OCTOBER, 1933

THE ULSTER MEDICAL JOURNAL

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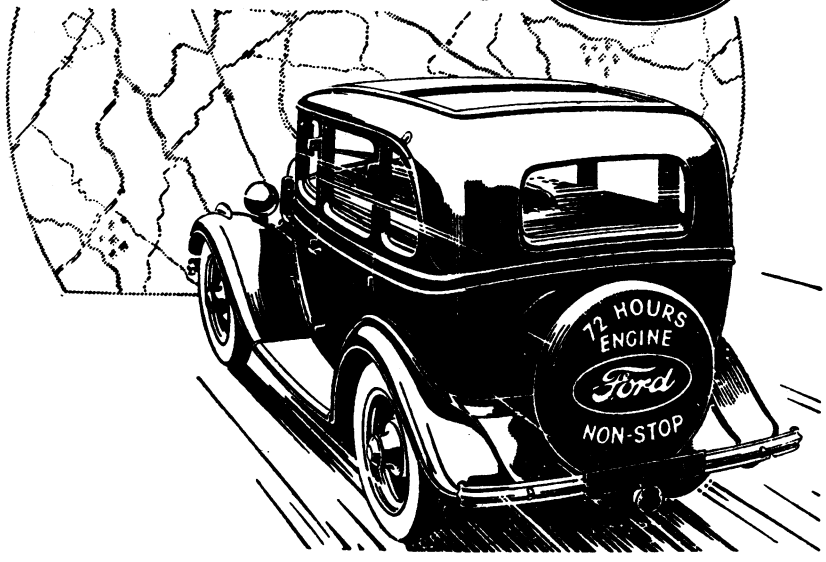

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The following books have been presented by Mrs. Campbell :—Spence's "Surgery"; Kelly's "Surgical Diseases in Children"; Bryant's "Operative Surgery"; Hare's "Practical Diagnosis"; French's "Index of Differential Diagnosis"; Sahli's "Diagnostic Methods"; Crandon's "Surgical After-Treatment."

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CONTENTS

Fellows and Members of the Ulster Medical Society receive the Journal free
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DATES OF PUBLICATION

1st January, 1st April, 1st July, 1st October.

THE ULSTER MEDICAL SOCIETY

THE MEDICAL INSTITUTE,
COLLEGE SQUARE NORTH,
BELFAST.

Dear Sir (or Madam),

If you are not a member of the Ulster Medical Society, we would appeal to you to give the question of joining your consideration. The Society has been in existence since 1862, and has always been active in keeping its members interested in the advances in medical science as well as in current professional affairs. The Medical Institute, situated in College Square North, belongs to the Society (through the generosity of Sir William Whitla), and is ideally adapted for meetings, committee meetings, and recreaton. There is a library with current medical periodicals, and facilities for reference to medical literature are available in conjunction with the library at the Queen's University. There is also a billiards-room available to members, and lighter periodicals are also provided. An annual dinner is held each year in December, and a golf competition in June. Meetings are held at intervals of a fortnight during the winter months, and papers are contributed by members. Distinguished visitors are occasionally asked to contribute papers on subjects upon which they are specially qualified to speak. THE ULSTER MEDICAL JOURNAL, the official organ of the Society, is issued to all Fellows and Members free of charge.

The subscription to the Society is one guinea for Fellows and Members living in the country; two guineas for Fellows living in Belfast; and one guinea for Members living in Belfast who are not qualified more than seven years. The payment of a sum of twenty guineas entitles one to election to Life Membership.

May we, therefore, appeal to you to join the Ulster Medical Society, and so enable us to widen its influence and sphere of usefulness still further? For your convenience a proposal form is attached, which, if filled in and sent to the Honorary Secretary, will ensure your name being put forward for election to membership of the Society.

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We remain,

Yours faithfully,

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J. A. SMYTH, *Hon. Secretary.*

F. M. B. ALLEN, *Hon. Treasurer.*

.....19.....

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and the anterior leg brought down first. If there is any difficulty in doing this, have the patient placed in the left lateral position, which encourages the presenting part to fall away out of the pelvis, and so gives more room. After bringing the arms down, the delivery is completed in the ordinary way. Smellie's method is the one most commonly employed.

Maternal complications may be classified as follows :—

1. *Lacerations.*—Extensive vaginal and perineal tears may be minimized by doing an epidiotomy at the beginning of the delivery. The laceration tends to occur when bringing down the arms and legs.

2. *Retained placenta, post-partum hæmorrhage, and obstetrical shock.*—It is not uncommon to get this complication, as the patient has usually had a long labour and a fairly long anæsthetic. After the placenta has been expressed or removed manually, the usual treatment for post-partum hæmorrhage should be commenced : hot douche at 118°F., pitourin ½ c.c. plus ergot aseptic (P. D. & Co.) 1 c.c.; raise foot of bed; warmth; morphia ¼ gr.

In addition to these routine measures, the intravenous injection of 50 to 100 c.c. of fifty per cent. glucose is good treatment. It should be given slowly.

Carmine 3 to 5 c.c. may be given intravenously if deemed necessary, or 3 c.c. of caffine sodium benzoate. If the patient has lost a large amount of blood and needs fluid, one pint of gum saline, or one pint of 5 per cent. glucose, should be given.

Fœtal complications may also occur. The baby may need to be resuscitated. All vigorous methods are contracted, as the baby is shocked. It should be wrapped up in a warm blanket and handled as little as possible. The air passages should be cleared. Brandy may be applied to the lips and gums. Carmine ½ to 1 c.c. may be given intra-muscularly or intra-cardially. CO_2 may be given to stimulate respiration, or a mixture containing ninety-three per cent. oxygen and seventy per cent. carbon-dioxide : with this it is impossible to give an overdose of CO_2. Lobeline by injection is sometimes used. Hot baths at 105°F. may be tried.

J. W. Peatt, *Hon. Secretary.*

Railway Street, Lisburn.

APPOINTMENTS

W. Waring Bassett, M.B., F.R.C.S.I., has been appointed Chief Medical Officer to the Lurgan Infirmary.

P. T. Crymble, M.B., F.R.C.S.Eng., has been appointed Professor of Surgery, Queen's University, Belfast.

H. P. Hall, M.B., M.Ch., has been appointed Visiting Surgeon to the new Dufferin Hospital, Belfast Infirmary.

H. P. Malcolm, M.B., M.Ch., has been appointed Lecturer in Operative Surgery, Queen's University, Belfast.

C. J. A. Woodside, M.B., F.R.C.S.I., has been appointed Lecturer in Applied Anatomy, Queen's University, Belfast.

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Ulster Medical Society.

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1932.

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Richard Ross, M.D.	1876–77
George F. Wales, M.D.	1877–78
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Robert F. Dill, M.D.	1879–80
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James Cuming, M.A., M.D.	1881–82
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Robert F. Dill, M.D.	1883–84
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Sir Alexander Dempsey, M.D.	1890–91
Henry O'Neill, M.D.	1891–92

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1896	FULLERTON, ANDREW, C.B., C.M.G., M.D., M.CH., F.R.C.S.I., F.A.C.S. ..	8 University Square, Belfast
1913	FULTON, T. F. S., M.B., D.P.H. ..	Pasadena, Knock Road, Belfast
1920	GAUSSEN, C. L., M.B., B.CH.	Hollydene, Holywood
1919	GEDDES, STAFFORD, M.B., B.CH. ..	Ulsterville House Lisburn Road, Belfast
1924	GIBSON, T. JORDAN, M.B., B.CH., F.R.C.S., Ed.	District Hospital, Banbridge
1931	GIBSON, W. S., M.B., B.CH.	16 Elmwood Avenue, Belfast
1916	GILLESPIE, JOHN R,, M.A., M.D., D.P.H.	28 Knockdene Pk. South, Belfast
1931	GILLESPIE, G. A. M., M.B., B.CH. ..	Ballygawley, Co. Tyrone.
1932	GODFREY, W., M.B., B.CH., D.P.H. ..	4 The Mount, Belfast
1914	GRAHAM, N. B., M.C., M.B., B.CH. ..	Purdysburn Villa Colony, Belfast
1920	GRAHAM, W., M.B., B.CH. ..	Redwing, Antrim
1932	GRAHAM, N. C., M.B., B.CH., D.P.H. ..	Bacteriology Dept., Queen's University, Belfast
1912	GRAY, D., M.A., M.D. (T.C.D.), F.R.C.P.I., L.M.	170 York Street, Belfast
1919	GRAY, HAMPTON A., M.D., D.P.H., L.M.	4 Charlemont Place, Armagh
1912	GRAY, HAROLD, L.R.C.P. & S.I. ..	Ridgmount, Malone Rd., Belfast
1926	GRAINGER, ROBERT, M.B., B.CH ..	Donard, Comber

Elected.		
1914	GREER, H. L.H., M.B., B.S., F.R.C.S., Ed. D.P.H., F.C.O.G.	8 College Gardens, Belfast
1921	HADDEN, E. M., M.B., B.CH.	Magharee House, Portadown
1921	HADDEN, WINIFRED E., M.B., B.CH., D.P.H.	Magharee House, Portadown
1928	HADDEN, R. E., B.A., M.B., B.CH. ..	Magharee House, Portadown
1914	HALL, H. P., M.B.,M.CH.	39 Wellington Park, Belfast
1919	HALL, HUGH E., M.D.	57 University Road, Belfast
1919	HALL, ROBERT (Jun.), M.B., B.CH. ..	1 Royal Terrace, Belfast
1930	HALL, T. S., M.D.	28 Fitzwilliam Street, Belfast
1903	HANNA, HENRY, M.A., M.B., B.SC. ..	30 University Square, Belfast
1932	HAYDEN, W. R., M.D., ...	100 Cliftonville Road, Belfast
1925	HEAZLEY, H. D., M.B., B.CH.	16 Cregagh Road, Belfast
1923	HENEY, J. W., M.D., D.P.H.	2 Mount Charles, Belfast
1922	HICKEY, EILEEN M., M.D., D.P.H. ..	485 Ormeau Road, Belfast
1912	HILL, ROWLAND, B.A., M.D., M.R.C.P., Lond. ; F.R.C.S., Ed.	17 Malone Road, Belfast
1921	HOGG, GERALD, M.B., B.CH.	26 University Square, Belfast
1913	HOLMES, T. S. S., M.B., M.CH., F.R.C.S., Eng., F.C.O.G.	5 College Gardens, Belfast
1931	HUEY, D., L.R.C.P., F.R.C.S., Ed., L.A.H.	Bella Vista, Bushmills, Co. Antrim
1921	HUNTER, R. H., M.D., M.CH., PH.D. ..	20 Haypark Avenue, Belfast
1907	HUNTER, S. R., M.D.	Rosemount Terrace, Dunmurry
1928	HUNTER, W. M., M.B., B CH. ..	Camcairn, Crumlin, Co. Antrim
1929	HUSTON, W. A., L.R.C.P. & S. ..	Greyabbey, Co. Down
1896	IRVINE, HUGH, L.R.C.P. & S.	Lonsdale, Strandtown, Belfast
1904	IRWIN, S. T., B.A., M.B., M.CH., F.R.C.S., Ed.	29 University Square, Belfast
1921	IRWIN, T. R. V., M.B., B.CH., D.P.H. ..	44 Cromwell Road, Belfast
1931	IRWIN, E. H., M.D., C.M., F.R.C.S., Ed.	Castleblayney, Co. Monaghan
1917	JAMISON, D., Jun., M.D., M.A.O., D.P.H., L.M.	Gt. Francis Street, Newtownards
1932	JAMISON, J. N., M.D.	40 Shankill Road, Belfast
1913	JEFFERSON, F., M.B., B.CH.	17 Wellington Place, Belfast
1920	JOHNSTON, J. G., M.C., M.D.	46 Castle Street, Lisburn
1923	JOHNSTON, J. A. L., M.D.	19 Clarendon St., Londonderry
1897	JOHNSTONE, R.J., B.A., M.B.,F.R.C.S., Eng., F.C.O.G., M.P.	14 University Square, Belfast
1931	KEAN, T. A., M.D.	9 Lower Crescent, Belfast
1926	KENNEDY, J. A., B.A., M.D.	Islay View, Portstewart
1913	KENNEDY, THOMAS, L.R.C.P. & S.I. ..	10 University Square, Belfast
1922	KERR, T. J., L.R.C.P. & S.I.	119 Gt. Victoria Street, Belfast
1914	KEVIN, R. G., M.D., D.P.H.	Wilmont Pl., Lisburn Rd., Belfast
1927	KIDD, CECIL W., M.B., B.CH.	180 Lisburn Road, Belfast
1912	KIDD, LEONARD, B.A., M.D.	Westbridge, Enniskillen
1911	KILLEN, S. J., M.D., D.P.H.	High Street, Carrickfergus

Elected.		
1910	KILLEN, THOMAS, B.A., M.B., F.R.C.S., Eng.	Arranmore, Larne, Co. Antrim
1890	KILLEN, W. M., B.A., M.D., M.CH., M.A.O.	32 College Gardens, Belfast
1931	KINLEY, D., M.B., B.CH.	31 Hughenden Avenue, Belfast
1890	KIRK, T. S., B.A., M.B., B.CH.	21 University Square, Belfast
1931	KYLE, J. T., M.B., F.R.C.S., Ed. ..	24 Ormskirk Street, St. Helens, Lancs.
1931	LANGSTAFFE, COL. J.W. D.S.O., L.R.C.P.I., L.M.	Royal Victoria Hospital, Belfast
1920	LEE, J. S. J., M.B., D.P.H.	121 Crumlin Road, Belfast
1931	LENNON, W., M.D.	5 Queen's Elms, Belfast
1932	LEVY, LAURA, M.B., B.S., L.M. ..	Beechwood, 70 King's Rd., Knock
1921	LEWIS, J. T., M.D., B.SC., M.R.C.P., Lond.	25 College Gardens, Belfast
1930	LEWIS, MRS. IDA, M.B., D.P.H. ..	25 College Gardens, Belfast
1932	LINTON, WALLACE, M.B., B.CH. ..	71 Woodvale Road, Belfast
1911	LOUGHRIDGE, J. C., L.R.C.P. & S., Ed.	Whitewell, Co. Antrim
1920	LOUGHRIDGE, J. C. (Jun.), M.B., B CH.	Templepatrick, Co. Antrim
1931	LOUGHRIDGE, J. S., M.D., B.SC., F.R.C.S., Eng.	52 Elmwood Avenue, Belfast
1928	LOWRY, H. C., M.B., F.R.C.S., Ed., M C.O.G.	3 Fitzwilliam Street, Belfast
1913	LYTTLE, G. G., M.B., B.S. M.R.C.S., Eng., L.R.C.P., Lond.	1 Windsor Park, Belfast
1921	MACAFEE, C. H. G., M.B., F.R.C.S., Eng., F.R.C.S.I., F.C.O.G.	18 University Square, Belfast
1931	MAGEE, EVELINE F., M.B., B.CH., D.P.H.	11 Crumlin Road, Belfast
1913	MALCOLM, H. P., M.C., M.B., M.CH. ..	27 College Gardens, Belfast
1913	MARSHALL, ROBERT, M.D., F.R.C.P.I., D.P.H.	9 College Gardens, Belfast
1931	MARTIN, J. C. M., L.R.C.P. & S.I., L.M.	Duncreggan, Portrush
1916	MARTIN, WILLIAM, M.B.E., M.B., Ed. ; D.P.H., J.P.	Promenade, Whitehead
1929	MAYRS, E. B. C., M.D., D.P.H. ..	Belair, Windsor Avenue, Belfast
1931	MILLER, S., D.S.O., M.C., M.A., M.D., B.SC., M.R.C.P., Lond., D.P.H. ..	1 Wordsworth Crescent, Harrogate, Yorks.
1902	MILROY, T. H., M.D., Ed., B.SC., LL.D., F.R.S., Ed.	Queen's University, Belfast
1914	MILROY, J. A., M.A., M.D.	Queen's University, Belfast
1898	MONYPENY, H. J., M.D.	Glastry Villas, Antrim Rd., Belfast
1909	MOORE, R. L., L.R.C.P. & S., Ed. ..	Redcliffe, Bangor, Co. Down
1909	MURPHY, H. A. J. S., L.R.C.P. & S.I., L.M.	45 Castle Street, Lisburn
1931	MURRAY, J. J., M.B., B.CH., ...	Ardnagale, Downpatrick
1931	MURRAY, P., L.R.C.P. & S., Ed. ..	118 Up. Newtownards Rd., Belfast
1911	MACKENZIE, W. R., L.R.C.P. & S., Ed. F.C.O.G.	6 University Square, Belfast
1922	MCCAW, IVAN H., M.B., B.CH. ..	10 College Gardens, Belfast
1913	MCCLOY, J. M., M.D., D.P.H.	15 Wellington Park, Belfast
1914	MCCOMB, S., M.B., B.CH.	Albertville, Crumlin Rd., Belfast

Elected.		
1913	McComb, S. Wilson, M.B., B.Ch. ..	Salisbury, Antrim Road, Belfast
1913	McConnell, R. J., M.B., M.Ch. ..	28 University Square, Belfast
1910	McCready, Wiclif, M.B., F.R.C.S.I. ..	20 College Gardens, Belfast
1926	McCready, Carlisle, M.D., ...	Skegoniel House, Shore Road, Belfast.
1916	McCulloch, R., M.B., D.P.H.	3 Queen's Elms, Belfast
1931	McDaniel, Eveline, M.B., D.P.H. ..	38 Ardenlee Avenue, Belfast
1932	McDonald, F., M.B., B.Ch.	Church Place, Portadown
1920	McDonald, Mark, B.A., M.B., B.Ch. ..	Portaferry, Co. Down
1899	McDowell, Robert, M.B., B.Ch. ..	98 Antrim Road, Belfast
1926	McFadden, G. D. F., M.B., F.R.C.S., Eng.	12 College Gardens, Belfast
1931	McKenna, T. H., M.B., B.Ch. ..	331 Crumlin Road, Belfast
1928	McKnight, Maud W., M.B., D.P.H. ..	256 Antrim Road, Belfast
1921	MacLaughlin, F. A., M.B., F.R.C.S., Eng.	67 University Road, Belfast
1931	MacLaughlin, J. N., M.D.	13 Queen Street, Londonderry
1904	McLorinan, Wm., L.R.C.P. & S.I. ..	103 Antrim Road, Belfast
1920	McMaster, H., L.R.C.P. & S., Ed.; D.P.H.	Ashbourne, Strandtown, Belfast
1915	McNabb, H. Russell, L.R.C.P. & S.I.	147 Donegall Street, Belfast
1919	McNeill, Mrs. Marjorie, M.B., D.P.H.	56 Wellington Park, Belfast
1920	McSorley, F., M.D., M.R.C.P.I. ..	Marinella, St. James' Pk., Belfast
1913	Nelson, M. K., M.R.C.S., Eng.; L.R.C.P., Lond.	Ballylesson, Belfast
1930	Nicholson, J. C., L.R.C.P. & S., F.R.C.S., Ed.	Hamilton Road, Bangor
1894	Nolan, M. J., L.R.C.P. & S.I., L.M. ..	Down County Mental Hospital, Downpatrick
1930	O'Connor, E. F., B.A., M.B., B.Ch. ..	Bessbrook, Co. Armagh
1906	O'Doherty, J., L.R.C.P. & S.I., F.R.C.S.I.	Windsor Gdns, 37 Malone Road, Belfast
1927	O'Prey, H. J., M.B. B.Ch.	Elsinore, Antrim Road, Belfast
1921	Patrick, N. C., B.A., L.R.C.P., M.R.C.S.	Ministry of Home Affairs, Belfast
1906	Patton, W., M.B., Ch.B., Ed. ..	86 Easton Street, High Wycombe, Bucks
1920	Peatt, J. W., L.R.C.P. & S., Ed. ..	14 Railway Street, Lisburn
1924	Pedlow, T. B., B.A., M.B., B.Ch. ..	61 Market Street, Lurgan
1920	Picken, S. E., M.C., M.B., B.Ch., D.P.H.	7 Richmond Crescent, Antrim Rd., Belfast
1929	Price, Muriel G., M.B., B.Ch. ..	31 Cyprus Avenue, Belfast
1914	Purce, G. R. B., M.C., M.B., M.Ch., D.P.H., F.R.C.S., Ed.	11 College Gardens, Belfast
1931	Poston, R. I., M.D.	Thorncliff, Failsworth, Manchester
1900	Rankin, J. C., M.D.	75 University Road, Belfast
1931	Ramsay, Mary F., M.B., D.P.H. ..	29 Cliftonville Road, Belfast
1902	Reid, Robert, L.R.C.P. & S., Ed., J.P.	Brookvale, Whiteabbey, Co. Antrim
1919	Ritchie, H. J., M.B., L.A.H., Dub. ..	257 Shankill Road, Belfast

Elected.		
1921	ROBB, ELIZABETH M., M.B., B.CH. ..	44 Ulsterville Avenue, Belfast
1920	ROBB, J. C., M.D., M.CH.	Infirmary House, Downpatrick, Co. Down
1932	ROBINSON, M., M.B., B.CH.	70 Up. Richmond Road, Putney, London, S.W. 15
1922	ROBERTSON, KATHERINE O., M.D., B.SC., (Glas.)	Main Street, Limavady
1925	ROSS, LESLIE N., M.B., B.CH. F.C.R.S., Eng.	King Edward Memorial Hospital, Ealing, London.
1931	ROSENBERG, M. L. M.B., B.CH.	20 Crumlin Road, Belfast
1923	RUTHERFORD, E.D., M.B., B.CH. ..	85 University Street, Belfast
1931	SCARLETT, GEO., M.B., B.CH.	74 Dublin Road, Belfast
1932	SCOTT, T. B. H., M.B., B.CH., Ed. ..	Avonmore, Antrim
1922	SEYMOUR, A. C., M.B., B.CH.	37 University Road, Belfast
1930	SHANE, W. B., M.B., B.CH.	Bengal Place, Lurgan
1931	SHANNON, W. A., B.A., M.B., B.CH., L.M.	Lodge Road, Coleraine
1924	SIMMS, S., M.D., B.SC., M.R.C.P.I. ..	235 Antrim Road, Belfast
1932	SINTON, D., M.B., B.CH.	Forster Green Hospital, Belfast
1920	SKILLEN, H. A., M.B, B.CH.	169 Duncairn Gardens, Belfast
1922	SMYTH, J. C., L.R.C.P. & S. I., L.D.S.	16 College Gardens, Belfast
1901	SMYTH, MALCOLM BRICE, B.A., M.B., B.CH., (T.C.D.)	20 University Square, Belfast
1932	STACK, H. T., O.B.E., B.A., M.B., B.CH., D.P.H., D.M.R.E. (Cantab), L.M.	2 Clarendon Street, Londonderry
1902	STEVENSON, HOWARD, B.A., M.B., F.R.C.S.I.	14 College Gardens, Belfast
1897	STEWART, J. HAMILTON, M.B., B.CH.,	198 Antrim Road, Belfast
1928	STEWART, H. HILTON, M.D., M.R.C.P., Lond	18 Malone Road, Belfast
1931	STEWART, HALL, M.B., B.CH., D.P.H. ..	Downings, Randalstown
1909	TAGGART, W. J., M.B., B.CH.	411 Antrim Road, Belfast
1905	TATE, T. M., B.A., M.D., Dub. ..	Killief, Strangford
1914	TAYLOR, D. R., B.A., M.B., B.CH., D.P.H.	20 Malone Road, Belfast
1894	THOMSON, R., M.B. B.CH.	Kelvin House, Botanic Av., Belfast
1909	THOMSON, W. W. D., B.A., M.D., B.SC., D.P.H., F.R.C.P., Lond.	25 University Square, Belfast
1910	THOMPSON, JOHN, M.D., ...	5 Dunelin, Malone Road, Belfast
1916	TRIMBLE, A., M.B., D.P.H., J.P. ..	Tuberculosis Institute, Durham Street, Belfast
1915	TURKINGTON, S. I., M.D., D.P.H. ..	41 University Road, Belfast
1920	TURNBULL, M. H., M.B., B.CH. ..	180 Grosvenor Road, Belfast
1929	UNSWORTH, JOHN, L.R.C.P. & S.I. ..	111 Cliftonville Road, Belfast
1894	WALLACE, JAMES, L.R.C.P. & S., Ed.	Laven House, Knockahollet, Belfast
1927	WALKER, PERCY S., M.D., D.P.H. ..	MunicipalSanatorium,Whiteabbey
1917	WARNER, CHARLOTTE E., M.D. ..	Shimna Rd., Newcastle, Co. Down

FELLOWS (Continued).

Elected.		
1932	WARNOCK, J. M., M.D.	34 Francis Street, Newtownards
1919	WARWICK, J., M.B., B.CH.	Kimberley Gardens, Bloomfield, Belfast
1933	WATSON, ANNA, M.B., B.CH., D.P.H. ..	The Hamlet, Earlswood Road, Belfast
1925	WATSON, ANNIE K., M.B., B.CH. ..	Redlands, Bloomfield Av., Belfast
1931	WATSON, JOHN, M.B., B.CH. ..	District Mental Hospital, Londonderry
1926	WHEELER, JAMES R., M.B., B.CH., D.L.O., D.O.M.S., F.R.C.S., Ed.	6 College Gardens, Belfast
1933	WHITE, J. B., M.D.	The Hill, Dunmurry
1894	WILLIAMSON, J. D., M.D., M.CH., M.A.O., J.P.	Albertbridge Road, Belfast
1916	WILSON, W. R. E., M.B., B.CH. ..	262 York Street, Belfast
1906	WILSON, W. JAMES, B.A., M.D., D.SC., D.P.H. (Cantab.)	Queen's University, Belfast
1931	WILSON, LIEUT.-COL. M. O., M.B., B.CH.	c/o Glyn, Mills & Co., Holt's Branch Kirkland Ho., Whitehall, London, S.W. 1
1920	WOODSIDE, C. J. A., M.B., F.R.C.S.I. ..	8 Elmwood Avenue, Belfast
1921	WRIGHT, P. P., M.B., D.P.H., F.R.C.S., Ed.	28 Crumlin Road, Belfast
1926	YOUNG, PEARL MCGREGOR, M.B., B.CH.	Crag Royston, Cherryvalley, Knock, Belfast
1931	YOUNG, J. S., M.C., M.A., M.D., B.SC. ..	Sunnymount, Malone Park, Belfast

MEMBERS.

1932	ALLEN, J. S., M.B., B.CH.	Mental Hospital, Antrim
1931	ATKINSON, F., M.B., B CH.	219 Springfield Road, Belfast
1930	BASSETT, W. W., M.B., B.CH.	Bawnmore Road, Belfast
1930	BAXTER, JAMES S., M.SC., M.B., B.CH., F.R.C.S.I.	110 Eglantine Avenue, Belfast
1929	BIGGART, J. H., M.D.	103 The Mount, Belfast
1928	BROWNE, J. W., M.B., B CH.	New Road, Donaghadee
1931	BOYD, J. D., M.SC., M.B., B.CH. ..	Greenisland, Co. Antrim
1932	BOYD, J., M.B., B.CH.	281 Ormeau Road, Belfast
1932	CALDER, A. M., M.B., B.CH.	12 Victoria Avenue, Newtownards
1928	CHISHOLM, H. M'B., M.B., B.CH. ..	Clare House, Ballyclare
1930	CLARKE, MAUDE, P. S., M.B., B.CH. ..	76 King's Rd., Knock, Belfast
1931	CORKEY, J. A., M.B., B.CH., F.R.C.S.I. ..	224 Lisburn Road, Belfast
1931	CRAIG, D. H., M.B., B.CH.	9 Waterloo Gardens, Antrim Rd., Belfast
1932	CRAIG, J. VICTOR, M.B., B.CH. ..	2 Manor Terrace, Cregagh Road, Belfast
1931	DEENY, J. A. D., M.D., B.SC.	22 Church Place, Lurgan
1932	DOHERTY, J., M.B., B.CH.	Abbey Hill, Whiteabbey, Co. Antrim

Elected.		
1931	DOWLING, CHAS. P., M.B., B.CH.	Church Place, Lurgan
1931	FORSYTHE, K., M.D.	40 Ranfurly Avenue, Bangor
1932	GETTY, J. H., M.B., B.CH.	156 Crumlin Road, Belfast
1930	GILLESPIE, J. H., M.B., B.CH.	16 Knockdene Park S., Belfast
1928	GIBSON, MRS. EILEEN, M.B., B.CH.	The District Hospital, Banbridge
1930	GLASGOW, A. D., M.B., B.CH.	Conway Square, Newtownards
1932	HAMILTON, J., M.A., M.B., B.CH.	52 Balmoral Avenue, Belfast
1931	HASLETT, T. B. H., M.B., B.CH.	5 Sharman Road, Stranmillis, Belfast
1932	HUSTON, R. J., M.B., B.CH.	Governor's Place, Carrickfergus
1931	JENKINS, C. R., M.R.C.S., Eng.; L.R.C.P. Lond.	Campbell College, Belfast
1929	KANE, F. F., M.D., D.P.H.	Fever Hospital, Purdysburn
1929	LINDSAY, H. C., M.B., B.CH.	Royal Victoria Hospital, Belfast
1932	LOWE, H. P., M.B., B.CH.	44 Crumlin Road, Belfast
1932	MONYPENNY, A. M'C. D., M.B., B.CH.	2 University Square, Belfast
1932	MORTON, W. R. M., M.B., B.CH.	42 Rugby Road, Belfast
1932	MORIARTY, J. J., M.B., B.CH.	77 South Parade, Belfast
1931	MUIR, A. IRENE, M.B., B.CH.	10 Lansdowne Road, Belfast
1930	MCCLENEHAN, J. MARTIN, M.B., B.CH.	"Commedagh," Newcastle, Co. Down
1928	MCCLURE, H. I., M.B., B.SC., F.R.C.S., Ed.	Elmwood Terrace, University Rd., Belfast
1929	MCCULLAGH, G. P., M.D., B.SC.	1 Dunelin, Malone Road, Belfast
1931	M'COLLUM, J. K., M.B., B.CH.	39 University Road, Belfast
1932	M'DANIEL, J., M.B., B.CH.	1 Wilmont Place, Lisburn Road, Belfast
1932	M'KINNEY, J. B., M.B., B.CH.	Bridge Street, Antrim
1928	MCNEILL, JANE L., M.B., B.CH.	Ravarnet, Lisburn
1932	MCVICKER, J. A., M.B., B.CH.	419 Antrim Road, Belfast
1927	NEVIN, S., M.D., B.SC.	Ardheim, Newtownards
1932	NORTHEY, J. H., M.B., B.CH.	Innisfail, Helen's Bay, Co. Down
1928	PRICE, J. A., M.B., B.CH.	31 Cyprus Avenue, Belfast
1931	POLLOCK, GRACE K., M.B., B.CH., D.P.H.	Lisbreen, Larne Harbour
1930	RODGERS, S., M.B., B.CH.	174 Albertbridge Road, Belfast
1928	SMITH, GEORGE, M.D.	Inisreen, Larne Harbour
1930	STRAIN, R. W. M., M.B., B.CH., B.SC.	9 University Square, Belfast
1932	TAYLOR, J. M., M.B., M.CH., F.R.C.S., Ed.	The Infirmary, Lurgan
1931	WALLACE, R. A. M'C. M.B., B.CH.	Cuan View, Lisbane, Co. Down
1930	WATSON, WM. SCOTT, M.B., B.CH.	U.V.F. Hospital, Craigavon, Belfast
1930	WHYTE, J. N., M.B., B.CH.	39 Stranmillis Gardens, Belfast
1926	YOUNG, J. CAMPBELL, M.B., B.S.	Crag Royston, Cherryvalley, Knock, Belfast
1932	YOUNG, M. C., M.B., B.CH., D.P.H.	69 Myrtlefield Park, Belfast

Fellows and Members will please forward any corrections to the Hon. Sec.
(Dr. J. A. SMYTH, 23 University Square, Belfast.)

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Pædiatrics

By F. M. B. ALLEN, M.D., M.R.C.P.LOND.

THE use of the term pædiatrics is becoming recognized in this country, and is regarded as general medicine limited to a definite age period, and not to a special organ or set of organs. It is not a speciality comparable to ophthalmology or radiology, but its activities are directed towards the care of children in health and the cure of them when ill. Its field covers the care of the newborn, the feeding and regulation of the diet and habits of the infant, the immunization against certain specific infections as well as the prophylaxis of others, the recognition of children who, while not ill, are not well, and, lastly, the diagnosis, prognosis, and treatment of disease occurring in children. The field is, therefore, a fairly wide one, not so far-flung as general medicine, but much less circumscribed than, say, gynæcology or ophthalmology. It has been estimated that there are 346 diseases which are met with in children, and of these some twenty-nine are preventable, fifty-five are amenable to specific therapy of some form, and the remainder account for twenty-three per cent. of the mortality under fourteen years.

The Editorial Board of THE ULSTER MEDICAL JOURNAL has recognized the importance of the subject of children's diseases in issuing this special number, and, in giving such attention, is following the commendable lead of the various examining bodies and universities in instituting special lectures and examinations upon the subject. It must be confessed that, as in so many other instances in history, public opinion has been clamouring for more interest in the health and welfare of the child, for the prevention of disease amongst children, and for the better care of the infant. Ignorance and tradition, not to speak of superstition, have often been the guidance of therapeutics rather than that intelligent understanding which knowledge and experience alone can provide.

The number of child and infant patients seen by the general practitioner varies very much with his success in treating them and the favourable (or otherwise) reputation which he acquires as a result of his advice. With the least successful probably they constitute twenty per cent. of his total clientele, but with the more popular and knowledgable upwards of sixty per cent. of general practice may be

concerned with patients under fourteen years. The pædiatrician is essentially a general physician, although some would, and do include surgery in their pædiatric practice. The most useful and successful pædiatrician is evolved from a good general physician, just as the successful general practitioner must be a sound pædiatrician.

When one considers the enormous mortality in child-life which used to be accepted with a measure of equanimity, it is little wonder that intelligent public opinion has demanded attention, and while much has still to be achieved, we should recognize that a very great deal of success has already been attained. Infantile mortality has been reduced enormously in the past century. It is striking when we consider that two centuries ago in a foundling hospital, of 10,272 children admitted in twenty-one years, only forty-five survived! Most large cities have reduced their infantile mortality to figures below, or in the region of, 100 per 1,000, but there is a figure below which we shall never be able to reduce the toll of mortality, largely on account of unavoidable circumstances, of congenital defects and such-like misfortunes. When knowledge has reached its Utopia, and all are omniscient and undefiled, an absent infantile mortality may be a possibility!

Ante-natal care will reap its reward in better and more successful midwifery, in the prevention of much avoidable maternal mortality, morbidity, and permanent invalidism. It will also do much in reducing death and disability in the newborn, in preventing many still-births, and eliminating a certain amount of crippling and disability. The understanding of the mechanism of intracranial hæmorrhage due to mechanical causes (apart altogether from gross trauma), the treatment of hæmorrhage neonatorum, the proper methods of resuscitation of the asphyxiated by the use of oxygen and carbon-dioxide gas, all come within the province of the practitioner who is the family obstetrician. Where the accoucheur leaves off and the pædiatrician enters the field is demarcated by the delivery of a living child, but, by the very nature of the problems the two activities are complementary, and more often the obstetrician must be his own pædiatrician. Excessive moulding and consequent strain on the tentorium, or sudden release of a prolonged increased intracranial pressure by precipitate delivery in the second stage of labour, is probably the most important factor in the occurrence of intracranial hæmorrhage, and frequently is not suspected in the otherwise normal and carefree delivery.

Dietetic problems have received much scientific attention during the past twenty years or more by such workers as the Mellanbys, Harriet Chick, Helen Mackay, and many others. Vitamines have been recognized, tried, and lauded—but it is important to realize that such preparations are very potent, and over-dosage is a real danger, and may cause irreparable and possibly fatal alteration in the functions of the human body. Manufacturers often ascribe fields of usefulness to their preparations which are not justified and which may be actually harmful. Discretion and discrimination are necessary adjuncts which should be cultivated by the over-credulous, and it should be remembered that a grain of scepticism is not an unworthy component of healthy judgment. This is not to say that vitamines are unworthy subjects for our attention, but rather to emphasize that our use of them should be

confined to such conditions as they are scientifically proved to help. And when they are used they should be in such a form that they are present in that state nearest to their natural condition and be pure in quality. Lay members of the public talk glibly of various vitamine products, of acidosis and acidity as modern topics of the drawing-room, often with an amusing contradiction of ignorance and knowledge. Acidosis is looked upon by parents as a fashionable, if somewhat troublesome, abnormality for which the sovereign remedy is glucose. They cast aside the grandmotherly horror and condemnation of sugar and sweets for children, but are confused and confounded in supplying the valuable fat soluble vitamines as contained in their most natural state in cod liver oil, rich milk, butter and eggs with the use of sugar, so that their course of avoiding the Scylla of excessive fat is extremely difficult in their attempt to avoid sailing too near the Charybdis of excessive sugar.

Infection and immunity have attracted some of the best scientific work of recent years. Of great practical value are the Schick and Dick tests for the identification of children who are susceptible to diphtheria and scarlet fever. The active immunization of children by the use of prophylactic sera is a course which will receive the assent, encouragement, and possibly the compulsion associated with vaccination against smallpox. The immunizing of susceptible children is not difficult, and is free of severe reactions except in a very small minority (in whom some temporary upset may occur). For diphtheria prophylaxis three intramuscular injections are necessary, and for scarlet fever four or five are given. The two injections can be made concurrently, and the whole course extends over a period of five or six weeks. It is impossible, on account of the recent date of this development in the field of immunity, to forecast the longest durations of such immunity, but it is reliably estimated that it will last from twelve to eighteen years. Many advantages of such immunization are obvious among school children, as, apart from the absence of the actual diseases and their complications, the quarantine and isolation insisted upon with each school outbreak (particularly in residential schools) can be disregarded.

The removal of tonsils has been an important feature of preventive pædiatrics, so much so that routine tonsillectomy seemed to be accepted by a long-suffering public. Within recent years it is evident that the pendulum is swinging towards conservatism and that certain criteria must be present to justify the operation. Nature never intended them to be regarded as a redundancy, as there is a very useful function to be performed by their presence, a fact often proved by the aggravation of an existing minor ailment to a major illness by ill-warranted interference. When it can be shown that the tonsils are the focus of infection in rheumatic fever or endocarditis, accompanied by chronic cervical adenitis, when there is recurring otitis media, recurring acute follicular tonsillitis, or when they are enlarged to such a degree that breathing is obstructed, then, and then only, should tonsillectomy be performed.

An intensive campaign is being carried out to satisfy the general public's demand for milk of good quality which is clean and free from tubercle bacilli. Such a supply

is eminently desirable and should prove one of the most valuable means of eliminating the "white scourge." But the extreme importance of personal contact with open tuberculosis most not be lost sight of, as it may be the more important agent in the dissemination of the disease. A parent cannot help feeling aggrieved when money and convenience have been sacrificed in procuring a tuberculin-tested milk, and at a later date it is found that a maid, or a nurse, or even an elderly grandparent, who was suspected of nothing more than being "subject to chronic bronchitis," is found one day to have hæmoptysis or some other startling evidence of tuberculosis.

Rickets and scurvy may be avoided by the use of the appropriate vitamines, and it would appear that pellagra and xerophthalmia are also preventable. There is accumulating evidence that the supply of vitamine A raises the resistance to infection, and yeast as a source of vitamine B has a catalytic action upon iron and copper in anæmia. The absence of vitamine D from the diet of the growing infant so disorganizes calcium and phosphorus metabolism, that various changes occur at the growing ends of bones and in other tissues. Until recent years the richest source of fat soluble vitamines was the liver of the cod-fish, although salmon and halibut liver may contain one hundred times the amount of vitamine A present in cod liver oil. The standardizing methods of vitamines D and (particularly) A are far from satisfactory to the chemist, and the unwary may easily be misled by the portentous display of advertisements of commodities standardized according to units. The inhabitants of the Atlantic seaboard have used cod-liver oil as a remedy for countless years, and it was the winter substitute for milk and butter. Its virtues receive striking testimony in the recent autobiography of Halliday Sutherland, who records that in the Isle of Lewis, "children were kept in 'black houses,' windowless, chimneyless, earthen-floored, and verminous, and did not cross the threshold until they could walk . . . and yet rickets was unknown." Incidentally the infantile mortality of Lewis (1923) was 28 per 1,000 births. In the absence of sunlight even, rickets was absent, probably because the dietary contained abundance of raw or cooked fish liver as well as cod liver oil in its natural fresh state.

The feeding of infants is too often regarded as a maze of shrubbery too complicated to be traversed by other than the fully initiated. The journey was certainly never supposed to be taken by the doctor! Such a view is now happily passing and the family physician is expected to guide the mother on her course of feeding for the infant, and not merely as an individual to treat the baby when it is sick. It is a tribute to nature that so many infants survive the ministrations of so many ignorant and helpless parents and nurses, but it is also a tragedy that so many sleepless nights are spent by indulgent parents for the lack of even a little knowledge of a subject which is within the compass of the averagely intelligent with half an hour's study. The bogey that infant-feeding is a difficult problem is passing with a new generation of doctors, and with the increase in breast-feeding in all classes, less difficulties arise. Artificial feeding should be kept within the simplest possible formulæ, which will succeed with the great majority of infants. Various systems composed of special percentages of the constituents of milk with the

additions of nauseating creams and emulsions are foreign to nature's intentions, for breast-milk varies in most of its constituents from feed to feed, from day to day, and even during the feed. Variety in food, as in life, is essential to human comfort and well-being. Withal it is more difficult to teach a mother to feed her infant during the second year than in the first, and again, a simple principle underlies the success.

Psychology has a more important place in pædiatric practice than is realized. Hysteria is not confined to the adolescent maiden, because contractures and paralyses do occur in children, and dramatic results are obtained in young subjects with appropriate suggestion. Enuresis is largely a functional disorder in the majority of children, and it can be influenced by psychological means, except in those cases which are due in whole to some organic abnormality of the genito-urinary tract. In the mental "make-up" of the child, vanity plays an important rôle, and the conscious or subconscious desire to be "in the centre of the picture," to attract attention, or to recover a lost position of sympathy or love, often explains a refusal of some article of food, lack of sleep, bed-wetting, stammering, tics, and some other functional disorders. For the "difficult child" an intimate understanding of the workings of the child mind is not given to many, but it is all important. A knowledge of the world, a renewal of child memories, good judgment, and sympathy, will help more than textbooks or guides to normal behaviour.

A most careful examination and good understanding of the features of disease are essential to elucidate the case of the child who is not "just right," and who is not ill, but is not well. Such problems are being constantly set by parents who see their child from day to day, and who notice that energy and good spirits are lacking, that good nature and pleasant temper have gone, and that duties which were full of interest are now unpleasant. The early manifestations of tuberculous infection and of rheumatic infection have to be considered. Infection of the middle ear and of the urinary tract must be excluded. Chronic indigestion and constipation may be the cause, or dental caries may make proper mastication well nigh impossible, and so lead to bad hygiene and habits of "bolting" food. The radiologist, oto-rhinologist, and bacteriologist are therefore essential members of the team, and their experience and help are often necessary.

There still remains a field of activity upon which we are only on the threshold, namely, the work of child guidance clinics. Such require the intelligent co-operation of a large staff of visitors and inquisitors, who, by the nature of the investigations, necessitate an expense, which must be provided either by philanthropy, the municipality, or the state.

The end-result of all these outlets for interest in the rising generations, interest taken from ante-natal life to young manhood and womanhood, will be a definite improvement in the health and conduct of the infant and child, which will be reflected in the adult population of the nation : an improvement which will wholeheartedly refute the pessimists who fear for our future ability and stamina.

The Feeding of Infants

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A SOUND knowledge of the methods used in the correct feeding of infants can probably save the lives of more infants in the first twelve months of their existence than can any special knowledge of medicine or surgery. By far the most important cause of death in infants is some disorder of nutrition, and the importance of correct ideas regarding the subject of infant-feeding can therefore hardly be over-rated. The problem is not one solely of saving life during the perilous first year by avoiding such immediate dangers of improper feeding as acute indigestion, diarrhœal diseases, infantile atrophy, and rickets, but also of ensuring such a degree of general nutrition as will permit of healthy growth and the highest physical and mental development of which the individual organism is capable.

The infant requires the same food elements, namely, protein, fat, carbohydrate, salts, and those accessory food substances—the vitamins—as does the adult, but the forms and relative amounts of the food elements required by the infant differ from those required by the adult. One reason for this is the delicate structure of the organs of digestion in infancy and their inability to assimilate certain forms of food.

There can be no doubt in the minds of those with even a limited experience of infants that the ideal food for them is human milk. As one American pædiatrician, Brenneman, has put it : "The more fully one learns the fundamentals of the science, and the more nearly one masters the art of feeding babies artificially, the more one is impressed with the fact that to the young infant mother's milk is a true specific, and that during this early period, at least, artificial feeding is a substitute that necessity alone imposes upon us."

It is largely owing to the increase of breast-feeding since the war that the continuous decline of infantile mortality of recent years is due.

Every mother of a newly-born child should, in the absence of any of the extremely limited number of contra-indications, be considered capable of suckling her child, and it should be pointed out that there are many advantages to the mother herself in so doing, namely, (*a*) suckling aids in the proper involution of the uterus, (*b*) it is cheaper, (*c*) it is much less laborious than preparing artificial feeds, cleaning feeding-bottles, etc., (*d*) there is a lower sickness-rate amongst the breast-fed, consequently in the aggregate her child will require less attention.

Further, the advantages to the infant itself are of outstanding importance. It has a fivefold greater chance of survival during the first twelve months of its existence, and it is much less likely to contract rickets, scurvy, or spasmophilia; consequently in later life it will probably have a better physique and fewer bony deformities than the artificially-fed child.

The only two disadvantages of suckling fall upon the mother; it may interfere with her employment, and it will certainly interfere with the social duties of the modern "society woman."

There are only two absolute contra-indications to breast-feeding on the part of the mother, namely, active tuberculosis and malignant disease of the breast. Relative contra-indications are latent tuberculosis, puerperal insanity, mastitis, or labour complicated by severe hæmorrhage, eclampsia, or septicæmia.

In the infant, certain deformities, such as hare-lip or cleft palate, may interfere with sucking, whilst acute coryza, pulmonary atelectasis, congenital heart-disease, or the debility due to prematurity, may render sucking extremely difficult. In these cases the milk should be drawn from the breast by means of a pump and fed to the child by a dropper, or, better still, by introducing a No. 8 soft rubber catheter into the child's stomach and running in the meal through a funnel.

Certain ante-natal measures may assist subsequent lactation. A generous diet during pregnancy, containing milk, greenstuffs, and fruit, will supply a sufficiency of vitamins and salts, especially calcium, both for the mother and the fœtus *in utero*. In the case of the mother's nipples being unduly retracted, measures of correction should be undertaken at least two or three months before the expected delivery. The breasts should be bathed daily with cold water, and the nipples should then be drawn out, either manually or by the suction of a breast pump.

The diet of the nursing mother should, with certain modifications, follow the same lines as those during pregnancy. Milk, porridge, green vegetables, salads, fruit, and meat should be allowed freely, but tea, coffee, spices, and other stimulants should be partaken of sparingly. Daily exercise in the open air, sufficient sleep, and freedom from worry and excitement, are essentials.

Frequency of Feeding.—The child should be put to the breast for the first time twelve hours after birth, and thereafter every six hours, both breasts being used, for twenty-four hours. For the next two days the intervals should be shortened, so that by the fourth day the infant is having three-hourly feeds. At the end of four to six weeks four-hourly feeds should be instituted, and shortly afterwards the infant should go for eight hours during the night without being fed.

Notwithstanding the measures just mentioned being carefully observed, certain difficulties may arise, e.g.—

(1) *Delayed Lactation.*—An ordinary healthy infant has sufficient reserves to go for forty-eight hours after birth without food. The secretion of milk in the mother's breast may be delayed until the fourth or fifth day after parturition. The most potent stimulus for the breast secretion is provided by the sucking infant. The infant should therefore be put to the breast at the intervals previously mentioned, even though there be yet no milk secretion, but immediately afterwards it should be given a little warmed water. If by the fourth day there is no appearance of milk in the breasts, or if the supply be deficient, a small artificial feed, consisting of two parts of cow's milk to one of boiled water, should be given. The vigorous

sucking of the child when put to the breast and the complete removal of any little secretion that may have formed is a powerful stimulus to further milk flow.

(2) *Insufficient Milk Secretion.*—This should be suspected if a breast-fed infant is restless and peevish immediately after suckling. Undernourished infants are inclined to vomit after feeding, owing to excessive swallowing of air whilst at the breast. A test-feed should be immediately carried out. The child is weighed in his clothes immediately before and immediately after his feed. The difference in the weights will show the amount of milk received. The weighings should continue for a whole twenty-four hours. The average daily amount of milk that an infant receives from the breast in the first few months of its life is between eighteen and twenty-five ounces. If it be found that the breast-milk is deficient, the following measures for increasing it should be taken :—

(*a*) See that there is strong suction at the breasts at regular intervals.

(*b*) Make sure that the breast is absolutely emptied after each feed; any residue left being expressed or withdrawn by a breast-pump.

(*c*) Give the mother sweet malted drinks twice a day, e.g., Horlick's Malted Milk, Bourn-Vita, or Ovaltine.

(*d*) See that the mother is having regular meals of ample quality and quantity thrice daily.

(*e*) Promote optimism and freedom from worry on the part of the mother.

(*f*) Have the breasts massaged twice daily for ten minutes, and douched with hot and cold water alternately.

Should these measures fail to increase the supply of milk, then complementary feeding should be resorted to. This consists in giving the child a small artificial feed immediately after each breast-feed. Two parts of cow's milk to one of water, with a level teaspoonful of sugar to each two ounces of the mixture, will be found a suitable meal. If the feed be made too sweet, the child will cease to take the breast.

I desire to emphasize that with care nearly every child can be given its natural food; breast-feeding for a month is better than none at all, and one eminent authority has laid down the dictum, "In all cases of doubt decide against weaning."

Artificial Feeding.

If human milk is not available, the most satisfactory substitute is fresh cow's milk. Some infants can digest and thrive on undiluted cow's milk, but the majority of infants require to have their milk suitably diluted with water, owing to the indigestibility of the fat and casein of cow's milk. The fat of cow's milk has a higher melting-point than the fat of human milk, and as received in the home contains a higher percentage of volatile fatty acids than human milk, and to this is due the tendency of cow's milk fats to "sour" in the stomach. Casein forms eighty per cent. of the protein in cow's milk, whilst only thirty per cent. of the protein of human milk is casein. Casein, when acted upon by acids and rennin,

produces large curds, and infants fed on undiluted cow's milk are liable to vomit these curds or to pass them undigested in their stools.

The Question of Boiling Milk.—Unless "certified milk" is available, it is on the whole much wiser to boil all milk that is to be given to an infant. The advantages of boiling are that it sterilizes the milk and destroys any tubercle bacilli that are present, and secondly, it modifies the casein, so that this forms much smaller curds in the child's stomach, thereby being more digestible. The disadvantages are that the vitamins, and especially the anti-scorbutic vitamin C, are liable to be destroyed, that there is a loss of a certain amount of the lactalbumen present, this being coagulable by boiling, and, lastly, that boiled milk is less palatable.

In practice, however, the vitamins can be replaced by giving a teaspoonful of orange-juice once daily, and half a drachm of cod-liver oil twice daily, and it is usually found that infants will take boiled milk quite readily.

Amount of Milk Required.—When an infant is fed on the breast it requires two and a half ounces of milk per pound of body-weight per day. Owing to the relatively greater amount of protein in cow's milk, it has been found by experience that the infant only requires, to satisfy his protein requirements, one and a half ounces of cow's milk per pound of body-weight per day. This amount of cow's milk will, however, be insufficient for his carbohydrate requirements. It will therefore be necessary to add sugar to the milk. It will be found that one level teaspoonful of brown sugar per pound of body-weight per day will satisfy most artificially-fed infants, and will enable them to maintain a steady gain in weight. Lastly, the passage of a large amount of urine of low specific gravity is one of the physiological requirements of the normal infant; it will, therefore, be necessary to add sufficient water to the cow's milk to provide for the infant receiving two and a half ounces of fluid per pound of body-weight per day, i.e., one ounce of water per pound per day will be required.

It is possible to set out these requirements graphically, thus—Body-weight in pounds × (1½ oz. of milk + 1 oz. of water + 1 level teaspoonful of sugar) = Amount of food for twenty-four hours.

Cow's milk is definitely deficient in iron, and most infants fed on it ultimately show signs of anæmia. It is therefore advisable to administer iron and ammon. cit. in gr. ½ doses in solution, thrice daily, to all infants so fed.

Difficulties in Artificial Feeding.

Vomiting.—This may be due to the infant being unable to digest the fat in cow's milk. When such is the cause, the vomiting occurs a considerable time after feeding, and the infant brings up large curds. To overcome this difficulty, a low fat-content milk should be used. If a pint of milk be allowed to stand in a vessel for four hours, and then the lower twelve ounces be syphoned off, a milk containing only two per cent. of fat will be obtained; if the lower eight ounces be alone syphoned off, a milk containing only one per cent. of fat will be obtained.

If vomiting of considerable quantities occurs just after feeding (in the absence of pyloric stenosis), too much food has been given.

Constipation.—This is generally due to too small an amount of sugar in the food. Use may be made either of Lactose or Maltose (dextrin-maltose) instead of cane sugar, as they are definitely more laxative. Maltose is cheaper than Lactose, but it accentuates any tendency there may be to vomiting.

Diarrhœa.—This is most commonly due to infection of the bowel with organisms which ferment carbohydrate. When it arises, one should cut down the amount of sugar in the feeds and add additional protein in the form of casec, calcium caseinate, or protosol.

Dried Milks.

These are extremely useful for the artificial feeding of infants in the tropical countries where fresh milk is not available. They are also of use in this country if for any reason the usual daily supply of fresh milk has failed. Dried milks have this advantage : they are sterile and their composition does not vary. The destruction of vitamins by most processes of drying is not complete, and any danger of deficiency of these substances can be overcome by administering orange-juice and cod-liver oil. Examples of dried milks obtainable locally are Cow and Gate, Trufood, and Glaxo.

Acid Milks.

The use of milk which has been modified by the addition of a dilute acid to it will be found valuable in some conditions. It has been found that premature infants and infants suffering from fermentative diarrhœa, acute gastro-enteritis, and other infections, tend to have hypochlorhydria. Owing to the buffer action of cow's milk (by this is meant the capacity of the curd of milk to absorb into itself acid or alkali without changing its reaction to litmus), the small amount of hydrochloric acid in these infants' stomachs is quickly mopped up or absorbed, leaving no free H.Cl. to raise the gastric contents to the optimum acidity for normal peptic digestion. The practice of adding dilute acids to cow's milk in order to fill the buffer before it reaches the infant's stomach has been found of the greatest advantage since it was first adopted some ten years ago. Many dilute acids have been used, e.g., lactic, hydrochloric, citric acid, pure or in the form of lemon-juice, and acetic acid in the form of vinegar. The one most commonly used in the British Isles is acid lactici dilut. (B.P.C.).

A pint of milk is boiled for two minutes and then rapidly cooled. When cold, the acid is added, drop by drop, stirring after the addition of each drop, until a fine curd just begins to form. In summer it will probably be found that the curd begins to appear after the addition of about forty-five drops; in winter, sixty drops may be needed. The milk is then set aside in a cool place. The amount necessary for a feed can be withdrawn at any time, the necessary water and sugar added, and after warming to blood-heat can be given to the infant.

Some Observations on Respiratory Diseases in Childhood

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Congenital Pulmonary Atelectasis is, in mild degree, rather a common condition. The factors responsible for the collapse or non-expansion of portions of the infant's lung are unknown, and we are ignorant of the nature of the stimulus responsible for initiation of respiration in the new-born. Certain cases are probably due to bronchial obstruction by mucus or meconium. The symptoms are snuffling, noisy breathing, inability to suckle properly, and cyanosis or actual blueness—of an extent roughly proportional to the amount of lung collapsed. Convulsions are not unusual, and, when present, call for a gloomy prognosis. Some believe that the convulsions and the atelectasis are both caused by intracranial hæmorrhage, but recent post-mortem investigation[1] does not indicate any relation between intracranial trauma and pulmonary atelectasis. Treatment is unsatisfactory. The infant should be kept warm and disturbed as little as possible. Expectorants are of no value. Attempts at hyper-ventilation of the lungs with oxygen-carbon-dioxide mixtures are not only useless, but physiologically unsound. The use of the Drinker respirator has received favourable report in this condition.

Broncho-pneumonia may be primary, or it may be secondary as a complication of some infective or debilitating disease. Whooping-cough, measles, and rickets are frequent predisposing causes. In infants the systemic disturbance is often out of all proportion to the extent of lung involvement, as judged by physical signs. Lobar pneumonia is usually a primary condition, and, in contrast to broncho-pneumonia, often attacks strong, healthy children. Broncho-pneumonia runs an ill-defined and variable course, whereas the lobar variety is simply an outcropping of the underlying pneumococcal septicæmia, which may simultaneously localize in pleura, middle ear, brain, or meninges.

A quiet, airy room should be selected for the pneumonic patient. The air of the room should be cool, and the bed-coverings light. Many believe that pneumonia cases do better if nursed in the open air, but few have the courage of their convictions. In no other disease is good nursing so important. The diet should be limited to milk and glucose lemonade during the first few days of the attack. Purgatives are contra-indicated. Unless in older children, and for severe pleural pain, poultices are useless, and are an embarrassment to the already overtaxed respiratory apparatus. A mixture containing citrate and ipecacuanha is as useful as any. Atropine is often given for broncho-pneumonia in the hope of "drying up" the lung, but it is a general experience that the drug will dry up every part of the patient—except the lung. Oxygen should be available, and in severe cases I prefer to use it as a routine from the commencement. Its use will then produce no apprehension in parents or child, and the latter will readily co-operate. A mixture of five per cent. CO_2 in O_2 is best, and it should be given through a rubber bag and

face-mask for fifteen minutes in every hour. An equally efficient but extravagant method is to bubble oxygen through a Woulff's bottle containing warm water, and administer by nasal tube. The anti-infective vitamin (A) has interesting possibilities in treatment of pneumonia. Next to the liver, the lung is the most important store-house of this vitamin, which, among other functions, is responsible for maintenance of the normal structure and nutrition of mucous membranes. The prophylactic and curative influence of vitamin A in other septicæmia conditions has been studied from both experimental and clinical standpoints, and there are good *a priori* grounds for the suggestion that this substance should have a useful therapeutic rôle in pneumonia. Large doses of such preparations as avoleum and adexolin sometimes give surprisingly good results in pneumonias of infancy.

The utility of specific treatment has been greatly extended by the introduction of Felton's concentrated sera for cases infected with the pneumococcus (types I and II). Where facilities for typing the organisms are not available, a mixed concentrate may be injected in repeated doses. Type I and II infections account for about two-thirds of the number of cases seen in adults. Unfortunately, children show a much smaller proportion of type I and II infections, and a higher proportion of the "indeterminate" type IV infections. Anti-serum cannot, therefore, be so efficacious in children as in adults. Vaccine therapy is sometimes useful in treatment of lobar pneumonia. A small dose, say five to ten million mixed stock pneumococci, if injected hypodermically in the initial stage of the disease, will sometimes abort it. In nearly all cases stimulants are called for, and best of these is coramine. Digitalis is often given in order to ward off cardiac failure, and its exhibition should be begun early in the course of the illness.

EMPYEMA is a common complication of pneumonia, particularly the fibrinous type. Small circumscribed collections of pus are often very difficult to diagnose or locate, and physical signs may be frankly misleading. In a child, slight dullness, increased breath-sounds, and coarse crepitations are occasionally found over a small empyema; larger exudates will generally give more definite dullness with loud bronchial or tubular breath-sounds. If the effusion be very extensive, the classical signs are present—displacement of the cardiac dullness, with loss of movement, resonance, and breath-sounds over the affected side of the chest. Irregular pyrexia with a rising leucocyte count is suggestive, and exploratory puncture will usually settle the question. Unfortunately, a failure to strike pus on repeated needling does not rule out the possibility of its presence. It is often impossible to distinguish between unresolved pneumonia and empyema, unless radiographic evidence is available.

With regard to treatment, all agree that the liquid should be evacuated, but there are two methods in vogue :—

1. Repeated aspiration, using Potain's or Poynton's apparatus.
2. Thoracotomy and drainage, with or without removal of one or more rib sections.

Late statistical investigation proves that the end-results of the open operation are as good as those obtainable by the more troublesome newer methods. Thoracotomy is based on sounder surgical principles, and the advantages of the aspiration treatment are more apparent than real. In streptococcal empyema, however, it is advisable to aspirate on several occasions before finally incising the pleura. This allows time for the thoracic muscle planes to become walled off before pleural drainage is carried over them.

PULMONARY FIBROSIS is a troublesome sequel of acute or chronic inflammatory lung disease. Pneumonia and bronchitis, especially when complicating influenza or whooping-cough, are very liable to lead to fibrosis. A common symptom is a series of colds in the chest, which never clear up completely, leaving a slowly progressive residuum of fibrosis. The physical signs are flattening and deficient expansion, harsh breathing with prolonged expiration, and coarse crepitations. Signs of consolidation supervene later. When bronchial dilatation and suppuration complicate the picture, finger and toe clubbing, cyanosis, dyspnœa, and pyrexial bouts make their appearance. Chronic bronchitis is an unsatisfactory diagnosis in a child, and many children so labelled are really suffering from a low-grade bronchiectasis or bronchiolectasis. These lesions should be suspected when a child ceases to thrive and has a chronic loose cough (children never expectorate unless taught to do so). Fœtid breath and the toxic signs enumerated above confirm the diagnosis. The extent of lung damage can be best appreciated by radiography after the introduction of lipiodol into the trachea. The outlook depends upon whether the disease is stationary or progressive, and whether it is unilateral or bilateral in distribution. In unilateral cases the disease can be dealt with fairly satisfactorily, but unfortunately there is a definite tendency for the sound lung to become bronchiectatic later.

An essential point in treatment is the eradication of septic foci from the nose, sinuses, teeth, and tonsils. The presence of inhaled foreign bodies, e.g., fishbones, tooth fragments, must be excluded by X-ray examination. The patient should be placed on a fresh-air routine as in pulmonary tuberculosis. Rest and exercise are prescribed according to the presence or absence of fever. Postural drainage is a useful method of emptying the cavities of retained secretion. (The patient is made to lie with his head and shoulders hanging over the edge of his bed several times daily.) Autogenous vaccines are sometimes of service in allaying the toxæmia. For unilateral cases, phrenic evulsion and artificial pneumothorax will often produce marked alleviation of the symptoms. Thoracoplastic operations produce a more effectual and permanent lung-collapse, and are useful in cases where the lung is extensively diseased, or where relapse has occurred in spite of pneumothorax treatment.

RECURRENT BRONCHITIS is most prevalent amongst children of junior school age, and is responsible for much loss of school time. Many cases are secondary to unhealthy conditions of the upper respiratory tract, and removal of diseased tonsils or adenoids usually produces swift and lasting improvement in the chest condition. A prolonged change of air and food will often break the cycle of bronchitic attacks when all other treatment fails. More is to be gained by general tonic treatment than

by dosing the child with expectorants. Cod liver oil, iron, and hypophosphites are all of service. Vaccines, either autogenous or stock, are of assistance in some cases. Pneumococcal vaccines should be used with great caution, however, as they appear to sensitize occasional patients, instead of immunizing them. For the acute attack, a simple expectorant mixture, enriched with a few grains of iodide, has no equal.

Certain cases of recurrent bronchitis are allergic in nature, and careful observation may connect the attacks with exposure to particular proteins, e.g., grass pollen, strawberries. Treatment aims at excluding the offending protein. In case of ubiquitous proteins like pollen or milk, exclusion is virtually impossible, and an attempt must be made at immunizing the patient by graduated dosage with the protein in question. In practice it is more usual to resort to non-specific immunization with peptone. It is certain that many cases are due to dietetic errors, and excessive carbohydrate intake is a most potent cause, and by no means an uncommon one, now that the glucose habit has become so fashionable. Child asthmatics present many interesting problems, and it is not unusual to find evidence of other allergic manifestations—urticaria, eczema, gastro-enteritis, etc., in the patient, or in other members of the family. Some cases seem to have their origin in an acute infection of the lung, such as bronchitis or pneumonia. It is not unusual to find a history of hyper-sensitiveness to some commonplace article of diet, and I have recently seen two cases where the asthmatic attacks were provoked by eating potatoes. The percentage of asthmatic children giving clear-cut skin reactions to foreign proteins varies enormously, according to the technique and enthusiasm of different workers. Probably not more than ten per cent. give definite and unambiguous skin tests. Such patients may be desensitized by a course of injections of the toxic protein, or they may be instructed to shun their particular poisons. Some asthmatic children give positive tuberculin tests; here tuberculin should be used as the desensitizing agent. I have found this a valuable remedy, even in children with negative skin tests. Maxwell[2] considers that there is a certain optimal dosage for each patient. This is gauged by the extent of the patient's Mantoux reaction. He emphasizes one important consideration, namely, that the courses of tuberculin should be lengthy. Weekly injections should be given for six months, followed by fortnightly injections for a like period.

Intrathoracic Tuberculosis of children is a daily problem with most practitioners. Within the past decade it has been recognized that characteristic childhood lesions occur. These lesions, as contrasted with adult lesions, show a much greater tendency to healing. This "first infection" of childhood renders the patient allergic or immune. A second infection during adult life will act as reactant, producing all the toxic signs of tuberculosis. There are two important portals of infection, the alimentary and the respiratory. In the former, the infection is usually milk-borne, and the resulting disease is abdominal or osseous in localization. In the latter case intrathoracic tuberculosis results. Blacklock,[3] in a recent investigation, found that ninety-seven per cent. of fatal cases of thoracic tuberculosis were infected with the human type of bacillus tuberculosis, whereas in fatal abdominal cases 19.6 per cent.

were infected with human, and 80.4 per cent. with bovine organisms. These figures prove that thoracic tuberculosis rarely follows entereal infection, despite many ingenious experiments tending to prove the contrary. That twenty per cent. of abdominal cases show human types of infection is not surprising when we consider the facilities available for contamination of foodstuffs with tuberculous sputum. The "seed" is therefore of much greater importance than the "soil."

The 'first infection' is usually a small broncho-pneumonic focus, which may be situated in any part of the lung. Infection and enlargement of the related intra-pulmonary and tracheo-bronchial lymph-glands is an immediate sequel, and at this stage the child's skin reaction becomes positive. If the resistance is poor, miliary spread through the lung parenchyma may occur, giving a fatal form of tuberculous pneumonia, or dissemination may follow, and the case terminate as tuberculous meningitis. If the child's resistance is more substantial, the original focus soon becomes quiescent, and no evidence will remain save a fibrous or calcified scar in the lung parenchyma, recognized radiographically as a Ghon's tubercle. Evidence of intrathoracic gland enlargement will often persist for years after the lung focus has healed. Post-mortem evidence suggests that tuberculous infection may spread from the intrathoracic glands upwards to the superficial and deep cervical lymphatics, and occasionally downwards to the abdominal lymphatics.

There is still some dispute as to whether adult cases of tuberculosis are due to "flaring" of childhood lesions, or to infection *de novo*. Consideration of the evidence outlined above, and of other important facts, such as the difference in localization between adult and childhood tuberculosis, forces us to the conclusion that adult infection usually means reinfection, and not merely recrudescence.

The following is a rough clinical classification of intrathoracic tuberculosis in children :—

1. Miliary, pneumonic, and broncho-pneumonic types.
2. Resolving parenchymal lesions.
3. Hilar or mediastinal adenopathic type.
4. Pleural type.
5. Adult type.

The pleural type has the same course and significance as in adult life, and requires no further consideration here.

The broncho-pneumonic and pneumonic types are found most frequently in infants, and in children up to the age of three years. They usually simulate ordinary pneumonia, and the true nature of the disease is suspected only when resolution and defervescence fail to make their appearance. Tuberculin reaction and radiographic evidence will settle the diagnosis. Search of the stomach washings and fæces often reveals the presence of bacillus tuberculosis.

Clinical evidence proves that even massive pneumonic tuberculosis may recede, and of late much has been written in pædiatric literature regarding the resolving

pulmonary tuberculosis of childhood, or "epituberculosis." Here the main features of the attack are the rapid development of intense pulmonary infiltration and consolidation. Pyrexia, toxæmia, positive cuti-reaction, and the isolation of tubercle bacilli from material obtained by lung puncture, are also characteristic. These cases resolve in a most dramatic fashion, and very little in the way of scarring remains. It has been suggested that a large area of non-specific inflammation and gelatinous œdema may form round a small tuberculous focus, and the term "perifocitis" has been tentatively adopted for the condition in pursuance of this view.

The adenopathic syndrome is very common in Northern Ireland. Here the damage to the lung parenchyma is comparatively insignificant, whereas the hilar glands remain large for an indefinite period afterwards. It is evident that no particular symptom complex can be attributed to the gland condition alone, and the description "adenopathic" allotted to this group is anomalous. If, however, the true pathology of the disease be borne in mind, the term "gland type" is a convenient clinical label. In this connection it is worth remembering that hypertrophy of the tracheo-bronchial lymphatics occurs in diseases other than tuberculosis. For example, they are constantly enlarged after lung infections such as bronchitis, pneumonia, and pertussis, while lymphatic leukæmia and Hodgkin's disease are general causes.

The symptomatology is very variable, the commonest complaints being lack of energy and failure to gain weight; anorexia, irregular pyrexia with night sweats, cough, and anæmia also are frequent. Diagnosis by clinical examination is admittedly difficult; at best the presence of enlarged intrathoracic glands may be detected, but no criteria of activity or latency can be elicited. The typical phthisical shape of chest may be observed. A tendency to "winging" of the scapulæ and the presence of a downy growth of hair on the back are suspicious. Dilated superficial veins on the upper thorax were found in thirty-seven per cent. of Fishberg's cases.[4] Dullness on percussion over the hylar areas—corresponding to the interscapular areas posteriorly, and to the first and second interspaces adjacent to the sternal margin anteriorly—is a useful sign, but one which can be appreciated only if the percussion stroke be light and precise. The breath-sounds are usually increased in intensity, with exaggerated expiratory murmur, but localized areas of bronchial breathing may be found, mainly in the interscapular and upper axillary regions. D'Espine's sign is difficult to interpret, and on the whole unreliable. Enlargement of the cervical glands is a concomitant in a fair proportion of cases. Radiographic examination together with the tuberculin reaction affords definite diagnostic evidence. The Mantoux intradermal tuberculin reaction is the simplest, safest, and most accurate of all methods. Human old tuberculin is diluted 1 :100, 1 :1000, and 1 :10000 as required, with a half per cent. phenol saline. After cleansing a patch of skin on the arm or forearm with spirit, .1 c.c. of the 1 :10000 dilution is injected into the skin through a fine hypodermic needle, so as to form a small weal. The test is read in seventy-two hours. "A positive reaction consists of an area of erythema or erythematous infiltration, the greatest diameter of which equals or exceeds 5 mm."[5] The infiltration is more important that the erythema. If negative or feeble positive,

the test is repeated, using the next stronger tuberculin, until the series has been given without reaction, or a definite positive has been obtained.

In an infant or young child a positive tuberculin test has great diagnostic significance. In older children the test becomes less useful, but on the other hand, a negative in an older child is of decided assistance. In "fulminant" cases of tuberculosis the skin reaction may fail to develop. In spite of these limitations, the skin reaction is an indispensable diagnostic measure.

Children over the age of eight years tend to develop the adult type of tuberculosis, i.e., subapical infiltration, tending to caseation and cavitation. In American literature this has been somewhat inaptly dubbed "the 'teens type of tuberculosis." Some of these show a healed lesion of the Ghon type, as well as the presumably second infection giving rise to subapical adult focus. The outlook in such cases is that pertaining to adult types of infection in general. The prognosis in childhood tuberculosis is, as already indicated, good. The adenopathic children do very well, and cases of the resolving type heal with surprisingly little lung scarring. The younger the child, the greater the danger of dissemination. The miliary types are, of course, desperate risks, but occasional recoveries occur.

The question of whether an infected child is likely to develop serious adult lesions later must be left an open one. It was formerly believed that bone or glandular tuberculosis indicated good resistance, and, conversely, it is a commonplace clinical observation that patients showing pulmonary tuberculosis are often free from evidence of osseous or gland involvement. It is possible that the immunity produced by such attacks is valuable, and to some extent enduring. Fishberg[6] summarizes present-day views on the subject thus :—

"Early tuberculous disease of the tracheo-bronchial glands is not necessarily followed by phthisis in later life, and there seems to be valid evidence that it may serve as an immunizing agent, as is the case with nearly all extra-thoracic tuberculous lesions during childhood."

The question of treatment raises the whole issue of tuberculosis as a national and economic problem. Tubercle-free milk supply, and the avoidance of contact with cases of open phthisis, are prophylactics of prime importance for infants and children. The French are devotees of the Grancher system, where infants born to tuberculous parents are promptly removed to healthy country homes. Tubercle carriers should be isolated in sanatorium, hospital, or colony. Another method of approach to the problem is the mass dosage of the infant population with an avirulent live culture of bacillus tuberculosis, such as the B.C.G. strain. This method has been given very extensive trial on the Continent. Calmette[7] states that the attenuation of his B.C.G. is hereditarily fixed, and that it possesses definite antigenic properties. He believes that none of the accidents which have attended its use can be rightfully attributed to the vaccine. On the contrary, he finds that the mortality from all causes in children up to one year old is only half as high in the B.C.G.-vaccinated infants as in those not so vaccinated. Calmette's conclusions have not found general acceptance, but the situation is promising, and possibly

mass immunization with strains of this type may in future prove a valuable weapon in the fight against tuberculosis.

When exposure has already occurred, the contacts should be subjected to clinical and radiographic examination, and the skin reactions done. These investigations are repeated at regular intervals, and children showing signs of infection are given prompt and prolonged treatment. It is helpful to distinguish two classes of reactors. Firstly, those who are the subjects of "tuberculous infection," and have a positive tuberculin reaction but no demonstrable lesions. These require observation. Secondly, we have the group of sufferers from "tuberculous disease"; these are reactors who have definite lesions, and require sanatorium routine, or a stay at a "sunshine school," according to the extent of their lesions.

The treatment of acute miliary and pneumonic types of tuberculosis can be summed up as absolute rest in bed, fresh air, and such aids to nutrition as cod liver oil. Collapse therapy is sometimes a life-saving measure, and gold treatment is credited with some brilliant cures.

In the adenopathic cases, fresh air, rest, and general hygienic measures are important. When the pyrexia subsides, graduated exercise should be ordered. Recurrence of fever is an indication for a slowing up of the exercise programme. Tonics such as syr. ferri iodid. and cod liver oil emulsion encourage a gain in weight. A salt-free diet and calcium intramuscularly are of undoubted benefit in almost all cases. In the early stages sunlight and artificial sunlight will often provoke undesirable reactions. This treatment must be ordered with great circumspection until the patient's tolerance expands, when, as a rule, the child may literally live in the sun. A prolonged holiday in the country or at the seaside will usually rehabilitate the patient's health and strength. Children of fortunately circumstanced parents should winter in Switzerland, or on the southern French or Italian seaboard, where it is possible to escape the respiratory infections engendered by our rigorous winter climate. Open-air schools should be available for children of artizan parents. Pending Utopia, a not inferior physical salvation can be worked out for these children by intelligent treatment on a farm, or even in a backyard of a city terrace.

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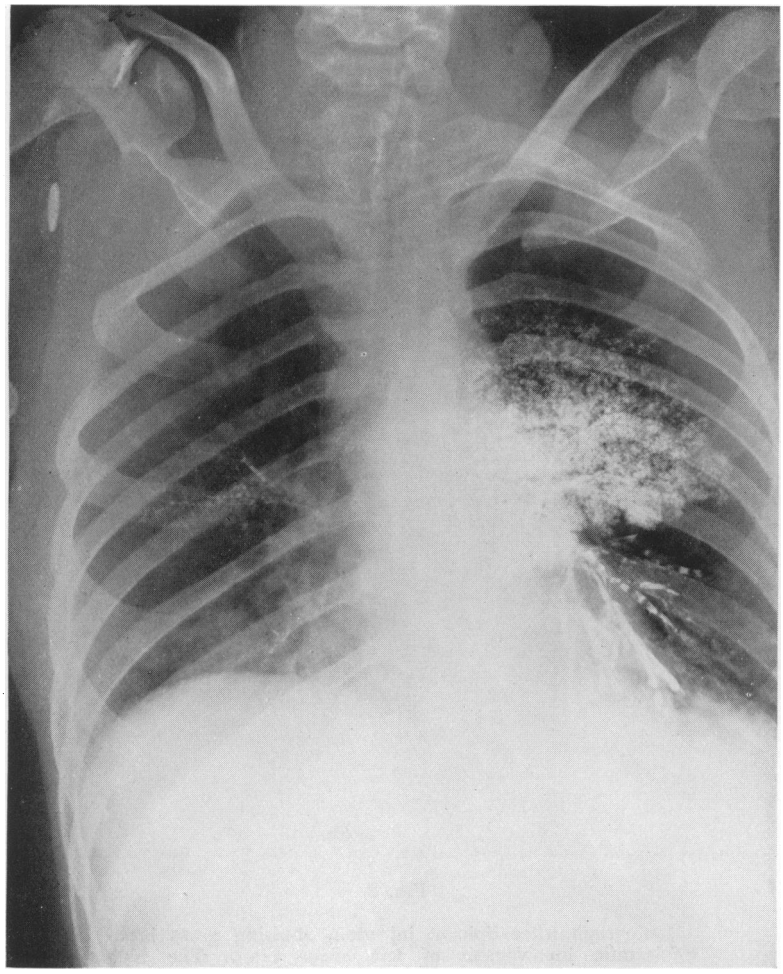

FIG. 1.

Radiograph after injection of lipiodol into trachea, showing bronchiolectasis of left base. Left middle zone shows well the appearance of normal lipiodol-filled lung.

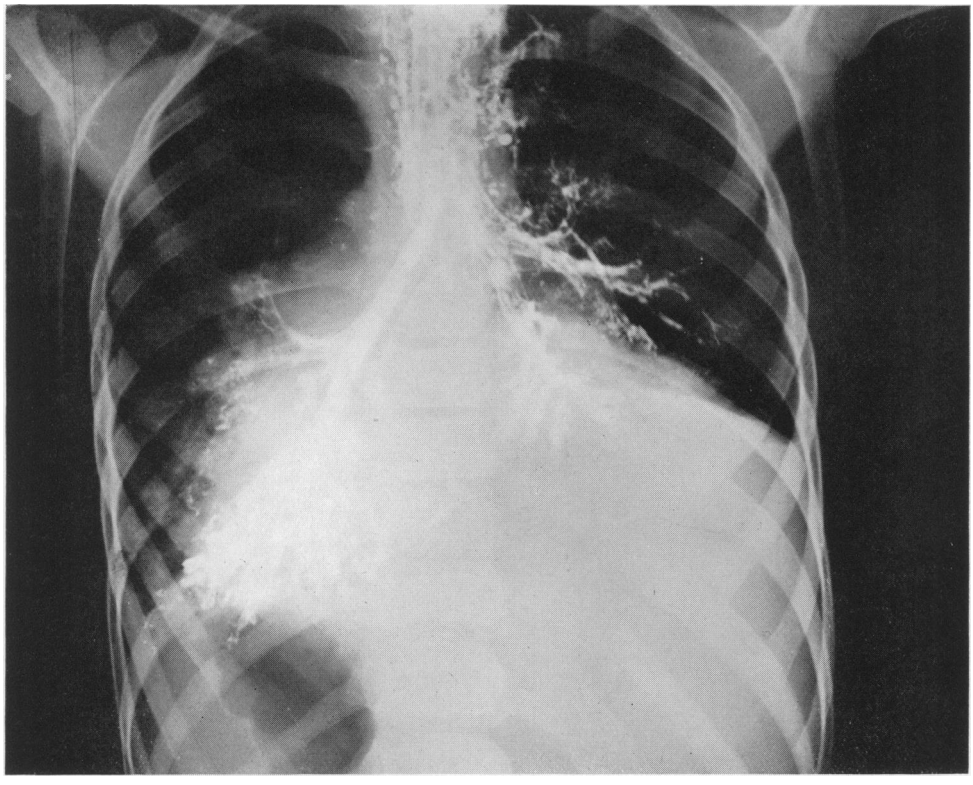

FIG. 2.

Radiograph after lipiodol injection, showing gross bronchiectatic involvement of left lower lung. The high diaphragm on the right side is due to phrenic evulsion done two years ago. In spite of the resultant lung collapse the dilated bronchi can still be seen.

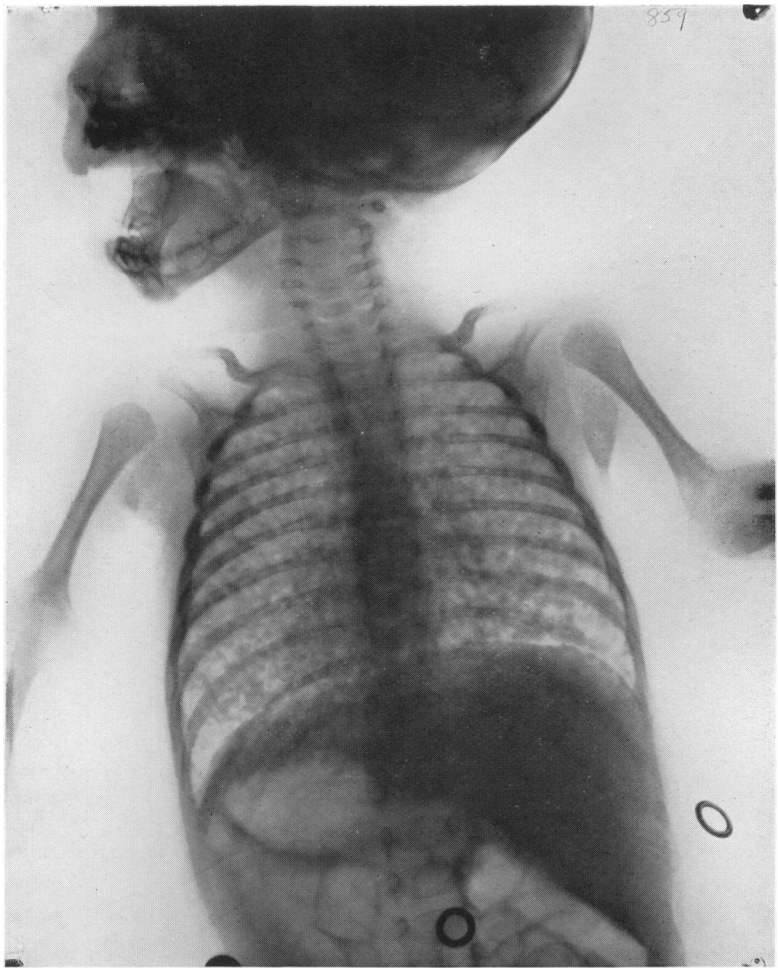

FIG. 3.

Radiograph showing widespread miliary tuberculosis.
(*Courtesy of Dr. R. M. Beath.*)

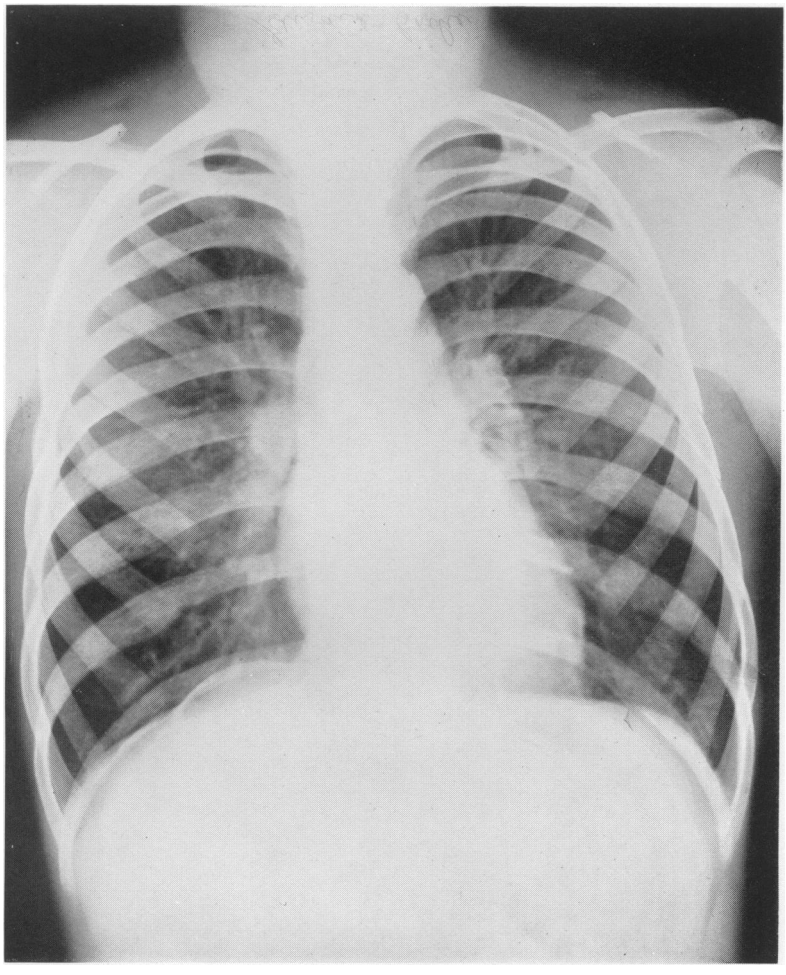

FIG. 4.

Childhood tuberculosis. Gland masses are visible at both hylar regions, also involvement of the adjacent lung tissue.

The Outlook for the Rheumatic Child

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THE problem of the future of the rheumatic child depends on various factors, the value of many of which is difficult to assess. In the first place, the presence of the rheumatic toxin is frequently difficult to prove : as Mackie[1] has said, "Rheumatism is a chronic and often progressive disease characterized by alternating periods of activity and quiescence, and the age at onset plays a dominant rôle in determining its clinical phenomena" : in the young child the early clinical phenomena may be at first so ephemeral that the presence of established carditis may be the first positive sign of its presence; there is, as yet, no serological test comparable with the Wassermann or tuberculin tests, and therefore the diagnosis rests on a purely clinical basis, as syphilis formerly did, but with, in many cases, less florid stigmata. It is interesting in this regard to note that, having educated the public to regard growing pains as rheumatic, there is a tendency amongst pædiatricians[2] to deny that growing pains are ever rheumatic. Personally I do not agree with this modern view.

Secondly, it is difficult to know whether the toxæmia is inactive. Smith and Sutton[3] suggest the following criteria of probable inactivity :—

(1) That the child is symptomless.

(2) That the temperature has been continuously normal for at least two weeks.

(3) That the leucocyte count is normal.

(4) That the pulse-rate is "fairly stable."

(5) That the weight is increasing.

(6) That the vital capacity is increasing.

Possibly the most informative pulse-rate is that obtained while the patient is asleep. In my experience, considerable patience is needed to obtain reliable spirometric readings of vital capacity in children.

To these may be added the more obvious points of facial complexion, tongue, appetite for food, and, possibly most important of all, inclination for exercise. The tendency to sit about rather than go out of doors is, to my mind, almost proof of toxæmia.

Intimately associated with this problem of the presence and the activity of toxæmia is the coincidence of foci of infection. Without reopening this vexed question, one may agree with Mackie's statement that "chronic foci of infection are more frequent in rheumatic cases than in normal children; appropriate treatment of these foci seems to reduce, but not to remove, the incidence of recurrences." One may also note that children with even severe valvular disease can stand tonsillectomy and its necessary anæsthesia with minimal risk. I have failed in

almost all my cases to elicit a history of an attack of tonsillitis occurring as a precursor of acute rheumatism as recorded by Schlesinger,[4] Sheldon,[5] and others.

Thirdly, it is difficult in some cases to say positively that a heart has escaped involvement; here the time-factor is important, at least in acute rheumatism: as Sir William Gull[6] pointed out in 1869, "there is evidence tending to show that it is part of the natural course of rheumatic fever for the heart to become diseased during the first few days of the fever, and if it does not then become diseased, it rarely does so in the second, third, or later weeks of the fever. We would here venture to observe that in acute rheumatism care is requisite in determining during the first week whether the heart is really diseased or not." But Sir William went on to add that "in nearly all the cases referred to in this paper there was some modification of the heart's sounds during some period of the rheumatic attack," usually the appearance of a bruit which, in some cases, soon subsided, but in others was the precursor of a definite murmur.

In his description of the auscultatory changes accompanying endocarditic invasion, Vaquez[7] writes as follows: "In the beginning the sole characteristic sign consists in muffling of the sounds of the heart, pointed out by Bouilland. It affects either the aortic or the mitral valve, notably the latter, lasts for a variable time, and is replaced by a ringing accentuation, the tabourka (Turkish drum) sound of Potain. Then if the endocarditis recovers, the sounds resume their normal characters, or in the contrary case, change to murmurs. The murmurs symptomatic of valvular lesions appear from the twelfth to the thirtieth day."

The detection of early carditis is therefore a comparatively simple matter in the case of acute articular rheumatism. In the child the arthritis is so slight, and its nature so often unrecognized, that it is only very rarely that one can trace the commencement of auscultatory change.

Possibly the greatest of all our difficulties is when we try to foretell what will happen to the child who shows signs of carditis. If his murmur is purely systolic, it may disappear completely; it may persist throughout life without any symptom or any other signs of cardiac disability; it may be the precursor of severe mitral or aortic disease, and the first indication of myocardial failure.

As a practical exercise in the consideration of the prognosis in young rheumatic subjects, I have recently sent a circular letter to 160 such patients, who were under my care for varying periods during the years 1923-30 at the Ulster Hospital for Children and Women, asking them to report for re-examination, or failing this to answer the following questions:—

(1) Have you suffered from any attacks of rheumatism or chorea (St. Vitus's dance) since you last attended hospital? If so, please say how many attacks and how long each lasted.

(2) Have you now any symptoms of heart trouble—palpitation, shortness of breath, or pain?

(3) Are you at present undergoing medical treatment?

As will be seen from table 1, seventy-five patients reported in person, forty-eight by letter or message conveyed by a parent or other person, seventeen did not report and could not be traced, and twenty had died.

In table 1 I have recorded the age and sex distribution in the various groups, and I have endeavoured to tabulate the various types of rheumatic infection according to each patient's history; I may say that the grouping into acute and subacute rheumatism is, of necessity, somewhat arbitrary, as the majority of these children were not seen at hospital during their first rheumatic attack.

In table 2 the cases are arranged according to the evidence of valvular lesions for each of the four groups.

Table 3 is based on the classification adopted by the American Heart Association, which is as follows:—

Class I—Cases of organic heart disease without undue fatigue, palpitation, dyspnœa, or pain.

Class II—Patients with organic heart disease unable to carry on ordinary physical activity without discomfort: (*a*) activity slightly impaired; (*b*) activity greatly limited.

Class III—Patients with heart failure at rest.

Class IV—Patients with possible heart disease (symptoms or signs referred to heart, but in whom the diagnosis is uncertain).

Class V—Cases of potential heart disease, i.e., where there is the presence of an ætiological factor such as rheumatism or chorea.

Table 4 shows the results of electro-cardiographic examinations in 136 of the 160 patients.

Group A.

This was much the most interesting group, as one had an opportunity to re-examine these patients after an average period of six years since their first attendance at hospital.

Reference to table 2 will show that twenty-three had no demonstrable valvular lesion, and in eight cases a mitral systolic murmur recorded in the notes had apparently disappeared. This was gratifying, but, on the other hand, in three cases cardiac lesions were found in patients who had been classed as normal when first seen: one (A. B.) had developed mitral regurgitation after repeated attacks of chorea; another (R. B.), a choreic, had developed aortic regurgitation and mitral stenosis during an attack of acute rheumatism; while a third (L. R.), employed as a waitress, complained of dyspnœa, and had developed a mitral leak in a heart which had been regarded as normal five years before. As well as these, no fewer than seven cases of mitral regurgitation had developed mitral stenosis, and three "mitral" cases had developed aortic lesions.

It is frequently possible to observe the transition from mitral regurgitation to mitral stenosis in an individual case: the apical murmur becomes longer in duration and is frequently followed by the so-called "reduplicated" second sound;

when heard at the apex of the heart this double sound consists, as Vaquez[8] states, of the second heart-sound followed immediately by the "opening snap" of the mitral valve; (true reduplication of the second sound, due to asynchronism of aortic and pulmonary valves, is only heard at the base of the heart). Apparent reduplication at the apex is most commonly an early sign of mitral stenosis, but is occasionally present in normal subjects: in such cases Smith and Sutton say that "the most plausible explanation is sudden stretching of the chordæ tendinæ of the mitral valve by blood rushing through the mitral orifice and tensing the chordæ before the ventricle contracts." As the stenosis develops, the second pulmonary sound intensifies, the pulse-volume lessens, and there appears the characteristic murmur, "the only sign justifying a diagnosis of mitral stenosis," which one may learn to know, as Lewis[9] says, "as one learns to know a dog's bark."

Three of these seventy-five patients had had pericarditis while in hospital: of these only one had recovered with a clinically normal heart.

One patient had developed chronic nephritis following an acute attack; one had tuberculosis of the lungs; and a third was found to be under treatment for congenital syphilis, which raised the obvious doubt as to whether she ever had rheumatism!

Thirty patients were working, and of these twenty-one had found suitable employment, while nine had drifted into occupations involving relatively heavy work.

Almost all looked well, and were considerably over the average height and weight for age.

Of this group thirty-one, or 41.3 per cent., had apparently escaped carditis.

I formed the opinion that in about two-thirds of the cases medical supervision and treatment had been reasonably adequate during the earlier years, but usually no doctor had been consulted after the age-limit for attendance at a children's hospital or school clinic had been reached.

It is noteworthy that only nineteen of the seventy-five admitted, on questioning, any cardiac symptoms whatever.

There are, I think, two reasons for this: first, that frequently after puberty a period of inactivity of the rheumatic toxæmia is reached; to what extent medical treatment, nursing, and maternal care are responsible for this I am not prepared to say, but I do claim that all three are of the utmost importance to the rheumatic child; second, that the cardiac muscle of the adolescent is capable for years to deal with the overload resulting from stenosed or incompetent valves. This freedom from symptoms is in itself a source of danger: not only because rheumatic recurrence is always possible, but because the patient, oblivious of his risk, undertakes work and play for which he is unfit. It is from girls of this type, with symptomless or almost symptomless mitral stenosis, that are recruited the tragic young wives with heart failure in pregnancy.

This comparative efficiency of the heart-muscle as a whole—in spite of rheumatic invasion occurring as part of a generalized carditis—is confirmed by the relatively

large number of cases in which a normal electro-cardiogram is recorded by an obviously diseased heart.

Group B.

Information regarding forty-eight patients was obtained by their replies to the questionnaire, or by the attendance of a relation at hospital. In twenty out of forty-eight no valvular lesion had been found, and in three a transient apical lesion had been found, and in three a transient apical systolic murmur had disappeared during the patient's attendance at hospital. Of the remaining twenty-five, all of whom had been diagnosed as having disease of the heart, only six admitted symptoms : four of the six have mitral stenosis and the other two have mitral and aortic lesions. Of the latter, one, T. S., was almost continuously under observation at the hospital from 1922, when, at the age of six years, he had severe aortic and mitral regurgitation, until 1930, when he refused to allow his mother to bring him back; during these years he had been repeatedly admitted with congestive heart failure, usually following some boyish exploit such as swimming in the Victoria Park pond. For some time he was a rivet-boy in the shipyards, and is now almost as precariously occupied in the street-cleansing department. This case is an exception to the statement that a child will never take exercise that will do him harm.

Classified according to the American scheme, this group had even better results than group A, as only six (12.5 per cent.) are classified under category II, and none in III. It is probable that twenty-three (47.9 per cent.) have, so far, escaped carditis.

Group C.

These seventeen cases could not be traced. Their numbers would have been larger if it had not been for the kindness of Dr. Thomson and the City Hall nurses, who were instrumental in rounding up a considerable number of patients into groups A and B, and in some cases finding out details of those who had died. I should here like to express my gratitude to them. The original diagnoses are classified in tables 1 and 2.

Group D.

Of the twenty patients now known to be dead, the duration of life from the time of their first attendance at hospital until death is known in fifteen cases, and averaged three years and four months. In many cases one is surprised at the length of time an apparently moribund cardiac cripple can live. All except four had gross cardiac lesions when first seen at hospital, and were immediately admitted as urgent cases. Of these four, two had severe and recurrent chorea, one had a six-months history of pains in hands, knees, and back, followed six years later by acute rheumatism, and the fourth had severe chorea and limb pains.

Electro-Cardiographic Examinations.

The electro-cardiographic findings are stated in table 4. Forty per cent. showed flat or inverted T in lead 3. This is a larger percentage than Shookhoff and Taran[10] found in normal children (24.8 per cent.), but on the other hand, Parry[11] found inversion of T wave in fifty-four per cent. of normal children. It was a remarkably constant finding in these children, and was unusually present in repeated records. In four of the five cases in group A where T_3 became upright in later records, it was coincident with clinical improvement. Inversion of T wave in leads 1 and 2 was seen only once, in a boy with progressive mitral stenosis. Inversion of T in leads 2 and 3 was seen in a girl with persistent tachycardia; in a later record T_2 had again become upright—no valvular lesion was detected. "Left ventricular predominance" was found in fifteen cases, sometimes when unexpected, as in two cases of mitral stenosis, and two cases where neither valvular lesion, cardiac enlargement, or arterial hypertension could be elicited. "Right ventricular predominance" was more rarely found, and like enlarged or notched P most often in association with mitral stenosis.

A P-R interval of more than 0.2″ was only recorded once. Shookhoff and Taran say that the P-R interval in healthy children averages 0.125″, and Parry gives 0.142″, 0.148″, and 0.146″ for leads 1, 2, and 3 respectively, so probably 0.2″ is not a true normal standard for children as it is in adults.

Distortion of QRS is commonly confined to lead 3, and is not regarded as being of pathological significance, but in one case of rheumatic mitral stenosis, in a youth now eighteen years old, who is working as a pastrycook's labourer, repeated electro-cardiograms from the age of eleven years have shown distortion of QRS in all three leads; this raises the question as to whether rheumatic lesion is superimposed on a congenital one. As Seham and Shapiro[12] have stated, "one of the uses of electro-cardiography in children is that it may help to distinguish a congenital lesion from acquired heart disease."

Gertrude Nicholson[13] writes: "In the electro-cardiogram of a cardiac child, startling differences from the normal are not to be constantly expected, though fine changes such as decrease in the amplitude of deflections and slight prolongations of transmission-time are not rare and are frequently overlooked. Thus a typical clinical mitral stenosis or aortic lesion will frequently show no right or left ventricular predominance in the electro-cardiogram, and often disappoints physicians who want positive evidence." This confirms my own impressions during the past ten years at the Ulster Hospital.

Prognosis.

The outlook for the rheumatic child is necessarily serious. If the figures quoted in the short series may be taken as a criterion, 101 (or sixty-three per cent.) have already developed cardiac disease, and twenty (or one in eight) are known to be dead. Of these eighty-one, the prognosis is certainly grave in the thirty-two cases of mitral stenosis and the fourteen cases of aortic disease.

The fifty-five cases in whom a diagnosis of mitral regurgitation has been made present a more uncertain problem. For them, as for the fifty-nine in whom there is, as yet, no obvious lesion, but who live in the shadow of probable recurrence, the outlook depends on many factors: the extinction of the "smouldering fire" of rheumatic infection, the uncertain workings of heredity, the amount of cardiac damage already done, the avoidance, if possible, of other infections, notably scarlet fever, which seems to have a grim affinity for the rheumatic heart, and the social position of the patient.

Amongst factors which are more or less within our control are the provision of rest, nursing, and medical care; the removal of infected teeth and tonsils; the adequate use of sodium salicylate, and of iron to combat the tendency to anæmia; the provision of suitable education and the supervision of games and exercises; in adolescence the choice of suitable employment, the avoidance of too strenuous sports; in adult life the avoidance of the misuse of alcohol and, in young women, of repeated pregnancies.

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TABLE 2.

CLASSIFICATION ACCORDING TO PRESENCE OF VALVULAR MURMURS.

	No Valvular Lesion	Transient Apical Systolic Murmur	Mitral Regurgitation	Mitral Stenosis	Aortic and Mitral Regurgitation	Aortic Regurgitation and Mitral Stenosis	Aortic Stenosis
GROUP A (reported in person).—75 Cases.							
(*a*) History of acute rheumatism -	0	3	4 (2 improved; 2 no change)	1 (definitely worse)	1 (definitely worse)	0	1
(*b*) History of subacute rheumatism -	15	2	14 (9 improved; others no change)	6 (3 worse; 1 improved)	0	3 (1 improved; 1 worse)	0
(*c*) History of chorea - - - -	4	3	4 (2 improved; 2 worse)	6 (1 improved; 5 worse)	0	1 (worse)	0
(*d*) History of chorea and rheumatism	4	0	1 (worse)	1 (worse)	0	1 (worse)	0
GROUP B (reported by letter).—48 Cases.							
(*a*) History of acute rheumatism -	1	0	2	3	0	2	0
(*b*) History of subacute rheumatism -	7	3	4	5	0	0	0
(*c*) History of chorea - - -	6	0	3	1	0	0	0
(*d*) History of chorea and rheumatism	6	0	4	0	1	0	0
GROUP C (did not report).—17 Cases.							
(*a*) History of acute rheumatism -	0	0	2 (1 pericarditis)	1	0	0	0
(*b*) History of subacute rheumatism -	2	0	5	0	0	1	0
(*c*) History of chorea - - -	2	0	3	0	0	0	0
(*d*) History of rheumatism and chorea	1	0	0	0	0	0	0
GROUP D (died).—20 Cases.							
(*a*) History of acute rheumatism -	0	0	3	3	1	2	0
(*b*) History of subacute rheumatism -	0	0	1	2	0	0	0
(*c*) History of chorea - - -	0	0	2	1	0	0	0
(*d*) History of chorea and rheumatism	0	0	3 (1 had pericarditis)	2 (both had pericarditis)	0	0	0
TOTALS	48	11	55	32	3	10	1

TABLE 1.

CLASSIFICATION ACCORDING TO TYPE OF RHEUMATIC INFECTION.

	Boys	Girls	Average age on first examination at U.H.C.W.	Acute Rheumatism	History of Subacute Rheumatism (including growing pains)	History of Chorea	History of Rheumatism and Chorea	Average age on re-examination in 1933
GROUP A.—75 patients who reported for re-examination in 1933	31	44	9	10	40	18	7	15
GROUP B.—48 patients who reported by letter	15	33	10.5	8	19	10	11	18
GROUP C.—17 patients who did not report	9	8	9	3	8	5	1	—
GROUP D.—20 patients who are known to be dead	9	11	9	9	3	3	5	—
TOTALS	64	96	—	30	70	36	24	—

TABLE 3.

CLASSIFICATION ACCORDING TO AMERICAN HEART ASSOCIATION'S FORMULA.

GROUP	I	IIa	IIb	III	IV	V
GROUP A.—75 patients who reported in person	23	14	4	2	9	23
GROUP B.—48 patients who reported by letter	19	2	4	0	3	20

TABLE 4.

ELECTRO-CARDIOGRAMS.

	Normal	Inverted T3	Flat T3	Inverted T in other Leads	Distorted QRS	L.V. Predominance	R.V. Predominance	Extra Systoles	P. +	P-R Interval Prolonged
GROUP A (reported in person). 70 patients electro-cardiographed -	20 (In 1 case T3 became flat)	15 (1 became L.V. plus; 1 became R.V. plus)	22 (5 became upright)	2	3	5	2	1	0	0
GROUP B (reported by letter). 36 patients electro-cardiographed -	20	9 (1 widened Q.R.S.; 1 became L.V. plus)	3	0	0	2	1	1	0	0
GROUP C (did not report). 15 patients electro-cardiographed -	4	2	2	0	0	4	2	0	1	0
GROUP D (died). 15 patients electro-cardiographed -	2	1	1	0	0	4	3	0	3	1
TOTALS	46	27	28	2	3	15	8	2	4	1

The Prevention and Attenuation of Measles

By V. D. Allison, M.D., D.P.H.,
Late Pathologist, Southern Group Laboratory, London County Council

The employment of convalescent measles serum for the prevention or attenuation of the disease has passed through the stage of experiment to that of established method, and it is now a recognized procedure in measles control. It is fitting, therefore, to review the present position in this country with regard to the collection, dosage, and administration of convalescent measles serum and adult measles serum, and to indicate the trend of experimental work for the immediate future.

Nicolle and Conseil (1918)[1] were the first to show that the serum of children convalescent from measles as a rule conferred complete protection on exposed susceptibles, with provisos as to the age of patient, dosage of serum, and period of incubation. Subsequent work in America and on the Continent confirmed these findings, and successful protection was obtained in over ninety-seven per cent. of upwards of 3,500 published cases. The most important work carried out in this country was that of Gunn (1928),[2] in the Infectious Fever Service of the London County Council, who investigated the prophylactic value in measles of three well-known immune measles sera (Tunnicliff, Degkwitz, and Ferry and Fisher's) derived from animals, and at the same time used convalescent measles serum for the purpose of comparison. The results indicated that convalescent serum was the only prophylactic agent which gave uniformly satisfactory results, showing apparent protection in sixty-six (95.7 per cent.) out of sixty-nine cases; it was, moreover, the only serum to show complete freedom from the unpleasant effects which may follow the injection of a foreign protein.

In the London epidemic of 1929-30, the use of convalescent measles serum in the fever hospitals of the London County Council was considerably extended, and the experience obtained enabled the conditions governing the collection, preparation, and use of the serum to be more accurately determined. The chief points regarding the collection of blood are—(*a*) The donor should be healthy and able to stand the loss of blood, (*b*) the attack of measles should be of moderate to marked severity, and recovery should take place without complications, (*c*) patients who may possibly be incubating any other infectious disease should be avoided, (*d*) blood should be withdrawn on the tenth to the fourteenth day after defervescence, when the antibody content is regarded to be at its highest. Amounts up to 20 c.c. of blood may be taken from a child of five years, 100 c.c. from a child of ten years, 250-300 c.c. from an adult. For the collection of amounts of blood up to 40 c.c., an all-glass syringe lined with liquid paraffin is recommended, and the contents are expelled into agar-lined test-tubes of 15 c.c. or 60 c.c. capacity; when larger amounts are taken, an intravenous needle with about six inches of rubber tubing attached, both paraffin-lined, may be used, and the blood is collected in an agar-lined screw-cap bottle of 300 c.c. capacity. The containers are agar-lined to promote separation and contraction of the clot, with a consequent larger yield of serum. The all-glass syringes, intravenous needle, and rubber tubing may be

sterilized by rinsing out with hot liquid paraffin at a temperature of 150°C.; the paraffin should be contained in a seamless vessel (not soldered), such as the cap of a vacuum flask or a "Record" syringe box. In the absence of a thermometer, the temperature of the paraffin may be gauged by dropping in a white bread crumb; when the crumb turns a golden-yellow colour the temperature of the oil is sufficiently high. Following withdrawal of the blood, the tubes or bottles are immediately incubated at 37°C. for one hour, and then left at room-temperature for twenty-four hours. For the preparation of the serum, the blood is then sent to the laboratory, taking care to avoid shaking. To avoid a milky serum, blood should not be withdrawn within two hours after a meal. If these precautions are followed, the yield of serum is usually greater than fifty per cent., and the serum is clear and free from hæmolysis. At the laboratory the serum is drawn off, and a sample set aside for the Wassermann reaction. Three or more samples are pooled, filtered through Seitz filters, and 0.5 per cent. of phenol is added. A sterility test is carried out, and the serum is then distributed in sterile ampoules of 5 c.c., 10 c.c., and 20 c.c. capacity, and these are stored at 4°C. until required for use.

Where complete protection is desired, a minimum dose of 5 c.c. serum is given to all cases, and for patients over three years of age the dosage is calculated by multiplying the age in years by two. The serum is given intramuscularly into the *vastus externus,* and contacts should be injected immediately after exposure to the disease, and in any case not later than the fifth day following contact. The protection usually lasts for three to four weeks. If attenuation is aimed at, the same dose is given between the fifth and ninth day following exposure, or if economy of serum is desired, half the dose may be given within the first five days. If these conditions are observed, and the antibody content of the serum is satisfactory, the result aimed at is obtained in one hundred per cent. of cases.

As the supply of convalescent measles serum was by no means commensurate with the demand, a further investigation was undertaken in 1931-2 in order to assess the value of adult serum in the prevention and attenuation of measles. For this purpose blood was obtained from volunteers among the medical officers and nursing staff of the L.C.C. hospitals, and also through Professor Okell from a number of students in the University of London. Care was taken that only donors who had had a previous attack of measles should be chosen, that they should be healthy and free from infectious disease, and the Wassermann reaction was carried out on all sera before pooling. Usually the serum from twenty to twenty-five donors was pooled to form a batch, in order to obtain as far as possible a uniform potency. Nearly twenty-six litres of serum were collected, and made available to the Council's infectious and general hospitals and residential schools. In all, nearly 1,500 children received injections of serum either to confer complete protection or to produce an attenuated attack. Complete protection was advised for all weak or debilitated patients, those suffering from any serious intercurrent disease, and all children under three years of age. The recommended dosage was exactly double that of convalescent serum, viz., a minimum dose of 10 c.c. of serum for all cases, and for patients over three years of age the dose is calculated by multiplying the

age in years by four. It was also recommended that an attenuated attack of measles should be sought for in contacts over three years of age, if the general health of the patient was satisfactory. The exposure injection intervals for prevention or attenuation were the same as those recommended for convalescent measles serum, viz., for complete protection, the estimated dose given within the first five days following exposure, and for attenuation the full calculated dose given between the sixth and ninth day of exposure, or half the calculated dose given within the first five days following exposure to infection.

A full account of the methods adopted, and an analysis of the results obtained, appears in the report of the Medical Officer of Health for London on the measles epidemic of 1931-2. The conclusions arrived at indicate that adult measles serum is a valuable weapon for the control of measles, only slightly inferior to convalescent serum. Adult serum confers substantial protection on children under five years of age, but is somewhat less potent than convalescent serum; both sera appear to be equally valuable when given to children between five and ten years of age, and to possess an equally poor protection value when given to contacts over ten years of age. A further important conclusion is that children under three years of age should be injected within the first three days following exposure if complete protection is desired.

Variations were observed in the protection and attenuation rates obtained with different batches of serum. The lower potency of some batches may have been due to the presence of serum from donors who either never had measles, or exhibited a long interval between the attack of measles and the withdrawal of blood. It will be a problem for future investigation, as to the relative value of sera from donors with attack bleeding intervals of 0-5, 5-10, and 10+ years. It will also be of value to determine how long serum may be kept at 4°C. without losing its potency; at present it is accepted that there is no decrease of protective power for at least twelve months. Determination of the time limit for the preservation of serum potency is of especial importance from the point of view of laying up a large reserve stock of serum during inter-epidemic periods, so that supplies may not fail when the demand is greatest. It would be of interest in this connection to investigate the value of serum in the dried state, in view of the success which has attended the drying of guinea-pig serum for complement, and the drying of agglutinating sera to maintain the potency of the immune bodies. Dried guinea-pig serum prepared at the Southern Group Laboratory, L.C.C., has now been used for nearly a year as a source of complement for the Wassermann reaction. Further points on which elucidation is desirable are the effect on serum potency of concentration, as is done with diphtheria and other antitoxins, and the possibility of raising the protective power of the serum by re-activation of the donor's blood before withdrawal.

In conclusion, it remains to mention a further method for the prevention or attenuation of measles of especial value to practitioners in outlying districts, where serum may not be readily available. It has been shown that the intramuscular injection into contacts of whole blood from donors who have previously had measles produces the desired result as successfully as by the use of adult serum. The blood

may be taken from either parent who has had a previous attack of measles, or from a suitable donor, care being taken to exclude syphilis or other infectious disease. A minimum dose of 20 c.c. of blood is given, and the dosage according to age is twice the amounts as recommended for adult serum.

I have to thank Dr. W. Gunn, Deputy Medical Superintendent, North-Eastern Fever Hospital, L.C.C., for access to his notes regarding the findings on the use of adult serums.

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Neuroses in Children

By H. HILTON STEWART, M.D., M.R.C.P.LOND.,
from the Ulster Hospital for Children, Belfast

WHILE neurotic disorders are of very frequent occurrence in adults, it is generally thought that they are rarer in the child, but if an outlook is kept for them, it will be found that they turn up with surprising frequency in the medical, surgical, and ophthalmic departments in such varied forms as eneuresis, paralysis, limps, habit spasms, blinking, etc.

I have no doubt that the so-called rarity of the condition is due to a number of causes, the chief being that one hesitates to make the diagnosis owing to the deep-rooted belief that "children do not have neuroses."

The advance of psychology has, however, made two points abundantly clear in the etiology of this condition. The first is, that neuroses may occur at any time in a person's life, and that, secondly, the seeds of the condition are sown from the age of two years and onwards usually by careless or misguided parents.

Various attempts have been made to try to find some physical force to account for the nervous child. An investigation into the carbohydrate and fat metabolism[1] only showed one definite case of starvation in a series of 169 cases; while another authority[2] states that sixty-nine per cent. of his cases showed normal body-weight and good tissue tone. He concludes by saying, "Apparently good nutrition is no safeguard against neurosis."

Observers, however, are in agreement that the most important factor in the etiology of these states is the inheritance of a neuropathic tendency. In reviewing a series of cases, I have failed to find an instance where the neurotic tendency in the parents was absent. As to the origin of the neuropathic diathesis in the adult, proof of any theory is difficult to find. Devine,[3] quoting Damaye, says that the etiology of mental affections is dominated by three great social scourges : syphilis, tuberculosis, and alcohol. Not only are the individuals morbidly affected by these agents, but this author believes that their descendants may inherit a "vitiation of

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Various attempts have been made to try to find some physical force to account for the nervous child. An investigation into the carbohydrate and fat metabolism[1] only showed one definite case of starvation in a series of 169 cases; while another authority[2] states that sixty-nine per cent. of his cases showed normal body-weight and good tissue tone. He concludes by saying, "Apparently good nutrition is no safeguard against neurosis."

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the vital processes of the organism upon which the development of personality depends."

In the same book support is given to this theory in the quotation of Ferrés' experiments on the incubation of eggs in an alcohol or absinthe vapour. In these carefully controlled experiments it was shown that, when allowed to hatch in an alcohol vapour, only some sixty-two per cent. were normal, and in the absinthe vapour twenty-five per cent. Numerous others have confirmed these findings, and Holmes has gone farther in showing that similar results occur after the mating of alcoholized guinea-pigs. This observer has noted also that the guinea-pigs of alcoholic parents produce a relatively defective progeny, even though they may not have been given alcohol themselves.

Next in importance to a psychopathetic heredity in the etiology of such conditions is the environment of the child. Hobhouse[4] states that this heredity is reinforced by the environmental influence, and "when this combination is present, it is impossible to estimate the exact proportions of the two components." This was illustrated in a recent case when a child was brought to me suffering from insomnia. On questioning the child, it transpired that her mother, to whom she was devoted, had been taken to hospital suffering from high blood-pressure. The neighbours had congregated to discuss the ailment, and as a result of the conversation the child had become convinced her mother was going to die. Removal from these surroundings and some reassurance was sufficient to cure the insomnia without the use of any drugs.

The guidance of the child in its earlier years is the third great factor in the etiology of these neuropathic conditions. Details of the various methods of guidance are many, and cannot be included in a short communication such as this, but there is no doubt that a lot can be done to counteract a bad family history by observance of certain fundamental rules. In dealing with these children one cannot but be impressed by Cameron's observation[5] that a child has four main characteristics—imitativeness, love of power, imagination, and reasoning powers. It is difficult to say which of these, if any, is the most pronounced in the normal child, but in my experience the second, or "love of power," i.e., power to get its own way, is the most striking in the psycho-neurotics. This is especially so of the hysterical case, and this phenomenon, which is usually controlled early in life, has been traced as the source of the patient's ailment. The following case illustrates some of the important factors I have emphasized.

A healthy boy of eleven years was playing football at school. He was struck on the head with the ball when he was not looking, and, being angry, rushed to strike the player who had kicked the ball. Just as he approached the offender, the injured boy dropped suddenly to the ground in a semi-comatose condition. His doctor examined him after he went home, and found no loss of consciousness, and no physical signs except some slight inequality of pupils. On the second day, while in bed, the boy had another attack. He got out of bed and fell "half on the bed and half on the floor." Again no physical signs were found, and recovery ensued, only to be supervened by a third attack. When seen on the eighth day of illness, the

boy presented no physical signs in the central nervous system, except increase of all reflexes. His co-ordination while lying in bed was perfect in every respect, but he was unable to stand. After some suggestion a stance was assumed and an attempt made to walk. Gross staggering movements were made, but it was noticed that the boy never fell. The diagnosis of hysteria was then made with confidence, and confirmation was forthcoming in a few minutes, when with a little further suggestion and persuasion the boy walked normally before an audience.

Inquiry was made to find out what the cause of the condition might be, and the boy's own story was that he disliked school, as he was required to work too hard, and got no time to play. This did not appear to be reasonable, as the first attack did not occur while at work. Investigation from a close friend showed that the patient's personality was not normal before the football incident. He is said to have been self-conscious, deep, and inclined to be deceitful. He would cry if he lost a game, and in fact he is a "spoiled boy."

Further investigation revealed a "nervous background." The father was said to be a teetotaler, a non-smoker, a very dour man who did not make friends easily. He was an inconsistent man who at one moment would order his son to go and do his home lessons, but a little protesting from the boy and the father commenced to do the lessons himself. The mother is said to be "abnormally nervous," and was up till lately afraid to cross the street.

In attempting, then, to reconstruct the whole case, we have an only child of parents who have provided an unstable neurological background. The inherent love of the child to occupy the "centre of the stage" has not been suppressed, and so the unconscious mind employs hysteria as a cloak in order to gain this end when ordinary methods appear to be failing. A few weeks in hospital made him quite well, but it is not surprising to hear the following story after his discharge. The boy was running a relay race, and was to take the finishing lap. He ran the race well, and after finishing he collapsed on the ground. He was not unconscious, and remembers everything, including the arrival of his doctor, who found nothing abnormal. Interpreting this in the same way as before, it would seem that the unconsciousness mind had added the note of drama to ensure "the limelight." I do not think that all cases of hysteria in children are reactions of the unconscious mind because of unsuppressed "love of power," but a large number I have seen have been of this nature, especially those exhibiting negative phenomena, such as paralysis of legs or arms, talipes equino-varus, etc. The difference between this type of case and that of the malingerer is quite definite. Whereas in the malingerer the symptoms and signs are only present when the patient is being watched or examined, those of the hysteria are present all the time, except of course during sleep.

Numerous other cases of hysteria might be quoted to further prove that the condition is independent of mimesis, and especially that no disease is ever exactly imitated by hysteria.

An excellent example of this latter statement is illustrated by the child presenting itself with an hysterical limp simulating joint disease. Such a case presented itself

at the Ulster Hospital some time ago. A child aged nine years was struck on the left side by a bicycle. The child was said to have been unconscious after the accident. She was X-rayed at another hospital, and discharged in three days' time apparently quite well. One week later she complained of great pain in her left flank, and was noticed to have a limp. Examination showed a healthy looking child walking as if her left hip, knee, and ankle were ankylosed. The pain complained of varied in position from time to time during examination, and was never like that complained of in joint disease. Close examination revealed that movement was perfect at all these joints, but that when left alone and asked to walk by herself, all the muscles of the left lower extremities contracted, extensors, flexors, abductors, adductors, and rotators, so that a rigid limb was the result. This condition, it will be noted, is not explained by any organic lesion. In differentiating one of these hysterical joints from that of true organic disease, one is helped by the fact that the position in which the limb is held is rarely the same as that which occurs as a result of organic disease. For example, in the case of the knee, the joint in a neurosis is usually held fully extended, whereas in a true arthritis the knee is usually slightly flexed. No joint disease shows at one moment cast-iron rigidity and full range of movement at the next. The only condition which will satisfactorily explain it is hysteria. Further inquiries were instituted to learn if the child had ever seen anyone suffering like this, and the answer was in the negative. Suggestion was sufficient to cure the case in a few minutes.

The history of injury elicited above is not uncommon, but the injury is usually trifling, and is only at most a precipitating factor in the disease. It is the outstanding factor emphasized by the parents. It would appear to act almost in trigger fashion, producing rigidity in all the muscles of the limb or around one particular joint.

The diagnostic criteria differ in no way from those used in diagnosing neuroses in the adult. There must be no physical signs of organic disease. Contractures must be differentiated by giving an anæsthetic, when in an hysterical subject they will disappear. Suggestion should cause appearance and disappearance of symptoms.

The treatment of these cases presents certain difficulties which are not seen in the treatment of adults. For example, one is denied the "dream interpretation" of Freud, and in most cases, owing to the children tending to being reticent, one cannot apply the tests for the "association of ideas."

The first essential is the complete removal of the child from its parents and home surroundings. This therapeutic test, often based on the etiology of the condition, amply justifies its practice, and some of the neuroses, notably eneuresis, often require no further treatment. The second essential in treatment is to gain the confidence of the patient; and, thirdly, suggestion methods should be used according to the particular type of neurosis presented. I have no experience of hypnosis, and with children it must always have a limited field of operation. Most cases I find recover rapidly with the suggestion method, but the "diathesis" always remains, and is liable to be the cause of a recurrence should other circumstances be suitable for it.

Drugs have little or no place in the treatment of these patients. General tonics are prescribed more as a placebo than for any psychological or physical benefit. The dictum of Robert Hutchison, however, still holds good, that bromide often acts like a charm, especially if given to the parents.

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The Role of the Anterior Hypophysis in the Child

By J. HENRY BIGGART,

Johns Hopkins University, Baltimore, Md., U.S.A., and Queen's University, Belfast

PERHAPS in no field of research has there been a greater advance in the last ten years than in that of endocrinology, more especially in our knowledge of the anterior hypophysis. Most of this work has been done on animals, and whilst it is still premature to apply many of the experimental findings to the human being, our interpretation of the functions of the human gland now rests upon more secure foundations.

As with every other endocrine gland, there are three possible degrees of function : (1) normal, (2) hypersecretion, and (3) hyposecretion. It is still impossible to measure the degree of secretory activity of the gland, but it is becoming more easy to interpret various structural changes in other organs as the result of hyper- or hyposecretion. It is well known that the gland secretes several hormones, and the problem at present is to trace the origin of each hormone to its mother cell in the anterior hypophysis. Once this has been done the interpretation of various pathological findings in the gland will be rendered more easy and less speculative.

On the basis of staining reactions, the cells of the anterior lobe are divisible into three types : (1) cells with alpha granules or eosinophils, (2) cells with beta granules or basophils, and (3) cells without any evident granulation or chromophobes. Studies on the Golgi apparatus lend support to this classification, as there is quite a distinct difference between this structure in the eosinophil and basophil. Whilst the French school led by Collin[1] maintains that all three are but transitional stages in the secretory activity of one cell type, the more general belief is that the chromophobe is the mother cell of the other types, and it is on this assumption that the pathological findings in this gland are interpreted. Certain syndromes are now definitely related to over-secretion of the eosinophils, whilst the syndrome due to changes in the basophils is still under debate. There also exists the possibility that the chromophobes may elaborate a secretion, but little attention

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has been paid to it, and most authors merely regard this cell type as the inactive progenitor of the secretory forms.

The gland controls at least two important vital functions of the body—growth and sex. In addition, in several species of experimental animals, hormones controlling the thyroid and adrenal have been demonstrated (Loeb,[2] Aron,[3] Schockært,[4] and Biggart and Friedgood[5]), but data are still wanting to help in the application of these new facts to human metabolism.

Since Cushing's monograph on acromegaly, it has been generally accepted that the growth hormone is manufactured in the eosinophil cell. The function of the basophil in the human is not so clear, though in the rat it seems to be definitely connected with the manufacture of the hormone controlling the development of the sexual apparatus. An analysis of the hypophysis in human castrates would seem to indicate that they play a somewhat similar role in man, at any rate as regards the manufacture of the luteinizing hormone (Biggart[6]).

It is now a well-known fact that a pituitary-like factor (A.P.L.) is found in the urine of pregnant women. This probably plays a role in fœtal metabolism, as it has been demonstrated by Bruhl[7] and Neumann and Peter[8] that it is also present in the urine of fifty per cent. of the new-born for two to eleven days following birth.

Certain autopsy findings in the new-born seem to be linked with this finding. The human male infant is the only one born with its testes normally in the scrotum. Human pregnancy is the only one characterized by the presence of A.P.L. in the urine. Injections of this substance in infantile rats cause descent of the testes.

Neumann[9] has also pointed out that the prostate and uterus of new-born infants are increased in size at birth, and subsequently undergo a certain degree of involution. The increase in size of the prostate and uterus in rats following injection of pregnancy urine is now well attested.

Injections of A.P.L. over some time into young female rats brings about a cystic condition of the ovaries. In a series of still-born female children cystic ovaries were found in over thirty per cent.

Whilst it is impossible to be sure that these findings in the human are to be interpreted in this manner, the analogies in experimental animals render this interpretation at any rate a possibility.

Some of the more important actions of the anterior hypophysis are related to growth. Thus syndromes involving both hyper- and hypo-secretion are recognized. The most important of these is gigantism, which is produced by an excessive supply of growth hormone. Examination of the hypophysis in these cases usually reveals an eosinophil adenoma. That this is the causative lesion has now been proved by the injection of anterior hypophyseal extracts into rats (Evans[10]) and dogs (*Benedict et al.*[11]). The effect of such an adenoma, however, is not merely confined to excessive growth, but there is also an increased basal metabolic rate, a lowered carbohydrate tolerance, and changes in the sex glands and hair indicating a widespread disturbance in the body's metabolism. At autopsy the thyroid, adrenals, heart, lungs, liver, spleen, and kidneys are greatly enlarged. The pancreatic islets are hypertrophied. The changes in the sex glands are not yet sufficiently analyzed, but

are thought to be due in part to the destruction of the basophils by the growth of the adenoma.

Gigantism usually begins between the ages of 15 and 25. If the adenoma develops later when the epiphyses are closed one gets the symptoms of acromegaly.

The evidence for the syndrome due to hyposecretion of the eosinophil cells is not so convincing, but in all probability many cases of dwarfism, especially of the Lorain type, are of hypophyseal origin. Thus Worster-Drought, Dickson, and Archer[12] report the case of a girl aged 19. The child's height was forty-nine inches, and her weight forty-seven pounds. No secondary sex characteristics had developed. The child had a mental age of 13. At autopsy a cyst of Rathke's pouch was found, which had almost completely destroyed the anterior hypophysis. Similarly Bailey[13] records the case of a dwarf aged 21 whose anterior hypophysis was replaced by several small cysts and only traces of abnormal gland tissue. He was well proportioned and moderately nourished, but only 111 centimetres in height. The beard, axillary, and pubic hair were wanting. The thymus weighed 2.5 gms., the thyroid 2 gms., and the adrenals 2 gms. The testes were like those of a baby.

Such cases are not uncomplicated failures of growth, but sexual development is also interfered with. Hypophysectomized rats not only fail to grow, but also remain sexually infantile. The pathology of such cases in the human is generally of such a nature that both eosinophils and basophils are destroyed, and it is so far impossible to state how much of the syndrome is due to the lack of secretion of either one of these cells.

In some cases of dwarfism the individual appears prematurely old. Such cases are often classified as examples of progeria. The resemblance of such a patient to the clinical syndrome in the adult known as hypophyseal cachexia or Simmond's disease, which is characterized by loss of weight, atrophy of the sex organs, loss of hair, premature senility, lowered metabolism, etc., suggests that hypophyseal deficiency in the child may be the underlying endocrine dysfunction in progeria.

Another juvenile disease in which most textbooks incriminate the hypophysis is Fröhlich's dystrophia adiposo-genitalis. Here the striking findings are the sexual infantilism and the adiposity. So far experimental evidence does not support a purely hypophyseal origin for this syndrome. Hypophysectomized animals do not become fat. Patients in whom the anterior hypophysis is destroyed by embolic processes lose weight. On the other hand, in a series of hypophysectomized animals a certain number do become fat. In such cases, however, hypophyseal transplants will not restore the animal to normal, which suggests that the occurrence of adiposity cannot be regarded as the result of an uncomplicated hypophysectomy. Camus and Roussy[14] have reproduced the syndrome by lesions in the hypothalamus. In a typical case of Fröhlich's dystrophia, which I was able to examine, the hypothalamus was completely destroyed by a cystic growth from the hypophyseal stalk, whilst the anterior hypophysis, though mainly composed of chromophobe cells, was not even compressed.

The not uncommon occurrence of obesity following encephalitis certainly suggests that some hypothalamic injury is more probable than a state of hyposecretion

of the hypophysis. According to Armstrong,[15] seventy per cent. of all supra-pituitary tumours present signs of dystrophia adiposo-genitalis.

From a physiological point of view, which has a great amount of support from pathological studies, one is forced to conclude that Frölich's syndrome is not one of uncomplicated hypopituitarism, but is rather of hypothalamic origin. Similar conclusions must be made in regard to the types of the syndromes described by Fearnsides and Brissaud.

Somewhat similar to Fröhlich's syndrome is the Lawrence-Moon-Biedl syndrome, characterized as it is by obesity, genital dystrophy, retinitis pigmentosa, polydactylism and its familial occurrence. However, such an extensive clinical picture can scarcely be interpreted on a purely endocrine basis, and its familial occurrence forces the pathologist to fall back upon some defect in the germ cells as the etiological factor.

Whilst cases of hyposecretion of both eosinophils and basophils result in a diminution in growth and development of the sex apparatus, and whilst hypersecretion of the growth hormone of the eosinophil cells produces gigantism, little is known of the state of hyper-secretion of the basophil cells. Basophil adenomata are rare, and though Cushing[16] has recently endeavoured to state a case for an accompanying clinical syndrome, the relationship is by no means proven. As yet no cases have been recorded in children.

Much of our knowledge of the functions of the anterior hypophysis is the result of animal experiment. In no subject, however, more than in endocrinology, is it dangerous to translate findings from one species to another. Hence our present position in regard to the human hypophysis must remain *in statu quo* until some method, chemical or biological, has been found to measure the activity of the various cell types of this gland. The urine test in pregnancy, the guinea-pig thyroid hyperplasia test suggested by Aron, are the beginnings in this final analysis.

To summarize :—The anterior hypophysis produces at least two hormones which are of vital importance to the development of the child. The first of these controls growth, and is probably manufactured in the eosinophil cells. Disturbances in its secretions are recognized clinically by the syndrome of gigantism (hyper-secretion) and some forms of dwarfism (hypo-secretion). The second of these is the hormone controlling the development of the sex apparatus. This is possibly, at least partly, manufactured in the basophil cells. Hyposecretion leads to sexual infantilism, and is usually accompanied in children by hyposecretion of the growth hormone, as in such cases the pathological lesion found is most often a hypophyseal duct cyst which destroys both types of secretory cells. Hypersecretion of the sex hormone would presumably lead to precocious development of the sex organs, but a clinical syndrome with this etiology has not yet been clearly defined.

Adiposity is probably not a symptom of hyposecretion of the anterior hypophysis, but rather of injury to the hypothalamus.

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Hip-Joint Disease

By H. P. MALCOLM, M.CH.,
from the Belfast Municipal Hospital, Graymount

THOUGH it may arise at any age, hip-joint disease is essentially a disease of childhood, the majority of cases occurring during the first ten years of life, and after the spine, the hip is the joint most frequently attacked by tuberculosis.

During the early years of life milk forms a large part of the diet, and probably explains the frequency of the disease in children, though statistics given by different investigators of the relative frequency of infection by the bovine type of bacillus vary greatly, from 2.7 per cent. up to 60 per cent. (Fraser).

The source of infection by the human type of bacillus is the presence in the house of another person actively infected. Congenital infection may be disregarded, though Fraser states that twenty cases of apparent true congenital tuberculosis have been reported.

Probably at most there is an inherited predisposition, and the disease is inaugurated by transmission of the germ after birth. The infection may enter the body either through the respiratory or the alimentary tract. In adults the lungs are the most frequently affected parts, the disease spreading sometimes to the bones. In children surgical tuberculosis is the most common. The bacillus in these cases enters by the alimentary tract and settles first in the mesenteric glands, or sometimes through the tonsil or carious tooth. Fraser states that in submaxillary gland infection he has frequently demonstrated the bacillus in the pulp of decayed teeth. Tubby believes that tuberculous dactylitis may result from direct infection of skin wounds caused by crawling on dirty floors. From the glands the infection is carried to the joint by the blood-stream.

The disease is practically always attributed to an injury, usually of minor degree, and it is always difficult to say if this is a predisposing cause, or if a slight injury which would ordinarily be forgotten is noticed because the joint is already becoming abnormal. Certainly injury can have no effect on the distribution of bacilli from the primary gland focus. It may, however, by causing vascular clotting in the bone

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and excluding the protective fluids and cells, or by breaking down an encysted and quiescent focus, encourage the development and propagation of bacilli which would otherwise have been quietly done away with. The cause of the disease, therefore, is the entry of the bacillus through the alimentary tract, infection of the associated glands and spread therefrom by the blood-stream to the joint. The exanthemata, influenza, bad feeding, and unhygienic surroundings, by lowering the vitality, play an important part in the development of the infection.

In the hip-joint the disease may begin in the synovial membrane or the bone, more frequently in the latter. The starting-point is usually in the under-surface of the neck of the femur, near the epiphyseal cartilage, at the site of entrance of the anastamosis between the synovial and the osseous vessels, but it may also be on the surface of the acetabulum in the neighbourhood of the Haversian pad of fat. At first the joint cavity is not affected, or at most there is a slight synovial effusion, and the symptoms are mild and intermittent. Soon the initial focus bursts into the joint cavity, and there is an immediate exacerbation of all the symptoms, which may be very acute and accompanied by severe pyrexia. All the constituent parts of the joint are then affected. Necrosis begins in the articular cartilage, the head of the femur, and the acetabulum. The head of the femur is eroded and distorted, and not infrequently disappears completely. The roof of the acetabulum, where the pressure is greatest, is gradually eaten away, successive buttresses of bone being laid down by nature's efforts to reconstruct the cavity, so that when finally the disease is arrested the femur lies at a higher level and the limb is shortened. Shortening also occurs from softening of the ligaments and actual dislocation of the head of the femur, from interference with growth, and from destruction of the head and neck of the femur. Occasionally the floor of the acetabulum is destroyed and the head of the femur penetrates into the pelvic cavity. Abscess formation is frequent, the swelling appearing at the outer and anterior aspect of the joint between the sartorius muscle and the tensor fasciæ femoris.

Other situations where the abscess may point are the adductor region, posteriorly below the lower border of the gluteus maximus, and, if the acetabulum has been penetrated, the pus may burrow up into the iliac fossa, simulating psoas abscess, or down to the ischio-rectal fossa. In the last instance the rectum may be perforated and fæcal matter be discharged through the sinuses.

In fatal cases death is usually due to prolonged toxæmia or amyloid disease following secondary infection, or to meningitis. In my experience, spread of the infection to the lungs has been very rare.

The symptoms are too well known to be mentioned at any length. Briefly, they are, pain, often referred to the knee; limp, varying degrees of rigidity due to protective muscular spasm, wasting of muscle, abscess formation, and the three stages of deformity. First, abduction, flexion, and external rotation, with apparent lengthening; next, flexion, adduction, and internal rotation, with apparent shortening; and finally, adduction, flexion, and internal rotation, with real shortening due to one of the causes mentioned above.

In a fully developed case the diagnosis is simple, but in the early stages, when the focus of disease is still small and localized, it is by no means easy. There may

only be a little discomfort and stiffness, most noticeable in the morning, and wearing off during the day, and X-ray may reveal no change. Well-marked muscular spasm is a sign which cannot be missed, but even in the early stages of the disease there is some spasm, and it is important to be able to recognize its presence. A slight degree of spasm can be demonstrated by the rotation test, when coarse movements of the joint may give no information. To test the external rotator muscles, place one hand on the anterior superior spine of the affected side, with the other hand grasp the knee firmly, and gently and slowly rotate the limb inwards until it will go no farther. Having reach the limit of movement, with a short, sharp jerk of the hand try to rotate the limb a little farther. If there is no spasm, nothing happens, but if spasm is present, the muscles resent the last movement and give a quick contraction. As the limb is firmly held and cannot move, the force of the contraction is transferred to the pelvis, and the anterior superior spine can be felt to rise suddenly under the hand. By placing one hand on the opposite anterior superior spine and rotating the limb outwards, spasm of the internal rotator muscles can in the same way be detected. This is the most delicate test for spasm. Minor spasm of the flexor muscles is most easily detected by placing the child's pelvis on the edge of a table and allowing the lower limbs to hang over; if spasm is present, the affected limb will hang at a slightly higher level than the sound limb. I am firmly convinced that symptoms, however slight, referred to the hip-joint in a child, provided that no definite diagnosis can be made, should be treated as tuberculous.

At this stage cure may be only a matter of months, but if the disease is allowed to develop it becomes a matter of years.

Two other chronic conditions which occur in children, and which may lead to an erroneous diagnosis of tuberculosis, are slipped epiphysis and Perthes' disease. Slipped epiphysis occurs between the age of ten and seventeen, usually in males. Pain and spasm are present, but the patient stands with the limb slightly adducted and rotated outwards, in contradistinction to any of the attitudes assumed in tuberculosis. If the epiphysis has already slipped, there will be half an inch of shortening, a condition which cannot be present in the early stage of tuberculosis, and an X-ray photograph will dispel all doubt.

It should not be forgotten, however, that slipping of the epiphysis may be a gradual and not a sudden event, and the patient may come for examination at a stage when X-ray reveals no change. This is well shown in figures 1A and 1B. A boy aged fifteen came complaining of limp and pain. On examination some spasm was found, but X-ray revealed nothing abnormal, and he was kept under observation as a possible case of hip-joint disease. Three months later (fig. 1B) an X-ray showed a slipped epiphysis, and accounted for the symptoms.

Perthes' disease occurs between the ages of five and ten, and is more common in males. In this condition also, limp, pain, and spasm are present, but the limitation of movement caused by the spasm affects most the movements of abduction and internal rotation, while in hip-joint disease all movements are affected. The child holds the limb in a position of slight flexion and adduction, a deformity which is not present in hip-joint disease until the later stages, and the X-ray appearances are quite distinctive. (Fig. 2.)

The head of the femur is flattened and broadened, and its structure irregular and fragmented in appearance. The neck is thickened so that the continuation of its lower border may pass below the upper border of the obturator foramen instead of being continuous with it. The roof of the acetabulum, according to Calot, is more oblique than normal, and shows a woolly and irregular appearance. The latter is not of much help in the diagnosis, as in children of seven to twelve years of age an irregular-fringed appearance of the upper part of the acetabulum may normally be present.

In hip-joint disease X-rays, though not of great value at the onset, are of the utmost importance during the progress of the disease for estimating the effect of treatment and deciding when cure may be presumed to have taken place. When studying X-rays of the hip-joint, there are some normal appearances which should be kept in mind. The head of the femur begins to ossify in the tenth month, and does not assume its semi-circular shape until the third or fourth year. Some translucency between the head and neck at the lateral and medial terminations of the epiphyseal line is normal. The acetabulum is formed by all three pelvic bones, and is not completely osseous until the seventeenth or eighteenth year. The cartilaginous junction between the pubis and the ischium is not visible in X-rays, but that between the ilium and these two bones is seen as a clear linear area dividing the acetabulum into an upper and a lower part. The roof, posterior wall, and floor of the acetabulum are visible, but the anterior wall is not. In the ilium over the roof of the acetabulum there is sometimes a brighter area, which is normal. In examining an X-ray for disease, one should note, first, if the outline of the bones is clear and regular: any interruption and raggedness means necrosis; secondly, the cancellous tissue should be examined for isolated areas of translucency or general loss of detail as compared with the sound side; and, lastly, the position of the head of the femur, whether normal or displaced, a point easily missed in children. There are three indications that the position of the head is normal. With the limbs in the straight position and the feet vertical, a continuation of the clear line caused by the cartilaginous junction of the pubis and ilium meets the mid-point of the osseous nucleus of the head of the femur; the termination of the inner margin of the neck is at the lower border of the acetabulum; and a production of the curved lower line of the neck coincides with the upper border of the obturator foramen.

At the onset of hip-point disease there is likely to be no X-ray change. Later a diffuse atrophy of structure in the head and neck is characteristic of the synovial type. In the primarily osseous form, the first appearance is a translucent area or areas in the neck, surrounded by normal bone. (Fig. 3.) As the disease develops, the later changes are the same in both types: progressive destruction of the head and neck of the femur with widening upwards of the acetabulum, or dislocation. (Figs. 4, 7, and 9A.)

In fig. 5 an interesting and rare condition is seen—penetration of the base of the acetabulum by the head of the femur, called "protrusio acetabuli" by Köhler, and "arthrokatadysis" by Jenner. According to Köhler, twenty cases have been described. He has found it after typhoid and in healed tuberculosis. It has been

attributed to abnormal gout, tabes, interruption of growth, ostitis deformans juvenilis, and gonorrhœa. In this X-ray the process is seen : during its development the head of the femur has penetrated into the pelvis, and a new floor is being formed to the acetabulum, but is not yet complete. I believe this case is tuberculous. Mercer in his book has published a series of photographs showing the development of this condition in a case which he also attributes to tuberculosis.

The most important function of the X-ray is the help which it gives in deciding when a cure has been effected, though the only absolute test is that of time. In looking for evidence of cure, one must distinguish between calcification and ossification. After a time many joints show massive new formation which is billowy and structureless in appearance. (Fig. 6.) This is due to deposit of calcium in the diseased tissues, and is not a sign of cure. If function is resumed in such a case, there will be further collapse of the bone and probably recrudescence of the disease. In a cured case with true ossification this billowy appearance is absent, the bones are dense, and details of structure can be seen. (Fig. 7.)

I used to think that when X-ray showed definite evidence of necrosis in the bones, a stiff joint was inevitable. I find that this is not the case, and that a number of patients in whom there was extensive joint destruction have regained excellent movement. (Figs. 8, 9A, and 9B.)

The mortality given by different authorities varies greatly. Huismans at Heidelberg gave a mortality of forty-six per cent. for non-operative, and fifty-eight for operative cases. Bruns gave twenty-three per cent. in non-suppurative and fifty-two in suppurative cases. In this country the mortality was much lower; ten to eighteen per cent. were the figures given by Fraser. Recently the New York Orthopædic Hospital gave the following end-results in one hundred and fifty cases. Twenty-four per cent. had died; in forty-seven per cent. the disease was active; in twenty-seven per cent. quiescent, and two were free from symptoms and had useful movement. Pattison out of three hundred patients had fifty-five per cent. with mobile joints.

During the ten years from 1921 to 1931 I discharged thirty-eight cases, all of whom had been treated conservatively. Three of these died, a mortality just under eight per cent. One was discharged improved, and one with incurable deformity. The remaining thirty-three were discharged as "disease arrested," and at the last investigation in 1932 it was found that one had died of appendicitis and thirty-two were still well. The times which had elapsed since discharge were as follows :— Nine had been discharged under one year, two over one year, two over two years, two over three years, three over four years, four over five years, four over six years, four over seven years, and two over eight years. These figures may not be final, and some of the more recent cases may still relapse. Ten (that is, twenty-six per cent.) of these cases had full movement. This number might be greater, as a few did not come for examination, but were traced by the visiting nurses and stated to be well.

The general treatment of surgical tuberculosis—fresh air, and a nutritious and well-balanced diet combined with rest—is well known to everyone. This enables

the patient to overcome the infection, and it remains for the surgeon to deal with abscesses, to prevent deformities, or to correct, as far as possible, deformities which have already arisen. Many things have been tried, but I do not think there is any medicinal or biological treatment of proved value. Bone regeneration is slow, and time is essential. It is one of the most important factors in the treatment, and the factor which is most often stinted, mainly owing to the insufficient hospital accommodation for this type of case. Children with surgical tuberculosis should be sent to an institution to be brought up, just as healthy children are sent to a boarding-school. Unfortunately, this is at present only possible for a few.

On diagnosing a case of joint tuberculosis, it is wise to impress on the parents at once that cure will be a matter of some years, otherwise one is constantly worried and may be persuaded into relaxing treatment too soon.

The points to be considered in the conservative treatment are :—The abscess, extension by weight and pulley, fixation of the joint without extension, and convalescence.

As long as a cold abscess is not enlarging and approaching the surface, it should not be interfered with. When it has approached the skin, however, the case is different, and every attempt must be made to avoid secondary infection, which greatly increases the gravity of the disease. If when the patient is first seen, the skin is inflamed, thin, and obviously going to break, it is best to make an incision as small as is compatible with emptying the abscess. A small clean incision is less likely to become infected than a jagged burst.

When secondary infection occurs in a closed abscess, as it sometimes does through the blood-stream, drainage is indicated. For the uncomplicated cold abscess which is approaching the skin, aspiration is the best treatment, and is of benefit even if the skin breaks down later. A patient in whose abscess a sinus forms without previous treatment, often develops a marked toxæmia and pyrexia, while the patient whose abscess has been aspirated repeatedly for some time does not as a rule do so. The reason probably is the state of the abscess wall, which consists mainly of sloughing tissue, and presents no barrier to invading organisms; while the abscess which has undergone repeated aspirations has been enabled to line its walls with granulation tissue, which is well known to be strongly resistent to infection. When the contents of the abscess are too thick to pass through the aspirator, an injection of one part thymol, two parts camphor, and three parts ether, may liquefy the pus sufficiently to allow of aspiration. If aspiration fails, a small incision may be made, the abscess cleaned out, and the incision closed. It will usually heal, and if the cavity fills again, as it frequently does, the new contents are fluid and can be easily aspirated. In aspirating, a point in the skin some distance from the swelling should be chosen, anæsthetized by a local anæsthetic, and a small opening made with a fine tenotomy knife for the reception of the aspirating needle. When an aspirator is introduced through the whole skin, the force which has to be used destroys all delicate sense of touch and direction, and it is more painful to the patient. If aspiration be performed through the skin immediately over the abscess, a sinus will almost inevitably follow.

Extension by weight and pulley is indicated at first in all cases. If there is no

deformity, the limb is placed in the position of about thirty degrees of abduction. When deformity is present, traction is applied in the line of the deformity, and as spasm diminishes the limb is gradually brought into the extended and abducted position. If the limb is finally shortened and the joint stiff, abduction minimizes the disability. The period for which extension is continued depends upon the progress of the case. In early cases with no bone destruction a mobile joint is to be hoped for, and extension is continued until all spasm, as shown by the rotation test, has disappeared. The patient is then kept in bed without any apparatus, and if there is no return of spasm after a month or two, is allowed up for gradually increasing periods. The instruction sometimes given, that extension should be continued until all pain has disappeared, is quite erroneous. Pain nearly always disappears rapidly under the influence of efficient extension, and its absence is no indication of the progress of the disease. When disintegration of the joint is present, extension should be applied until the deformity has been reduced, and maintained until all symptoms have subsided. Then, if one decides that a stiff joint is unavoidable, the limb should be placed in plaster of paris from the toes or knee to the axillæ, and the joint kept fixed until consolidation of the bone is complete. A plaster case which does not extend well on to the chest-wall will not keep the joint abducted.

I am now continuing extension for longer periods, and using plaster less, since finding that some apparently hopelessly disorganized joints in children eventually recover a useful range of movement.

When the joint has become permanently stiff, the ankylosis is always fibrous in uncomplicated cases, but frequently bony in those who have had secondary infection. The latter, therefore, though more dangerous from a survival point of view, may give a better result, as in fibrous ankylosis the limb may adduct again, even after complete cure, and require sub-trochanteric osteotomy. Hibbs arthrodesis is designed to prevent this. In it the great trochanter is separated from the femur, turned up and inserted into the pelvis just above the acetabulum; it fuses with the pelvis and the neck of the femur, forming a bony ankylosis. Its function is to prevent deformity and supply an internal splint, and it should not be performed until the disease is quiescent.

Operation during the active stages is likely to reveal a soft and porous bone useless for grafting purposes.

At the conclusion of recumbent treatment, the stiff joint should be protected for a year or more from weight-bearing, either by a patten on the sound foot and crutches or by a walking caliper. The duration of the whole treatment will be three to five years or more.

There are many varieties of fixation apparatus and methods of extension. The simplest is that shown in fig. 11. A long Liston splint with a foot-prop is applied to the sound side to keep the patient flat, and a bandage and webbing extension to the affected limb. A layer of soft crepe bandage is first applied over the skin, a length of upholsterer's webbing is then laid along each side of the limb and fixed by a figure-of-eight bandage of crepe or flannelette from three fingers breadth above the maleoli to the groin. This will take up to nine pounds extension, a weight which is seldom necessary, as one pound per year of age up to about seven is usually

sufficient. The extension has to be reapplied about twice weekly, and for that reason it is useless in the treatment of fractures. Sometimes when the bandages are not firmly enough applied they slip a little, and irritate the skin just above the patella. An adhesive extension can then be used for a few days until the skin is normal again. Adhesive extensions cannot be used for the prolonged periods necessary without causing still more serious irritation of the skin.

With the extension no splint is necessary for the affected limb, which merely lies flat on the bed, and is held abducted by the weights. When flexion is present the simplest splint is an inclined plane of wood, which can be lowered gradually as spasm subsides, and discarded when the limb comes down to the bed.

At one time there was much difference of opinion in regard to operative versus non-operative treatment. For some years conservative treatment has been regarded as the best treatment, but now again there is a tendency on the part of some surgeons to resort to operation. Bankart believes that the disease always starts in the pelvis, and is doubtful if it is ever cured permanently by conservative methods. He is trying complete excision of the acetabulum and the upper end of the femur. His first nine cases did well, and the next two died, an immediate mortality of eighteen per cent. The operation is severe, requiring blood transfusion, and some years must elapse before its relative value can be assessed.

The Treatment and Prognosis of Acute Osteomyelitis

By Ian Fraser, M.D., M.Ch., F.R.C.S.Eng., F.R.C.S.I.,
from the Belfast Hospital for Sick Children

Of the surgical treatment of osteomyelitis in the acute stage, as in other phases of everyday life, the fashion is constantly changing.

It is doubtful if, with all changes in technique, more lives are saved or the period of illness in an individual case is reduced. At times the only motive might almost appear to be as in Tennyson's King Arthur: "Lest one good custom should corrupt the world." The pendulum keeps swinging from, on the one hand, the side which advocates doing as little as possible to an acutely ill patient, to, on the other, the side whose motto is: Do as much as possible at the first operation, so that the long, tedious convalescence and period of discharging sequestra will be very much shortened. Pus is present in two places—(1) under the periosteum, and (2) in the medullary cavity. The former probably causes the tenderness and the bone destruction chiefly, and the latter the deep-seated pain and the toxæmia. The two are in communication by very small channels through the substance of the bone.

The least that can be done in an acute case is a long and deep incision down to the bone through the periosteum. Subperiosteal pus thus escapes and drains well; and the internal pus slowly makes its way to the surface through minute channels in the bone.

The bone deprived of its periosteum is dead, and will later be separated from the living along a line of demarcation.

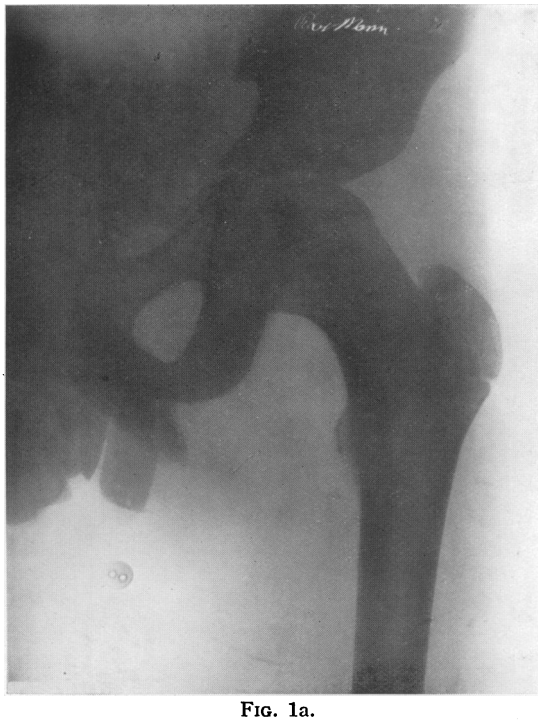

FIG. 1a.
Boy aged fifteen. Pain limp, and slight spasm of hip. No X-ray change.

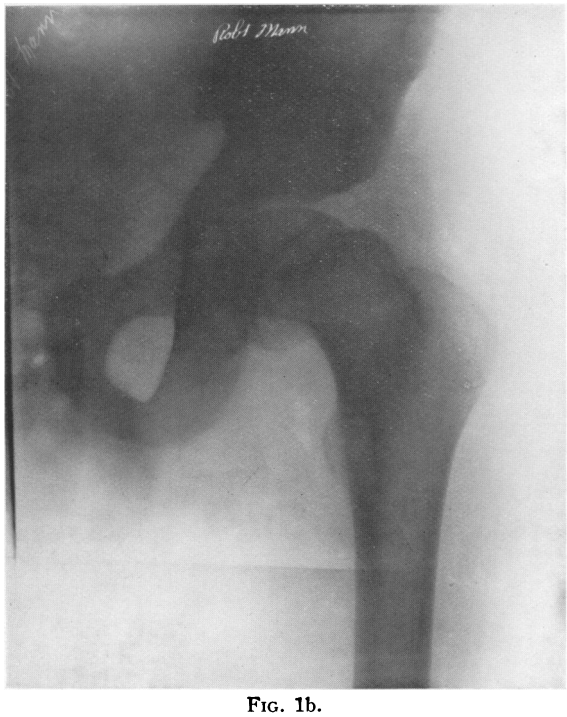

FIG. 1b.
The same boy three months later. X-ray shows slipped epiphysis.

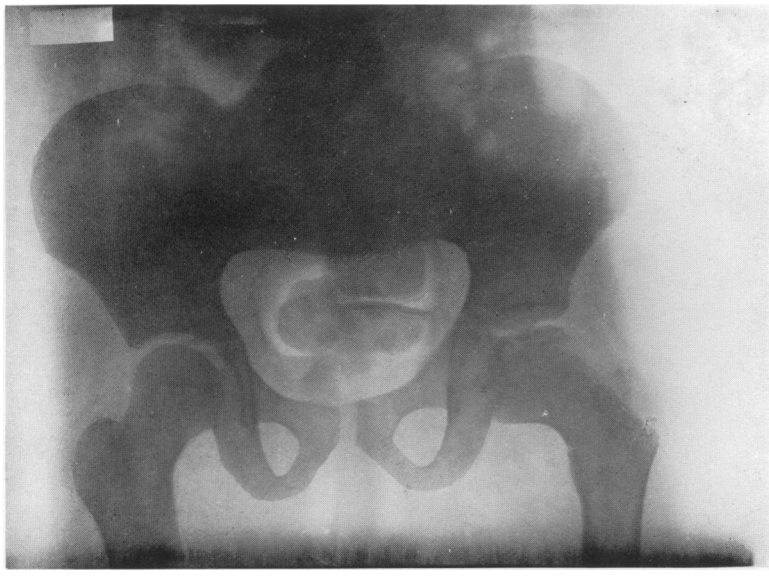

FIG. 2.

Perthés disease. The head of the femur is flattened, broadened, and fragmented. The neck is thickened, and its lower border produced would pass below the upper border of the thyroid foramen.

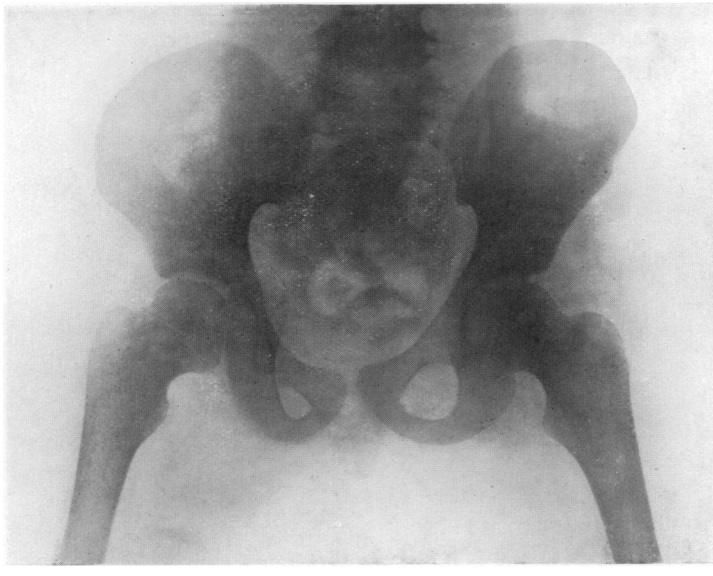

FIG. 3.

Hip-joint disease in a girl aged ten years. Onset, February, 1932. X-ray taken August, 1932, shows translucent tuberculous foci in the neck of the femur. The head and acetabulum are not yet affected.

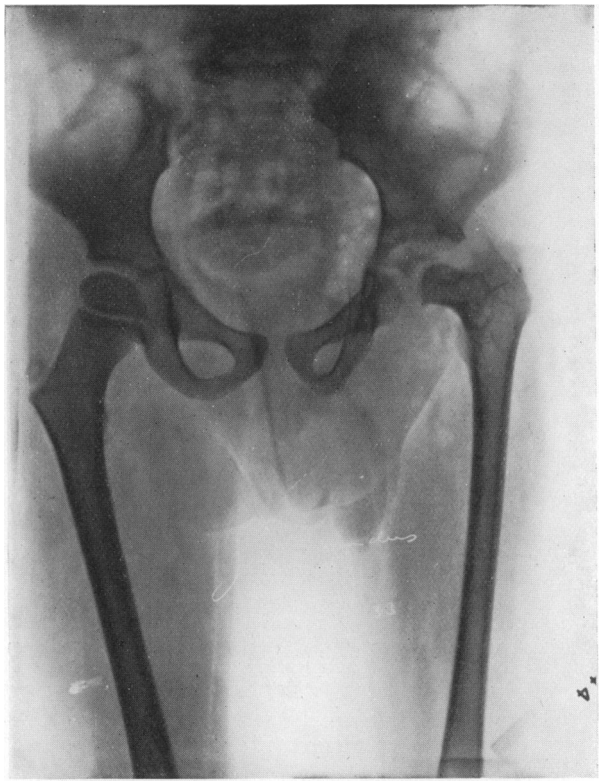

FIG. 4.

Advanced changes due to tuberculosis in a boy aged six years. Onset at the age of eighteen months. X-ray taken in June, 1933, shows almost complete destruction of the head of the femur and part of the neck. He has been under treatment for four years for this and spinal caries. In spite of the great destruction of the joint, there is now forty-five degrees of free movement. Extension is being continued, and it is possible that the surfaces will be smoothed out and mobility permanently maintained. There is considerable shortening due to widening of the acetabulum and destruction and lack of growth in the neck.

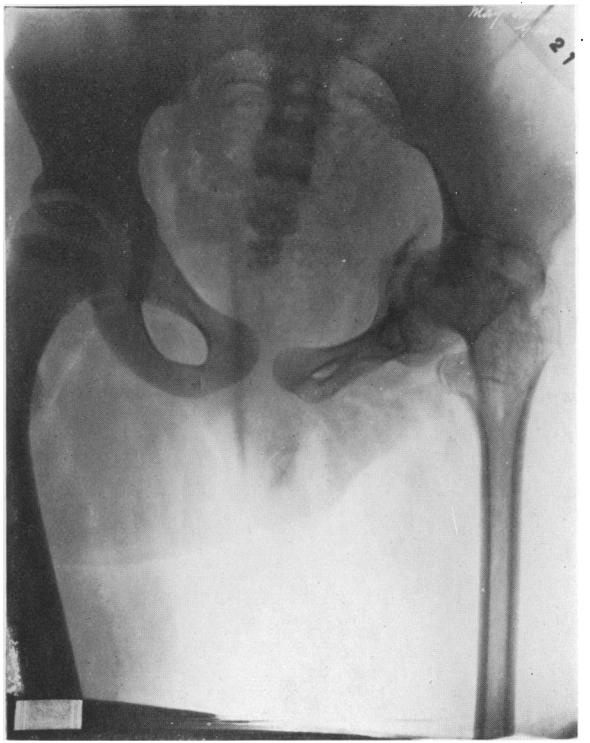

FIG. 5.

X-ray taken on 4th November, 1932, of a girl aged eleven. Onset 1927. In hospital from 1929 till 1933. The acetabulum is widened and the head of the femur has penetrated its base, a rare condition. Bony consolidation is good, and a new base to the acetabulum is being laid down.

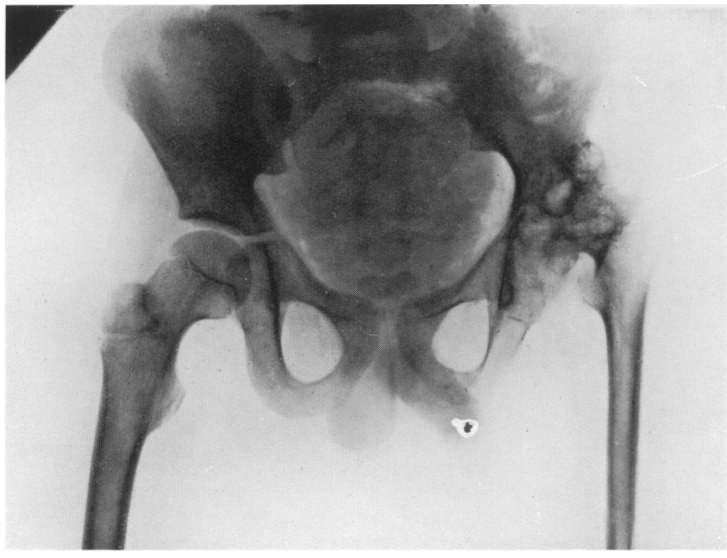

FIG. 6.

X-ray of a boy aged thirteen who has been under treatment for eight years. Massive calcification is present, but true ossification is incomplete.

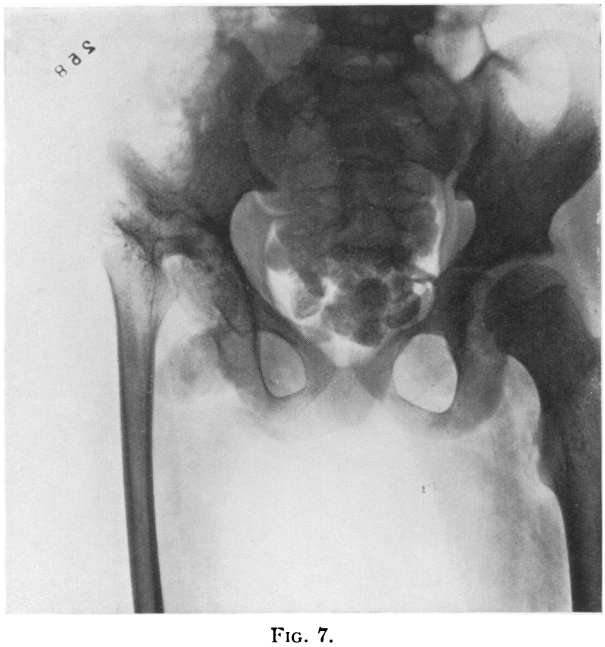

FIG. 7.

X-ray of a boy aged nine who has been under treatment for seven years. The acetabulum is much enlarged, and the great trochanter has fused with its upper border. Compare with Fig. 6. Ossification is present as opposed to mere calcification, and the structure of the bone is becoming visible.

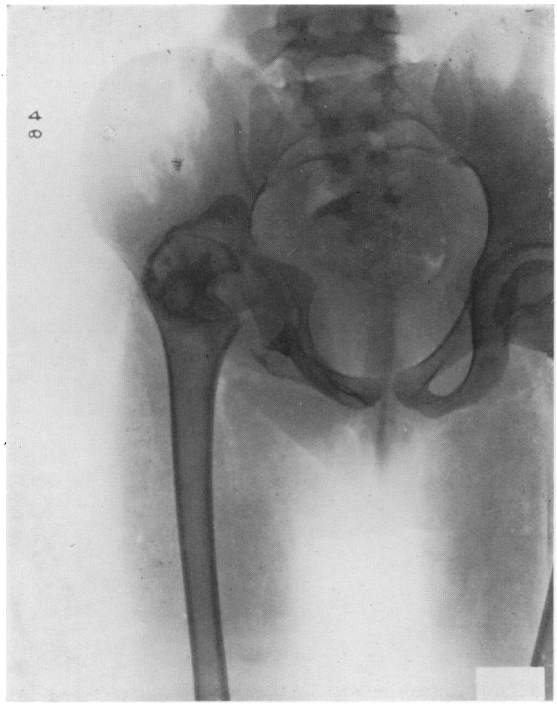

FIG. 8.

X-ray of a girl aged eleven who is also suffering from spinal caries, and who has been under treatment for five years. The acetabulum is enlarged upwards and the head of the femur has disappeared. Disease is arrested, and the opposed bony surfaces have become smooth, allowing nearly ninety degrees of free and painless movement.

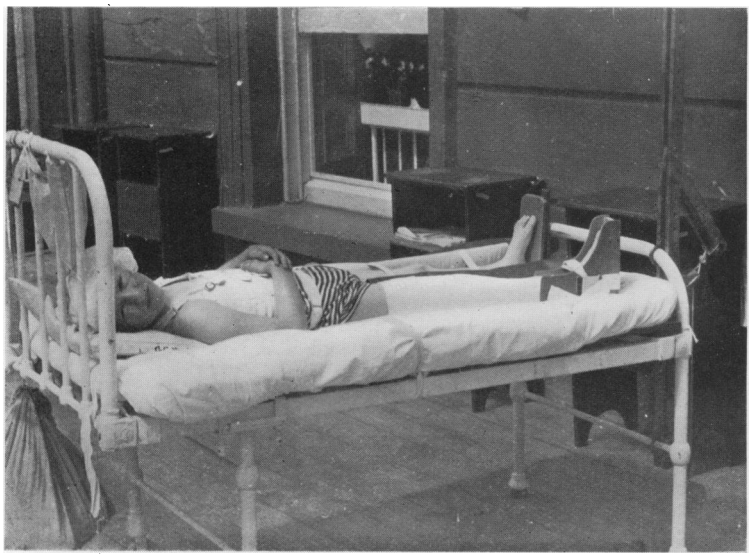

FIG. 11.

Extension applied to the right leg by webbing and bandages. A footpiece of three-ply wood serves to keep the foot vertical, to keep the heel off the bed, and the bedclothes from resting on the toes. A long Liston splint with a footprop is applied to the sound side to keep the patient in position.

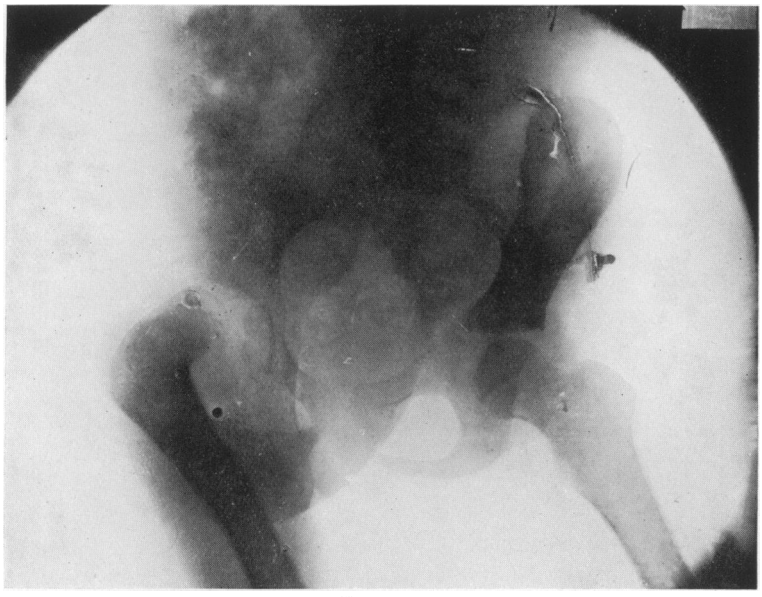

FIG. 9a.

X-ray taken in 1921 of a girl aged eight years, showing rarefaction amounting almost to cavitation in the head, neck, and great trochanter, and partial dislocation. Five years' treatment in hospital. She also had lumbar caries, dactylitis, and disease of a rib. In all four situations there was abscess formation.

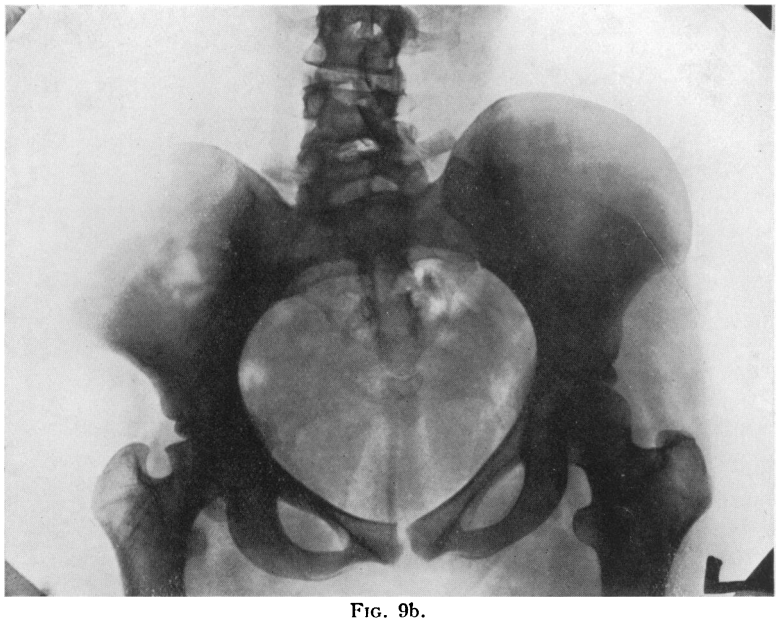

FIG. 9b.

X-ray taken in 1933 of the same girl shown in Fig. 9a. The neck of the femur is shortened, but the structure of the bone is normal and the head has maintained its smooth convexity. There is full movement in every direction, and in spite of about two inches shortening she walks without any noticeable limp. Photographs of this girl on admission and on discharge are shown in Figs. 10a and 10b.

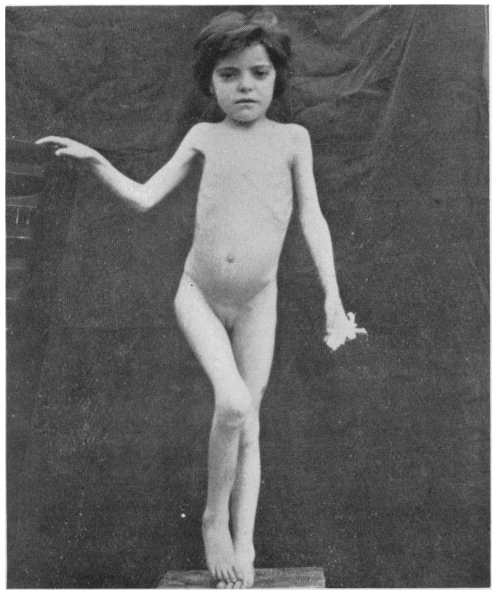

FIG. 10a.

On admission, 1921. Hip-joint disease, spinal caries, dactylitis, and disease of a rib. The hip is rigid in the adducted and flexed position.

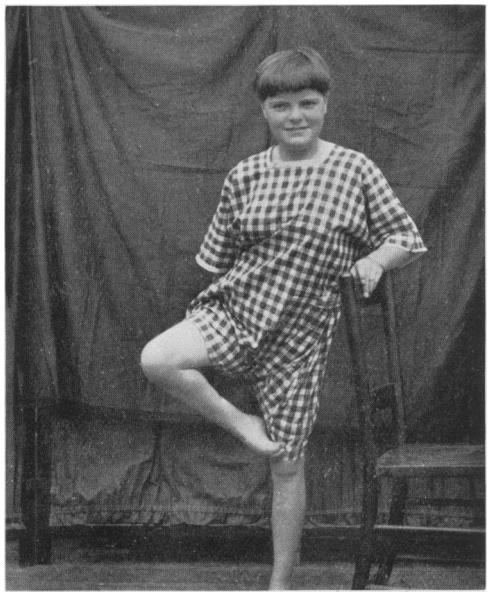

FIG. 10b.

The same girl on discharge, 1926. There is full mobility of the hip. All lesions are healed.

sufficient. The extension has to be reapplied about twice weekly, and for that reason it is useless in the treatment of fractures. Sometimes when the bandages are not firmly enough applied they slip a little, and irritate the skin just above the patella. An adhesive extension can then be used for a few days until the skin is normal again. Adhesive extensions cannot be used for the prolonged periods necessary without causing still more serious irritation of the skin.

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The least that can be done in an acute case is a long and deep incision down to the bone through the periosteum. Subperiosteal pus thus escapes and drains well; and the internal pus slowly makes its way to the surface through minute channels in the bone.

The bone deprived of its periosteum is dead, and will later be separated from the living along a line of demarcation.

By this method the pus in the medullary cavity is not directly reached : it can only work to the surface with difficulty. Better and quicker drainage can be achieved by trephining or drilling through the bone cortex. This is the technique employed by many.

A natural advance upon the last treatment was when it was decided to remove at the primary operation much of what was going to be dead bone, and so doing in a few minutes what nature would take weeks or months to perform. Thus anything up to one-half of the cortex of the bone was removed, and the medullary cavity left open as a gutter. This certainly reduced convalescence, but withal a dirty wound remained, and small spicules of bone discharged at their own slow rate, extending into months. This probably is the technique most universally employed at the moment.

The next and obvious advance was when it was decided to remove the entire shaft of the bone, leaving the periosteum. It was rightly agreed that if the periosteum could replace one-third to one-half of the bone when the latter was removed, why would it not do the same when the entire bone was removed?—and thus the operation of "diaphysectomy" came into popular favour. Difficulties arose naturally, e.g., in the case of a single bone like femur and humerus—the soft tube of periosteum was unsupported, and could bend with the weight of the limb or become telescoped by the pull of the muscles; thus the new bone was found to grow both crooked and shortened. This was not so likely to happen in the case of parallel bones, e.g., radius and ulna, as the sound bone remained as a splint. To prevent the shortening, methods such as extension, or the insertion—temporarily—of a glass rod of equal length to the bone removed, have been tried. Occasionally it happens that bone regeneration does not take place, and a bone-graft is needed. Such failures naturally make the cautious surgeon prefer the older operation. It entailed a longer period of illness with the discharge of dead bone, but the possibility of a flail limb was never to be feared.

The immediate treatments thus can be summed up under (1) incision down to the bone, (2) incision and drilling into the medulla, (3) incision and gourging a large gutter, (4) incision and removal of the shaft (leaving the periosteum).

Various modifications of the above to suit the individual surgeon, to suit the patient or the particular bone involved, represent the immediate treatment as generally practised.

The sterilizing of the cavity and the subsequent healing of the wound again offers scope for surgical enterprise. In this again the extremes are found : on the one hand is the surgeon who insists on the part being dressed every two, three, or four hours, or even with continuous irrigation; and on the other the man who fills the cavity with sterile vaseline, encases the limb in plaster of paris, and the wound is untouched for six weeks or more. With the latter, since no nursing is required, the child is sent home.

The obvious disadvantage is the appalling foul smell of the undressed wound, which must make life uncomfortable for the patient and the home uninhabitable for its friends. As one surgeon says, the case is heralded in the extern by its odour

long before it is visible, and memories remain in an appreciable form long after it has gone.

In hospital the case can only be dealt with satisfactorily if treated on the balcony. Contrary to expectations, the result of this treatment—Winnett Orr method; Nebraska—are good. The wound, when cleaned of the pus in which it is bathed, shows a healthy granulating surface, and usually epithelializes quickly. For those who favour frequent dressings, the substance used varies with the personal tastes of the operator. Glycerine, vaseline, B.I.P.P., eusol, hydrogen-peroxide, iodoform in paraffin, etc., are used, to mention a few.

At the moment the Orr method of infrequent dressings at six-weekly intervals is practised widely, either as advocated or in a modified form. For the mal odour, cyanide gauze or B.I.P.P. seems to "temper" the air. To the patient the daily terror of dragging adherent dressings from a painful wound is done away with, and any sequestra left behind seem to work their way to the surface. Those in favour speak in glowing terms, but it has firm opponents in men such as Elmslie, whose opinion, after many years of experience in such matters, naturally must carry much weight. The outcome of the treatment is that it has modified greatly the dressing of these cases, and although many do not carry out the Orr treatment to the strict letter of the law, they have embodied it in their own practice. Although this method of treatment goes by a special name, and is often referred to as a new technique, it is not so. It was originally employed by Sir Wm. Arbuthnot Lane in the Children's Hospital, Great Ormond Street, London, and still is the underlying basis of their method of treatment.

For the cases in which sinuses persist to discharge for months, many methods are tried. Probably to "give it a scrape" is the most popular, although the least satisfactory. With good drainage, removal of sequestra and sclerosed bone, and the filling of the cavity with a mobilized flap of soft tissue, persistent sinuses should not occur.

In the "débridement" of these wounds since mechanical and chemical means so often fail, the use of living maggots as scavengers has been employed. This treatment goes back as far as Ambroise Parré, and farther. It was practised during the American War of Secession, but it was an incident during the last war which—again—brought it to the forefront. Two soldiers with compound fractures of femur and extensive abdominal and scrotal wounds were found lying untreated and undressed after seven days, with the wounds filled with maggots. The men were in good general condition (comparatively), and the wounds in a healthy healing state. The late Dr. Baer of Baltimore tried to put this method upon a sound basis. The maggots used are the larvæ of the Blowfly—the green-bottle and blue-bottle. Naturally they must come from a cultured strain to prevent pathogenic bacteria being carried into the wound in addition. For five days these larvæ wriggle in the wound, working their way into the farthest recesses, there to eat dead tissue. At the end of that they pupate and are killed and replaced by another "team," and so on until the wound shows clean granulating tissue. The maggots will not as a rule attack an intact surface, although in inaccessible regions like the nose and ear they may do great damage. Usually they can be controlled by chloroform or

turpentine. The treatment has found little, if any, support in this country, for many reasons. It is expensive and difficult. It may cost anything up to £14 in a single case, and the incubating and production of the eggs, all through the cold as well as the hot months of the year, naturally entails trouble and expense.

The repulsive appearance to patient and surgeon of a wound filled with these animals makes the treatment have a limited application, and so older methods possibly entailing a slightly longer convalescence are still likely to hold the field.

Prognosis.

The prognosis in acute osteomyelitis is always serious, as the disease is essentially the local manifestation in a child of a blood-borne infection. This is evidenced by the multiplicity of the lesions in many cases in which parts, although widely separated, are simultaneously affected, and, secondly, by the fact that many succumb to a general septicæmia. The mortality varies between twenty and sixty per cent., depending upon the source. The greatest number succumb within the first four days, and many about the tenth day. At autopsy a pericarditis or other signs of a septicæmia or pyæmia are a frequent finding.

The prognosis in the acute stage is influenced by several factors :—(1) The duration of the patient's illness prior to the liberation of the pus, (2) the site of the lesion, (3) the degree of toxæmia, (4) the type of bone, (5) the presence of septicæmia.

Taking these seriatim, the first is self-evident. Pus unless liberated usually tends to accumulate and spread. Subperiosteal spread always means further bone destruction and greater toxæmia.

Of the bones involved, each carries with it its own prognosis. It is obvious that the disease when present in the fibula—a bone whose entire absence from the body appears to cause little, if any inconvenience—is very different from that when present in the body of a vertebra. From this a spread backwards may involve the cord, and forwards may pass into the mediastinum or other areas beyond surgical reach. The extremities, which are the easiest to diagnose and treat, carry the best prognosis—certain exceptions exist, such as os calcis and the bony prominences adjacent to joints. In the pelvic girdle and scapula, the diagnosis is often overworked. In the spinal column, as mentioned, the disease is difficult to diagnose, and treatment, if the lesion is present in the bodies, is fraught with such technical difficulties that the prognosis is correspondingly bad.

Toxæmia with a high temperature is a feature of the disease; but in those cases with acute toxæmia, with a burning skin, a temperature of 105-107, a patient very ill and wildly delirious, the outlook, even to the lay mind, is obvious. The patient is unable to assist in localizing the affected area, and if physical examination can reveal nothing, a definite diagnosis cannot be made. In such cases it is customary to assume that osteomyelitis is the cause. By some it is advocated that as many as possible of the bones most frequently involved should be drilled. I have done this same three or four times, but cannot yet claim to have had any success. General measures such as sera and blood transfusions would appear to be a more rational treatment, but have had disappointing results.

The prognosis will vary with the type of the bone—as distinct from the site of the bone. Of bones developed in cartilage, whether ivory or cancellous in texture, the progress of the disease runs a similar course, but it is in those bones developed from membrane that bone destruction and bone construction differ. In the acute stage this may not be such an important point, but in the chronic stage it is most noticeable when one remembers the poor and slow attempt that membrane bones make to produce new bone—this is most noticeable in the skull and mandible.

For the presence of septicæmia one must always be in readiness. A personal case upon which I operated within thirty-six hours died after running a swinging temperature for a week. At autopsy the original bone infection was satisfactory, but death was due to multiple abscesses, including a large one in the interventricular septum of the heart. This often cannot be foreseen at the operation, but it makes the prognosis guarded for some time, no matter with what satisfaction to the surgeon the operation has been performed. Upon the above points one may base an immediate prognosis, but the disease is not finished there, and before the patient goes home the parents should be warned regarding the further points—

(*a*) The limb may be shorter than its fellow, due to the disease having destroyed the epiphyseal cartilage upon which growth in length depends.

(*b*) The limb may be longer than its fellow, because the inflammation subjacent to the epiphyseal cartilage has stimulated the latter to greater than normal bone-growth.

(*c*) The limb may not grow straight, because one side (medial or lateral) of the epiphyseal plate was stimulated (or restricted) while the other side was allowed to grow normally.

(*d*) A fracture—spontaneously or due to a trivial injury—may occur. This is due obviously to the bone which is weaker at the site of the operation, and, secondly, to the new periosteal bone, which has not the elasticity and resilience of the child's normal bone.

(*e*) The possibility of "flare-ups" or recrudescences must always be borne in mind. As time progresses the liability to this diminishes, but everyone has seen cases—considered healed—relighting after ten, twenty, or thirty years. A minor injury or a slight sprain seems to be the deciding factor. But no mother should ever take her child home without realizing such a possibility.

(*f*) A dull ache—more common in the adult—in a healed osteomyelitis is frequent. The pain is greatly affected by the temperature of the body, and is probably due to the impeded circulation of the blood through the very dense bone. The appearance presented in an X-ray of chronic osteitis, while it helps one to realize that this is a very real and troublesome disability, also helps to dispel the fear that the dull ache is a residual abscess or a further sequestrum.

P.S.—Since writing the above there has appeared in the "British Journal of Surgery" a critical survey of 262 consecutive cases of acute osteomyelitis. The chief conclusion drawn is that the best results are obtained with the more conservative surgical methods.

The Treatment of Congenital Talipes Equino-Varus

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Talipes Equino-Varus is the most common orthopædic problem which the newly-born child presents to the doctor or surgeon. It has been estimated that one baby in every thousand is born with some variety of foot deformity. The vast majority of these deformities belong to the type we are at present considering. The analysis, given below, of 118 cases of congenital foot deformities treated at the Ulster Hospital during the past five years, is fairly well in keeping with the figures published from other centres :—

Talipes equino-varus - - - - -	99
Talipes calcaneo valgus - - - - -	14
Talipes equinus - - - - - -	1
Talipes equino-varus on one side associated with calcaneo valgus on the other - - - - -	2
Talipes equino-varus on one side associated with pes planus on the other - - - - -	1
Metatarsus varus - - - - -	1
	118

Thus, out of 118 cases of congenital foot deformities, 102 (or eighty-six per cent.) had either a unilateral or a bilateral equino-varus present.

Importance of the Condition and Necessity for Early Treatment.—The condition is for various reasons an important one. Treatment commenced early in life and properly applied over a long period will end with the complete cure of many cases, and in all great improvement can at least be promised. When treatment is neglected in the early weeks or months of life, or is inadequately carried out, a very unsightly deformity may result. Even the early case may prove very resistent to all methods of treatment, and it may be that years of careful work on the part of the surgeon will in the end result in a moderate or only fairly good foot. How much more then may the neglected case be expected to present a long and difficult problem, painful for the patient, expensive for the parents and for the hospital, and trying and time-consuming for the surgeon.

Nature of the Condition.—Before discussing the treatment of congenital talipes equino-varus, a short reference must be made to some of the more important points concerning the nature of the condition. We shall here consider only those cases in which the deformity has occurred in an otherwise normally developed child. Space would not permit of reference to the treatment of cases of equino-varus associated with other congenital abnormalities, such as spina bifida, congenital absence of the tibia, absence of some of the toes and fingers, or of failure of development of the calf, anterior tibial, and peroneal muscles, etc.

There is some difference of opinion in regard to the part played by the bones, by the muscles and tendons, and by the ligaments and fasciæ, in the production and maintenance of the equino-varus deformity. It is now fairly generally agreed that the cartilages and bones of the foot are primarily normal in shape, and that such deformity as may occur in them is a secondary adaptation to a long continued abnormal position of the foot. Likewise, it is felt that although the everting and dorsiflexing muscles of the foot are elongated and weak, they are not the underlying cause of the trouble and will recover when the position and function of the foot are restored to normal.

ANALYSIS OF THE DEFORMITY.—Whatever the ætiology may be, the deformity can be best understood by considering it as a compound one, made up of a combination of abnormal positions of fixation existing at certain joints of the foot and sometimes at the ankle. Thus the deformity is a combination of :—

(1) Plantar flexion at the ankle-joint. The degree of fixed plantar flexion or equinus at the ankle-joint always appears very much greater than it really is. A careful study of X-ray pictures of talipes equino-varus feet and a comparison of the position of the head of the astragalus before and after correction show that a fairly marked condition of equinus deformity may appear to exist at the ankle-joint on external examination of the foot, when in reality none is present. This will be mentioned further when discussing treatment by tenotomy.

(2) Inversion of the os calcis underneath the astragalus at the sub-astragaloid joint carrying with it the fore-foot. (The hind-foot is the part lying behind the midtarsal joint and made up of the astragalus and os calcis.) In this the foot is rotated inwards around an axis parallel with the long axis of the os calcis, so that the plantar surface of the foot comes to face inwards.

At the midtarsal joint situated at the junction of the fore-foot and the hind-foot—

(3) Plantar flexion
(4) Adduction
(5) Inversion

} of the fore-foot on the hind-foot.

In any individual case of talipes equino-varus, the degree to which any one of these above deformities may be present in combination with the rest varies considerably. One case may have a severe grade of adduction associated with relatively mild grades of inversion and plantar flexion, whilst in other cases the inversion or the equinus may be the predominant element in the compound deformity.

VARIABILITY OF TYPE.—Clinically, it is found, that of two cases, each suffering from a similar degree and type of deformity and commencing treatment at an early age, one may respond readily to manipulative efforts and the other may be extremely resistant to or even impossible to correct by manipulation alone. It may therefore be said that the degree of rigidity present varies from case to case. There have been many theories advanced to explain this varying rigidity. The wide divergence of opinion existing in regard to its causation affords an explanation for the many different types of operation that have been recommended for the rigid foot.

General Principles of Treatment.—Before referring to the various methods of treatment in more detail, it may be well to consider some of the general principles that must be observed if success is to follow our efforts.

It is most important that *treatment should commence as soon after birth as possible.* Delay in commencing treatment carries with it many disadvantages—the necessity for more forcible and more numerous manipulations; more applications of strapping and plaster; more visits of the parents to the hospital; more anæsthetics; more open operations; and in the end a greater proportion of poor or bad results.

The necessity for *over-correction of the deformity* is also of importance. It is not enough simply to correct the deformity: it must be over-corrected if a perfect result is to be obtained. Before being satisfied that over-correction has been sufficiently carried out, one should be able to push the foot into such a position with light pressure of the finger on the sole.

After obtaining satisfactory over-correction, it is important to maintain it until the stretched and weakened muscles on the front and outer side of the leg have regained their power to such an extent that over-correction can be produced by the patient's own unaided efforts. If treatment is stopped before restoration of muscular balance has been obtained, relapse of some degree is certain to follow. Massage and exercises to improve the tone of the weakened muscles after further retention in plaster or strapping is no longer necessary; the provision of boots with strong uppers, the use of a club-foot shoe for a time, and the wedging up of the outer border of the boot, are important adjuncts to treatment while the weakened muscles are gradually recovering their tone.

When restoration of normal position and function have been obtained after a few months' treatment, and the child has not yet reached the walking age, it is important to insist on the *necessity for regular re-examination* until the child has begun to walk and for some months after this. It is not uncommon to see children, who have been declared cured at the age of six to nine months, return after an absence of two or three years with a severe grade of recurrence. These recurrent cases are proverbially difficult to cure. It may be that the trauma of former manipulations has resulted in the production of scar tissue around the joints, and that this now adds to the difficulty of reduction.

Methods of Treatment.—These may be tabulated as follows:—

Correction by means of splints.
Manipulative reduction.
Operations on tendons.
Operations on ligaments and fasciæ.
Bone operations.

Correction by Means of Splints.—Many splints have been devised to secure correction of the deformity, but the majority of orthopædic surgeons are agreed that no splint is entirely satisfactory for the purpose. I have not had any experience of this method, and am not competent to give an opinion regarding it.

MANIPULATIVE REDUCTION.—Repeated manipulations, with retention of the foot, between the treatments, in adhesive strapping, plaster of paris casts, or various splints, is now generally considered the best method of treatment. In the majority of cases in which manipulations are commenced early and properly carried out, complete cure can be obtained. In neglected or late cases manipulative treatment alone can be expected to yield a fair percentage of good results. A certain number of the early cases do not yield to manipulation, and the older the patient and the more severe the deformity the less likely is such treatment to be successful. Nevertheless, manipulative treatment should be tried in every case before proceeding to more drastic measures. It is impossible to say that because a child is nearing adolescence, and has a gross degree of deformity, that correction by manipulation is impossible.

When manipulating a deformed foot, one has two objects in view :—

(*a*) The stretching out of the shortened soft structures on the inner and under surfaces of the foot;

(*b*) The restoration of the bones at the tarsus to their normal relationships to one another at the various joints.

It is desirable to secure these objects, if possible, long before the child begins to walk. During manipulations, considerable force is required, and the aid of a wrench is often necessary. It is important to avoid any strain on the ligaments of the knee by flexing it and having the upper part of the leg grasped firmly by a nurse or assistant. The limb being steadied in this way, the operator grasps the heel firmly with one hand, the thumb or fingers of which act as a buttress to the outer side of the os calcis. With the other hand the adduction of the fore-foot is corrected by stretching out the soft structures on the inner border of the foot. After overcoming the adduction deformity, the foot is now grasped around the back of the heel, over the insertion of the tendo achilles, with one hand, the index finger or the thumb of which supports the back of the external malleolus; with the other hand strong pressure is brought to bear on the inner and under surface of the fore-foot at the level of the metatarsal heads, dorsiflexing, everting, and abducting it. By this manœuvre the structures on the inner border of the foot are further stretched, and at the same time the longitudinal arch is opened out. During these manipulations a twisting strain may fall upon the fibula, and unless the back of the external malleolus is strongly supported a fracture of the fibula may result.

General anæsthesia is often required when much rigidity of the foot exists.

In the equino-varus position, the head of the astragalus forms a marked prominence on the dorsum of the foot in front of and medial to the external malleolus. With over-correction, the interval between the sustentaculum tali and the tuberosity of the scaphoid is increased, and this permits of the head of the astragalus sinking down on the inner border of the foot. Now one finds, instead of the prominence, a distinct hollow in front of the external malleolus, and this is a good index of over-correction. Another reliable index is the appearance of the heel when

viewed from behind. As long as any deformity remains uncorrected, the os calcis will be inverted. Even a slight degree of inversion of this bone is readily noticeable, and if it exists when treatment is discontinued, more or less of the equino-varus deformity will recur (unless a fusion operation on the tarsus has been performed).

Manipulations are usually required once or twice per week for many months. In the intervals between the manipulations it is essential to maintain correction as far as is possible within the limits of safety. Adhesive strapping, plaster of paris, or malleable splints may be used for this purpose. In the majority of cases adhesive strapping is the most effective and most readily applied method, though in some a plaster cast is more suitable. Adhesive strapping may be applied effectively in various ways. Good fixation can be secured by three strips, each from one to two inches wide, according to the size of the foot. The first strip is used to fix the heel and maintain it in eversion. Beginning over the internal malleolus, it passes down from this transversely across the under surface of the heel and, having been made taut, is fixed to the outer side of the leg as far as the knee. The second strip maintains dorsiflexion, eversion, and abduction. It begins on the inner side of the dorsum of the foot at the level of the first metatarsal head, passes slightly obliquely outwards and backwards across the sole, and, being made tight, is fixed obliquely across the back of the calf of the leg. The third strip, beginning over the heel, passes obliquely across the antero-lateral surface of the leg, and fixes the first two strips more firmly in place. Because of the delicacy of the infant's skin, there is a tendency for sores to form on the outer border of the foot under the strapping, and the preliminary application of a piece of lint or adhesive felt is often useful in this position. If the foot is strapped too tightly it is possible to interfere with the circulation of the foot as a whole. One should be satisfied to fix the foot in a position of correction a little less full than that obtained by manipulation.

Operations on Tendons.—Tenotomies have long been practised in the treatment of clubfeet. Tenotomy of the tendo achilles has had in the past a wide popularity. It was hoped that by means of this simple operation correction of the equinus deformity might be obtained. It is common in a severe case of talipes equino-varus for the patient when walking to be unable to put the under surface of the heel any nearer than from one to two inches from the ground. On examination of such a case, it would appear that most of the equinus deformity is present at the ankle-joint, but in reality this is not so. Careful determination of the position of the head of the astragalus, lateral X-ray pictures taken with the foot dorsiflexed as much as possible, and accurate measurements, will show that the equinus deformity is practically always largely due to plantar flexion of the fore-foot on the hind-foot. Tenotomy of the tendo achilles for a condition such as this, by causing a dropping of the heel, will increase the plantar flexion at the midtarsal joint. Further shortening of the already contracted soft structures will result, and increase rather than diminish the difficulty experienced in getting rid of the plantar flexion and inversion present at the midtarsal joint. The number of cases in which tenotomy of the tendo achilles is ever required is very small indeed. As a first step, or indeed anything else than a very last resort in the treatment of congenital talipes equino-

varus, it should be condemned as being usually unnecessary and frequently adding greatly to the difficulty of subsequent correction.

Tenotomy of the tibialis posticus or anticus has not been so commonly practised. Their division may help in some cases. However, such operations are seldom necessary and at best of doubtful value.

TENOTOMY OF THE PLANTAR FASCIA AND MUSCLES as an aid to manipulation is worthy of a trial for the very rigid foot that has failed to respond, before embarking on more drastic operative procedures. It may be helpful when the plantar fascia and muscles are felt to be very taut during an attempt to flatten out the longitudinal arch of the foot.

BONE OPERATIONS.—When the above procedures have failed to correct the deformity, recourse must be had to more radical measures. Numerous operations have been devised and practised for those difficult feet that resist all manipulative efforts. It is not possible here to more than mention some of the most important ones. These radical operations may be classified into three main groups :—

(*a*) *Operations confined to division of shortened soft structures.*

1. Phelps operation : Division of all the soft tissues on the inner border of the foot through a vertical incision just in front of the internal malleolus.
2. Ober's operation : Detachment of the deltoid ligament from the internal malleolus and the sustentaculum tali, and removal of the internal and inferior calcaneo-scaphoid ligaments, with sometimes division of the plantar fascia as well.
3. Brockman's operation : Division of the plantar fascia and separation of all the structures from the under surface of os calcis, together with removal of the internal and inferior calcaneo-scaphoid ligaments.

(*b*) *Operations on bone.*

1. Astragalectomy.
2. Removal of bony wedges from the outer side of the foot.
3. Modifications of Dunn's operation, or arthrodesis of the midtarsal and subastragaloid joints.
4. Osteotomy of the tibia.
5. Division of the external malleolus.

(*c*) *Various combinations of* (*a*) *and* (*b*).

Elmslie's operation : Removal of the anterior two-thirds of the deltoid ligament, the internal part of the astragalo-scaphoid capsule, and also the inferior calcaneo-scaphoid ligament plus osteotomy of the neck of the os calcis and, if necessary, the neck of the astragalus.

In discussing the more radical operations as a whole, it should be pointed out that bone operations should, as far as possible, be avoided. When manipulative measures have failed to correct the deformity, the next step should be subcutaneous tenotomy of the plantar fascia and muscles followed by further manipulation if plantar flexion forms a large part of the deformity. Should success still not be

secured, then more radical operations on the soft structures, such as Brockman's or Ober's, should be given a thorough trial. With these latter procedures correction of the deformity can be obtained in practically all cases. Personally, of these operations, I prefer Brockman's, because I feel that it permits of more thorough division of the contracted structures on the under surface of the sole. Bone operations may sometimes be required in the late or very deformed case. They should be undertaken only when, by manipulation and division of the soft structures on the inner and under surface of the foot, as much correction as possible has been obtained. When a bone operation is necessary, every effort should be made to remove as little bone as possible. The operation of astragalectomy, where a large piece of bone is removed, is almost invariably followed by a relapse of the foot into a more exaggerated position of equino-varus than ever, and the operation should be condemned as a method of treatment for a congenital equino-varus deformity of whatever grade. The dreadfully deformed foot that may and often does follow this operation is almost impossible to improve by any means. It is only after the deformity at the midtarsal joint has been corrected and some equinus deformity is still found to exist, that lengthening of tendo achilles should be performed.

RESULTS OBTAINED AS FOUND BY FOLLOW-UP IN 99 CASES OF CONGENITAL TALIPES EQUINUS-VARUS TREATED AT THE ULSTER HOSPITAL OVER A PERIOD OF FIVE YEARS.

TREATMENT	RESULTS*		
	Bad or Poor	Fair	Good or Perfect
Manipulation alone (56 cases)	31.3%	14%	54.7%
Treatment commenced within a few months of birth	28%	12%	60%
Treatment commenced later, but before walking age	50%		50%
Treatment commenced a varying time after walking began	27%	23%	50%
Tenotomy of tendo achilles early in life, plus manipulation (15 cases)	66%	20%	14%
	(Cases)	(Cases)	(Cases)
Astragalectomy (8 cases)	7	1	0
Wedge removed from outer side of foot (1 case)	0	1	0
Brockman's Operation (12 cases)	2	1	9
Elmslie's Operation (1 case)	0	0	1
Ober's Operation (4 cases)	3	0	1
Knocker's Operation (2 cases)	0	2	0

**Bad Result* - Walking very badly with boots on or off. Unable to run.

Poor Result - Intoeing with boots on, but walking fairly well. Runs badly in bare feet.

Fair Result - Walking well in boots. Fairly obvious deformity, but runs and walks fairly well in bare feet.

Good Result - Practically no deformity. Only intoeing a little when running on bare feet.

Perfect Result - Indistinguishable from normal.

Inguinal Hernia in Infancy

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Two opposing views are held regarding the nature of congenital inguinal hernia, one championed by Hamilton Russel and the other by Sir Arthur Keith. Russel[1] believes that the presence of a developmental pocket of peritoneum is all that is required to produce a hernia. But in mammals, other than man, the cavity of the processus vaginalis remains patent throughout life, and in them congenital inguinal hernias are almost unknown. The sac itself, therefore, cannot be the only factor involved. Keith,[2] on the other hand, holds that a pocket of peritoneum is not essential, that with or without a pocket of peritoneum a hernia can be produced by the gut forcing its way into the inguinal canal, through the shutter-like action of the internal abdominal opening between Poupart's ligament and the conjoined tendon. When the conjoined tendon is relaxed, Keith states, there is an interval between its lowest border and Poupart's ligament, and if a strain is put upon the two muscles which form the tendons, they will contract, close the opening and prevent the protrusion of the gut. Failure of contraction, according to this view, will lay the groin open to a hernia. Doubt is cast on the truth of this view by the study of the comparative anatomy of lower mammals. Man is the only animal which possesses a Poupart's ligament, except perhaps the gorilla. In these lower forms the external oblique muscle of the abdomen is carried into the thigh as a flattened sheet, attached to the anterior border of the ileum, and it is continuous with the deep femoral fascia, the fascia over the psoas, and through it with the fascia transversalis. Keith's view of the shutter-like action of the conjoined tendon and Poupart's ligament cannot therefore account for the absence of hernias in these animals. And even in the human subject there is such a wide interval between Poupart's ligament and the lower border of the internal oblique muscle, that it does not seem possible for a closing action between them to occur.

If a careful dissection is made of the inguinal region of a newly-born male infant, the most striking feature seen is the relatively wide arch made by the fibres of the internal oblique muscle, which sweeps medially to join the aponeurosis of the transversalis muscle. The lower border of the internal oblique muscle is separated from the inguinal ligament by such a wide interval that it is not possible to think that they could meet as Keith suggests, when the abdominal muscles contract. Behind the internal abdominal opening, lateral to the point of exit of the spermatic cord, is a strong sheet of transversalis fascia. This fascia is attached to Poupart's ligament laterally, and curves upward and medially over the spermatic cord. It appears in the dissection as a stout, well-developed falciform fold, continuous with the sheet of fascia attached to that part of Poupart's ligament which lies to the medial side of the internal abdominal opening.

If this same region is dissected from the deep aspect, the concave lower border of the posterior wall of the rectal sheath is seen to end medially in continuity with

the linea alba, and through it to an attachment on the pubic bone. The outer extremity of the fold is continued downward to the deep surface of Poupart's ligament at the inner side of the internal abdominal opening. These latter fibres constitute the ligament of Hesselbach,[3] and are described as a thickened part of the transversalis fascia. The lower border of this band forms a distinct internal pillar to the internal abdominal opening, where its fibres divide and pass in varying directions. Some of these fibres are attached to the ligaments covering the horizontal ramus of the pubis and to the pectineal fascia, others pass around the internal abdominal opening, and form an inferior or horizontal pillar, the fibres of which are continuous with the psoas fascia.

If a "window dissection" is made in the inguinal region of a fresh unpreserved infant, the transversalis fascia can be seen to present varying degrees of tautness, according to the position in which the thighs are placed. When the thighs are flexed and abducted, it is slackened; when they are extended and adducted it is tightened, and in this position it can be seen to guard the internal abdominal opening.

A living infant lies with its thighs flexed, abducted and externally rotated, i.e., in the position in which the transversalis is slack, and in which its guarding action at the internal abdominal opening is of the slightest. But when the child learns to walk, its thighs are extended, adducted and internally rotated, i.e., in the position in which the transversalis fascia is put in its most taut position, and the internal abdominal opening closed and firmly guarded. Now, it has been stated by Keith[1] that forty-four in every thousand infants suffer from hernia in the first year of life, and only nine in every thousand in the succeeding four years. As the child in his first year lies in the position of least advantage to produce a closure of the internal abdominal opening, is it not suggestive that the large percentage of hernias in the first year is directly related to this posture?

Then again, it is a common practice among the poorer classes to tie a skein of woollen yarn around the groin of an infant which shows a palpable hernia, as a kind of simple rupture belt. By this means, they say, the hernia is "cured" by the time the child begins to walk. A history of this kind is frequently elucidated in questioning patients who come to hospital with what is considered an "acquired" inguinal hernia, which developed suddenly on making an effort whilst at work. Here there seems to be no question of doubt whatever, that the hernia was not "cured" by the skein of wool, but that the hernia had been restrained from enlarging, and that when the transversalis fascia had been stretched by the extension, abduction, and medial rotation of the thighs, when the patient began to walk, the hernia was restrained within the abdominal cavity, while the congenital hernial sac had remained patent, but empty. The raising of the intra-abdominal pressure in making a violent effort overcame the closing action of the fascia, and the gut shot out, as it were, into the sac which had contained it during early infancy.

A point strongly in support of this view is found in the study of comparative anatomy. In all mammals other than man, the transversalis muscle of the abdomen passes deep to the rectus abdominis muscle. Man is the only animal in which it

passes superficially to that muscle in the lower part of the abdomen. The fascia transversalis is intimately related to its overlying muscle, and receives strengthening fibres from it, and when the two—muscle and fascia—become separated from one another by the rectus abdominis, the fascia is left relatively thin and lax, and man alone commonly suffers from congenital inguinal hernia.

A consideration of these facts seems to suggest clearly that the form and position of the transversalis fascia, in its relation to the internal abdominal opening, has a direct bearing on the development of congenital inguinal hernia. It can readily be understood that a crying and wailing infant, lying with its legs flexed, abducted and externally rotated, by intermittently raising and lowering the intra-abdominal pressure, can easily cause a protrusion of gut into any abnormal pocket of peritoneum present. It would seem, therefore, that infants born with this crippling condition should have operative treatment to remove the sac at the earliest possible moment, before the transversalis fascia and the other parts immediately surrounding the internal abdominal opening have become stretched and injured. In early cases these structures quickly take up their natural positions, and no permanent incapacity will result in later life.

The safety and ease with which this simple operation can be performed is shown by the published results from different clinics. Fullerton[4] performed 300 operations within a short period, with not a single death; Styles[5] performed 360 operations with five deaths; Salzer[6] performed 350 operations with three deaths; Kovaces[7] performed 232 operations with three deaths; and Pfaehler[8] 46 operations with no deaths.

The hernial sac is a development anomaly associated with the phenomenon known as the descent of the testes. A short account of this phenomenon will therefore be given in order that the formation of the hernial sac may be understood.

The genital gland of each side is united at first to the deep surface of the anterior abdominal wall by a fold of peritoneum, the inguinal fold. A condensation of mesenchyme occurs between the layers of this fold, passing from the lower pole of the genital gland, mesonephros, and Wolffian duct, through the abdominal wall at the lateral side of the umbilical artery. This condensation is the anlage of the gubernaculum, and it ends in an indefinite manner in the region of the scrotum. It occurs, in the human embryo, at the 13mm. stage, i.e., at a time before the abdominal musculature is differentiated. When the latter begins to form, it must, therefore, grow around the cord, and the inguinal canal thus contains the gubernaculum from the beginning, and it marks out the path for the out-pocketing of the peritoneum which is known as the processus vaginalus.

About the middle of the twelfth week the gubernaculum grows rapidly, and the part immediately below each testis becomes bulbous in shape, with a fan-shaped base. At the fourth month the fibres of the fan-shaped portion separate into three distinct bundles, each of which again divides, and six bundles are formed. The first of the three primary bundles passes towards the saphenous opening, its secondary division passing towards the inguinal ligament. The second of the primary divisions passes towards the pubis, its secondary division passing towards the root of the

penis. The third primary bundle passes towards the scrotum, its secondary division passing to the perinæum. These bundles are unequal in size, and in the human subject the largest bundle normally passes towards the scrotum. (In the pig the largest bundle passes towards the perinæum, and in the kangaroo to a pre-penile position. In these animals the testes are found in the perinæl and pre-penile positions respectively.)

The gubernaculum reaches its greatest development about the middle of the sixth month of fœtal life, and then gradually becomes smaller and less easily defined, until at birth all the fibres have disappeared except those which pass to the scrotum. The structure of the gubernaculum is said to be composed of unstriped muscle tissue, but this is not in agreement with observable fact. Sections stained by Mallory's selective connective tissue stain demonstrate that it consists of true fibrous tissue.[9] Lying amongst this tissue can be seen a stream of rounded cells, each containing a small amount of cytoplasm. These cells first appear in the neighbourhood of the genital gland, and stream outward through the inguinal fold, along the main strand of gubernaculum. With the appearance of these cells the processus vaginalis begins to form as an outpocketing of peritoneum, and it also follows the line of the main strand of gubernaculum.

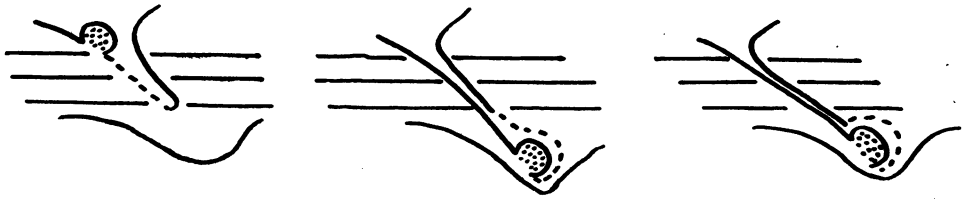

FIG. 1.

Diagrams to illustrate normal growth of the processus vaginalis, and the changes which occur during the descent of the testis.

The cavity of the processus vaginalis in a human fœtus of the sixth month is cone-shaped. Its apex ends in the midst of the muscular fibres of the internal oblique and transversalis muscles. Its base corresponds to the position of the internal ring, where its lumen is directly continuous with the general peritoneal cavity. If an attempt is made to raise the processus vaginalis, it will be found that some fibres of the gubernaculum are attached to its apex, and if this part is examined microscopically, the large rounded cells, already described, can be traced streaming along the main bundles of gubernaculum towards the scrotum. The processus increases in length until the end of the seventh month, when growth in length apparently ceases, its apex then reaching the level of the junction between the scrotum and the anterior abdominal wall.

When the testis descends, the peritoneum which is adherent to it is carried down into the scrotum, and normally there is just sufficient peritoneum to form a simple tube-like diverticulum continuous with the general peritoneal cavity (fig. 1), and this soon becomes obliterated along the length of the cord. But when the processus

vaginalis is longer than normal, there is an excess of peritoneum, and instead of a simple tube, its wall is folded upon itself, so that a secondary tube of peritoneum is found in any of the positions shown in fig. 2. It is a secondary fold of peritoneum of this nature which forms the sac of a congenital inguinal hernia.

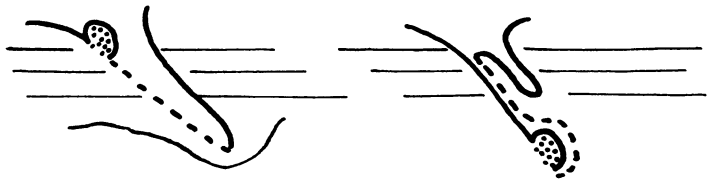

FIG. 2.

Diagrams to illustrate abnormal growth of the processus vaginalis, and the changes during the descent of the testis, which end in a potential hernial sac.

The transition of the testes from the abdominal cavity to the scrotum has been ascribed to a contraction of the gubernaculum. This, however, is not a satisfactory explanation, for it implies that the lower fixed end of the gubernaculum is *adherent to the lax scrotum,* and if the gubernaculum were to contract, the scrotum merely would be invaginated. It would appear that the view advanced by Bland-Sutton[10] is more in keeping with the known facts. He considers that the descent of the testes is an involuntary herniation, due to a raised intra-abdominal pressure. This occurs after the formation of a complete diaphragm between thoracic and abdominal cavities. Under such conditions, with the development of an active life involving running and jumping, high degrees of intra-abdominal pressure are produced, due to the fore-limbs and hind-limbs being in opposite phases of motion.[11] The two halves of the elongated trunk are upheaved when their respective limbs are brought into contact with the earth, and depressed in the interval of time between successive contacts. If an organ is embedded or in any way firmly fixed to the main bulk, it will readily share both the elevations and depressions incurred in locomotion, but if, on the other hand, an organ is merely suspended from the main mass of the body by a mesentery or ligament, it will be brought under the influence of the periodically recurring strains. The latter condition is applicable, to a large extent, to the testes within the abdominal cavity. It is not surprising, therefore, to find that the testes remain within the abdominal cavity in animals with a slow method of progression where there are no marked changes in intra-abdominal pressure, and protruded in those animals living an active existence, coming to lie in the position of greatest safety and are not subject to constant changes of pressure.

Among the monotremes, activity is of the lowest degree, and the testes retain their primitive position within the abdomen close to the kidneys.

Progression among the marsupials is by a series of leaps from the hinder limbs, and as a consequence each bodily depression is marked by a prolonged termination as compared with ordinary running movements. The testes are thrown forward and the pre-penile marsupial position is explained.

The testes of the sloths are retained within the abdomen, lying close together on the rectum.

In the aard-vark the testes occupy an intermediate position within the inguinal canal, but here there is reason to believe that they descend into the scrotum during the mating season.

The testes of active creatures like ungulates, carnivores, and primates lie within a post-penile scrotum, where they are free from violent changes in pressure and are kept in the position of greatest safety.

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THE ROYAL MATERNITY HOSPITAL, BELFAST

The great medical unit which is rapidly taking shape around the Royal Victoria Hospital, Belfast, is carried one stage nearer completion by the erection of the new Royal Maternity Hospital. This Hospital, which is to be officially opened, on the invitation of Her Grace the Duchess of Abercorn, by Mrs. Stanley Baldwin, on 21st October, 1933, replaces the incorporated Belfast Maternity Hospital founded in 1793. The old hospital did excellent work, but the immense advances in the science of obstetrics, and the growing need of an increasing population, rendered a new and modern hospital an absolute necessity. The plans of the new buildings were drawn up only after exhaustive study of modern practice both at home and abroad, and after the very latest maternity hospitals had been inspected in the U.S.A., Canada, the Continent, and Great Britain. And it is the firm belief of those competent to judge that the new Royal Maternity Hospital is in advance of anything at present in existence.

The hospital has been designed for one hundred maternity beds, and has been erected at a cost of £104,000. Unfortunately, sufficient funds have not been forthcoming to build a Nurses' Home, and the Committee of Management has been compelled, until this sum is provided, to allocate a portion of the building for the accommodation of the nursing and domestic staffs, thereby reducing the number of beds for maternity cases to sixty. Even with this regrettable reduction it is

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pleasant to know that the new hospital has more than twice the number of beds available at the old hospital in Townsend Street.

The hospital includes the following :—

A large Ante-Natal Out-Patient Department, where expectant mothers attend for advice and treatment. Here also mothers are encouraged to bring their babies periodically after they leave the hospital, for examination and advice.

Labour Suites where the confinements take place. These constitute a separate nursing unit with its own staff. There are four one-bed wards in which patients may spend the early stage of labour. In addition to the labour wards, which are equipped in all respects like operating-theatres, there is a theatre for cases which require operation, such as cæsarean section, etc.

There are two maternity ward units, each of twenty-one beds, and a number of small wards. Each bed has its own light, and there is a push-bell which causes an indicator to buzz in the nurses' duty room, and lights an electric lamp to indicate from which bed the call comes.

A series of nurseries for infants is also installed. The nurseries form a separate unit under the charge of a sister, and are under the general supervision of a pædiatrician. As no visitors are allowed to enter the nurseries, inspection windows are provided for interested relations. There are four nurseries for healthy babies, one for premature infants, and two for sickly infants who for any reason require isolation. Specially constructed trolleys carry the infants, in groups of four, to their mothers at stated intervals. The design of the nurseries is new in this country.

A special feature of the hospital is the complete separation of septic and other abnormal cases in isolation wards. These wards are provided with their own theatre, treatment room, nursery, and staff. They have been named after the late James Rea, of Belfast, who through his executors bequeathed the sum of £25,000 towards the erection of the hospital.

The educational side of the hospital has been fully developed. It comprises a classroom for the instruction of nurses and students, study rooms, accommodation for post-graduate workers, and laboratory for bacteriological and biochemical examinations.

The entrance hall, too, is worthy of special mention. It has been made beautiful by a carved stone plaque, by Miss Praeger of Holywood, symbolical of Maternal Love. This plaque is the gift of Mrs. Maitland Beath, as a memorial to her father and mother, Mr. and Mrs. R. M. Young.

R. H. H.

THE LISBURN AND DISTRICT MEDICAL GUILD

THE annual meeting of the Guild was held during April at Dr. Hunter's, Dunmurry. Dr. Colquhoun was elected president, and Dr. Peatt was again elected secretary and treasurer. The report and statement of accounts having been accepted and passed, Mr. H. C. Lowry was called upon to deliver his paper : "Some Obstetric

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Difficulties in General Practice." Mr. Lowry dealt with the breech with extended legs, and the difficulties which arise in these cases. With regard to the etiology, he declared that in his experience it occurred practically always in primigravidæ. In a series of twenty-three cases delivered personally by him, all but two had been primigravidæ. Contracted pelvis seemed to be a factor in a number of cases, but not contraction of a major degree. The diagnosis, both before and during labour, is difficult for the following reasons : (1) The presenting part is deep in the pelvis, and is therefore difficult to palpate per abdomen; (2) fœtal heart is usually heard at the same level as in a vertex presentation; (3) when the legs are extended, the arms are usually extended also and tend to mask the head. There is, however, a stiffness and want of mobility about the fœtus which is not present in a vertex or in an ordinary breech presentation.

Except in a very difficult patient, a vaginal examination should settle the question in the majority of cases, whether the legs are extended or not.

If an attempt at external version fails, one should be very suspicious that it is an extended breech, and an X-ray picture will be very helpful : it will confirm or disprove extension of legs.

During labour the diagnosis per abdomen is very difficult, and a vaginal examination is essential. If the cervix will admit one or two fingers, a definite diagnosis can be made.

With regard to the treatment, some obstetricians, particularly American, regard this complication as an indication for cæsarean section. This is hardly justifiable except in cases where there is some other factor present, such as a contracted pelvis, elderly primigravida, large child, or where for some reason one must make as sure as possible of delivering a living child. Induction of labour ten to fourteen days before term is sometimes useful, and should be considered. There is no doubt that the chances for the baby are diminished if it weighs much over eight pounds.

During labour the patient should be kept in bed and given a sedative, either hyosine or morphia and mogresium sulphate followed by rectal ether. These cases are always prolonged, and the patient becomes tired out. Sedatives, if judiciously used, do not prolong labour and do not cause post-partum hæmorrhage.

In the actual management of these cases there are three lines of treatment :—

(1) Leave alone and allow patient to deliver herself with the legs still extended, or deliver by traction in the groins. This is not recommended, as the patient seldom delivers herself unless the body is very small. Groin traction is very tiring to the accoucheur's fingers and wrists. A piece of stout rubber tubing may be used for traction, but it is difficult to place in position.

(2) Bring down a leg as soon as sufficient dilatation occurs, and leave to nature. The drawback to this method is that it requires two anæsthetics and two interferences.

(3) The best method is to leave until fully dilated, then bring down the legs and proceed with the delivery; in these cases the arms are usually extended whether the legs are pulled or not. In bringing down legs, the patient must be well anæsthetized

and the anterior leg brought down first. If there is any difficulty in doing this, have the patient placed in the left lateral position, which encourages the presenting part to fall away out of the pelvis, and so gives more room. After bringing the arms down, the delivery is completed in the ordinary way. Smellie's method is the one most commonly employed.

Maternal complications may be classified as follows :—

1. *Lacerations.*—Extensive vaginal and perineal tears may be minimized by doing an epidiotomy at the beginning of the delivery. The laceration tends to occur when bringing down the arms and legs.

2. *Retained placenta, post-partum hæmorrhage, and obstetrical shock.*—It is not uncommon to get this complication, as the patient has usually had a long labour and a fairly long anæsthetic. After the placenta has been expressed or removed manually, the usual treatment for post-partum hæmorrhage should be commenced : hot douche at 118°F., pitourin ½ c.c. plus ergot aseptic (P. D. & Co.) 1 c.c.; raise foot of bed; warmth; morphia ¼ gr.

In addition to these routine measures, the intravenous injection of 50 to 100 c.c. of fifty per cent. glucose is good treatment. It should be given slowly.

Carmine 3 to 5 c.c. may be given intravenously if deemed necessary, or 3 c.c. of caffine sodium benzoate. If the patient has lost a large amount of blood and needs fluid, one pint of gum saline, or one pint of 5 per cent. glucose, should be given.

Fœtal complications may also occur. The baby may need to be resuscitated. All vigorous methods are contracted, as the baby is shocked. It should be wrapped up in a warm blanket and handled as little as possible. The air passages should be cleared. Brandy may be applied to the lips and gums. Carmine ½ to 1 c.c. may be given intra-muscularly or intra-cardially. CO_2 may be given to stimulate respiration, or a mixture containing ninety-three per cent. oxygen and seventy per cent. carbon-dioxide : with this it is impossible to give an overdose of CO_2. Lobeline by injection is sometimes used. Hot baths at 105°F. may be tried.

J. W. Peatt, *Hon. Secretary.*

Railway Street, Lisburn.

APPOINTMENTS

W. Waring Bassett, M.B., F.R.C.S.I., has been appointed Chief Medical Officer to the Lurgan Infirmary.

P. T. Crymble, M.B., F.R.C.S.Eng., has been appointed Professor of Surgery, Queen's University, Belfast.

H. P. Hall, M.B., M.Ch., has been appointed Visiting Surgeon to the new Dufferin Hospital, Belfast Infirmary.

H. P. Malcolm, M.B., M.Ch., has been appointed Lecturer in Operative Surgery, Queen's University, Belfast.

C. J. A. Woodside, M.B., F.R.C.S.I., has been appointed Lecturer in Applied Anatomy, Queen's University, Belfast.