wish to emphasize my belief that, if we are to understand diphtheria aright, and to cope successfully with its spread, we must, in the first instance, bring ourselves to recognise, or at least to admit, its insidious and often trivial forms, and seek not to split up, but to unify our classification of its varieties.

The association of apparently simple sore-throats with outbreaks of diphtheria has been often recognised. Attention was especially drawn to them some nine years ago, when the outbreak in the Grand Ducal Palace at Darmstadt was attributed to infection from cases of this sort.* In Germany, indeed, the term catarrhal diphtheria seems to be fully accepted, and its identity with the typical disease allowed. In France they were described long since as "diphtherite sine diphtheria".† In this country their identity has not been so fully allowed.

It has been suggested, notably by our President,‡ that these debatable cases are essentially non-specific quinsies from which true diphtheria is evolved by conditions unknown.

The remarkable experiments of Dr. Burdon Sanderson§ seemed to lend especial support to this position. Starting from an apparently simple inflammation induced by some chemical irritant, Sanderson was able to produce by successive inoculations from subject to subject, what Sir John

* Oertel, *Brit. Med. Journ.*, 1879, i, p. 36; Eigenbrodt, *ib.*, 112.
† Michel Peter, *Thèse de Paris*, 1859, No. 270; cited by Mackenzie.
‡ Thorne Thorne, *Trans. Epidem. Soc.*
§ *Rep. Med. Off. Privy Council*, New Series, No. VI.

Simon justly termed one of the most tremendous poisons which the mind of the pathologist can conceive. Others, applying the Darwinian theory, have cited to similar effect observations such as those of Buchner, in which ordinarily harmless organisms had apparently become transformed in successive generations into deadly pathogenic forms of life.*

Now, *à priori*, I venture to think that the general facts of evolution are opposed to a theory of development of diphtheria from non-specific sore-throat. I will pass by arguments which might be founded on the antiquity of diphtheria, for antiquity is but a relative term; and one must concede to the evolutionist, as to the geologist, whatever he wishes in point of time. But though it is true that tendency to variation is an attribute of all living things, and is the basis of the Darwinian theory, it must be remembered that continued natural or artificial selection is necessary for the fixing of a variation and the production of a new species. It is conceivable, for example, that, by continually selecting those cultivations of anthrax which show the greatest virulence, we might obtain a stock far more deadly than any now known; but, substituting for selection the natural conditions of chance infection, any casual increase of virulence would assuredly be lost in a few removes. It is the old experience—*Naturam expellas . . . tamen usque recurret.*

I need scarcely remind this Society that Buchner's supposed conversion of the hay bacillus into anthrax has not stood the test of time, that Dr. Klein† has shown the fallaciousness of Sattler's belief in a cultivated infectiveness of *bacillus subtilis* in jequirity infusion, or that the same observer has demonstrated that Grawitz *aspergillus* had possessed its pathogenic qualities *ab initio.*

To Dr. Sanderson's experiment, again, I think we may apply Koch's criticism on the kindred work of Coze, Feltz, and Davaine.‡ There is nothing to show that the results are not simply due to the survival and increase of a particular pathogenic micro-organism to the exclusion of others in successive cultivations. Dr. Sanderson, it is true, says that the irregular and sudden augmentations of virulence in his experience is not comparable to a process of cultivation which would be gradual. But the context shows that he is thinking of quite another matter, namely, the progressive modifications which might be induced in the properties of a particu-

* Airy, *Trans. Epidem. Soc.*

† *Micro-Organisms and Disease*, cap. xvii, third edition, 1886; and *Rep. Med. Off. Local Govt. Board*, 1883.

‡ "Traumatic Infective Diseases" (*New Syd. Soc. Trans.*), pp. 9, 71, etc.

lar organism cultivated in a soil differing from that to which it is naturally habituated. Such acclimatisation would necessarily be gradual; but the extinction of other microbes by the development of some particular species may occur in a single cultivation. I think, then, that the arguments from analogy have hitherto afforded no support to the doctrine of evolution of specific diphtheria from non-specific sore-throat. On the other hand, there is, I submit, strong evidence of the absolute generic identity of diphtheria and the throat-illness to which I have referred as associated with it.

In the first place, mild cases devoid of the characteristics of the fully developed malady are met with in every epidemic disease. Mr. Spear* has recently drawn attention to the insidious onset of typhus epidemics through mild and unrecognised, though specific, cases; and probably many of the supposed *de novo* outbreaks of this disease might have been explained in the same way. Scarlet-fever may be of every degree of mildness or severity; none can draw the line between cholera and choleraic diarrhœa, or say when enteric fever ceases to be "ambulant"; nor does anyone doubt the identity of variola and varioloid, even though this be *sine variolis*.

Then there is the argument, of proverbial and common-sense sort, that such constant companions as diphtheria and its attendant throat-illness must be *ejusdem generis*.

Finally, there is direct and positive evidence that one may give rise to the other. On the one hand, one constantly sees the introduction of a typical case into a household followed by every degree of throat-illness in other members of the family.† On the other hand, a trivial case may initiate the most virulent outbreak.

One rarely sees greater intensity of infection than appeared in an outbreak which has lately come under my notice in a family residing in the Essex parish of Great Leighs. Within a brief space of time, four children died, and two, who survived, escaped after a most dangerous illness, which in one of them was attended by what one only sees, I believe, in the most intense forms—diphtheria of the skin, commencing apparently without previous lesion. Even the cat, a young healthy animal, died with all the symptoms of laryngeal diphtheria two days after licking a basin which had received the vomit of a diphtheric child. Yet the seventh

* *Rep. Med. Off. Local Govt. Board*, 1886.

† This every-day sequence, and its reverse, appear to me to dispose of the theory which one sometimes hears, that the attendant throat-illness of epidemic diphtheria merely predisposes to, and affords a nidus for, the graver disease.

and final case in this family was seemingly only a common sore-throat, and the original infection was derived from some cases of "quinsy" of so slight a kind that the medical attendant, a man of large experience of diphtheria, had no idea of its diphtheric character till some paralytic sequelæ supervened in one of the cases. He had an excellent opportunity of observing these quinsies from first to last, and he is confident that no specific symptom made its appearance until the paralysis supervened.

These slight cases infected also, during last December, two children in an adjoining cottage. I saw them repeatedly, and satisfied myself of the absence of any visible membrane. The children ailed very little, and played about the house. One day, in January of this year, the mother, Mrs. C——, asked me to examine her throat. On one tonsil was a patch of membrane about as large as a sixpence. She felt unwell, but did not take to her bed, and in two or three days was well again. Her only other child, aged two, was thought to have a little sore-throat for a few days.

Under the same roof lived a widow and her daughter, Phœbe; the cases I have just mentioned were, in spite of my warning, thought of little account, and the neighbours saw a good deal of each other. On January 30, Phœbe sickened of diphtheria, had a bad attack, and died. During her illness, Mrs. C—— came in to assist, bringing with her the two-year-old child above mentioned. On February 20 this child was attacked by laryngeal diphtheria, and died in a few days. I should have said also that two brothers-in-law of Mrs. C——, living a mile away, had "quinsy" after coming to see her. No membrane was visible in their case.

I will not take up the time of this Society by multiplying examples of this sort: they are not peculiar to my experience; and I will merely add the practical lesson which I deduce from it, as embodied in the following extract taken from my circular of instructions to school-managers.

"*Scarlet-fever* ('Scarlatina' is identical) and *Diphtheria*. When prevalent, watch for and exclude all cases of sore-throat (no matter how slight or what called), or of suspicious rash, or of recently swollen neck-glands (often erroneously called mumps by parents), or of peeling of scarf-skin of hands or body (after Scarlet-fever), or of altered tone of voice or impaired vision (after Diphtheria). Children in either disease must be considered unsafe to others so long as any of the above-named symptoms remain. At such times 'croup' needs the same precautions as diphtheria, of which it is usually only a form."

III.—*Rural Epidemics.*

Epidemics of diphtheria in rural districts are in their general progress broadly of two classes. The one I may term the "smouldering", the other the "explosive" kind. Either may precede or follow the other.* The former is leisurely in its movements, hangs about a limited area, usually a parish, for months or even years, invading families singly or in small groups in irregular sequence, mostly during the colder months. With the warmth and drought of summer it is little heard of, though it may probably reappear in the succeeding autumn.

Careful inquiry will usually show that minor throat-illness, not coming under medical treatment or attracting public notice, bridges the gaps which may exist between the severer cases. I may, in fact, compare a prolonged prevalence of diphtheria to a wall-surface painted in monochrome, picked out here and there with irregular shadings and dots or blotches. "Catarrhal diphtheria" forms the groundwork of this monochrome, but they whose vision is limited by the orthodox diphtheria of the text-books will only see the severer cases standing out without connection in exaggerated relief.

The "explosive" type, on the other hand, is a sudden outburst affecting a number of families within a brief limit of time. Where it is not preceded by the smouldering sort, it is probably occasioned by some recent importation of infection. I have elsewhere† adduced evidence that these explosions of diphtheria may centre around, and apparently be occasioned by, an exceptional intensification of infectiousness of some particular case. I say exceptional, because the ordinary transmissibility of diphtheria would seem not to be great; it has indeed been ranked as the lowest of our current infections in this regard. Even in districts where diphtheria is endemic, and diphtheric children frequently mingle with their fellows, the common rule of its spread is insidious, slow, and not by leaps and bounds; and in epidemic diphtheria of fatal kind it has been found that in quite 50 per cent. of the infected houses there has been no extension from the first sufferer to the rest of the household.‡

Epidemic explosions of zymotic disease are commonly

* An example of each may be found in the Oaksey outbreak reported in *Rep. Med. Off. Local Govt. Board* for 1883, p. 63.

† *Practitioner*, xxxii, xxxv.

‡ Thorne Thorne, "On Diphtheria at Coggeshall", *Rep. to Local Govt. Board*, 1876; Downes, *Practitioner, ib.*

occasioned in an indirect manner by the introduction of infection into some new field, lying all ready for its spread, or by some swift medium of transit, as milk or water. I think, however, that history affords examples of sudden exaltation of direct infectious virulence, and spreading power analogous to that of diphtheria. I refer to the famous outbreaks of typhus known as the Black Assizes.

The ordinary spreading power of typhus fever is not great. Haygarth* estimated its infection-distance to be less than "half-a-yard"; and Murchison observes that this opinion has been confirmed by all subsequent observations. Pringle† states that though he had "seen some instances of a high degree of contagion attending it, yet the common course of the contagion is slow"—a description which might be applied *verbatim* to diphtheria. Typhus was practically endemic in the jails of that time, yet the Black Assizes can be reckoned on the fingers.

If it is said that these outbursts were simply an outcome of the combination of infectious prisoners and crowded and ill-ventilated courts, it may be replied that, unless the history of that day is altogether exaggerated, such a combination must have been a common event. Moreover, at the Old Bailey, in 1750, it will be remembered that those persons were especially attacked on whom a current of air from an open window is said to have blown. Typhus is a disease of the poor and miserable, yet the victims of the Black Assizes included judges, sheriffs, grand-jurymen, and county notables.

It does not seem certain that the statement is correct that there was, in some of these Assizes, no typhus among the prisoners, and that therefore the infection was borne by *fomites*, or arose *de novo*. Murchison is in error when he cites‡ Pringle in support of his assertion, that there had been no typhus among the Old Bailey prisoners in 1750. Pringle§ expressly says the matter was uncertain, and adds that it was the custom to remove all the malefactors from other jails into Newgate some days before every sessions. What does seem more certain to my mind, from the accounts which we possess, that any preceding fever among the prisoners had not been of great intensity; and this would be in accordance with what I have observed in diphtheria, for the cases to which I have traced explosive outbreaks have in every instance been of moderate severity. Another point of resemblance lies in the

* *On the Prevention of Infectious Fevers*, London, 1801, cited by Murchison; *Continued Fevers*, first edition, p. 64.

† *Diseases of the Army*, fourth edition, 1764, p. 296.

‡ *Ibid.*, p. 87.

§ *Loc. cit.*, pp. 339, 340.

rapid decline of several of the outbreaks of Black Assize and the sudden subsidence of infectiousness. At Oxford, in 1577, when more than 600 are said to have sickened in a single night, the plague ceased as suddenly as it arose; its whole mortality of 510 persons was comprised within five weeks, and, to use the words of the contemporary Register of Merton College, quoted by Anthony à Wood,* "that which is most to be admired is that no women were taken away by it, or poor people, or such that administered physic, or any come to visit."

So also of the Old Bailey outbreak in 1750, it is noted that the disease spread little, if at all, beyond those who had been attacked in court, and that it disappeared in less than six weeks.† I have observed a corresponding rapidity of decline following diphtheria outbreaks of great intensity.‡

IV.—*Variability.*

Diphtheria is, of zymotic diseases, one of the most variable. What we may in one place see as being little more than an affair of sore-throat, may elsewhere number its victims by hundreds and thousands.§

The virulence of an acute infectious disease and its spreading power are usually associated attributes, but not always. We must draw a distinction between them when we see, as in recent London experience of scarlet-fever, an epidemic spreading actively, yet not raising the special mortality above the seasonal average of the country.

A well-defined outbreak at Broomfield, Essex, last year, afforded an example of diphtheria with high spreading power within small limits of time and space, yet devoid of virulence throughout. On October 6, Mr. Waller of Chelmsford informed me that he had just seen two cases of diphtheria in a gardener's family at Broomfield, and that there was reason to believe there were many more in that parish. He kindly gave me permission to see his patients. I found one child with slight pharyngeal paralysis after a sore-throat, which dated from Sept. 28, and the other child with a typical patch of membrane on the left tonsil. I found also that fourteen other families had been attacked by throat-illness between September 23 and October 6. Every case, with one exception (which was otherwise accounted for, and need not be

* *Hist. et Antiq. Univ. Oxon., sub* 1577.

† *Murchison, l. c.*, 102; *Pringle*, 342. ‡ *Practitioner, l. c.*

§ *E.g.*, the Russian and Chinese epidemics. (Hirsch, *Handbook of Geographical Pathology*, New Syd. Soc., 1886, iii, 93, 99.)

further referred to here), was of a child who had been attending the National School, lately reopened after the harvest holidays. Further inquiry revealed the fact that "just before these holidays" some relatives of the schoolmaster living next door to him had had "bad throats", one of them being "very bad", though no medical man had been called in. It is noteworthy, by the way, that diphtheria had occurred in this same house and family about five years ago. From their relatives the schoolmaster's children in turn had taken an infection, which by an easy transition passed into the school. Closure of the school brought the outbreak to an abrupt conclusion. Now, the point I wish you to note is, that, although altogether there were thirty-five cases in fifteen families, and although the outbreak had so suddenly arisen, in only one-fourth of these cases—and I examined them all myself—was there evidence derivable from the symptoms that the disease was diphtheria. Yet its real nature was evident from the following considerations:—

1. Several cases were followed by characteristic paralyses.
2. Cases with and cases without pathognomonic symptoms arose and subsided *pari passu* within the same limits of time and space.
3. Cases seen separately by three medical men were by each diagnosed as diphtheria.

Epidemic influences doubtless resolve themselves into two classes, accordingly as they affect the seeds of contagion or their soil. Of the inter-dependence of these, a laboratory experiment, recorded* by Dr. Klein, affords an illustration. Dr. Klein had already cultivated anthrax bacillus and its spores on gelatine, medicated with $\frac{1}{40000}$ part of corrosive sublimate, but had found that throughout ten subcultures the crops were as virulent as ever to guinea-pigs. One day, however, some accidental difference in their preparation provided him with two sets of agar-agar tubes, obviously distinct in appearance; the one set was pale, the other dark in colour. He sowed the tubes of each set with the blood from a guinea-pig dead of virulent anthrax. There was no difference in the sowings, but there was a remarkable difference in the results. Guinea-pigs inoculated from the pale agar-agar crops died, as usual, in two days; while guinea-pigs inoculated from the dark-coloured agar-agar did not die until the sixth, or even the seventh day. Evidently growth in the dark-coloured cultivation medium had induced a decided attenuation of virulence. But this was not all. Dr. Klein then made in gelatine, medicated with $\frac{1}{40000}$ part of corrosive sublimate as

* *Rep. Med. Off. Local Govt. Board*, 1886, pp. 443, 444.

before, a series of subcultures from the crops on the pale and on the dark agar-agar respectively. The outcome was that, throughout the six generations to which he carried the experiment, crops descended from the pale agar-agar remained as virulent to guinea-pigs as ever, while those from the dark agar-agar, though outwardly as luxuriant, were from first to last innocuous. Finally, he found that any one of these six innocuous generations at once regained its full virulence by a single cultivation in ordinary unmedicated gelatine.

The field-work of epidemiology does not afford us the minute precision of a laboratory experiment, but on the broad scale we constantly see the counterpart of this observation. Indeed, we start on equal terms; for I presume that Dr. Klein could give no better account of the conditions which modified his anthrax in that dark-coloured gelatine than we can offer when, of two persons exposed to the same source of infection, the one has a bad, the other a trivial attack; or why one case should prove highly infectious, while another should be to all appearance sterile. But, once started, an acute infectious disease maintains, on the whole, throughout an epidemic, its type of mildness or severity with quite as much constancy as we can expect, where the conditions are so variable, and where each patient may be regarded as a new culture in an unknown medium.

The public recognise this constancy, speak broadly of a good or bad "sort" of measles and the like, and frequently prove the sincerity of their belief by exposing their children to what they deem a favourable infection. This sort of philosophy is, of course, risky enough at all times; it would be especially dangerous in so variable a disease as diphtheria, even if one attack were protective against a second.

Of the causes which promote intensity of diphtheria we have little knowledge as yet; of their relation to the contagium itself we know nothing; nor can we speak with certainty of their influence through the individual attacked.

Hereditary predisposition is generally admitted. Of the physiological influences of *age* and *sex*, I have nothing to add to what I have elsewhere written,* except that in the five "explosions" of diphtheria in which I have, as I believe, traced the initial case, such case was in each instance a female.

Recent attacks of other zymotics, more especially of scarlet-fever, are generally considered as predisposing to diphtheria; it is less certain how long the predisposition may remain. On more than one occasion I have noted epidemic diphtheria

* *Practitioner, ubi supra.*

following, at six months' interval or more, in the wake of mild scarlet-fever, and picking out to great extent the same families. The family referred to on page 199 as being so severely afflicted had suffered smartly from scarlet-fever eight months before.

A curious case of intermixed diphtheria and scarlet-fever has lately come under my observation, where a girl Annie, aged fourteen, had come home from service on or about January 1st with a mild attack of diphtheria, followed by slight paralytic sequelæ. She was getting well, when, on January 17th, her sister Eliza, aged twelve, also came home from another situation—a public-house—with scarlet-fever. Next day Annie was attacked with scarlet-fever, four smaller children had it within a week, and a lad aged eight went down soon after. The parents escaped, and it is uncertain whether the only other inmate of the house, a son aged sixteen, had scarlet-fever or not. On February 19th, however, the youngest child, aged three, died, according to the death-certificate, of "scarlet-fever and *croup*"; and on March 9th I found that within the previous three weeks the whole family had been attacked by diphtheria except the girl Annie, who had first introduced it, and except the parents, and that each one was then suffering from paralytic sequelæ, which in the lad of sixteen have lasted to the present day.

In this outbreak the rapidity with which Annie took scarlet-fever illustrates, I think, Sir John Rose Cormack's remark,* that diphtheria may predispose to scarlet-fever as well as scarlet-fever to diphtheria. A neighbour who came in to help when the three-year-old child died, conveyed scarlet-fever, but not diphtheria, to her own children. At the public-house where Eliza caught scarlet-fever two children and a lodger had it, but no diphtheria followed.

Seasonal conditions are not without importance. Diphtheric explosions usually occur in late autumn, and on several occasions I have noted their association with heavy rain-fall.

Previous attacks of diphtheria, more especially of a mild kind, often seem to predispose to subsequent attacks, frequently of a severer character. I make it, wherever practicable, a rule that no nurse who has had diphtheria shall be selected to attend on cases of this disease. A nurse who had already had two attacks nearly lost her life in a third, through contagion taken from a case at Tiptree a few years ago.

In the Broomfield outbreak, to which I have previously

* *Clinical Studies.*

alluded, it is noteworthy that one-fourth of the cases were of persons who had had diphtheria in previous years; in two or three families, indeed, such persons seemed to be selected by the infection.* In this respect diphtheria resembles erysipelas. The possibility must be borne in mind, however, that these repeated attacks may be simply due to an original constitutional disposition to diphtheria. Such disposition may perhaps be mainly anatomical; and when one examines the hypertrophied tonsils, with their enlarged follicles, of these patients, one is reminded of Fricke and Günther's observations on predisposition to chancre.†

In my paper in the *Practitioner* I referred to these recurrent cases in reference to a suggestion of Dr. Jacobi's, with which I inclined to agree, that in some of them at least the diphtheric contagium is stowed away for occasional resuscitation, as in syphilis. I regretted that I was not then able to adduce evidence either for or against a remark previously made by Dr. Squire, that such cases are, as in whooping-cough, non-infectious. Indeed, I cannot do so now, but evidence of their apparent infectiousness has been more recently brought forward by the able contribution of Dr. Gresswell to our *Transactions*, and I think that all who have read his paper will admit that he has given to Jacobi's hypothesis a greatly increased importance, and that no investigation into the origin of an epidemic will be complete if it does not take into account this possibility. If we allow that the well-known and often fatal recurrences of diphtheritic symptoms without obvious re-infections are really revivals of true diphtheria, it seems to me that the storage of the diphtheric virus within the organism, and its revival, must of necessity be admitted.

V.—*Mode of Spread.*

In the rural epidemics which I have investigated I have been able to prove only one mode of spread of diphtheria, viz., personal infection, direct or by families. The connection with school attendance has usually been very close.

It is a fortunate attribute of the diphtheric contagium that it is not very diffusible; commonly, indeed, it would seem to require considerable effort to dislodge it from the fauces or air-passages.

* One of these patients is now (Dec. 1888) again suffering from diphtheria.

† "Above all, those who had the mucous follicles of the vagina highly developed were peculiarly liable to the formation of chancre." Quoted by Graves, *Clinical Medicine*, vol. ii, p. 482, New Syd. Soc. edition.

This may possibly explain a curious point, which has been noticed by others, namely, that diphtheric children often seem to be more infectious at school than at home. I believe that the forced expiratory efforts, which are apparently essential to the rudiments of village education, play a part in this. Consequently, in times of epidemic throat-disease, I advise school-managers to space out the children, to dispense with singing exercises as far as possible, or, at any rate, to so dispose the children as to minimise the evil. Possibly, a similar explanation, among others, may apply to an undue incidence of diphtheria on chapel-goers as compared with church-goers in my districts.

Faucial and nasal diphtheria are, in my experience, more "catching" than laryngeal. This may be explicable partly by the earlier and greater fatality of the latter, partly perhaps by physical conditions impeding the expulsion of infectious particles when the disease is deep-seated. This is noteworthy in connection with the supposed non-infectiousness of croup, according to those who hold that croup and diphtheria are distinct diseases. That the laryngeal stuff, however, is itself virulent enough, is unhappily too clearly proved by the death-roll of our profession.

In its relation to puerperal fever diphtheria is similar to scarlet-fever. Again and again I have seen women delivered amidst infection without harm from either disease; on the other hand, I have seen fatal puerperal fever unmistakably attend on each. I believe that it will be found that either contagium may become associated with septic organisms, and that it is to this unholy alliance, and not to the contagia of either scarlet-fever or diphtheria pure and simple, that we must look for an explanation of the disastrous results which sometimes occur.

One occasionally sees a curious connection between diphtheria and erysipelas. In my Maldon Annual Report, for 1887, I have recorded a series of cases where an old woman having erysipelas, her next-door neighbour, who had helped to nurse her, died of puerperal fever; and a good-hearted lady of the neighbourhood, who had administered an enema to this poor thing, had diphtheria.* It is noteworthy that some regard the erysipelatous epidemics of North America as essentially diphtheritic, and it would seem, I think, highly probable that some such alliance with erysipelas as I have suggested may occur in those more "malignant" forms of diphtheria, the danger of which to puerperal women cannot be over-estimated.

* I have now (Dec.) under observation a mother with diphtheria and a son with erysipelas.

The conveyance of diphtheria by fomites is perhaps not so fully recognised by the medical profession as it deserves. I have seen it apparently conveyed to a washerwoman by infected linen, and by the clothes of visitors.

A remarkable outbreak occurred in the Maldon district in 1883. Diphtheria had been brought from Halstead to a family at Goldhanger, in the neighbourhood of which village no throat-illness was then known. Two children were attacked at Goldhanger, of whom one died. On October 19, their mother took some needlework to an isolated farmhouse, some two miles distant. On October 22, two boys at the farmhouse sickened with diphtheria. One of them had been in the kitchen at the time of the needlewoman's visit, but had not spoken to her; the other was away at a day-school, a mile distant, in another direction. The needlework was sent straight to the washtub, and the boys never touched it; but the brown paper in which it was wrapped was given, it was believed, to the two boys on the evening of the 19th, and was cut up by them into patterns for their amusement.

Another case of apparent retention of infection by fomites, although the evidence is not quite so clear, occurred last year in my Chelmsford district. A child, aged three, had died on May 27, in a well-drained roomy cottage with good water-supply, of diphtheritic croup, contracted, there is reason to believe, at school. The family consisted of parents, and five other children aged from two to fifteen years. The house was disinfected and fumigated with sulphur, and all went well until July 12, when the youngest child, who had not been off the premises, sickened, and in a few days died also of diphtheritic croup. In the interval none others of the family had suffered the slightest ailment, and the strictest inquiry revealed no way in which fresh infection could have been conveyed to the house; only one other case of diphtheria was known in the parish, and with that there had been no communication, direct or indirect.

It is true that a hen, of which the child, now dead, had been fond, had "got very thin after rearing chickens", had drooped gradually, and had died a few days before the child fell ill; unfortunately, the carcase was not forthcoming. On the other hand, I elicited the fact that, on July 11, the day before she sickened, this child had, for the first time, occupied the cot (with the same mattress and bedclothes) in which the first child had died. The mattress—a chaff one—had been fumigated with sulphur, and placed in the open air; all bedclothes had been washed; the iron-work of the cot had been exposed to the sulphur-fumes, but had not

been washed. I have not seen any milk diphtheria, but my experience has been chiefly rural, and among classes to which cow's milk is a rarity.

Nor have I ever met with any sufficient evidence of the propagation of this disease by water. I believe, indeed, that no such evidence exists. So much good work, however, results from the popular belief on this point, that I should be sorry to undermine it by merely negative indications. The same may be said as to the connection of true epidemic diphtheria with drainage defects. I have, however, seen some very definite evidence of the relation of some kind of membranous throat-illness to defects of the sort. The following case, for example, is a remarkable one.

A family of about eighteen persons, all adults or nearly so, residing in a good-class house, had for five years literally continually suffered from recurrent attacks of "diphtheria". The medical attendant, in asking me to investigate the matter, told me that these attacks were attended by great anæmia and depression; were best treated by such drugs as bark, steel, and chlorate of potash; but that nothing was so efficacious as change of air. Return home, however, was so often followed by relapse that he was convinced that the house was at fault. The attacks were independent of diphtheria in the locality; they had not spread beyond the family, nor had they ever been followed by paralytic sequelæ. I found that they dated from the time of some "improvements" in the house drainage. Further, that they had always been restricted to the ladies of the family.

The men had not suffered, nor had the servants. This gave me a clue. Differentiating the daily life of these two classes, I found that those who suffered habitually used a particular water-closet, and that those who did not suffer used other closets. Further, it was found that a bad smell was frequently noticed in the particular closet. An examination of this closet showed that the apparatus was a valve discharging without the intervention of any trap into a 10-foot drop of soil-pipe. The soil-pipe was fully ventilated by a long continuation, upwards to above the roof, but it was trapped at the foot. The result was that each time a flush of water entered, a reflux of foul air passed into the closet. The trap was removed from the bottom of the soil-pipe, and a disconnecting trap with ventilating inlet provided elsewhere. A simple lead P-trap was fitted under the valve-closet, and the whole length of drains was charged with sulphur-fumes. It is now five years since this was done, and there has not been a single recurrence of the old throat-illness.

This is, I think, an instructive case; but, gentlemen, although, as you may have gathered from this paper, I am ready to admit as diphtheria any kind of throat-illness, it is only so long as it is associated directly or indirectly with paralytic sequelæ.* I have still my doubts in regard to cases in which we are not able at any time to trace that association. And with this confession of my faith I will conclude what I feel to be a disjointed tissue of observations which must have taxed your patience and good-nature.

* The occasional occurrence of paralysis after other acute diseases (see Gubler, "Des Paralysies dans leur Rapport avec les Maladies Aiguës", *Archives Générales de Médecine*, 1860, i, p. 551) does not practically affect this position.

THE CONDITION AS TO VACCINATION OF PERSONS SCARRED BY SMALL-POX.

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(*Read: June 13th*, 1888.)

THE subject-matter of this paper was suggested to me as the result of a conversation a few years ago with Dr. Brailey, Assistant Ophthalmic Surgeon of Guy's Hospital. Dr. Brailey had observed and recorded the condition as to vaccination of patients who had lost eyes through small-pox, and it occurred to me that further evidence as to the value of vaccination might be obtained if this point were observed in connection with persons scarred with small-pox. Inquiries of this sort have to be undertaken for the reason that the law prohibits the most complete demonstration of the efficacy of vaccination, viz., the inoculation with small-pox virus of persons who have been previously vaccinated. It will be seen on reference to Table I, giving account of Dr. Brailey's cases, and which will be found appended to this paper, that of sixteen persons who had each lost an eye as the result of small-pox, seven were admittedly unvaccinated, or 43 per cent.; and the average age at which these contracted small-pox was 8.9 years; while the average age of four at time of attack who had no vaccination scar, but were nevertheless stated to have been vaccinated, was 8.2 years; two others, with one vaccination scar, had an average age of 20 years when attacked by small-pox; while two, bearing evidence of having been vaccinated in two or more places, had an average age of 28.5 years at time of their small-pox. These sixteen cases of loss of eye through small-pox, of which Dr. Brailey has kindly given me particulars, were collected from a total of 763 patients coming under his observation, and having from a variety of causes suffered from this misfortune.

It will be noted that of these sixteen persons not a single one had been revaccinated, nor, indeed, had a single one been vaccinated to the extent required of vaccinators under con-

tract by the instructions of the Local Government Board. In fact, those who had been submitted to this operation had been imperfectly vaccinated. Judged by the figures Dr. Brailey supplies, neglect of vaccination or the perfunctory performance of this operation may be credited with 2.1 per cent. of the total cases in England in which the eye has been lost.

Turning now to the cases of persons scarred with small-pox who have come under my own observation, and of which account will be found in Table II appended, I desire to state that this list includes the total number which have come before me. The table relates almost entirely to women attending at the obstetric out-patient department of St. Thomas's Hospital, and at the vaccination stations with which I am connected, women being, with few exceptions, the only adults thus coming before me; the cases are arranged in the table in order according to the age the individual had attained when attacked by small-pox.

In what follows hereafter, cases 5, 26, 84, 95, and 119 are eliminated from consideration for the following reasons. Case 5 was probably vaccinated during the period of her incubation of small-pox, but the exact date could not be fixed. Case 26 had some indistinct mottling, but it was impossible to conclude with any certainty that this was the result of vaccination. Case 84 was not examined. Case 95, for the same reason as case 26. Case 119, because the vaccination took place during the period of incubation of small-pox.

The number of cases to which the paper refers are therefore $152 - 5 = 147$. Three other cases marked † as shown on the table are not included, as the observations relating to them were too incomplete to admit of their being used.

Table III shows these 147 cases classified according to their condition of vaccination, and arranged according to the age they had attained before contracting small-pox.

This series of cases appears to indicate that if a person, before reaching the age of six, has small-pox which leaves scars, that person has never been thoroughly vaccinated; further examination of the series shows that of the total 147, 83 were admittedly unvaccinated, or 56.6 per cent. Now, if vaccination afforded no protection against small-pox, there can be no reason why unvaccinated persons, constituting only some 5 per cent. of the community, should contribute to our table more than 5 per cent. of those who are scarred with small-pox; yet we find that, instead of contributing 5 per cent., as many as 56.6 per cent. are derived from this unvaccinated section of the population. The difference between

5 and 56.6 per cent. is the more striking when we recollect that the scarred persons who have come under my observation are the survivors from a larger number who have been attacked with small-pox, and that the proportion of vaccinated persons who survive attack by small-pox is much greater than the proportion of unvaccinated persons who recover from this disease. Gayton, indeed, shows that as many as 43.7 in every hundred unvaccinated persons attacked by small-pox die, while vaccinated persons when thus attacked die at the comparatively low rate of 10.57 per cent.

It is now many years since Marson proved that the mortality of vaccinated persons from small-pox was in inverse proportion to the number of vaccination scars which they possessed. The cases I have collected demonstrate clearly enough that the greater number of vaccination vesicles produced in infancy, the longer is the duration of protection against attack by small-pox which scars the face. This will be conveniently seen by the following figures compiled from Table II; Table III further showing this in greater detail.

	No. of cases.	Average age when attacked by small-pox.
Unvaccinated	83	6.6
Having no vacc. scar, but alleged to have been vaccinated	19	11.4
Having one vacc. scar	19	17.1
Having two vacc. scars	13	17.7
Having three vacc. scars... ...	9	19.1
Having four or more vacc. scars ...	4	19.7

The reader will of course bear in mind that the average age of those attacked does not indicate the earliest age at which susceptibility to small-pox which scars recurs after infantile vaccination; and that, further, the results given above are dependent upon the chance of exposure to small-pox infection. This chance may be assumed to be equal in regard to each class, but in those in which the number of cases is small, there is of course greater opportunity for error.

An interesting relation is found to exist between the death-rate from small-pox after one and another kind of vaccination, and the length of time during which such vaccination confers immunity against attack by small-pox which leaves scars.

To appreciate this, the rate of mortality after such vaccination must be stated, and the following table, compiled from

Marson's and Gayton's statistics, may be regarded as trustworthy.

	Mortality per cent.
Unvaccinated	40.4
Alleged to be vaccinated, having no cicatrix ...	26.3
Having one cicatrix	10.13
Having two cicatrices	6.9
Having three cicatrices	4.9
Having four or more cicatrices	1.9

If the rate of mortality of those alleged to have been vaccinated, but having no cicatrix, is deducted from the rate of mortality of those admittedly unvaccinated, and the remainder be divided by 2.84, the result will be found to be equal to that which is obtained when the average age at time of attack by small-pox of those alleged to have been vaccinated, but having no cicatrices, is deducted from the average age of those admittedly unvaccinated. Thus:—

$$\frac{40.4-26.3}{2.84} = 11.5-6.6$$

The figures 2.84 give an arbitrary number, which may be applied as a constant to the rest of the series of these two tables, with results sufficiently close to demonstrate the relation between rate of mortality from small-pox and duration of immunity from disfiguring disease which is given by the different kinds of vaccination.

I would submit that the table I am able to put before the Society may be regarded as further evidence that the amount of protection afforded by infantile vaccination against small-pox is directly proportioned to the number of vesicles which have been produced by the operation; and I hold that the practitioner who supplies to his patient an inferior vaccination cannot escape responsibility if this patient becomes subsequently disfigured or blind as the result of small-pox.

The instruction of the Local Government Board in their requirement of four vesicles is, I am satisfied, founded on an ample experience, and is supported by every honest investigation of this subject. In the absence of any legal requirement as to the amount of vaccination which should be produced, I believe advantage would be gained if the form of vaccination certificate were so altered as to necessitate record of the number of vesicles which the operation has produced. Should this paper come under the observation of the insufficient Vaccinator, I would desire to caution him that I have dealt solely with persons who have been scarred by the small-pox from which they have suffered; that if I had been considering

the average age attained at the time of attack by that small-pox which does not disfigure, I am convinced it would have been found below the age-period 6.6 years of my table.

Since this paper was written, and in the interval before its publication, I have collected an additional thirty-six cases of persons scarred by small-pox. When these are added to the preceding 148, the following is the average age at time of attack by small-pox in respect of each class:

Admittedly unvaccinated.	Stated to have been vaccinated, but without vacc. cics.	Having one vacc. cic.	Having two vacc. cics.	Having three vacc. cics.	Having four or more vacc. cics.
Years 7.05	10.8	17.1	18.4	19.5	19.0

TABLE I.

A record of cases who lost eyes from small-pox out of a total of 763 cases who lost eyes from all causes, as observed at Moorfields Hospital by Dr. Brailey.

	No. in Case Book.	Age when attacked.	Whether vaccinated with effect before having small-pox.	If vaccinated, number of scars.	Age of patient at date of observation.	Date of removal of eye.	Year patient had small-pox.	REMARKS.
1	346	Under 1 year	Not	.....	12	Nov. 12, 1878	1866	Both eyes lost.
2	*	1 year ...	Not	.....	18	?	1862	
3	712	1 year ...	Not	.....	30	?	1851	
4	841	1 year ...	Vacc. during incubatory period	.....	62	April 22, 1882	1820	
5	944	4 years ...	Vacc. produced only a pimple	.....	4	Oct. 1882	1882	
6	798	7 years ..	Not	.....	8	Feb. 3, 1888	1881	
7	741	12 years ...	In inf.	No scar	13	?	1880	
8	903	15 years ...	Not	.....	29	Aug. 5, 1882	1869	
9	748	16 years ...	In inf.	No scar	16	1881	1880	Holds certificate of vaccination.
10	486	17 years ...	In inf.	1 scar	21	June 1881	1877	
11	489	19 years ...	Not	.....	24	Mar. 14, 1880	1875	
12	768	19 years ...	Not	.....	37	Dec. 7, 1881	1863	
13	498	20 years ...	In inf.	2 scars	22	?	1878	
14*								
15	222	23 years ...	In inf.	1 scar	24	July 11, 1878	1877	
16	158	37 years ...	In inf.	Has scars, but No. not stated.	38	July 3, 1887	1876	

* Imperfectly observed.

TABLE II.

Patients at St. Thomas' Hospital and elsewhere observed by Dr. Cory to be scarred by small-pox (inclusive of all *those observed).*

	No. in Case Book.	Age when attacked.	Whether vaccinated with effect before having small-pox.	If vaccinated, number and area of scars, the average diameter being indicated by a line.	Age of patient at date of observation.	Date of observation.	Year patient had small-pox.	REMARKS.
1	83	5 weeks ...	Not	.....	24	July 13, 1887	1863	Not deeply pitted.
2	32	6 weeks ...	Not	.....	56	June 27, 1887	1831	Somewhat pitted.
3	49	6 weeks ...	No evidence	No scars	21	Mar. 25, 1887	1886	Said to be vaccinated. Severely pitted and eye damaged.
4	16	2 months	Not	.....	32	June 29, 1885	1853	Eye with serious cornial damage. Face scarred.
*5	122	3 or 4 mths.	In inf.	3 / fov.	23	Oct. 20, 1887	1864	Scarcely marked.
6	39	3 or 4 mths.	Not	.....	28	Feb. 15, 1887	1859	Much scarred on face.
7	129	6 months	Not	.....	36	Nov. 1, 1887	1851	Much pitted.
8	92	6 months	Not	.....	52	Aug. 4, 1887	1835	Slightly marked.
9	148	6 months	Not	.....	32	May 1, 1888	1859	Not much scarred.
10	12	7 months	Not	.....	28	April 12, 1885	1858	Face pitted.
11	77	7 months	Not	.....	30	July 3, 1887	1857	Pits deep, but not numerous.
12	62	9 months	Not	.....	35	June 2, 1887	1852	Face scarred.
13	117	9 months	Not	.....	32	Sept. 21, 1887	1856	Pitted.
14	48	10 months	Not	.....	39	Mar. 23, 1887	1849	Much pitted.
15	154	10 months	Not	.....	25	May 31, 1888	1862	Sparely, though deeply scarred.
16	72	11 months	Not	.....	49	June 23, 1887	1839	Perceptibly scarred.
17	96	1 year ...	Does not know	No scars	62	Aug, 11, 1887	1826	Deeply scarred.
18	42	1 year ...	Not	.....	28	Feb. 18, 1887	1860	Much scarred on face.
19	128	1 year ...	Not	.....	28	Nov. 16, 1887	1860	Distinctly marked.

20	106	1 year ...	In inf.	No scars	46	Aug. 24, 1887	1842	Face scarred.
21	104	1 year ...	Not	.....	24	Aug. 23, 1887	1864	Face scarred.
22	149	1 year ...	In inf.	No scar	24	May 8, 1888	1865	Deeply scarred.
23	120	1½ years ...	Not	.....	58	Oct. 6, 1887	1831	Severely pitted.
24	133	1½ years ...	In inf.	No scars	36	Dec. 14, 1887	1853	Slightly pitted.
25	91	1½ years ...	Not	.....	38	Aug. 2, 1887	1851	Scarred on face.
*26	64	1½ years ...	In inf.	Doubtful scars	22	June 9, 1887	1867	Not recorded.
27	65	1½ years ...	Not	.....	29	June 9, 1887	1860	Much scarred.
28	124	2 years ...	Not	.....	49	Nov. 1, 1887	1840	Scarred.
29	143	2 years ...	Not	.....	65	Feb. 21, 1888	1825	Much marked.
30	6	2 years ...	In inf.	No scars	27	Mar. 11, 1887	1859	Face pitted.
31	107	2 years ...	Not	.....	40	Aug. 27, 1887	1849	Much pitted.
32	115	2 years ...	Not	.....	62	Sept. 13, 1887	1826	Badly pitted.
33	36	2 years ...	Not	.....	25	Feb. 9, 1887	1864	Severely pitted.
34	116	2 years ...	Not	.....	21	Sept. 21, 1887	1868	Badly pitted.
35	10	2 years ...	Not	.....	39	April, 1885	1848	Scarred on face.
36	63	2½ years ...	Not	.....	41	June 8, 1887	1848	Slightly pitted.
37	71	3 years ...	Not	.....	22	June 23, 1887	1868	Deeply pitted.
38	86	3 years ...	Not	.....	42	July 1, 1887	1848	Face pitted, but not badly.
39	45	3 years ...	Not	.....	23	Mar. 10, 1887	1867	Markedly pitted.
40	90	3 years ...	Not	.....	24	Aug. 2, 1887	1886	Deeply, but sparsely pitted.
41	108	3 years ...	Not	.....	25	Aug. 27, 1887	1865	Much pitted.
42	109	3 years ...	Not	.....	26	Aug. 27, 1887	1865	Pitted, not severely.
43	69	3 years ...	Not	.....	24	June 14, 1887	1866	Slightly scarred.
44	95	3 years ...	Not	.....	53	Aug. 10, 1887	1837	Markedly pitted.
45	151	3 years ...	Not	.....	49	May 10, 1888	1842	Much scarred.
46	46	3½ years ...	Not	.....	35	Mar. 22, 1887	1856	Markedly pitted.
47	8	4 years ...	In inf.	4 indistinct and plain scars	22	Mar. 17, 1885	1866	Face not badly, but distinctly marked.
48	27	4 years ...	In inf.	No scars	24	Dec. 27, 1886	1866	Pitted distinctly.
49	53	4 years ...	Not	.....	35	May 3, 1887	1856	Slightly pitted.
50	67	4 years ...	Not	.....	50	June 2, 1887	1841	Much pitted.
51	125	4 years ...	Not	.....	24	Nov. 2, 1887	1867	Slightly scarred.

	No. in Case Book.	Age when attacked.	Whether vaccinated with effect before having small-pox.	If vaccinated, number and area of scars, the average diameter being indicated by a line.	Age of patient at date of observation.	Date of observation.	Year patient had small-pox.	Remarks.
52	127	4 years ...	Not	.....	30	Nov. 10, 1887	1861	Face scarred.
53	141	4 years ...	Not	.....	45	Jan. 25, 1888	1847	Marked on face.
54	142	4 years ...	Not	.....	56	Feb. 1888	1836	Much marked.
55	147	4 years ...	In inf.	No scar	25	April 26, 1888	1867	Sharply scarred.
56	54	5 years ...	Not	.....	29	May 3, 1887	1853	Pitted, but not deeply.
57	105	5 years ...	Not	.....	28	Aug. 23, 1887	1864	Scarred.
58	126	5 years ...	Not	.....	67	Nov. 3, 1887	1825	Slightly scarred.
59	134	5 years ...	Not	.....	34	Dec. 14, 1887	1860	Slightly scarred.
60	153	5 years ...	Not	No scar	32	May 29, 1888	1861	Slightly scarred.
61	38	5½ years ...	Not	.....	46	Feb. 15, 1887	1866	Slightly scarred.
62	50	6 years ...	Not	.....	33	Mar. 25, 1887	1870	Pitted, but not severely.
63	57	6 years ...	Not	.....	27	May 10, 1887	1866	Slightly pitted.
64	88	6 years ...	Not	.....	34	July 30, 1887	1859	Not severely scarred.
65	98	6 years ...	Not	.....	28	Aug. 17, 1887	1865	Scarred on face.
66	114	6 years ...	In inf.	1 plain very indistinct	30	Sept. 13, 1887	1871	Small scattered, but deep pits.
67	121	6 years ...	In inf.	1 slightly fov.	29	Oct. 20, 1887	1864	Slightly pitted.
68	155	6 years ...	In inf.	1	35	May 31, 1888	1859	Deeply scarred. Damage to left eye.
69	40	7 years ...	In inf.	1	35	Feb. 15, 1887	1859	Not recorded.
70	79	7 years ...	Not	.....	31	July 7, 1887	1863	Deeply scarred.
71	110	7 years ...	Not	.....	31	Aug. 30, 1887	1863	Severely scarred.
72	137	7 years ...	Not	.....	47	Jan. 10, 1888	1847	Markedly pitted.
73	28	8 years ...	Not	.....	20	July 23, 1886	1870	Deeply pitted.
74	52	8 years ...	Not	.....	28	Mar. 30,	1867	Pitted.
75	68	8 years ...	In inf.	1 very faint and indistinct	43	June 14, 1887	1852	Slightly marked.

76	102	8 years ...	Not	.....	28	Aug. 23, 1887	1867	Face much scarred.
77	11	9 years ...	Not	.....	9	April 10, 1885	1885	Both eyes lost entirely; face disfigured; most of hair gone.
78	24	9 years ...	In inf.	2 faint and indistinct	30	Dec. 30, 1885	1881	Scarred on face.
79	29	9 years ...	Not	.....	30	Jan. 8, 1887	1866	Much marked.
80	118	9 years ...	Not	.....	25	Sept. 27, 1887	1878	Badly pitted.
81	135	9 years ...	Not	.....	33	Dec. 24, 1887	1863	Considerably marked.
82	43	10 years ...	Not	.....	32	Feb. 24, 1887	1867	Slightly scarred.
83	73	10 years ...	In inf.	No scar	25	June 23, 1887	1872	Slightly scarred.
*84	130	10 years ...	In inf.	Not examined	23	June 29, 1887	1874	Slightly marked.
85	5	11 years ...	Not	.....	34	Mar. 3, 1885	1861	Much pitted on face.
86	33	11 years ...	In inf.	2 fov.	33	Feb. 1, 1887	1865	Pitted perceptibly.
87	80	11 years ...	In inf.	1	24	July 7, 1887	1874	Not deeply pitted.
88	144	11 years ..	In inf.	1 indistinct scar	38	Feb. 22, 1888	1861	Not much marked. This patient is said to have had a second attack of small-pox in 1881.
89	7	12 years ...	Not	.....	36	Mar. 17, 1885	1860	Face much and deeply scarred.
90	37	12 years ...	In inf.	No scars	34	Feb. 12, 1887	1866	Slightly scarred on face.
91	41	12 years ...	In inf.	No scars	32	Feb. 16, 1887	1867	Slightly scarred.
92	47	12 years ...	In inf.	3	36	Mar. 22, 1887	1863	Slightly pitted.
93	59	12 years ...	In inf.	2 indistinct	20	June 2, 1887	1879	Very slightly pitted.
94	75	12 years ...	In inf.	3 fov.	21	June 28, 1887	1878	Heavily scarred.
*95	150	12 years ...	In inf.	Doubtful scar	28	May 8, 1888	1872	Scarred.
96	22	13 years ...	In inf.	3 faint cicatrix	13	Nov. 1885	1885	Much pitted.
97	31	13 years ...	In inf.	2 very faint.	29	Jan. 25, 1887	1867	Markedly pitted.
98	74	13 years ..	In inf.	1 fov.	23	June 28, 1887	1877	Pitting of face thickly distributed, but small scars.
99	81	13 years ...	In inf.	1 good fov.	31	July 12, 1887	1870	Slightly scarred.
100	99	13 years ...	In inf.	1 in unusual position on back of shoulder	19	Aug. 17, 1887	1881	Scarred on face.

	No. in Case Book.	Age when attacked.	Whether vaccinated with effect before having small-pox.	If vaccinated, number and area of scars, the average diameter being indicated by a line.	Age of patient at date of observation.	Date of observation.	Year patient had small-pox.	Remarks.
101	19	14 years ...	In inf.	4	27	Aug. 5, 1887	1872	Face scarred.
102	51	14 years ...	Not	No scars	33	Mar. 25, 1887	1868	Slightly pitted.
103	78	14 years ...	Not	.....	25	July 6, 1887	1876	Scarred very perceptibly.
104	93	14 years ...	In inf.	2 plain & indistinct	35	Aug. 4, 1887	1866	Slightly marked.
105	136	14 years ...	Not	scars	23	Dec. 27, 1887	1878	Very badly pitted.
106	17	15 years ...	Not	.....	29	July 6, 1885	1871	Face scarred, ectropion of lower eyelid.
107	84	15 years ...	Not	.....	23	July 13, 1887	1879	Much pitted.
108	94	15 years ...	In inf.	No scars	50	Aug. 10, 1887	1852	Slightly, but distinctly marked.
109	30	16 years ...	In inf.	6 plain, well marked	33	Jan. 25, 1887	1870	Considerably pitted.
110	146	16 years ...	In inf.	1	33	April 25, 1888	1871	Scarred.
111	157	16 years ...	In inf.	No scar	40	May 23, 1888	1864	Slightly scarred.
112	2	17 years ...	Not	.....	45	Jan. 20, 1885	1844	Face much marked.
113	44	17 years ...	In inf.	1	33	Mar. 10, 1887	1870	Face scarred.
114	85	17 years ...	In inf.	1 very indistinct and plain scar	32	July 16, 1887	1872	Pitted, especially in regions of freckles of face.
115	87	17 years ...	In inf.	No scars	32	July 19, 1887	1870	Scarred, but not badly.
116	89	17 years ...	In inf.	3 distinct	24	Aug. 2, 1887	1880	Slightly, and almost imperceptibly marked.
117	131	17 years ...	In inf.	1 fov.	37	Nov. 22, 1887	1867	Marked.
118	145	17 years ...	In inf.	3	30	May 1, 1888	1859	Not much scarred.
*119	21	18 years ...	In inf.	5 days before small-pox attack	27	Aug. 1885	1876	Pitted on face.
120	34	18 years ...	Not	.....	31	Feb. 1, 1887	1869	Face pitted.
121	132	18 years ..	In inf.	No scar	33	Dec. 13, 1887	1872	Much pitted.
122	23	19 years ...	In inf.	No scar	44	Jan. 1886	1860	Not much scarred.

123	66	19 years ...	In inf.	2 fov.	34	June 16, 1887	1872	Slightly pitted.
124	70	19 years ...	In inf.	1 fov.	28	June 22, 1887	1878	Thickly, but not distinctly scarred.
125	97	19 years ...	In inf.	2 fov. distinct	40	Aug. 16, 1887	1866	Slightly scarred.
126	18	20 years ...	Not	.....	45	July 1885	1860	Face scarred.
127	60	20 years ...	In inf.	2 fov.	28	June 2, 1887	1879	Slightly pitted.
128	113	20 years ...	In inf.	2 fov.	36	Sept. 13, 1887	1871	Slightly pitted.
129	123	20 years ...	In inf.	3 fov.	28	Oct. 27, 1887	1877	Not scarred.
130	100	21 years ...	Not	.....	27	Aug. 18, 1887	1881	Face scarred.
131	103	21 years ...	In inf.	2 slightly fov.	37	Aug. 23, 1887	1871	Face scarred.
132	20	22 years ...	In inf.	3	36	Aug. 1885	1871	Pitted.
133	35	22 years ...	In inf.	2 faint and plain	35	Feb. 4, 1887	1874	Slightly scarred on face.
134	82	22 years ...	In inf.	No scar	29	July 14, 1887	1880	Face frightfully scarred.
135	119	22 years ...	In inf.	5	31	Oct. 4, 1887	1878	Slightly scarred.
136	139	22 years ...	In inf.	1 indistinct scar	39	Mar. 11, 1887	1870	Markedly pitted.
137	15	24 years ...	In inf.	No scar	24	May 7, 1885	1885	Face much marked with recent small-pox.
138	58	26 years ...	In inf.	2 plain	36	June 2, 1887	1877	Pitted.
139	9	27 years ...	Not	.....	27	Mar. 25, 1885	1885	Severe keratitis; face much pitted; hair gone.
140	25	27 years ...	Not	.....	35	July 28, 1886	1878	Scarred obviously on face, but not deeply.
141	112	27 years ...	In inf.	5	36	Sept. 13, 1887	1878	Slightly marked.
142	56	29 years ...	In inf.	1 faint	34	May 11, 1887	1882	Pitted.
143	140	29 years ...	In inf.	No scars	49	Jan. 17, 1888	1868	Sight of right eye gone, and deaf in left ear from small-pox. Very much disfigured.
144	1	30 years ...	Not	.....	52	Nov. 1, 1884	1862	Face much scarred.
145	4	30 years ...	In inf.	3	30	March 1885	1884	Scarred.
146	26	30 years ...	In inf.	3 faint	34	Dec. 8, 1887	1884	Scarred on face, not deeply.
147	138	30 years ...	In inf.	1 fov.	33	Jan. 14, 1888	1885	Severely pitted.

	No. in Case Book.	Age when Attacked.	Whether vaccinated with effect before having small-pox.	If vaccinated, number and area of scars, the average diameter being indicated by a line.	Age of patient at date of observation.	Date of observation.	Year patient had small-pox.	Remarks.
148	101	33 years ...	In inf.	2 quite plain and indistinct	39	Aug. 20, 1887	1881	Face much pitted.
149	111	33 years ...	In inf.	No scar	40	Sept. 3, 1887	1880	Slightly marked.
150	61	34 years ...	In inf.	1 indistinct	45	June 4, 1887	1876	Slightly pitted.
151	55	36 years ...	Not	.....	62	May 10, 1887	1861	Pitted; the scars not deep, but white.
152	76	37 years ...	In inf.	1 fov. distinct	41	June 29, 1887	1883	Slightly marked.
†	3	?	Could not say as to vacc.	No scar	18	Mar. 3, 1885	?	Much pitted on face.
†	13	?	Not	.....	4½	April 28, 1885	?	Left eye destroyed.
†	14	?	?	?	?	April 28, 1885	?	Blind from small-pox.

TABLE III.

Showing the 148 *cases classified in regard to conditions as to vaccination.*

Admittedly unvaccinated.	Stated to be vaccinated, but without vaccination cicatrices.	Having one vaccination cicatrix.	Having two vaccination cicatrices.	Having three vaccination cicatrices.	Having four or more vaccination cicatrices.
5 weeks ...	...	...	...	...	...
6 weeks ...	6 weeks	...	...	...	...
2 months	...	...	...	...	...
3 or 4 mths.	3 or 4 mths.	...	...	...	...
6 months	...	...	...	...	...
6 months	...	...	...	...	...
6 months	...	...	...	...	...
7 months	...	...	...	...	...
7 months	...	...	...	...	...
9 months	...	...	...	...	...
9 months	...	...	...	...	...
10 months	...	...	...	...	...
10 months	...	...	...	...	...
11 months	...	...	...	...	...
1 year ...	1 year	...	...	...	...
1 year ...	1 year	...	...	...	...
1 year ...	...	...	...	...	...
1 year ...	...	...	...	...	...
1⅓ years ...	...	...	...	...	...
1½ years ...	1½ years	...	...	...	...
1½ years ...	...	...	...	...	...
2 years ...	...	...	...	...	...
2 years ...	2 years	...	...	...	...
2 years ...	...	...	...	...	...
2 years ...	...	...	...	...	...
2 years ...	...	...	...	...	...
2 years ...	...	...	...	...	...
2 years ...	...	...	...	...	...
2½ years ...	...	...	...	...	...
3 years ...	...	...	...	...	...
3 years ...	...	...	...	...	...
3 years ...	...	...	...	...	...
3 years ...	...	...	...	...	...
3 years ...	...	...	...	...	..
3 years ...	...	...	...	...	...
3 years ...	...	...	...	...	...
3 years ...	...	...	...	...	...
3 years ...	...	...	...	...	...
3½ years ...	...	...	...	...	...
4 years ...	4 years	...	...	...	...
4 years ...	4 years	...	...	...	...
4 years ...	...	...	...	...	...
4 years ...	...	...	...	...	...
4 years ...	...	...	...	...	...
4 years ...	...	...	...	...	...
5 years ...	...	...	...	...	...
5 years ...	...	...	...	...	...
5 years ...	...	...	...	...	...
5 years ...	...	...	...	...	...
5 years ..	...	...	...	...	...
5½ years ...	...	...	...	...	...

Admittedly unvaccinated.	Stated to be vaccinated, but without vaccination cicatrices.	Having one vaccination cicatrix.	Having two vaccination cicatrices.	Having three vaccination cicatrices	Having four or more vaccination cicatrices.
6 years ...	6 years	...	...	...	...
6 years ...	...	...	...	...	...
6 years ...	...	6 years	...	...	...
6 years ...	...	6 years	...	...	...
7 years ...	...	...	...	...	...
7 years ...	...	7 years	...	...	...
7 years ...	..	...	...	...	...
7 years ...	...	...	...	...	...
8 years ...	...	8 years	...	...	...
8 years ...	...	...	...	...	...
9 years ...	...	...	9 years	...	...
9 years ...	...	...	...	...	...
9 years ...	...	...	...	...	...
9 years ...	...	...	...	...	...
10 years ..	...	...	...	...	...
10 years ...	...	...	...	...	...
11 years ...	...	11 years	11 years	12 years	...
12 years ...	12 years	11 years	12 years	12 years	...
14 years ...	12 years	13 years	13 years	13 years	14 years
14 years ...	...	13 years	14 years	...	...
14 years ...	...	13 years	...	...	...
15 years ...	...	...	...	...	...
15 years ...	15 years	...	...	...	...
17 years ...	16 years	16 years	...	17 years	16 years
17 years ...	...	17 years	...	17 years	...
18 years ...	18 years	17 years	...	...	...
20 years ...	19 years	17 years	19 years	...	...
21 years ...	22 years	19 years	19 years	...	...
27 years ...	24 years	22 years	20 years	20 years	...
27 years ...	29 years	29 years	20 years	..	...
...	...	30 years	21 years	22 years	...
30 years ...	33 years	34 years	22 years	30 years	22 years
36 years ...	...	37 years	26 years	30 years	27 years
...	...	...	33 years	...	...
Average age in years 6.6	In years 11.8	In years 17.2	In years 18.4	In years 19.2	In years 19.7