TRANSACTIONS

OF THE

ROYAL ACADEMY OF MEDICINE IN IRELAND.
CONTENTS

List of Officers, - ix
List of Fellows, - xiii
List of Members, - xxiv
List of Student Associates, - xxv
Rules, - xxvii
Annual Report, - xxxv
Treasurer’s Report, - xxxvii

SECTION OF MEDICINE.

Hodgkin’s disease occurring in twins - Dr. G. J. Peacocke - 1
Syringomyelia, with account of an uncommon case - Dr. Joseph O’Carroll - 6
Typhoid and paratyphoid fever - Dr. Parsons - 16
Large white kidney - Dr. G. J. Peacocke - 25
Sequel to a case of innominate aneurysm reported in 1898 - Dr. James Craig - 31
Pneumonia in pregnancy - Dr. Henry C. Drury - 35
Some miscellaneous clinical experiences - Dr. R. Travers Smith - 42
Some colour tests for the chief nitrogenous constituents of urine - Dr. Walter G. Smith - 50
Bacillus coli communis as a cause of septicemia - Dr. T. Gillman Moorhead - 54
Abstracts of meetings of Medical Section: -
  Sporadic cretinism - Dr. T. P. C. Kirkpatrick - 70
  Caisson disease - Dr. J. B. Coleman, C.M.G. - 71
  Hysterical chorea - Dr. James Craig - 73
  Syringomyelia - Dr. H. T. Bewley - 74
Tuberculosis of lungs and of knee treated by Professor Deny’s Tuberculin - Dr. F. J. Dunne - 76

SECTION OF SURGERY.

Inefficient operations for gastric ulcer - Mr. A. B. Mitchell - 81
The healing of wounds—aseptic versus antiseptic methods - Mr. William S. Haughton - 91
Contents.

New methods for the performance of herniotomy for inguinal and femoral hernia
Case of intestinal obstruction by a gall stone
Cholecystotomy for acute cholecystitis during convalescence from enteric fever
Conservative perineal prostatectomy
The application of Plaster of Paris facilitated by a new apparatus
Oblique fracture of the tibia
Some cases of joint excision
Painless haematuria
On the treatment of purulent cavities
Operation for closure of cleft palate in infants
Notes on a case of volvulus of the cecum, secondary to malignant disease of the sigmoid flexure of the colon
Abstracts of Meetings of the Section of Surgery

SECTION OF OBSTETRICS.

Two cases of cystic endometritis with remarks on treatment
Observations on Rossi’s dilator, with notes of four cases
Puerperal convulsions
Curative operation for procidentia uteri
Two cases of wounds of the female genitals
Notes on a case of labour in a unilateral synostotic pelvis
Clinical report of the Rotunda Hospital
Abstracts of Meetings of the Section of Obstetrics:
Influence of fibromyomata on pregnancy and parturition

SECTION OF PATHOLOGY.

Haematological observations—A case of chronic lymphemia
A case of acute yellow atrophy of the liver
A case of glanders
A case of acute lymphæmia

Mr. Edward H. Taylor 111
Mr. T. E. Gordon 118
Mr. R. C. B. Maunsell 125
Mr. C. Arthur Ball 134
Mr. W. I. de Courcy Wheeler 152
Mr. Edward H. Bennett 160
Mr. Denis Kennedy 163
Mr. L. G. Gunn 169
Mr. R. H. Woods 182
Sir Thornley Stoker 196
Dr. R. D. Purefoy 217
Dr. A. J. Smith 223
Dr. R. J. Kinkead 230
Dr. E. Hastings Tweedy 240
Dr. R. J. Kinkead 245
Dr. Henry Jellett 250
Dr. E. Hastings Tweedy 256
Dr. E. J. McWeeney 297
Dr. W. J. Thompson and Dr. E. J. McWeeney 307
Dr. E. F. Stephenson 313
Dr. E. J. McWeeney 315
Contents.

Note on a large fibromyoma from the rectum - Dr. H. C. Earl - 330
Note on a case of cirrhosis of liver Dr. George Peacocke - 332
A case of infective endocarditis Dr. George Peacocke - 336
On a case of primary actinomycosis of the right kidney with embolical cerebral abscess - Dr. H. C. Earl - 339
Melano-sarcoma of the conjunctiva Dr. Arthur H. Benson and Dr. H. C. Mooney 347
Fractures of first costal cartilage Mr. E. H. Bennett - 349
Abstracts of Meetings of the Section of Pathology:
- Aortic aneurysm perforating the oesophagus - Dr. J. A. Matson - 354
- Aortic aneurysm rupturing into pleura Dr. T. G. Moorhead - 354
Glioma of the retina - Mr. J. B. Story and Dr. H. C. Earl - 355
Glioma of the retina Dr. H. C. Mooney - 355
Anaemic infarction of liver Dr. O'Sullivan - 356
Endothelioma of uterus Dr. H. C. Earl and Mr. R. C. B. Maunsell - 356
Pott's caries Mr. L. G. Gunn - 357
Gastric ulcer Mr. L. G. Gunn - 357
Carcinomata Sir Thornley Stoker - 357
Anomaly of cardiac valve Dr. T. G. Moorhead - 358
Tuberculous disease of caecum Mr. R. A. Stoney - 358
Recurrent ulceration of stomach Dr. W. J. Thompson - 359
Congenital sarcoma of the eyelid Mr. A. H. Benson and Dr. H. C. Mooney - 360

SECTION OF STATE MEDICINE.

Earth temperature and diarrheal diseases in Dublin during 1904 Sir John W. Moore - 361
Sicily as a winter health resort Dr. E. Parlato - 372
A case of carbon monoxide poisoning Dr. W. J. Thompson - 376

SECTION OF ANATOMY AND PHYSIOLOGY.

The anatomy of a sirenomelician monster Dr. T. G. Moorhead - 382
Histogenesis of the grey matter of the cerebellum Dr. D. J. Coffey - 396
Irregular form and position of the colon Mr. Alec. Fraser - 399
# LIST OF ILLUSTRATIONS

<table>
<thead>
<tr>
<th>Illustration</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Syringomyelia (Dr. J. F. O'Carroll)</td>
<td>6</td>
</tr>
<tr>
<td>Conservative perineal prostatectomy (Mr. C. Arthur Ball)</td>
<td>134</td>
</tr>
<tr>
<td>The application of Plaster of Paris facilitated by a new process (Mr. W. I. de Courcy Wheeler)</td>
<td>152</td>
</tr>
<tr>
<td>On the treatment of purulent cavities (Mr. R. H. Woods)</td>
<td>182</td>
</tr>
<tr>
<td>Operation for closure of cleft palate in infants (Sir Thornley Stoker)</td>
<td>196</td>
</tr>
<tr>
<td>Notes of a case of labour in a unilateral synostotic pelvis (Dr. Henry Jellett)</td>
<td>250</td>
</tr>
<tr>
<td>Charts of earth temperature and diarrhoeal diseases in Dublin during 1904 (Sir John W. Moore)</td>
<td>361</td>
</tr>
<tr>
<td>The anatomy of a sirenomelian monster (Dr. T. G. Moorhead)</td>
<td>382</td>
</tr>
</tbody>
</table>
ROYAL ACADEMY OF MEDICINE IN IRELAND.

Established 1882.

SESSION 1904-1905.

President:
SIR THORNLEY STOKER, 3 Ely-place.

General Secretary and Treasurer:
JAMES CRAIG, 18 Merrion-square.

Secretary for Foreign Correspondence:
SIR JOHN W. MOORE, 40 Fitzwilliam-square.

General Council:

SIR THORNLEY STOKER, President.
SIR JOHN BANKS, K.C.B., Ex-President.
E. H. BENNETT, Ex-President.
JAMES LITTLE, Ex-President.
LONBE ATTHILL, Ex-President.
W. J. SMYLY, President of Medical Section.
ARTHUR CHANCE, President of Surgical Section.
A. J. SMITH, President of Obstetrical Section.
H. C. EARL, President of Pathological Section.
EDWARD H. TAYLOR, President of Section of Anatomy and Physiology.
SIR JOHN W. MOORE, Secretary for Foreign Correspondence, and President Section of State Medicine.
JAMES CRAIG, General Secretary and Treasurer.
F. TRAVERS SMITH, Secretary Medical Section.
EDWARD H. TAYLOR, Secretary Surgical Section.
T. HENRY WILSON, Secretary Obstetrical Section.
A. H. WHITE, Secretary Pathological Section.
A. F. DIXON, Secretary Section of Anatomy and Physiology.
F. C. MARTLEY, Secretary Section of State Medicine.


List of Officers.

Council of Medical Section:
President—W. J. SMYLY, President of the Royal College of Physicians.
1. Secretary—R. TRAVERS SMITH, 20 Lower Fitzwilliam-street.
2. J. B. COLEMAN,
3. W. R. DAWSON,
4. H. C. DRUBY,
5. T. P. KIRKPATRICK,
6. G. J. PEACOCKE,
7. F. C. PURSER,
8. W. LANGFORD SYMES,
9. W. J. THOMPSON,
10. W. A. WINTER.

Council of Surgical Section:
President—ARTHUR CHANCE, President of the Royal College of Surgeons.
1. Secretary—EDWARD H. TAYLOR, 77 Merrion-square.
2. ALEXANDER BLAYNEY,
3. T. E. GORDON,
4. G. JAMESON JOHNSTON (co-opted),
5. J. LENTAIGNE,
6. R. B. M'CAUSLAND,
7. R. C. B. MAUNSELL,
8. SIR LAMBERT ORMSBY,
9. WILLIAM TAYLOR,
10. J. B. STORY.

Council of Obstetrical Section:
President—A. J. SMITH, 30 Merrion-square.
1. Secretary—T. HENRY WILSON, 33 Upper Merrion-street.
2. PAUL CARTON,
3. R. H. FLEMING,
4. ROBERT A. FLYNN,
5. J. H. R. GLENN,
6. A. J. HORNE,
7. H. JELLETT,
8. F. W. KIDD,
9. W. C. NEVILLE,
10. R. D. PUREFOOT.

Council of Pathological Section:
President—H. C. EARL, 10 Leinster-street.
1. Secretary—A. H. WHITE, Royal College of Surgeons.
2. A. H. BENSON,
3. E. J. McWEENEY (co-opted),
4. H. C. MOONEY,
5. T. G. MOORHEAD,
6. A. C. O'SULLIVAN,
7. A. R. PARSONS,
8. F. C. PURSER,
9. J. A. SCOTT,
10. J. B. STORY.
List of Officers.

Council of Section of Anatomy and Physiology:

President—EDWARD H. TAYLOR, 77 Merrion-square.

1. Secretary—A. F. DIXON, Anatomical Department, Trinity College.
2. A. BIRMINGHAM, 5. W. H. THOMPSON (co-opted)
4. A. FRASER, 

Council of Section of State Medicine:

President—SIR JOHN W. MOORE, 40 Fitzwilliam-square.

1. Secretary—F. C. MARTLEY, 32 Upper Merrion-street.
2. SIR C. A. CAMERON, C.B. (co-opted), 4. J. M. REDMOND.

Reference Committee:

1. A. H. BENSON, 3. W. A. WINTER.
2. R. A. FLYNN, 4. T. E. GORDON.
HONORARY FELLOWS.

1903 BERGMANN, PROFESSOR VON, Berlin.
1885*BILLROTH, PROFESSOR T.
1903 BROUARDDEL, PROFESSOR, Paris.
1885*CHARCOT, PROFESSOR.
1885*EMMET, THOMAS ADDIS, 9 Madison Avenue, New York.
1885*FLINT, PROFESSOR AUSTIN.
1904 FUCHS, PROFESSOR E., Vienna.
1885 HUTCHINSON, JONATHAN, F.R.S., London.
1885*JENNER, SIR WILLIAM, Bart., F.R.S.
1885*KEITH, THOMAS.
1899 KELLY, PROFESSOR HOWARD, Baltimore.
1899 KOCH, PROFESSOR, Berlin.
1899 KÖCHER, PROFESSOR, Bern.
1885*KÖLLIKER, PROFESSOR.
1899 LEBER, PROFESSOR Th., Heidelberg.
1885*LUDWIG, PROFESSOR.
1885*MACCORMAC, SIR W., Bart., K.C.V.O.
1899 MARTIN, PROFESSOR, Berlin.
1899 NOTHNAEL, PROFESSOR.
1899 OSLER, PROFESSOR, Oxford.
1885*PAGET, SIR JAMES, Bart., F.R.S.
1885*PASTEUR, PROFESSOR.
1900 POLITZER, PROFESSOR ADAM, Vienna.
1903 PYE-SMITH, P. H., F.R.S., London.
1885 RECKLINGHAUSEN, PROFESSOR VON, Strasburg.
1885*SCHEIDER, PROFESSOR.
1885 SIMON, SIR J., F.R.S., London.
1903 TREVES, SIR FRED., Bart., London.
1899 TURNER, SIR W., F.R.S., Edinburgh.
1885*VIRCHOW, PROFESSOR.

* Dead.
Fellows.

[The figures prefixed denote the date of election. The figures appended to Names denote the number of Communications. Original Fellows are marked +.]

1903 Ahern, W. F., M.D., Columba House, Howth.
1893 Allworthy, S. W., M.D., Assistant Physician Hospital for Skin Diseases, The Manor House, Antrim-road, Belfast.
+ Atthill, Lombe, M.D., Ex-President R.C.P., L.M.O.S., late Master Rotunda Lying-in Hospital, Monkstown Castle, Co. Dublin.
+ Baker, Arthur Wyndowe Willert, M.D., F.R.C.S., Surgeon to the Dental Hospital of Ireland, Consulting Surgeon Royal Victoria Eye and Ear Hospital, 59 Merrion-square, Dublin.
+ Ball, Sir Charles Bent, M.D., F.R.C.S., Regius Professor of Surgery, Univ. Dub., Surgeon Sir P. Dun's Hospital, 24 Merrion-square, N., Dublin.

1903 Ball, Charles Arthur K., M.D., F.R.C.S., Assistant Surgeon Sir P. Dun's Hospital, 12 Lower Mount-street. [1]
+ Banks, Sir John, K.C.B., M.D., Ex-President R.C.P., Ex-President British Medical Association, Physician-in-Ordinary to His Majesty the King in Ireland, 45 Merrion-square, East, Dublin.
+ Barton, John, M.D., F.R.C.S., Secretary of Council, Royal College of Surgeons, 10 Mount-street Crescent, Dublin.

+ Beatty, Joseph, F.R.C.S., Surgeon Monkstown Hospital, 3 Howard-place, Kingstown.
+ Beatty, Wallace, M.D., F.R.C.P., Physician Adelaide Hospital, 38 Merrion-square, E., Dublin.
+ Bennett, Edward Hallaran, M.D., Ex-President R.C.S., Surgeon to Sir P. Dun's Hospital, Professor of Surgery Trinity College, 26 Lower Fitzwilliam-street, Dublin. [2]
+ Benson, Arthur Henry, F.R.C.S., Ophthalmic Surgeon Royal City of Dublin Hospital, Surgeon Royal Victoria Eye and Ear Hospital, 42 Fitzwilliam-square, W., Dublin. [2]
+ Benson, J. Hawtrey, M.D., F.R.C.P., Consulting Physician Royal City of Dublin Hospital, 57 Fitzwilliam-square, N., Dublin.

1887 Berry, Wm., L.R.C.P. Edin., F.R.C.S., Surgeon Royal Albert Edward Infirmary, Park House, Wigan.
List of Fellows.

1904 BEVERIDGE, W. J., L.R.C.P. & S., Brough, Westmoreland.

1887 BEWLEY, HENRY T., M.D., F.R.C.P., Lecturer on Medical Jurisprudence and Hygiene, Trin. Coll., Physician Adelaide Hospital, 89 Merrion-square, Dublin. [1]

1905 BEWLEY, A. W., Major R.A.M.C., L.R.C.P.I., Temporary Fellow, Royal Hospital.

1887 BURY, Henry T., M.D., F.E.C.P., L.R.C.P.I., Brough, Westmoreland.


1905 BARRY, A. W., Major R.A.M.C., L.R.C.P.I., Temporary Fellow, Royal Hospital.


1891 BOYD, J. ST. CLAIR, M.D., Gynaecologist Ulster Hospital, 27 Victoria-place, Belfast.

1898 BRADSHAW, SAM. J. M., M.D., 1 Tempè-terrace, Dalkey.

1891 BROWNE, HENRY PETER, M.D., LL.D. (hon. causa), F.R.C.S.E., M. & L.M., R.C.P.I., L.R.C.S.I., ex-Medical Officer and Medical Officer of Health, Rathdown Union, West Malvern, Delgany, Greystones.

1883 BROWNE, J. WALTON, A.B., M.D., Surgeon to the Royal Victoria Hospital, 10 College-square, North, Belfast.

1884 BURGESS, JOHN J., F.R.C.S., late Assistant Surgeon to the Richmond Hospital, 22 Westland-row, Dublin.

1884 BURKE, JOHN RICHARD, M.D., Deputy Inspector-General Hospitals and Fleets, R.N.

1891 BYRNE, HERBERT U., M.B., B.Ch., Univ. Dublin, Medical Officer No. 4 Dispensary District, South Dublin Union, Physician Cork-street Hospital, 15 Upper Merrion-street, Dublin.

1891 BYRNE, LOUIS A., L.R.C.P., F.R.C.S., Surgeon Jervis-street Hospital, 79 Harcourt-street, Dublin.

1898 CALWELL, W., M.A., M.D., M.Ch., L.M.R.C.P.I., Physician Royal Victoria Hospital, Belfast, Consulting Physician to Ulster Hospital for Women and Children, 1 College-square, N., Belfast.

† CAMERON, SIR CHARLES A., C.B., M.D., ex-President R.C.S., Professor of Chemistry Royal College of Surgeons, Superintendent Medical Officer of Health, City and County Analyst, Dublin, 51 Pembroke-road, Dublin.

1895 CAMPION, THOMAS SPREAD, M.B. Univ. Dubl., Crescent, Lucan.

1900 CANILLO, GONZALO, L.R.C.P. & S., José Luiz Diez No. 5, Jerez de la Frontier, Spain.

1898 CARABIN, MORE, L.R.C.P. & S., Collon, Co. Louth.

1900 CARTON, PAUL, M.D., Ex-Assistant Master Rotunda Hospital, 35 Rutland-square.

1884 CHANCE, ARTHUR, President R.C.S., L.R.C.P., Surgeon to Mater Misericordiae Hospital, 90 Merrion-square.

List of Fellows.

1890 COADY, D. P., F.R.C.S.I., Medical Officer Naas Union Infirmary, Tintern, Naas.
1897 COADY, Ed. T., F.R.C.S., Medical Officer Clane and Timahoe Dispensary District, Clane, Co. Kildare.
1902 Cockey, Walter, M.D., Anaesthetist Metropolitan Ear, Nose and Throat Hospital, 1 Elm Villas, Ealing Green, London, W.
1891 Coffey, Denis J., M.B., Professor of Physiology Catholic University Medical School, Dublin. [1]
1897 Colahan, Nicholas Whistler, M.D., M.Ch., Professor of Materia Medica and Therapeutics Queen's College, Villa, Galway.
1891 Coleman, James Byrne, C.M.G., M.D., F.R.C.P., Physician to Rich mond, Whitworth, and Hardwicke Hospitals, and to Nat. Hosp. for Consumption, 9 Merrion-square, Dublin. [1]
+ Coppinger, Charles, M.D., F.R.C.S., Surgeon to the Mater Misericordiae Hospital, 17 Merrion-square, Dublin.
+ Cosgrave, E. MacDowell, M.D., M.Ch., F.R.C.P., Professor of Botany and Zoology, R.C.S., Physician to Drumcondra Hospital, 5 Gardiner's-row, Dublin.
1883 Cox, Michael F., F.R.C.P., Physician St. Vincent's Hospital, 26 Merrion-square, Dublin.
1889 Craig, James, M.D., F.R.C.P., Physician Meath Hospital, 18 Merrion-square, Dublin. [2]
1898 Crawley, Frank Chetwode, M.D., Univ. Dub., F.R.C.S., Clinical Assistant Royal Victoria Eye and Ear Hospital, 41 Lower Baggot-street, Dublin.
1884 Cronyn, John G., L.R.C.S., L.R.C.P., Medical Officer South Dublin Union Workhouse, 4 Clare-street, Dublin.
1897 Dargan, W. J., M.D., Assistant Physician to St. Vincent's Hospital, 45 St. Stephen's-green.
1899 Dempsey, Alex., M.D., Q.U.I., Physician Mater Infirmorum Hospital, Clifton-street, Belfast.
1891 Dempsey, Martin J. P., M.D. R.U.I., F.R.C.P., Professor of Materia Medica and Therapeutics, Catholic University, Physician to Mater Misericordiae Hospital, 35 Merrion-square, Dublin.
1900 Dempsey, Pat. J., F.R.C.S., Throat Surgeon, Mater Misericordiae Hospital, 7 Merrion-square, Dublin.
+ Denham, J. Knox, F.R.C.S., Medical Officer to Donnybrook Dispensary, 67 Lower Baggot-street, Dublin.
List of Fellows.


1895 Drury, H. C., M.D., Univ. Dub., F.R.C.P., Physician Sir P. Dun's Hospital and Cork-street Fever Hospital, 48 Fitzwilliam-square, Dublin. [1]


1885 Dwyer, F. Conway, M.D., F.R.C.S., Surgeon Meath Hospital, Professor of Surgery Royal College of Surgeons, 9 Rutland-square, Dublin.

1889 Ellis, George, F.R.C.S., M.B., Physician Sir P. Dun's Hospital, King's Professor of Practice of Medicine, School of Physic, Ex-President R.C.P., 36 Merrion-square, Dublin.

1891 Finlayson, Henry Lindo, M.D., F.R.C.S., Dunedin, New Zealand.

1900 Fannin, Edward M., M.B., B.Ch. Dubl., 3 Rutland-square.


1884 Fleming, Robert H., M.B., Gynaecologist Royal City of Dublin Hospital, 30 Lower Baggot-street, Dublin.

1891 Flynn, Robert Alexander, F.R.C.P., Gynaecologist Drumcondra Hospital, 55 Merrion-square, Dublin.

1886 Fotherell, Wm. Joseph, L.R.C.S., Medical Officer North Dublin Union Workhouse, 2 Rutland-square, Dublin.
List of Fellows.

† FRANKS, Sir Kendal, C.B., M.D., F.R.C.S., late Surgeon to Adelaide Hospital and to Throat and Ear Hospital, Dublin, Kilmainry, Johannesburg, South Africa.

1887 FRASER, Alec, M.B., F.R.C.S., Professor of Anatomy Royal College of Surgeons, Ireland, 18 Northbrook-road, Leeson Park. [1]

1891 GLENN, John Hugh Robert, M.D., F.R.C.P., Gynaecologist Mercer’s Hospital, 24 Lower Baggot-street, Dublin.


1893 GORDON, Thomas Eagleson, M.B., F.R.C.S., Surgeon Adelaide Hospital, 8 Fitzwilliam-square, Dublin. [1]

† GREENE, Thomas Wm. Nassau, L.R.C.S., 45 Dartmouth-square, Dublin.

1905 GREEN, J. S., Major, R.A.M.C., Temporary Fellow, 83 Lower Leeson-street.

1902 GUNN, L. G., M.D., Dub., F.R.C.S.I., Assistant Surgeon Adelaide Hospital, 43 Fitzwilliam-square. [2]

1892 HAMILTON, Wm. Cope, L.R.C.P. & S., late Resident Surgeon Steevens’ Hospital, 120 St. Stephen’s-green, W., Dublin.

† HARLEY, Robert William, L.R.C.S., 21 Pembroke-road, Dublin.

1899 HAUGHTON, W. S., M.D., Surgeon to Steevens’ Hospital, 30 Lower Fitzwilliam-street.

† HAYES RICHARD ATKINSON, M.D., F.R.C.S., Physician Steevens’ Hospital, Physician for Diseases of Throat, Royal Victoria Eye and Ear Hospital, 82 Merrion-square, S., Dublin.

1901 HENNESSY, Mrs. H. L., L.R.C.P. & S., M.D. (Brux.), 56 Fitzwilliam-square.


† HESTON, Francis TAYL, F.R.C.S., Surgeon Adelaide Hospital, 15 St. Stephen’s-green, N., Dublin.

1904 HOLMES, Arthur N., M.B., Univ. Dub., Assistant Master Rotunda Hospital.

1905 HOLT, M. P., Major R.A.M.C., D.S.O., Temporary Fellow, Royal Infirmary.

† HORE, Andrew John, F.R.C.P., Master National Lying-in Hospital, 94 Merrion-square, W., Dublin.

1883 JACOBS, David Baldwin, M.D., F.R.C.S., Surgeon Queen’s County Infirmary, Visiting Physician Maryborough District Lunatic Asylum, Surgeon Queen’s County Prison, Port Leix, Maryborough, Queen’s Co.

1890 JELLETT, Henry, M.D., F.R.C.P.I., Gynaecologist Steevens’ Hospital, 61 Lower Mount-street. [1]
List of Fellows.

1885 Jenings, Ulick A., M.D., Brigade Surgeon, M.S., Retired, Military Prison, Roche House, Cork.


1903 Johnston, Henry M., M.B., B.Ch., Univ. Dub., Chief Demonstrator of Anatomy T.C.D., School of Anatomy, Trinity College.

1893 Joynt, Richard Lane, M.D. Univ. Dub., F.R.C.S., Surgeon Meath Hospital, 84 Harcourt-street, Dublin.

1899 Kennedy Denis, F.R.C.S., Surgeon Jervis-street Hospital and Children's Hospital, Temple-street, 39 Harrington-street. [1]

1884 Kidd, Fred. W., M.D., ex-Master Coombe Hospital, Gynaecologist Meath Hospital, 17 Lower Fitzwilliam-street, Dublin.

† Kinkead, Richard John, M.D., L.R.C.S., Lecturer on Medical Jurisprudence, Professor of Obstetric Medicine, Queen's College, Galway, Foster House, Galway. [2]

1899 Kirkpatrick, T. Percy C., M.D. Dub., F.R.C.P.I., Demonstrator of Anatomy T.C.D., Physician to Steevens' Hospital, 23 Lower Baggot-street. [1]

† Knott, John, M.D., F.R.C.S., 34 York-street.

1901 Law, S. Horace, M.D., F.R.C.S., Throat Surgeon Adelaide Hospital, Surgeon Dublin Throat and Ear Hospital, 48 St. Stephen's-green.

1884 Ledwich, Edward J.E., L.R.C.P. & S.I., Physician to Mercer's Hospital, College Anatomist Royal College of Surgeons, 30 Upper Fitzwilliam-street, Dublin.

1900 Leeper, Richard R., F.R.C.S., St. Patrick's Hospital, Dublin.

1891 Lennox, Edward Emmanuel, F.R.C.P., Physician Meath Hospital, 22 Merrion-square, Dublin.

† Lentaigne, John, F.R.C.S., Surgeon Mater Misericordiae Hospital, 42 Merrion-street, Dublin.

1897 Letters, Patrick, M.D., Valentia Island.

1895 Lindsay, James A., M.D., F.R.C.P. London, Professor of Practice of Medicine Q.C.B., Physician Royal Victoria Hospital, Belfast, 13 College-square, E., Belfast.

† Little, James, M.D., ex-President R.C.P., Regius Professor of Physic Univ. Dub., 14 St. Stephen's-green North, Dublin.

1897 Lumsdon, John, M.D., Physician to Mercer's Hospital, 4 Fitzwilliam-place.

† Macan, Sir Arthur V., M.B., ex-President R.C.P., King's Professor of Midwifery T.C.D., 53 Merrion-square, Dublin.

† M. Arble, John Stephen, F.R.C.S., Surgeon St. Vincent's Hospital and the Mullen Convalescent Home, 72 Merrion-square, Dublin.
List of Fellows.

1887 McCausland, Richard Bolton, F.R.C.S., Surgeon Steevens' Hospital, 79 Merrion-square, Dublin.

1890 M'Evy, Thomas, L.R.C.P. & S., 1 Prince Edward-terrace, Blackrock, Co. Dublin.

1904 McGrath, James Joseph, L.R.C.P., St. Helens, Dunfanaghy, Co. Donegal.


1902 McIsack, H. L., M.D., Physician Royal Victoria Hospital, 15 College-square, Belfast.

1904 Macneece, J. G., Lt.-Col., R.A.M.C., Temporary Fellow.

1903 McVitie, R. B., M.D., 62 Fitzwilliam-square.

1897 McWeeney, E. J., M.D., F.R.C.P.I., Pathologist Mater Misericordiae Hospital, 84 St. Stephen's-green, Dublin. [3]

1901 Magennis, E., M.D., 37 Harcourt-street.

1900 Maguire, Katharine M. X., M.D., 67 Merrion-square, South.

1883 Marques, L. Pereira, M.R.C.P., L.R.C.S., late Medical Officer Victoria Gael Hospital, Macao, China.

† Martin, William James, M.D., F.R.C.P. Edin., ex-Physician Jervis-street Hospital and St. Joseph's Infirmary for Children, 17 Harcourt-street, Dublin.


1897 Maunsell, R. Charles B., M.B., B.Ch., B.A.O., Dub., F.R.C.S.I., Surgeon to Mercer's Hospital, 32 Lower Baggot-street. [2]


1904 Mills, John, M.B., B.S., R.U.I., District Asylum, Ballinasloe.

1901 Mitchell, A. B., F.R.C.S., Surgeon Royal Victoria Hospital, 4 College-square, Belfast. [1]

1894 Montgomery, Robert John, M.A., M.B. Univ. Dub., F.R.C.S., Ophthalmic Surgeon Drumcondra Hospital, Assistant Surgeon Royal Victoria Eye and Ear Hospital, 4 Gardiner's-row, Dublin.
List of Fellows.

1897 **Mooney, H. C., M.B., F.R.C.S.,** Ophthalmic Surgeon Children's Hospital, Temple-street; Assistant Surgeon Royal Victoria Eye and Ear Hospital, 22 Lower Baggot-street. [3]

1894 **Moore, Henry, L.R.C.P. & S.,** Surgeon Royal City of Dublin Hospital, 40 Lower Baggot-street.

† **Moore, Sir John William, M.D., ex-President R.C.P.,** Physician Meath Hospital, Professor of Practice of Medicine Royal College of Surgeons, 40 Fitzwilliam-square, West, Dublin. [1]

1901 **Moorhead, George, F.R.C.S.,** Tullamore.

1904 **Moorhead, T. Gillman, M.D., M.R.C.P.,** Physician City of Dublin Hospital, 12 Lower Fitzwilliam-street. [4]

1897 **Morrison, F. Saunders, F.R.C.S.,** Hollaton, Leicestershire.

1883 **Murphy, John, F.R.C.P.,** Physician Mater Misericordia Hospital, 13 Merrion-square, Dublin.

† **Murphy, John Joseph, L.R.C.P.,** Physician City of Dublin Hospital, 12 Lower Baggot-street.

1904 **Murphy, W. L., B.A., Cantab., L.R.C.P. & S.I.,** Mater Misericordia Hospital.

1886 **Myles, Sir T., ex-President R.C.S.,** Surgeon Richmond Hospital, 33 Merrion-square, Dublin.

† **Nixon, Sir Christopher, M.D., ex-President R.C.P.,** Physician to Mater Misericordia Hospital, 2 Merrion-square, N., Dublin.

1889 **Nolan, Michael James, L.R.C.P., L.R.C.S.,** Resident Medical Superintendent Down District Lunatic Asylum, Downpatrick.

1885 **Norman, Conolly, F.R.C.P.,** Medical Superintendent Richmond District Lunatic Asylum, Dublin.

899 **O'Brien, C. M., M.D., L.R.C.P. & S.,** Physician to City Hospital for Diseases of the Skin, 29 Merrion-square.

† **O'Carroll, Joseph Francis, M.D., F.R.C.P.,** Physician Richmond, Whitworth, and Hardwicke Hospitals, 43 Merrion-square, Dublin. [1]

1883 **Oliphants, J. Wybrants, M.D.,** Medical Officer Downpatrick Dispensary District, The Villas, Downpatrick.

1900 **O'Neill, Henry, M.D., J.P.,** Consulting Surgeon Royal Victoria Hospital, Curator Museum of Sanitary Science, 6 College-square, Belfast.

1883 **O'Neill, William, M.D., M.R.C.P. Lond.,** Physician Lincoln Lunatic Hospital, 2 Lindum-road, Lincoln.

† **Ormsby, Sir Lambert Hepensal, M.D., ex-President R.C.S.,** Surgeon Meath Hospital, Surgeon National Children's Hospital, 92 Merrion-square, West, Dublin.

1894 **O'Sullivan, A. C., M.D., F.T.C.D., F.R.C.P.I.,** Lecturer on Pathology Trinity College, 43 Ailesbury-road, Dublin. [1]

† **Quilton, Henry W., M.D. Dublin, F.R.C.S.I.,** Chief Surgeon Dublin Metropolitan Police, 17 Upper Fitzwilliam-street, Dublin.
Parsons, Alfred Robert, M.D. Univ. Dub., F.R.C.P., Physician
City of Dublin Hospital, 27 Lower Fitzwilliam-street, Dublin. [1]

Patten, Charles J., M.D., D. Sc., B. Ch. Dub., University College,
Sheffield.

Peacocke, Geo. J., M.D., F.R.C.P.I., Assistant Physician Adelaide
Hospital, 14 Lower Fitzwilliam-street. [4]

Peacocke, Reginald C., M.D. Dub., Medical Officer G. P. O., Blackrock
District, 2 Avoca-terrace, Blackrock.

Pearson, Charles Yelverton, M.D., F.R.C.S. Eng., Professor
of Surgery, Queen’s College, 1 Sydney-place, Cork.

† Pechev-Phipson, Mary Edith, M.D., L.R.C.P., Dasak Bungalow,
Nasik-road, Bombay.

Penny, Alfred Ferguson, L.R.C.P. & S., Hon. Visiting Physician
Chelsea Prov. Dispensary, 10 Oakley-street, Chelsea.

† Pollock, James Ferrier, M.D., F.R.C.P., Medical Officer Meath
Industrial Schools, Avoca House, Blackrock.

Pringle, Seton, M.B. Univ. Dub., F.R.C.S.I., Surgeon Mercer’s
Hospital, 17 Lower Baggot-street.


† Purefoy, Richard Dancer, F.R.C.S., Ex-Master Rotunda Hospital,
62 Merrion-square, Dublin. [1]

Purser, Frank C., M.D., F.R.C.P., Assistant Physician Richmond,
Whitworth, and Hardwicke Hospitals, 20 Lower Baggot-street.

† Redmond, Joseph Michael, M.D., F.R.C.P., Physician to Mater
Misericordiae Hospital, 41 Merrion-square, Dublin.

Ridley, George P., L.R.C.P., L.R.C.S., Surgeon King’s Co. In-
firmary, Tullamore.

Roche, Anthony, M.R.C.P., L.R.C.S., Professor of Medical Juris-
prudence and Public Health Catholic University Medical School,
60 Lower Baggot-street.

Rowlette, Robert J., M.D., Pathologist Rotunda Hospital, 17 Lower
Mount-street.

Rutherford, William, M.D., F.R.C.P.Ed., Visiting Physician District
Asylum, Ballinasloe.

Scott, C. Burnett, M.D., 35 Clarinda Park, Kingstown.

† Scott, John Alfred, M.D., F.R.C.S., Professor of Physiology Royal
College of Surgeons, 33 Lower Baggot-street, Dublin.

Sinclair, F. Howard, M.D., Rostrevor Sanatorium, Warrenpoint.

Smith, Alfred J., M.B., F.R.C.S.I., Gynecologist St. Vincent’s
Hospital, 30 Merrion-square, Dublin. [1]

Smith, R. Travers, M.D., F.R.C.P.I., Physician Richmond, Whit-
worth and Hardwicke Hospitals, 20 Lower Fitzwilliam-street. [1]
<table>
<thead>
<tr>
<th>Year</th>
<th>Name</th>
<th>Position and Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>1901</td>
<td>Smith, Trevor N.</td>
<td>F.R.C.S.I., late Assistant Master Coombe Hospital</td>
</tr>
<tr>
<td></td>
<td></td>
<td>34 Upper Fitzwilliam-street</td>
</tr>
<tr>
<td></td>
<td>Smith, Walter George</td>
<td>M.D., Ex-President R.C.P., King's Professor of Materia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Medica School of Physic, and Physician to Sir Patrick</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dun's Hospital, 25 Merrion-square, Dublin [1]</td>
</tr>
<tr>
<td></td>
<td>Smyly, Sir William J.</td>
<td>M.D., President R.C.P., late Master Rotunda Lying-in</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hospital, 53 Merrion-square, S., Dublin</td>
</tr>
<tr>
<td>1903</td>
<td>Staunton, Michael C.</td>
<td>M.D., Visiting Surgeon Children's Hospital</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Temple-street, 22 North Frederick-street</td>
</tr>
<tr>
<td></td>
<td>Stoker, Sir Thornley</td>
<td>M.D., ex-President R.C.S., Surgeon Richmond Hospital</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Surgeon Swift's Hospital for Lunatics, 8 Ely-place, Dublin [2]</td>
</tr>
<tr>
<td>1902</td>
<td>Stoker, Graves</td>
<td>F.R.C.S., Surgeon Drumcondra Hospital, 46 Rutland-square</td>
</tr>
<tr>
<td></td>
<td>Story, John Benjamin</td>
<td>M.B., F.R.C.S., Surgeon Royal Victoria Eye and Ear</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hospital, Ophthalmic and Aural Surgeon Steevens' Hospital</td>
</tr>
<tr>
<td></td>
<td>Story, W. G.</td>
<td>M.B., B.Ch., 8 Upper Fitzwilliam-street, Dublin</td>
</tr>
<tr>
<td>1900</td>
<td>Stratton, Michael</td>
<td>L.R.C.S., Medical Officer No. 2 North City Dispensary</td>
</tr>
<tr>
<td></td>
<td></td>
<td>District, 33 Rutland-square, Dublin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Officer Westmoreland Lock Hospital, 17 North Great George's-st.</td>
</tr>
<tr>
<td></td>
<td>Swan, Robert Lafayette</td>
<td>Ex-President R.C.S., Surgeon Steevens' Hospital and</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dublin Orthopaedic Hospital, 32 St. Stephen's-green, N., Dublin</td>
</tr>
<tr>
<td></td>
<td>Swandyke, Henry Rosborough</td>
<td>M.D., Dublin, hon. causà, F.R.C.S., Surgeon Royal</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Victoria Eye and Ear Hospital, Dublin, Ophthalmic Surgeon</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Adelaide Hospital, 23 Merrion-square, North, Dublin</td>
</tr>
<tr>
<td>1893</td>
<td>Symes, Wm. Langford</td>
<td>F.R.C.P., M.D., Assistant Physician Dublin Orthopaedic</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hospital, Physician Homes for Destitute Children, 74</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Merrion-square, S., Dublin</td>
</tr>
<tr>
<td>1893</td>
<td>Symington, Johnson</td>
<td>M.D. Edin., Professor of Anatomy Queen's Coll., Belfast</td>
</tr>
<tr>
<td>1895</td>
<td>Taylor, Edward Henry</td>
<td>M.D., Dub., F.R.C.S., Surgeon Sir Patrick Dun's Hospital</td>
</tr>
<tr>
<td></td>
<td></td>
<td>77 Merrion-square, Dublin [1]</td>
</tr>
<tr>
<td>1893</td>
<td>Taylor, William</td>
<td>M.B., F.R.C.S., Surgeon Meath Hospital, and Cork-street</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fever Hospital, 32 Harcourt-street</td>
</tr>
<tr>
<td>1887</td>
<td>Thompson, S. M.</td>
<td>L.R.C.P., L.R.C.S., 34 Harcourt-street, Dublin</td>
</tr>
<tr>
<td>1897</td>
<td>Thompson, W. H.</td>
<td>M.D., F.R.C.S., Eng., Kings Professor of Institutes of</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Medicine, Trinity College, Dublin</td>
</tr>
<tr>
<td>1894</td>
<td>Thompson, W. J.</td>
<td>M.D., Univ. Dub., Physician Jervis-street Hospital,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>27 Harcourt-street, Dublin [2]</td>
</tr>
</tbody>
</table>
List of Fellows.

† Thomson, Sir William, C.B., ex-President R.C.S., Surgeon Richmond Hospital, 54 St. Stephen's-green, E., Dublin.

† Tobin, Richard Francis, F.R.C.S., Surgeon St. Vincent's Hospital, 60 St. Stephen's-green, Dublin.

1889 Todd, H. Ross, F.R.C.S., Bembridge House, Bembridge, Isle of Wight.

1889 Tweedy, Ernest Hastings, F.R.C.P., Master Rotunda Hospital. [2]

1900 Tweedy, Herbert, L.R.C.P. & S., Colonial Surgeon.

† Usher, Isaac William, L.R.C.S., Medical Officer Dundrum No. 1 Dispensary, Rathdown Union, Tudor House, Dundrum, Co. Dublin.

1901 Watson, Edward J., M.D., M.R.C.P., Anæsthetist and Medical Officer in Charge of the X-ray Department Sir P. Dun's Hospital, Demonstrator in Anatomy T.C.D., 25 Fitzwilliam-place.

1902 Wayland, R. S., F.R.C.S., Physician National Children's Hospital, 54 South Richmond-street.


1900 White, Arthur H., Professor of Pathology Royal College of Surgeons, Derrybawn, Rathgar.

1902 White, Reginald J., F.R.C.S.I., Physician National Maternity Hospital, 39 Lower Baggot-street.

1903 Wigham, Joseph T., M.D., Albany House, Monkstown, Co. Dublin.

1898 Wigoder, Selik, M.D. Leipzig, L.R.C.P. & S. Edin., 5 Harrington-street.

1894 Wilson, T. Henry, F.R.C.P., late Assistant Master Rotunda Lying-in Hospital, 33 Upper Merrion-street, Dublin.

1899 Wilson, G. O'Keeffe, F.R.C.S., Physician Drumcondra Hospital and to Cork-street Fever Hospitals, 34 North Frederick-street.


1905 Woods, Charles R., Colonel R.A.M.C., M.D., Dub., Temporary Fellow, Royal Hospital.


1894 Wynne, Albert E., L.R.C.P. & S. Edin., late House Surgeon Mercer's Hospital, 27 Westland-row, Dublin.

1884 Yourell, M. J., M.R.C.P., 10 Ailesbury-road, Merrion.
MEMBERS.


† Boyce, Jos. W., M.B., Medical Officer Blackrock Dispensary District, St. Kilda, Blackrock, Co. Dublin.

1897 Boyd, Alfred E., M.B., 4 Fitzwilliam-square, East

1904 Boxwell, William, M.E., Dub., Clinical Assistant Meath Hospital.

1903 Charles, Andrew, L.R.C.P. & S., 62 Harcourt-street.

1887 Cope, Geo. Patrick, L.R.C.P., L.R.C.S., late Assistant Resident Medical Superintendent Richmond District Lunatic Asylum, Medical Officer No. 3 Dispensary District, South Dublin Union, 36 Harcourt-street.

1900 Cullinan, Henry, L.R.C.P. & S., Richmond Asylum.

1892 Day, J. Marshall, M.B. Univ. Dublin, Resident Medical Officer Cork-street Fever Hospital, Dublin.

† Delahoyde, O'Connell J., F.R.C.S., Medical Officer No. 2 District North Dublin Union, 47 Rutland-square, Dublin.


1896 Fagan, P. J., F.R.C.S., Demonstrator of Anatomy Catholic University Medical School, 31 North Frederick-street.

1897 Fleury, Eleonore Lilian, M.D., R.U.I., Richmond District Asylum, Dublin.

1903 Glenney, Edmund, F.R.C.S., 14 Harrington-street.

1900 Goff, A. S., L.R.C.P. & S., Lynton, Dundrum.

1889 Goulding, H. Benson, F.R.C.S., 16 Rathmines-road.

1900 Harvey, Robert James, F.R.C.S., Assistant Surgeon Richmond Hospital, 7 Gardiner's-row.

1898 Hatch, Richard, L.R.C.P. & S., 166 Pembroke-road.

1897 Hughes, Charles, L.R.C.P. & S., 16 Lower Fitzwilliam-street.

1903 Keegan, J. F., F.R.C.S., 2 Pembroke-road. [1]

1902 Lambe, Francis W., M.B., B.Ch., B.A.O., R.A.M.C., 7 Cornwall-road, Dorchester, Dorset.


1903 Lynn, K., M.B., B.Ch., B.A.O., 9 Belgrave-road, Rathmines.

List of Student Associates.

1905 Pasley Claude B., L.R.C.P. & S.I., 63 Dalymount, N.C.R.

1898 Redington, John, F.R.C.S., Richmond Asylum.

1885 Shaw, James, L.R.C.S., 93 Talbot-street, Dublin.

1904 Stokes, Henry, M.B., Dub., Clinical Assistant Meath Hospital, Harcourt-street.

1904 Stoney, R. Atkinson, M.B., B.Ch., B.A.O., Surgeon City of Dublin Hospital, 115 Lower Baggot-street. [1]

1900 Tennant, Elizabeth A., L.R.C.P. & S., 15 Harrington-street.

1895 Turner, David, F.R.C.S., late Resident Medical Officer Royal Hospital for Incurables, 69 Grosvenor-road, Rathmines.


STUDENT ASSOCIATES.

Baker, Miss Madeleine S., 173 Rathgar-road.

Boyce, W. W., St. Kilda, Blackrock.

Brown, R., 1 Stamer-street.

Clarke, Miss G. W., 16 Granby-row.

Devane, J. F., Mater Misericordiae Hospital.

Hargrave, Miss, 8 Upper Mount-street.

Hayden, P. E., Mater Misericordiae Hospital.

Haves, R. F., Mater Misericordiae Hospital.

Leighton, W. J., Richmond Hospital.

Murphy, R. X., 18 Harcourt-street.

Nash, James, Medical School, Cecilia-street.

O’Driscoll, T., Mater Misericordiae Hospital.

O’Meara, Miss, 3 Churchill-terrace, Glasnevin.

O’Reilly, Alfred, 75 Harcourt-street.

Talbot, Sidney W., Iverston, Tivoli-road, Kingstown.

Vasquez, John, 18 Harcourt-street.

Woolcombe, Robert Lloyd, LL.D., 14 Waterloo-road, Dublin.
RULES.

1. The name shall be, "Royal Academy of Medicine in Ireland." (1887.)

Constitution.

2. The Academy shall consist of Fellows, Honorary Fellows, Members, and Student Associates.

Management.

3. The affairs shall be managed by a Council, consisting of the President, Ex-Presidents (1893), the six Presidents of Sections, the General Secretary and Treasurer, the Secretary for Foreign Correspondence, six Secretaries of Sections, and eight Councillors, being two representatives from the Medical, Surgical, Obstetrical, and Pathological Sectional Councils respectively.

Meetings.

4. The Meetings shall be General and Ordinary.

Publication of "Transactions."

5. The "Transactions" shall be published by the Council, subject to the provisions hereinafter contained.

Original Fellows and Members.

6. All the Members of the present Societies (Medical, Surgical, Obstetrical, and Pathological) shall be Original Fellows or Members, without entrance fee, on payment of the annual subscription on or before 31st December, 1882.

Fellows.

7. Fellows of the Royal College of Physicians of Ireland, and of the Royal College of Surgeons in Ireland, shall be admitted, without ballot, on payment of the entrance fee and the subscription for the current year. All others, being Registered Medical Practitioners not directly or indirectly engaged in the sale of drugs, shall be proposed by two Fellows, and elected by ballot by the Council.

8. Candidates shall be proposed at one Meeting of the Council, and balloted for at the next—one black bean in four to reject.

8a. That all Rules referring to the admission of Fellows, Members, and Student Associates shall be interpreted as referring to Ladies as well as Gentlemen.

Privileges of Fellows.

9. Fellows only shall be eligible for office in the Academy. They shall have the privilege of attending all Meetings of the Academy, of making Communications, and of voting and speaking at such meetings. They shall also receive a copy of the "Transactions."

* Those who have paid a Life Subscription to any of the above Societies will be admitted to the privilege of Fellows on payment of Member's subscription.
xxviii

Rules.

10. These privileges shall not be exercised by any Fellow in arrear with his subscription.

Honorary Fellows.

11. Honorary Fellows, limited in number to 25, may be nominated by the Council, and elected, on motion at a General Meeting of the Academy by a majority of at least two-thirds of those present and voting.

Members.

12. Any Registered Medical Practitioner may be elected as a Member, the election to be conducted in the same manner as that of Fellows.

Privileges of Members.

13. Members shall have the privilege of attending the Ordinary Meetings of the Academy, of making Communications, and of taking part in debate. They can purchase the "Transactions" at cost price.

Student Associates.

14. Registered Medical Students may be elected Student Associates for the period of one year. The Subscription must be paid before election, and the Council may elect without notice of motion.

15. Student Associates shall have the privilege of attending the Ordinary Meetings of the Academy.

Annual Subscription.

16. Fellows shall pay £2 2s., and Members £1 1s. Student Associates shall pay 5s. The Subscription shall become due on the 1st of October in each year, and if the Subscription be not paid on or before the first Meeting in February, the defaulter shall cease to belong to the Academy, unless the delay shall be accounted for to the satisfaction of the Council. No Fellow shall vote at the Annual General Meeting who has not paid his subscription for the year. Registered Medical Practitioners not residing within 15 miles of Dublin are eligible as Fellows of the Academy on payment of the entrance fee, and an annual Subscription of £1 1s. Medical Officers on the active list of the Royal Navy and Army are eligible for election as "Temporary Fellows" of the Academy on payment of an annual Subscription of One Guinea. Such Temporary Fellows shall enjoy all the privileges of Fellows, except that of voting at General Meetings. No Fellow who has not paid his Subscription shall vote for any Candidate for Office at the Annual General Meeting, nor can the name of any Fellow be received by the General Secretary for insertion on the ballot paper as a Candidate for Office unless his Subscription shall have been paid for the current year.

Entrance Fee.

17. After admission of Original Fellows, all Fellows shall pay an entrance fee of £1 1s.

Council.

18. The Council shall meet on the first Wednesday in the month throughout the Session, or oftener should they see occasion—five to form a quorum.
19. Notice of all Extraordinary Meetings shall be transmitted by the Secretary to every Member of the Council. The President or any five Members of Council may call an Extraordinary Meeting of the Council. The Council shall determine questions by vote, or by division if so demanded, the President having a casting vote only. Any regulation of the Council shall have the force of a law, until submitted to the next General Meeting. The Council shall have the power of filling up any vacancies which may occur in the list of Officers of the Academy, except that of President, before the Annual General Meeting. If a vacancy in the office of President should occur, the General Council shall summon a Special General Meeting of the Academy to fill such vacancy. (1888.)

Sectional Councils.

20. There shall be six Sectional Councils elected by the Annual General Meeting in October, termed respectively—the Medical, the Surgical, the Obstetrical, the Pathological, the State Medicine, and the Anatomical and Physiological Councils.

21. No Fellow shall be eligible as a candidate for election on more than two Sectional Councils, but no Fellow shall be eligible as a candidate for election on both the Medical and Surgical Sectional Councils. (1888.)

22. Each Sectional Council shall consist of the President of the Section and ten Members, one of whom shall act as Secretary to the Section; except the State Medicine and Anatomical and Physiological Councils, which shall each consist of a President and six Members. (1888.)

Meetings of Sectional Councils.

23. Each Sectional Council shall meet on a fixed day at least one week before the Ordinary Meeting of their Section, three to form a quorum.

Powers.

24. Each Sectional Council shall have the power of making any such arrangements as it thinks necessary to carry on the work of the Ordinary Meetings which are under its charge, provided that such arrangements do not interfere with the general laws of the Academy; and any Rules laid down by such Council shall have the force of laws at the Ordinary Meetings under its charge, until submitted to the General Council.

25. Each Sectional Council shall have the power of filling up any vacancies that may occur among its Members until the Annual General Meeting.

Committee of Reference.

26. The Council shall appoint a Committee of Reference, to report upon morbid growths and other specimens exhibited before the Academy; of this Committee the Exhibitor shall, for the occasion, be a Member.

Officers.

27. A President, to be elected by the Annual General Meeting in October, and to hold office for three years.

28. The Presidents of the Colleges of Physicians and Surgeons for the time being shall be the Presidents of the Medical and Surgical Sections. The Presidents of the other Sections shall be elected by the Fellows at the Annual General Meeting, and shall hold office for two years. (1888.)
29. One General Secretary and Treasurer to be elected at the Annual General Meeting.

30. It is expedient that a fixed salary (of one hundred guineas) shall be paid yearly to the General Secretary in consideration of the fact that the editing of the "Transactions" is part of his duties.

31. One Honorary Secretary for Foreign Correspondence to be elected at the Annual General Meeting. (1888.)

32. The Councillors for each Section to be elected at the Annual General Meeting. Each Sectional Council shall elect two Members to act on the General Council, except in the case of the Sections of State Medicine and Anatomy and Physiology. (1888.)

33. Two Members in each Sectional Council shall retire annually, and be ineligible for re-election for one year, except in the Council of the Section of Anatomy and Physiology, in which only one shall retire. (1896.)

34. Six Secretaries, one for each Section, to be appointed by the Sectional Councils.

35. At all elections after the year 1882, any Fellow desirous of nominating a candidate for election shall, at least ten days before the Annual General Meeting, forward an application to the General Secretary to enter the name of such Fellow on the list of candidates for office, provided that the Fellow so nominated shall have consented to act, and shall have paid his subscription at the time of nomination. Should there be an insufficiency of regularly nominated candidates, the vacancies so created shall be filled up by the Sectional Councils at their first meeting after election. (1891 and 1901.)

36. That all elections shall be by ballot, but Fellows residing more than 15 miles from Dublin, and those incapacitated by illness (to be certified), may record their votes by ballot papers, sent to the presiding officer in sealed envelopes provided for that purpose. A Fellow cannot avail himself of this privilege unless his subscription for the current session has been paid. (1896 and 1901.)

37. That in all elections to the Sectional Councils there shall be affixed to the name of each candidate the number of meetings that he has attended of that particular Section of the Council for which he is now a candidate. (1898.)

Duties of Officers.

38. The President shall preside at the Annual and Special General Meetings and at General Council Meetings. In the absence of the President, the Chairman shall be appointed by the meeting. (1888.)

39. The Presidents of Sections shall preside at the Ordinary Meetings of the Academy, and shall also preside at the Sectional Council Meetings. In the absence of the President, the Chairman shall be appointed by the meeting. (1888.)

40. The General Secretary shall attend all General Meetings of the Academy and General Council. He shall take minutes of such meetings, to be read at the following meeting.

41. He shall receive and have charge of all papers intended for publication in the "Transactions" of the Academy, after they have been handed over to him by the Secretaries of the several Sections.
42. He shall, on receiving notice from the Secretary of a Section, send out to all the Members notices of the title or titles of the paper or papers for the next Ordinary Meeting, with the name or names of the authors, and, so far as possible, of the subjects for Exhibition, with the names of the Exhibitors.

43. He shall arrange for the Exhibition of specimens and the reading of papers, which are to be forwarded to the Academy by those who are absent, or are not members.

44. The General Secretary and Treasurer shall receive all moneys, and lodge the same in bank to the account of the Academy, and all cheques shall be signed by the Treasurer and one other Councillor.

45. The Accounts shall be audited by two Fellows, not Members of Council, to be appointed by the President at some meeting previous to the Annual Meeting.

Duties of Secretaries of Sections.

46. To attend the Meetings of the Council of the Section and the Ordinary Meetings of the Academy, under the management of said Council, and to take minutes at such meetings, to be read at the next following meeting of that Section.

47. To keep such papers as the Sectional Councils recommend for publication, for the purpose of handing them over to the General Secretary.

48. To inform the Secretary of the Committee of Reference of any specimens referred to that Committee, and to transfer the specimens to that Secretary.

49. To give notice to the General Secretary, one week previously to the meeting, of the titles of papers for the evening, the names of the authors, and, so far as possible, the objects for Exhibition, with the names of Exhibitors, so that the General Secretary may inform the Members.

Meetings.

50. The Annual General Meeting to take place on the second Friday in October, for the election of Officers and Members of Council, and for the general business of the Academy.

51. Due notice of the meeting shall be given by the Secretary to all Members at least three weeks previously. (1891.)

52. No motion involving a change of these Rules shall be brought before this meeting except one week's notice thereof shall have been given by the Secretary to each Member.

53. The President may—and shall forthwith, on receiving a requisition signed by seven Fellows, at any time—on giving one week's notice, summon a Special General Meeting, for the consideration of particular business, the nature of which must be specified in the letter of summons convening the meeting, and at such meeting no other business can be transacted. In the event of the President being unable, from any cause, or declining, to summon a Special General Meeting of the Academy, it shall be in the power of the General Council to summon such meeting. (1888.)
Ordinary Meetings.

54. The communications to be submitted to the Ordinary Meetings shall be grouped under the following heads:—Medicine, Surgery, Pathology, Obstetrics, State Medicine, and Anatomy and Physiology; and the conduct of such meetings shall be in the hands of the several Sectional Councils, each Sectional Council to have the management of the Ordinary Meeting in rotation, as arranged by the General Council. (1888.)

55. The Ordinary Meetings shall be held on every Friday evening, from the last Friday in October until the last Friday in May, inclusive, at eight o'clock, except during the Christmas and Easter recesses.

56. All Fellows, Members, and Student Associates attending the meetings shall write their names in the attendance book.

57. Any Fellow or Member may introduce two Visitors by cards obtained from the Sectional Secretaries.

58. Officers of the Army or Navy Medical Departments shall, on presenting their cards, be admitted to the Ordinary Meetings of the Academy.

59. No communication shall exceed twenty minutes in its delivery, nor any speech thereon ten minutes, except by permission of the Chairman. No one shall speak twice upon the same communication, except the author, who has the right of reply.

60. A paper by any other than a Fellow or Member of the Academy shall not be read before the Academy unless the author of such a communication shall have obtained permission to do so from the Council of the Section before which the communication is proposed to be read. (1892.)

Ordinary Meetings.—Order of Business.

61. (1.) Chair to be taken at 8 30 p.m.
(2.) Chairman to read list of specimens, &c., exhibited by card, together with the names of the Exhibitors.

(3.) No Pathological Specimen shall be exhibited at any Section other than the Pathological and Obstetrical, except by card. This Exhibition shall not exclude any subsequent communication regarding it at the Pathological Section.

(4.) There shall be no Exhibition of Specimens by card in the Pathological Section.

(5.) Any member shall have liberty to exhibit any recent specimen at any of the meetings of the Obstetrical Section, provided it illustrates any question in gynaecology.

(6.) At the meetings of the Obstetrical Section recent specimens may be exhibited, and the President may invite discussion thereon, provided that such exhibition of specimens or discussion, if any, thereon, must terminate at 9 o'clock, p.m., but that, if necessary, they may be resumed after the papers for the evening have been read and discussed.

(7.) Chairman to ask if any member has any observations to make or motion to propose relative to any living specimen on the List of Exhibition.
(8.) Chairman to call upon the author of the first paper on the list to read his paper.

(9.) Chairman to call upon members to discuss the paper, or, at his discretion, to take any other paper or papers on the list relating to the subject, and have the discussion subsequently on all such papers collectively.

(10.) When the last paper has been discussed, the Chairman to ask if any member desires to speak upon any of the specimens exhibited by card.

(11.) After the discussion upon any specimen, the Exhibitor has the right of reply.

Regulations regarding the Exhibition of Specimens by Card.

62. (1.) Any member may exhibit by card at any Ordinary Meeting, except at the meeting of the Pathological Section. At the meetings of the Pathological all specimens must be presented and described _viva voce_, and debate may be invited thereon.

(2.) Notice shall, if possible, be given to the General Secretary, or the Secretary of the Section, on or before the previous Ordinary Meeting.

(3.) Specimens must be in the room at 7 45 on the night of Exhibition.

(4.) Specimens for Exhibition by card shall be open for inspection at 8 p.m.

(5.) A card, containing all particulars for publication, shall be placed with the Specimen. Cards for this purpose are to be obtained from the Secretary.

(6.) The Exhibitor should be present, and he shall furnish further details if asked for.

(7.) Every Exhibitor shall submit the Specimen or Specimens on view to the Committee of Reference, if the meeting so decide.

Exhibition of Pathological Specimens.

63. No lengthened reference to treatment shall be allowed upon any Specimen, except by the express permission of the Chairman. Whenever it has been agreed that a Specimen exhibited at a Sectional Meeting of the Royal Academy of Medicine in Ireland shall be sent to the Reference Committee to report thereon as to its nature, the Exhibitor is to retain the custody of the specimen until he shall be summoned to a meeting of said Committee to be convened by its Secretary, on an early day, when he will attend and submit it for examination. (1889.)

By-laws concerning "Transactions."

64. The "Transactions" shall consist of such Communications made to the Academy by or through Fellows or Members as may be deemed by the General Council suitable for publication; also, of discussions of importance or interest arising out of such Communications.
65. All Communications accepted by the Academy become the property of the Academy, but authors may also print their Communications, subsequent to the reading of the same before the Academy, in any publication in addition to the "Transactions." Papers shall be handed to the Secretary of the Section immediately after they have been read. (1891.)

66. The "Transactions" for the year shall be presented to all Fellows of the Academy who have paid their Annual Subscriptions.

67. The "Transactions" may be purchased by Members at cost price.

68. The Publication Committee of each Section shall meet not later than the Tuesday after each meeting of the Section, for the purpose of abstracting the proceedings—the abstract to be placed in the printer's hands on the same evening, and forwarded to the editors of medical journals with the least possible delay. (1888.)

69. Contributors of papers may send their papers to the Academy printer early enough to allow of their being put in type before the meeting, provided the author be responsible for the cost of same should the General Council deem the communication not suitable for publication in the "Transactions."

70. That on the evening of the day of meeting of the Sectional Council, when the papers for the next meeting have been decided upon, a circular be sent to each contributor informing him:

(1.) That he is expected to be ready or else take his place at the bottom of the list.

(2.) That he must have an abstract ready with his paper, otherwise he will be noted in the published proceedings in such form as the Publication Committee think fit.

71. The General Council is empowered to defray the expenses in whole or in part of any illustrations which it may consider advantageous to the elucidation of the papers published by the Academy.

72. An abstract (prepared by the author) of each communication made at the Academy, along with a report of the discussions thereon, shall be furnished to the editors of such medical journals as may desire to publish them, and the authors of such communications shall be empowered to publish their papers in extenso in any periodical or periodicals they may think fit, such communications also to appear in the "Transactions," provided the Council consider them worthy of insertion.

Expulsion of Fellow or Member.

73. Expulsion of a Fellow or Member can take place only at a General Meeting of the Academy, on the motion of the Council, if two-thirds of the Members present shall vote for the same by ballot. Of such ballot the Council must give at least fourteen days' notice in writing to every Fellow of the Academy.

New Laws.

74. New Laws, or alterations in existing Laws, can be proposed only at the Annual General Meeting. Any Fellow proposing such alteration shall give notice to the General Secretary at least ten days before the General Meeting in October.
REPORT.

The General Council reports that the number of Fellows for the Session 1903-4 was 219, of Members 32, and of Student Associates 11. The Fellows decreased by 13, the Members increased by 1, and the Student Associates decreased by 1.

Notwithstanding this falling off in the number of Subscribers, and a corresponding reduction in the amount of Subscriptions, the financial condition of the Academy compares favourably with that of the last Annual Report.

The overdraft at the Bank of £25 18s. 3d. has been cleared off, and the Academy has now a credit balance instead of £84 6s. 1d., a result mainly due to the action of the Finance Committee.

The attention of the Council was directed by the Obstetrical Section to the injustice done to our countrywomen by the Midwives Act, and a resolution was adopted by the General Council submitting that any woman holding the Nursing Certificate of the Irish Chartered Maternity Hospitals should be deemed to have complied with the Rules of the Central Midwives' Board regulating the course of training, and should be eligible to present herself for the Examination of the Central Midwives' Board. This Resolution was forwarded to the Secretary of the Board, with a letter pointing out the injustice to which the Irish Chartered Hospitals would be subjected under the present regulations. After a long correspondence, on 26th May a Resolution was passed by the Central Midwives' Board, to the effect that the Privy Council should be asked to sanction the appending of a note modifying the existing Rule, and of an alternative form of
Certificate, so as to facilitate the admission of Irish trained midwives to the Examinations of the Board.

The arrangements for reporting the Proceedings adopted last Session were not found satisfactory, and the Council decided to leave the matter altogether in the hands of the Sectional Councils, who have been empowered to arrange for the reporting of their meetings at a cost not exceeding 10s. 6d. per meeting in each case.

With reference to the report of an Inquest held in the Royal City of Dublin Hospital on 8th April, 1904, and to a statement made on that occasion by the Coroner, a Resolution was adopted by the Council that the circumstances connected with this case were such as to interfere with the course of justice, to reflect on the conduct of the Medical Profession, and to hamper its members in the discharge of their professional duties, and recommending that the matter should be brought under the notice of the Government, with a view to inquiry being made into the facts. A Resolution dealing with this matter will be submitted to the General Meeting.

The Council reports that at the meeting in April, 1904, a letter was received from the General Secretary intimating that it was not his intention to offer himself for re-election next Session.

The Council records with regret the loss the Academy has sustained by the death of the following Fellows—Sir Philip C. Smyly, Austin Meldon, J. J. Cranny, and J. L. Lane.

<table>
<thead>
<tr>
<th>RECEIPTS</th>
<th>£</th>
<th>s.</th>
<th>d.</th>
</tr>
</thead>
<tbody>
<tr>
<td>To Subscriptions and Entrance Fees</td>
<td>-</td>
<td>419</td>
<td>0 0</td>
</tr>
<tr>
<td>&quot; Dividends on £596 2s. 4d. 2½ per cent.</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Consolidated Stock and £155 1s. 10d.</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>New 2½ per cent. Government Stock</td>
<td>18</td>
<td>1</td>
<td>8</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>EXPENDITURE</th>
<th>£</th>
<th>s.</th>
<th>d.</th>
</tr>
</thead>
<tbody>
<tr>
<td>By Balance due to Bank</td>
<td>-</td>
<td>25</td>
<td>18</td>
</tr>
<tr>
<td>&quot; General Secretary</td>
<td>-</td>
<td>105</td>
<td>0</td>
</tr>
<tr>
<td>&quot; Printing, Stationery, and Postage</td>
<td>-</td>
<td>50</td>
<td>4</td>
</tr>
<tr>
<td>&quot; Transactions, Vol. XXI.</td>
<td>-</td>
<td>103</td>
<td>0</td>
</tr>
<tr>
<td>&quot; Royal College of Physicians</td>
<td>-</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td>&quot; Royal College of Surgeons</td>
<td>-</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td>&quot; Reporters</td>
<td>-</td>
<td>5</td>
<td>15</td>
</tr>
<tr>
<td>&quot; Servants</td>
<td>-</td>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>&quot; Groceries and Milk</td>
<td>-</td>
<td>12</td>
<td>18</td>
</tr>
<tr>
<td>&quot; Lantern</td>
<td>-</td>
<td>0</td>
<td>12</td>
</tr>
<tr>
<td>&quot; Professor McWeeneey, Index</td>
<td>-</td>
<td>26</td>
<td>5</td>
</tr>
<tr>
<td>&quot; Interest on Account</td>
<td>-</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td>&quot; Sundries</td>
<td>-</td>
<td>9</td>
<td>15</td>
</tr>
<tr>
<td>&quot; Balance in Bank</td>
<td>-</td>
<td>84</td>
<td>6</td>
</tr>
</tbody>
</table>

Total                          | -  | -  | £467 1 8 |

The Capital is invested in the names of Sir William Thomson, Dr. Walter G. Smith, and Dr. James Craig.

We have examined the Accounts and Vouchers, and certify the same to be correct.

J. Lumsden.
R. Charles B. Maunsell.

3rd October, 1904.
Volume XXII. of the "Transactions" has been forwarded to the following:—

**IRELAND:**
- Medical Press - - - - Dublin.
- National Library - - - - Do.
- Royal College of Physicians - - - - Do.
- Royal College of Surgeons - - - - Do.
- Royal Irish Academy - - - - Do.
- Royal Dublin Society - - - - Do.
- Royal University - - - - Do.
- Trinity College - - - - Do.
- Queen's College - - - - Do.
- Do. - - - - Do.
- Do. - - - - Do.

**ENGLAND:**
- Birmingham Medical Review - - - - Birmingham.
- Medical Institute - - - - Do.
- Bristol Medico-Chirurgical Journal - - - - Bristol.
- Durham College of Medicine - - - - Newcastle-on-Tyne.
- University - - - - Durham.
- Liverpool Medico-Chirurgical Journal - - - - Liverpool.
- British Medical Journal - - - - London.
- Jenner Institute of Preventive Medicine - - - - Do.
- Journal of Obstetrics and Gynaecology, 26 Queen Anne-street, W. - - - - Do.
- King's College - - - - Do.
- Lancet - - - - Do.
- Library, British Medical Journal, 429 Strand - Medical Magazine - - - - Do.
- Medical Review (Med. & Surg. Review of Reviews) - - - - Do.
- Royal Medical Chirurgical Society, Hanover-square - - - - Do.
- Public Health, 19 Bloomfield-road, Maida Vale - - - - Do.
- Royal College of Physicians - - - - Do.
- Royal College of Surgeons - - - - Do.
- University of London, Burlington Gardens - - - - Do.
- University College - - - - Do.
- Victoria University - - - - Do.
- Official Year Book of Scientific and Learned Societies, Exeter street, Strand - - - - Manchester.

**SCOTLAND:**
- University - - - - Aberdeen.
- University College - - - - Dundee.
- Royal College of Physicians - - - - Edinburgh.
- Royal College of Surgeons - - - - Do.
- University - - - - Do.
- Faculty of Physicians and Surgeons - - - - Glasgow.
- University - - - - Do.
- Do. - - - - Do.

**EUROPE:**
- University - - - - Amsterdam.
- Naturforschende Gesellschaft - - - - Basel.
EUROPE—con.—

University Library - - - - - Berlin.
University College - - - - - Bologna.
Académie Royale de Médecine de Belgique - - - - - Brussels.
L’Année Chirurgicale - - - - - Do.
Société Belge de Chirurgie - - - - - Do.
Université Libre - - - - - Brussels.
University College - - - - - Christianity.
Académie de Médecine - - - - - Copenhagen.
La Grèce Medicale, Syra - - - - - Greece.
University College - - - - - Madrid.
Archivio di Ortopedia - - - - - Milan
Académie de Médecine - - - - - Paris.
Archives Orientales - - - - - Do.
Revue de Chirurgie - - - - - Do.
University of Paris - - - - - Do.
Imperial University - - - - - St. Petersburg.
Royal Carolinska Med. Chir. Institution. - - - - - Stockholm.
University Library - - - - - Strassburg.
Archivio Italiano di Otologia - - - - - Turin.
Medical Society, Royal University - - - - - Upsala.
University - - - - - Vienna.

Naturalforschende Gesellschaft, Dr. Rudolf Martin, Seefeldstrasse 119 - - - - - Zurich.

AMERICA:—

Johns Hopkins University - - - - - Baltimore.
Journal of Medical Research - - - - - Boston, Mass.
Brooklyn Medical Journal - - - - - Brooklyn.
McGill University - - - - - Montreal.
Academy of Medicine, 17 West 43rd Street - - - - - New York.
University - - - - - Do.
Journal of Mental Pathology - - - - - Do.
Medical Brief - - - - - St. Louis, Mo.
American Journal of the Medical Sciences - - - - - Philadelphia.
Association of American Physicians - - - - - Do.
College of Physicians - - - - - Do.
University of Pennsylvania - - - - - Do.
University Lavel - - - - - Do.
University of Toronto - - - - - Toronto.
Charlotte Medical Journal, Charlotte, N. C. - - - - - United States.
Library, Surg.-General’s Office, Washington - - - - - Do.
Journal of Comparative Neurology, Denison University, Granville, Ohio - - - - - Do.
Medical Library Association, 19th and Stout Streets, Denver, Colorado - - - - - Do.

AUSTRALASIA:—

University - - - - - Adelaide.
Do. - - - - - Auckland.
Do. - - - - - Melbourne.
Inter-Colonial Medical Journal - - - - - Do.
University of Otago - - - - - New Zealand.
The Australasian Medical Gazette, - - - - - Sydney, N.S.W.
University of Sydney, care of Young J. Pentland, 38 West Smithfield, London, E.C. - - - - - Do.

ASIA:—

Bombay University - - - - - Bombay.
University - - - - - Calcutta.
Imperial University Library - - - - - Tokio, Japan.
HODGKIN'S DISEASE OCCURRING IN TWINS.

By GEORGE PEACOCKE, M.D., F.R.C.P.I.;
Assistant Physician to the Adelaide Hospital.

[Read in the Section of Medicine, November 11, 1901.]

Since the year 1832, when Dr. Hodgkin first described the main clinical features of this disease, which now bears his name, numerous contributions on the subject have appeared, and many single cases and collections of cases have been published; but as yet the aetiology and pathology of the disease remain obscure.

Although the two cases which have recently come under my care throw little fresh light on the subject, one or two points in connection with them are, I think, of some special interest.

Frank and Charles W., twins, aged four years, came first under my notice in the early part of this year. Their father and mother are both alive and healthy, and, as far as I have been able to ascertain, there is no history of tuberculosis or any other so-called hereditary disease on either side. They have had six children. The first child died at birth, having reached full term; the second lived to be four years old and then died after an illness of about
twelve days from "gastric fever, followed by water on the brain." The third and fourth, both girls, aged eleven and seven respectively, are alive and in good health, and the remaining two children are the subject of the present communication. Both these children had been very healthy until the summer of 1902, when they contracted measles—as a result of which they both suffered for some time from enlargement of the cervical glands. I may add that the two girls also had measles at the same time, but in their case there was no glandular enlargement. Though this enlargement of the glands subsided there seems to have been some tendency to slight temporary enlargement on subsequent occasions, but in April, 1903, the glands of the right side of the neck of Frank enlarged and gradually increased in size. In August of the same year he was brought to the Adelaide Hospital, and the glands were removed by Mr. Gordon. They were examined microscopically at the time, and no evidence of tuberculosis was found. He left the hospital in three weeks' time with a slight puffiness behind the ear. This apparently increased in size, and in about two months time his mother noticed some fresh glands enlarging. In January of the present year she brought him to the out-patient department of the hospital, and he was admitted again under Mr. Gordon's care. As the child was pale, and there was considerable enlargement of the glands on the right side of the neck, I was asked to see him, and from that time on took charge of the case. I had him under observation until the middle of March, when, against my advice, he was taken home. His condition while in hospital remained much the same. He was bright and cheerful, and had a good appetite. The glands on the right side of the neck as far down as the clavicle were very much enlarged, of the soft variety, but showed no sign of breaking down or of inflammatory change. No other superficial glands were involved, and there was no evidence of any enlargement of the internal lymphatic glands. The spleen was, however, increased in size, but not to any great extent. The temperature was, as a rule, normal, but on two occasions, with exactly a month's interval, he had pyrexial attacks which lasted a few days, and from the account his mother has given me I believe these attacks continued, with somewhat shorter intervals, until the time of his death. A blood count made on the 2nd February showed—red cells 3,500,000 per c.m.
colour index normal; white cells 48,000 per cm. A differential count kindly made by Professor Scott, showed polymorphonuclears 15 per cent., lymphocytes 83 per cent. On the 20th February the white cells were only 8,200 per cm., and the differential count showed 57 per cent. polymorphs and 40 per cent. lymphocytes. Numerous counts were made during his stay in hospital, but on no occasion did the white cells again rise higher than 12,000, and the differential count resumed the normal condition. After his removal from hospital I did not again see him until a few weeks ago when he was brought to the hospital, but his mother would not allow him to be left there. The disease had advanced considerably. He was unable to walk, extremely anaemic, listless and fretful at times, his face puffy, the lips and conjunctive almost colourless, his pulse small and feeble, and it was quite evident he could not live long. I visited him subsequently at his home and examined him carefully. There was little increase in the glandular enlargement; a few glands in the right axilla were involved, but the left sides of the neck and axilla and both groins were not implicated. The spleen was greatly enlarged, and reached as low as the umbilicus. Professor Scott kindly made a blood count for me and found red cells 1,300,000. The haemoglobin percentage could not be accurately obtained as the pipette got accidentally broken before the examination was completed, but as nearly as he could ascertain it was about 10 per cent., certainly considerably below 20 per cent. The white cells were 8,400. A differential count showed polymorphs 76 per cent., lymphocytes 21 per cent. His temperature was 101°. he had a short cough, but I could detect no evidence of any pulmonary mischief. He very rapidly got weaker and died on the 30th October, eighteen months after the disease first definitely made its appearance. I was unable to obtain a post-mortem examination.

The other boy, Charles, came under my care on the 22nd February. About three weeks previous to this his mother noticed a swelling behind the left ear, which was soft at first but had got harder, and increased in size. His general health was good, he was in the best of spirits and had a very good appetite. With the exception of some enlargement of the glands on the left side of his neck there was nothing else abnormal found on examination. His spleen was not enlarged, and during the three
weeks he was in hospital he had no pyrexia. Examination of the blood showed a normal condition. He was also taken home last March, and I lost sight of him until early last month, when he was brought to the hospital to have the enlarged glands removed. I examined him carefully, and could detect no evidence of glandular enlargement, with the exception of the group of glands on the left side of the neck. His spleen was not enlarged, and the blood count showed 4,000,000 red cells per c.m. haemoglobin 85 per cent. white cells 8,500 per c.m. Mr. Heuston removed all the enlarged glands, and Professor Scott has kindly furnished me with the following report on the histological characters of the glands:—

"The enlargement of the lymph glands is due to a general enlargement of the cellular portion of the glands. A few of the larger trabeculae remain, but the smaller ones are very inconspicuous. There appears to be a slight increase of the fine connective tissue between the cells, visible by special staining, which gives the whole gland an unusual hardness. He is still in hospital, and it will be interesting to watch the further progress of the case.

The chief point of interest in these two cases appears to me to lie in the fact that the disease manifested itself in the two boys, who were twins.

This may have been a mere coincidence, or possibly it may point to some constitutional weakness shared by each, which rendered them more liable to contract the disease, or it may be taken as a factor in favour of the infectivity of this disease—the two boys being all their lives playmates together, and being, as I know, very much attached to one another.

Another curious point was the selective manner in which the glands were affected. In one boy the glandular enlargement was entirely confined to the right side of the neck and right axilla. In the other, the left side of the neck was alone affected.

The boy who has died, and who was apparently first affected by the disease, had enlargement of the spleen the first time I saw him, last January, and the increase in anaemia depended, as far as clinical observation can tell, more on the increase in
size of this organ than in an increase in the glandular enlargement of the neck.

The other boy, who is now alive and in good health, and, as far as his blood count shows, little if at all anæmic, had certainly as great an enlargement of the cervical glands, but as yet no palpable splenic enlargement.

If the account of the mother can be fully relied upon, and I have no reason to doubt it, the first damage to the glands resulted from an attack of measles, six months before definite signs of the disease appeared.

In the *Philadelphia Medical Journal* T. C. Ely describes a case of Hodgkin's disease in a child, four years old, the first sign of glandular enlargement having occurred eighteen months previously, after an attack of whooping-cough.

In 1865 Trousseau pointed out that in some cases the enlargement of the lymphatic glands was, in the first place, due to some local source of irritation in the neighbourhood of those glands which first became affected.

While these facts point to a definite predisposing factor in many cases of Hodgkin's disease, and possibly, if careful examination was made in all cases, some source of irritation could always be found, it does not answer the question, What is the cause of the disease?

Hodgkin's disease is, comparatively speaking, rare. Glandular enlargement, whether as a result of some of the infectious diseases or due to tubercular, syphilitic or other poisons, is very common.

A considerable amount of work has recently been done in connection with this and similar affections, by Flexner, Moorhead, and many others, and it would seem as if we were on the threshold of important discoveries, which will demonstrate not only the rôle of the lymphocyte, but also the pathology of Hodgkin's disease and those other diseases apparently so nearly allied to it.
SYRINGOMYELIA, WITH ACCOUNT OF AN UNCOMMON CASE.

BY JOSEPH O'CARROLL, M.D., F.R.C.P.I.,
Physician to the Richmond, Whitworth, and Hardwicke Hospitals.

WITH PATHOLOGICAL NOTE BY
H. C. EARL, M.D.,
Fellow of the Royal College of Physicians,
Pathologist to the Hospitals.

[Read in the Section of Medicine, December 9, 1904.]

It has been my fortune to come across in my wards four cases which fall under the designation of hydromyelia or syringomyelia: that is, a more or less tubular excavation of the spinal cord, the cavity being filled with clear watery fluid. One of these cases is still living, and the final evidence of the accuracy of the diagnosis is still lacking. But the clinical features of the case were so characteristic of syringomyelia, and have remained so for so many years, that there can be no reasonable doubt that the condition is present. The man was presented to the Academy by Dr. Coleman and myself in 1892. Before that time I had come across in the post-mortem room a spinal cord in which the central canal was dilated in a perfectly even manner in almost its whole length, the greatest diameter being about four millimetres. The man had been under my observation for only a day or two. He had arrived in Dublin from Limerick in a condition of such acutely occurring muscular helplessness and such mental confusion and excitement that he was arrested for supposed alcoholic intoxication before being brought to the hospital. There he presented a condition which suggested some acute cerebral inflammation;
Drs. O'Carroll and Earl. — "Syringomyelia."

Fig. 1.—Case of G. J. Section of Cord in Cervical Region.
Drs. O'Carroll and Earl.—"Syringomyelia."

Fig. 2.—Case of J. M·K. Section of Cord at origin of 3rd Cervical Nerve.
Dr. O’Carroll and Earl.—"Syringomyelia."

Fig. 3.—Case of J. M. K. Section of Cord at origin of 6th Dorsal Nerve.
By Drs. O'Carroll and Earl.

delirium, paralysis, acute bedsores, and hyperpyrexia: and died in a few days. Large plaques of haemorrhage were found in the pia-arachnoid structures, and the dilatation of the cord already referred to. The case is described more fully as a case of hydromyelus in the “Transactions” of the Academy for 1892.

A third case (G. T.), which I have not hitherto recorded, was that of a tall, raw-boned countryman, who came into hospital in April, 1894, for some ataxic symptoms. I think he really only felt that he wanted a day or two of rest, for he remained only that time with us at first. I saw him only for a few moments one day, and noticed that he had lost the end joint of one index finger, and was losing the tip of the thumb by a painless necrosis. The name “Morvan’s Disease,” with a note of interrogation after it, was put at the head of his chart, and a full examination deferred till the morrow. But on the morrow he had left the hospital for his home. About eight months later he arrived at the hospital in a state of rapidly-increasing paralysis, and when I saw him next day he was dying by failure of the muscles of respiration, so that a detailed examination would have been not merely cruel but impracticable. His spinal cord was found to contain a very large unsymmetrical cavity quite unlike that of my previous patient. (Fig. 1.)

These two fatal cases exemplify a distinction which is drawn by many writers between dilatation of the normal central canal of the cord, named hydromyelus (on the analogy of hydrocephalus, a dilatation of the normal cavity of the brain), and excentric excavation, for the most part unsymmetrical, to which the name syringomyelia is applied. I show sections of these two cords.

Although it has not been absolutely proven that the distinction is a natural one, it may have its use in
Syringomyelia, with account of an Uncommon Case.

descriptive morbid anatomy, and possibly in helping to separate clinical types. But in using the term syringomyelia in the description of the following case I shall apply it in the general sense of a longitudinal internal excavation of the spinal cord.

I think it well to call to mind the clinical signs which, in a majority of cases at least, characterise syringomyelia. Taking the motor system first, the greater or less compression by the fluid at certain levels may determine trophic changes in the cells of the anterior horns, with consequent progressive atrophic changes in the muscles related to them. At the same time descending degeneration in the pyramidal tracts may determine more or less spasticity in the muscles of levels inferior to those undergoing atrophy. Thus the clinical picture of progressive muscular atrophy in the arm and hand muscles and a more or less spastic condition of the legs is not uncommon. This spasticity is accompanied by increased knee-jerks, ankle clonus, and Babinski's toe-extension sign.

Certain trophic changes may occur in the skin of the atrophic limbs, such as blue, chilblainy hands, solid oedema, glossy skin, &c. (la main succulente of Marinesco).

The hand not infrequently after a time assumes a peculiar shape; the inner three fingers becoming flexed into the palm, while the thumb and index remain extended, and facing one another in just such a position as they occupy when holding a pen. In fact the whole hand may be said to occupy the ordinary writing posture with the pen dropped out. To this position of hand Marie applies the name main en pinces. I owe to this distinguished neurologist the personal demonstration of another trophic peculiarity in this disease—namely, the
thorax en bateau. This consists of a hollowing of the upper part of the chest anteriorly in such wise that a line drawn from the point of one shoulder to the point of the other, across the upper part of the sternum, shows such increasing depression from each side towards the sternum as to suggest the concavity of a canoe, hence the name suggested by Marie. No doubt this deformity—by no means remarkable till one sees its recurrence in a number of cases—is determined by the loss of bulk in the upper muscles of inspiration, and a consequent lapse of the bony cage into a position of more or less complete expiration.

The motor picture resembles closely that presented frequently by a chronic compression myelitis such as that met with in vertebral caries, or, to a still greater extent, amyotrophic lateral sclerosis.

But it is on the sensory side that the changes are supposed to be really characteristic. Tactile anaesthesia, anaesthesia to pain, to heat and cold, and various other stimuli may all be absent in certain areas; but of all of these the sensation of simple touch is commonly the least affected. Further—and this is the classical mark of syringomyelia—the sensibility for touch may frequently be keen in areas marked by absolute insensibility to pain and heat stimuli. Thus a man so affected may be able to hold a match rightly to light his pipe, which he could hardly do without fair sense of touch, and may let the match burn away till the finger tips undergo a severe destructive burn. This is the dissociation of sensibility which is the prime character of syringomyelia. The case described by Dr. Coleman and myself showed this dissociation very markedly. His left arm was keen in touch sensation, and absolutely insensitive to pain stimuli. The outer part of the right arm and hand showed con-
Syringomyelia, with account of an Uncommon Case.

Considerable loss of tactile sensibility, and only slight insensibility to pain. Thermal sensibility seemed to be disturbed on both sides in the anaesthetic and analgesic areas. In company with this loss of pain sense, there may be painless necrosis, or painless whitlows of some of the finger tips, the condition originally described by Morvan. It is to be noted that the atrophic, spastic, and sensory signs are rarely quite symmetrical on both sides—a point which is of importance. And lastly, as in most other diseases, no one case is likely to present all the characteristics just described.

It will thus be seen that the clinical picture of syringomyelia is very special and characteristic: but the following case shows that the disease may exist without presenting such a conjunction of them as to make the diagnosis feasible:—

J. M'K., aged thirty-two years, unmarried, marble polisher, was admitted to the Whitworth Hospital on 30th June, 1903, complaining of weakness in the left leg and right arm, and of difficulty in walking. This had begun about five months previously. Going home from his work he had sometimes been so fatigued that he had to sit down in the street. Previous to this onset he had been an active man. About a month after the onset of weakness in walking he had a painful suppurating whitlow of the right index finger, followed by cellulitis of the forearm. About a month later weakness set in in the right hand, so that he found it difficult to use a knife at meals. The inability to get to work and to use his right hand gradually increasing, he sought admission to hospital. He had been very temperate with regard to alcohol for the past seven years, had never had syphilis, and had never been abroad. No history of injury. His parents died at an advanced age. Three sisters and one brother are alive and well. A sister died of fever.

Patient is of medium height, dark complexion, keen expression, and has bad teeth. There is a very slight upper dorsal scoliosis, with convexity to the right. Pressure on the 2nd and 3rd
dorsal spines, and on the 2nd right rib, in front and behind, causes keen pain. The right interscapular area at the level of 2nd and 3rd ribs looks as if slightly swollen, but this may be due to the "muscular defence" of the rhomboid muscles over curious vertebral and rib ends (?). The gait is spastic; the left leg in particular being so rigid in extension that the toes catch the ground to such an extent as frequently to threaten to trip him up. There is no ataxy as he stands. Knee-jerks are excessive; rectus and ankle clonus are present. Babinski's toe-extension sign is slightly marked. The shoulder muscles and those of the thumb and digital interossei are much wasted, especially on the right side. Fibrillary twitchings are present in these groups. The right index finger in which the whitlow occurred shows no loss of tissue and no deformity other than a little thickening, such as remains after a common paronychia. The ordinary sensory plantar reflex is keen; no sensory disturbance in trunk, legs, arms, or face. This fact, and the history of the painfulness of the whitlow, seemed sufficient evidence against the diagnosis of syringomyelia, and a diagnosis of spinal caries, probably tubercular, was arrived at. The heart, lungs, and kidneys seemed healthy.

In November he is noted as having constant and excessive sweating in right axilla. In January, 1904, he begins to have a chronic painful dry pleuritis on the right side, and about the same time it is noted that almost all the muscles of upper and lower limbs are tender to pressure. Sensation of touch and pain are unimpaired.

Jan. 19—Light stroking of soles causes extension of big toes; Babinski's sign. Heavier stroking causes flexion of all the toes. Pressure in the 7th and 8th spaces in nipple line causes strong adduction of the arms to the side of the chest. By this time he is completely confined to bed, in part by weakness and the muscular rigidity of lower limbs, in part as a result of the almost constant pleuritic pain. Respiration is almost wholly diaphragmatic.

In May an examination de novo results in affirming the diagnosis of chronic compression myelitis, due to vertebral caries. In June the legs are noted as being very rigid in extension, with the foot tending to get into line with the leg. Irritation of the soles produces strong flexion of the toes—that is to say, Babinski's sign has been
Syringomyelia, with account of an Uncommon Case.

lost. As the stimulus is continued and intensified, however, the right big toe relaxes to the resting position, as if in fatigue, and after a few seconds, if irritation be kept up, the position of strong flexion is resumed. There is no defect of sensation of touch or pain. In the upper limbs the elbow-jerks are pretty keen; those of the wrist are absent. There is no rigidity in the arms, but much wasting of muscles especially in the hands. The thenar and hypothenar muscles and the interossei are much wasted, and the hands show a slight amount of the pose en griffe. The adductor pollicis on each side seems to have survived the general wasting better than the other muscles. There are no changes in the skin or subcutaneous structures of the hands.

Such was the condition, together with frequent recurrences of the pleuritic pains in the right side, till the end of September, 1904. He then began to suffer from rather sudden attacks of dyspnoea, or rather of apnoea, lasting only a few seconds, and due probably to momentary paresis of the diaphragm. On the 15th October rales were present over all the chest, and breathing was exceedingly shallow. Next day he was unable to cough, owing to paralysis of the respiratory muscles, and on the 17th October he died.

The post-mortem findings were immaterial, except for the following points:—The right pleural cavity was annihilated by pleural cohesion. There was a gentle curve of the spinal column as already noted, but otherwise absolutely nothing wrong with the structures of the spinal column or with the ribs, and the spinal cord showed a wide and long dilatation, filled with clear fluid.

Dr. Earl's report is as follows:

The spinal cord is broad and flattened in the cervical region. A cavity exists in it which, as seen by the naked eye, reaches from the origin of the third cervical nerve to that of the seventh dorsal nerve, but can be traced a little further above and below in microscopic sections. The shape and size of this cavity, which is in all regions provided with an incomplete epithelial lining, vary a good deal in sections made at different levels. At the origin of the third cervical nerve it is a narrow transverse slit, involving the grey commissure, and extending
slightly into the anterior horn on both sides, and also into the posterior horn on one side. The epithelial lining occurs on much of the anterior surface and on a little of the posterior surface of the slit. Behind the cavity there is marked sclerosis of the inner anterior part of the posterior columns. A thin layer of fibrous neuroglia lies outside the epithelial lining, and also lines the parts which lack epithelium, but medullated nerve fibres run to the edge of the cavity, and at one of its sides a bundle of medullated fibres is seen in sections projecting into the cavity. (Figures 2 and 3.)

At the origin of the sixth cervical nerve the cavity is much larger, and appears as a narrow transverse cleft, involving the grey commissure, and extending into both anterior horns, which it involves to a considerable extent, and into the left posterior horn, which is also extensively involved, so much so that the cavity is only slightly separated from the surface. The epithelium covers a great deal of the anterior and a little of the posterior surface. The neuroglia layer is better marked than at the third cervical nerve, and a very few medullated fibres approach the lining of the cavity.

The cavity, down to the end of the cervical enlargement, shows little variation from the condition just described as occurring at the sixth cervical nerve, but its antero-posterior diameter becomes greater, and the anterior horns become more extensively involved. About the level of the fourth dorsal nerve its antero-posterior diameter has considerably increased, its lateral diameter has diminished; the anterior horns are extensively involved, so that they are represented by only a very thin layer of grey matter, containing very few nerve cells. The anterior commissure has disappeared. Below this level the cavity narrows, and at the level of the seventh dorsal nerve is represented by a transverse cleft, which is almost entirely lined by epithelium, from which a diverticulum, without any epithelial lining, can be followed into the right posterior horn for some distance. This diverticulum in some sections appears to have no connection with the central cavity. Below this no cavity can be made out with the naked eye, but a slight cleft can be followed in microscopic sections a little further. The central canal above the cavity presents several peculiarities. At about the second cervical nerve there are two small canals lined with columnar epithelium, and lying
Syringomyelia, with account of an Uncommon Case.

side by side in the position of the central canal. In sections below the level of the cavity the central canal is abnormally large. In sections through the lumbar enlargement a diverticulum runs from the canal into the posterior median fissure for a short distance. The ependyma is very plentiful round the canal, and is in the lumbar enlargement arranged in two rounded masses, occupying the extremities of the grey commissure. Both these are connected with the epithelium of the central canal by a thin column of ependyma cells, and in some sections one of these round masses contains a tube lined with columnar epithelium.

In sections through the conus the ependyma is very abundant in the position normally occupied by the central canal, and three cavities lined with epithelium occur lying close together, and in sections, appearing quite independent of each other. The crossed pyramidal tracts are more or less degenerated throughout the region in which the cavity exists, and in the cord below the level of the cavity.

It is thus evident that syringomyelia may exist without disturbance of sensation, and with little or no atrophic manifestations other than what one meets with in ordinary progressive muscular atrophy. Further, this case shows that when in spastic paraplegia, associated with sclerosis of the lateral columns, rigidity becomes excessive and extension so great that the foot is pointed as far as possible away from the head, Babinski’s sign, previously in evidence, may be lost or supplanted by a vigorous flexion movement.

Dr. Coleman said they had to thank Dr. O’Carroll for having thrown much light on this disease, he having first described it in this country. He thought that the case would not have been absolutely diagnosed as syringomyelia from the symptoms, and he believed that more of these cases would be discovered if thorough post-mortem examinations were made, as Dr. O’Carroll
was in the habit of doing. He himself had had a case of the
disease under observation for the last ten years. It began when
the patient was a boy of ten, and the muscular symptoms were
then well marked, with the characteristic dissociation of sensations.
The anaesthesia and sensory symptoms were present in the arms
now exactly as they were ten years ago.
TYPHOID AND PARATYPHOID FEVER.

BY ALFRED R. PARSONS, M.D., F.R.C.P.I.;
Physician to the Royal City of Dublin Hospital and to the Royal National Hospital for Consumption.

[Read in the Section of Medicine, January 20, 1905.]

It falls to the lot of every physician occasionally to meet cases of continued fever of which the ætiology is obscure for some time. The onset of the fever has been gradual; the patient has continued at his usual work for some days, complaining, however, of headache, loss of appetite, dizziness, constipation—which he has usually treated by taking some purgative pills—and disturbed sleep. Several days, perhaps a week, have elapsed before any medical advice has been obtained. The patient's own diagnosis is a bilious attack, or an attack of indigestion. On examining such a case the patient does not look very ill; his pulse may not exceed 90, the skin is moist, the respirations are not materially increased, and the physician is rather surprised at finding that the temperature is 103° F. The tongue is coated. Careful examination of the lungs discloses no gross organic disease; the heart sounds are pure; there is no distention of the abdomen; no tenderness on palpation, no rose spots are visible, and the spleen is not enlarged. The urine is febrile, but contains no albumen and no sugar. The following morning the temperature is a little lower, say 101.5° F., but the same evening it is again 103° F. This condition of affairs, notwithstanding a careful regulation of the patient's diet and a satisfactory evacuation of the bowels, continues for the following three or four days, and the medical attendant is pressed for a diagnosis. He thinks it is probably a case of
typhoid fever, having arrived at that diagnosis chiefly by a process of exclusion, but he cannot be confident that the symptoms are not due to a concealed tubercular focus.

It is in cases like this that the Widal test is such a boon, and enables one to say with practical certainty that the case is one of typhoid fever, provided that the patient has not previously suffered from that disease, and that a definite reaction has been obtained with highly diluted serum. Three instances of obscure continued pyrexia have recently come under my own observation. The patients lived in the same institution, and the bedrooms which they occupied were in the same part of the building, and not more than ten yards from one another. The following are the clinical records:

Case I.—Mr. A. B., aged twenty-eight years, a tutor, a powerfully built, very athletic man, felt a little chilly on 27th September, and for the following week was not in good form. His appetite was not so good as usual, and he had occasional headache, but he continued to discharge his tutorial duties till the 3rd October, when, at 1 p.m., he felt rather fatigued, and lay down for a little on his bed. In the afternoon he felt so much better that he was starting for a walk when he was met by the Head Master, who thought it advisable to take his temperature. It was 104° F. At 7 p.m., when I saw him, he was perspiring profusely; his pulse was only 90 per minute, and on careful physical examination I failed to find anything sufficient to account for his pyrexia. On subsequent inquiry I ascertained that he had never been ill previously, and that his family history was excellent. On the morning of the 4th October his temperature was 101° F., but in the evening it was again 103° F.; and on the 5th October, the temperature being 103.2° at 12 p.m., I decided to remove him to the Royal City of Dublin Hospital. On the 11th October Dr. Symes obtained a positive Widal (1 in 50 dilution). About the same time some spots appeared over the abdomen, but the spleen was never palpable. He went through a severe uncomplicated attack of typhoid fever, the temperature not becoming normal till the 50th day of his illness. Though his evening temperature was often almost 104° F., his pulse on only three occasions
throughout his entire illness rose to 100 per minute. Its average was 90. Constipation was the rule during the pyrexial stage, the bowels being moved by enemata. Cultures were made on one occasion from the patient’s urine, but only cocci were obtained.

Case II.—C. D., aged thirteen years, a school-boy; got a headache on 21st October, and felt chilly. A couple of days later he had pain below the margin of the right costal arch. This pain diminished in the course of three or four days and disappeared. The temperature, however, varied from 99° F. to 100.4° F. As physical examination was negative—except that his tongue was coated in the centre and red at the tip and edges, and his pulse, though only 90 in frequency, was intermittent—I allowed him a little custard. Post hoc or propter hoc, his temperature rose to 103° F., and continued between 102° F. and 103° F. for three days. I gave some of his blood to Dr. Earl, who had no hesitation in pronouncing Widal negative. Still in favour of a diagnosis of typhoid fever were—(1) The occurrence of the case just described; (2) the want of proportion between the pulse and temperature, 90 to 103° F.; (3) the insidious onset; (4) the appearance of the tongue; and (5) the elevation of the temperature after the use of a little baked custard. However, physical examination still disclosed no enlargement of the spleen, and an absence of spots: the bowels were constipated, but had moved after a mild aperient.

He was admitted to the Royal City of Dublin Hospital on the 3rd November—i.e., 13th day of his illness. His temperature gradually settled down, and became normal on the 12th November, exactly three weeks from the commencement of the illness. Dr. Symes and Dr. Wigham also obtained the same negative result when his blood was tested with Eberth’s Bacillus typhosus. But Dr. Wigham found a positive result with Gärtner’s Bacillus enteritidis on 5th November—i.e., the 15th day of the patient’s illness. On the 12th November the blood was still positive to Gärtner, and even several weeks after the subsidence of all fever it was still positive to a different strain of Gärtner, though the reaction was not quite so definite.

Blood counts made on the 12th and 18th by Dr. Symes gave the following results:—Hæmoglobin, 78 per cent.; red
blood corpuscles, 3,140,000; white blood corpuscles, 6,428. Differential count—neutrophiles, 66 per cent.; eosinophiles, 0 per cent.; small lymphocytes, 12 per cent.; large hyaline cells, 22 per cent. Agar plates were made from the urine on 19th November, but no bacilli were found, only cocci. On the 29th November the urine was practically sterile, as in three plates there was only a solitary colony.

We had, therefore, in this case pyrexia lasting for three weeks, a slow pulse, a coated tongue, and abdominal distention—a combination of signs suggestive of typhoid fever. However, three examinations by three different observers gave a negative Widal with the Bacillus typhosus, but on three occasions with two different strains a positive reaction was obtained with Gärtner's Bacillus enteritidis.

Case III.—E. F., aged fifteen, a school-boy, felt poorly on 19th October. He suffered from headache, chilliness, slight sore throat, not sleeping well, pain referred to the end of the sternum, and some cough. His temperature varied from 100° F. to 103° F. during the following six days, but his pulse was remarkably slow, generally 70 per minute. Physical examination was negative. Still, on account of Case No. 1, and the combination of a slow pulse with considerable fever and the absence of any physical signs, I considered typhoid fever was a probable diagnosis, and admitted the patient to the Royal City of Dublin Hospital on 26th October. Neither his previous nor family history threw any light on his illness. He had always enjoyed good health, with the exception of attacks of measles, tonsillitis and influenza, and there was no tubercular history. After his admission to hospital a further careful examination was made. His tongue was coated with thick brown fur; there was a systolic murmur in the second left intercostal space close to the sternum. The area of hepatic dulness was diminished from some distention of his colon; there were no spots, and the spleen was not palpable. His temperature was 102° F. on evening of admission, but, dropping approximately a degree each day, became normal on the 13th day of his illness. His pulse never exceeded 72 per minute. A Widal test, with Bacillus typhosus
was made by Dr. Symes on 1st November, and was found negative. Five days later Dr. Wigham found Widal negative with Eberth's bacillus, but obtained a positive result with Gärtner. Urine passed into a sterilised vessel on 23rd November—i.e., three weeks after this attack had ceased—was inoculated on agar plates, and gave a very abundant growth of bacilli, closely resembling in their appearance the typhoid group. Dr. Wigham made sub-cultures from these, and proved they were not Gärtner, but Bacillus coli, by their power to ferment lactose. On 26th November some of the faeces were spread on Comrasi and Drigalski plates by Dr. Wigham. Practically all the colonies turned the medium red, and those examined were Bacillus coli. There were a few very minute colonies which remained blue. On 28th November agar plates were inoculated with the urine, which on this occasion was practically sterile.

A blood count made on the 22nd November by Dr. Symes gave the following result:—Hæmoglobin, 75 per cent.; red blood corpuscles, 4,818,750 per c.mm. Differential count of leucocytes—neutrophiles, 41 per cent.; eosinophiles, 0 per cent.; lymphocytes, 42; hyaline cells, 17.

The duration of this case was shorter than the preceding one, as the total duration of the pyrexia was only thirteen days. The temperature, accompanied by a slow pulse and an absence of any definite physical signs, was, in the earlier part of the illness, suggestive of typhoid fever. The blood was negative to Widal, according to two independent observers working with different strains of Bacillus typhosus. The blood, however, gave a positive reaction on two occasions with two different strains of Gärtner's Bacillus enteritidis.

Through the kindness of Sir J. W. Moore I shall briefly refer to a case which he had under his care at the Meath Hospital:

Case IV.—Mr. E. H., a resident in the Meath Hospital, felt rather poorly on 24th October, 1904. He was tired, and had lost of appetite, pain in the back, and headache. The bowels moved regularly each day. He continued at his work as resident pupil for a week with these symptoms, but every second day, when off
duty, lay down when he could find a friend to relieve him. By the end of a week he was, however, so poorly that he had to give up work and take to his bed. His temperature was 102.4° F., but it settled down in the course of a few days to normal, and he was able to leave the hospital at the end of the week—i.e., 14 days after the commencement of his illness.

The Widal test had been applied while he was in the hospital. It was negative. Having heard that Sir John Moore had diagnosed the case as one of paratyphoid fever, I thought it would be interesting to try whether his blood would react with the same organisms as in Cases II. and III. I obtained some blood four weeks after the temperature had become normal, and Dr. Wigham informed me that it produced some clumping.

In the American Journal of Medical Science for August, 1902, Dr. Johnston, one of the assistants at Johns Hopkins Hospital, reports four cases of paratyphoid which occurred in the hospital during the year 1901, and republishes Dr. Gwyn’s case, which occurred in the hospital in 1897, and in which Dr. Gwyn obtained paratyphoid bacilli from the patient’s blood.

In these five cases the Widal test was negative. In some of them there were rose spots, and the spleen was palpable. Three of the cases were diagnosed by isolating the organisms from the blood, and two by the agglutinating action of the patient’s sera on Gwyn’s bacillus, and on the above germs isolated by Dr. Johnston.

In the same journal A. W. Hewlett reports a case of paratyphoid fever from the New York Hospital, and Longcope reports a fatal case from the Pennsylvania Hospital. The post-mortem appearances were indicative of an acute general infection, and there was no swelling of Peyer’s patches.

The number of reported cases has been steadily increasing since 1902, and R. T. Hewlett states that a hundred or so cases have now been recorded.
Typhoid and Paratyphoid Fever.

At a meeting of the Pathological Section of the Royal Academy of Medicine in Ireland held on 2nd May, 1902, Dr. White discussed the bacteriology of the Gärtner group in describing an investigation which he made into a fatal case of continued pyrexia simulating typhoid, reported by Dr. Craig.

In conclusion, it seems certain:

1. That cases clinically indistinguishable from typhoid fever do not react with Widal.
2. That it is desirable in such cases to try blood reaction with one or more members of the Gärtner group.
3. That such cases run a milder course than typhoid.
4. That they are not so liable to a fatal termination.
5. That there is no swelling or ulceration of Peyer's patches.
6. That in such cases it is desirable to isolate the specific organism during life from the blood, urine or faeces.
7. That such organisms should be kept for purposes of comparison, and for testing the sera of suspected cases.

REFERENCES.

3 Longcope, W. T. Ibid. P. 207.

Dr. Craig described minutely the fatal case of paratyphoid which he had seen. He admitted the man from his dispensary, who was then looking very like typhoid. His temperature was 103.2°, pulse 100, and respirations 24. He had three liquid yellow motions, and developed a rash, which at first consisted of rose spots, but later became dark, like typhus. The tongue was thickly coated, cerebration slow, and he refused food. The abdomen was distended. He became delirious, and 17 days after the rigor which had ushered in the illness he died.
pulse averaged 95 to 100, and respirations 24. The spleen was
easily noted to be enlarged. Post mortem there was no evidence
of enlargement or ulceration of Peyer’s patches; the greater part
of the ilium was in a state of acute inflammation. Dr. White
examined the blood on the 4th day after admission. He tried a
Widal, and got clumping with a dilution of 1 in 25. There was
no evidence of Eberth’s bacillus, but one of the forms of Bacillus
enteritidis had caused the illness. In a number of cases hæmorr-
rhage from the bowel had been observed.

Dr. Travers Smith said he had seen two cases which he thought
were paratyphoid. One was a woman, aged thirty-two. On
admission her temperature was 103°, pulse not markedly quick,
no diarrhcea, enlarged spleen, or rose spots; but there was great
abdominal distention, and she was rather livid. The blood was
negative to Widal. She died in about a week, and post mortem
there was not a trace of disease in Peyer’s patches, nor was the
spleen enlarged. In the other case the tongue was coated. spleen
a little enlarged, pulse rather quick, and the temperature up.
The blood was negative to Widal. The fever gradually subsided:
but for ten days the spleen remained large, then it subsided, and
she got perfectly well. He suspected paratyphoid, but had no
proof of it, as the special blood reaction had not been tested for.

Dr. Pugin Meldon thought that in paratyphoid one should be
just as careful with regard to diet as in typhoid, as in some cases
there had been hæmor rhages, showing disease of the intestine. The
presence of a large ulcer had been reported in one case.

Dr. Fannin said he had had a case of a boy, aged seventeen,
who had a continued fever, the characters and course of which
were the same as typhoid. There were no rose spots, nor was there
enlarged spleen. He had acute laryngitis. At that time Widal
was negative. Ten days after the temperature had become
normal a relapse occurred. It followed a typhoid course, the
laryngeal symptoms were renewed, rose spots appeared, and the
spleen became palpable. He became very ill, and was removed
to hospital. Two or three days afterwards symptoms of per-
formation occurred. An operation was performed, and a per-
fornated ulcer discovered, but the patient did not recover. Dr.
W. J. Thompson was satisfied that the ulcer was like an ordinary
typhoid one. In that case, then, repeated examinations of the
blood had failed to give the Widal reaction. The course of the
illness resembled typhoid, and a fatal result occurred after perforation, therefore it was possible that some forms of paratyphoid could have the definite ulceration of Peyer's patches.

Dr. Kirkpatrick said he would like to ask whether any differentiation had been made in the clinical history of the fevers which were due to the different groups of paratyphoid bacilli?

Dr. McWeeney discussed the cases from a pathological point of view. He said that one would be inclined to call Dr. Parsons' 2nd and 3rd cases, cases of Gärtner infection. It had been mentioned that the blood serum failed to agglutinate typhoid, but did agglutinate Gärtner's bacillus. That carried an indication of Gärtner infection, which was not quite the same thing as paratyphoid. The Gärtner bacillus was not quite the same type as the alpha and beta groups of paratyphoid, but presented certain differences. It was quite possible that the 2nd and 3rd cases were paratyphoid, but the bacilli would have had to be isolated and their characters fully tested. What he found lacking in the pathological description was a statement of the different dilutions which were employed.

Dr. Parsons, during his reply, said that intestinal lesions had been mostly found to be in abeyance during paratyphoid, and thought it was not so necessary to restrict the diet as in typhoid. There was no difference in the clinical course of the fevers produced by the alpha and beta groups. In his communication he had used the term paratyphoid in its widest sense, and had included Gärtner's bacillus in the term, although he was aware that there was a tendency to restrict the term to a small group of bacilli.
LARGE WHITE KIDNEY.

By GEORGE PEACOCKE, M.D., F.R.C.P.I.;
Assistant Physician to the Adelaide Hospital.

[Read in the Section of Medicine, January 20, 1905.]

On the 25th of May, 1903, a young man, aged thirty, was admitted to the Adelaide Hospital under my care. He stated that he had been in his usual health up to the previous Christmas, when he caught a severe cold and suffered from pain in his head and back. He continued at his work, that of a carpet layer, until February, when, shortness of breath and swelling of his feet coming on, he sought advice at the Meath Hospital, and was admitted under the care of Sir J. W. Moore. His case was regarded as one of acute nephritis. He improved under treatment, and left the hospital on the 27th of March feeling quite well. Some time about the middle of May he noticed his face, hands and feet were again becoming swollen, and also that his abdomen was increasing in size. From the 25th of this month until his death, on the 25th of September, I had him under my care in hospital. His symptoms during this period were briefly as follow:—He was very anaemic, and universally dropsical. He suffered much from dyspnoea, but there was no discoverable pulmonary or cardiac disease. Diarrhoea, amounting on some days to as many as eight motions, was a constant symptom all through his illness. His urine was scanty, sp. gr. 1020 or less, loaded with albumen, occasionally contained blood, and the abundant deposit consisted largely of hyaline, granular, epithelial and fatty casts—a typical case clinically of chronic tubal nephritis. At the post-mortem the kidneys presented the appearance usually described as large white kidneys. Professor Scott made sections of them, and he handed me the following report:—“The connective tissue capsules of the Malpighian corpuscles are thickened in many cases, and some of the corpuscles are turned into spheres of connective tissue. There are many inflammatory granulomata in the intertubular tissue. The tubes are dilated, and contain albuminous casts.”
Large White Kidney.

The second case was that of a young man, aged twenty-four years, a painter by trade. He was admitted to the Adelaide Hospital under my care on the 23rd of March, 1904. About two month s before his admission he had noticed his feet and ankles were swollen; previous to this he had enjoyed excellent health. On admission he was found to be very anemic, and universally dropsical. Like the previous case, he suffered from diarrhoea. He had considerable dyspnoea and cough. His chief complaint was of pain in the right side of his chest, which was evidently pleuritic in origin. His urine was diminished in quantity, sp. gr. 1015, and contained a large amount of albumen, with numerous hyaline, granular, epithelial and fatty casts. He remained in hospital until his death on the 24th of May. His kidneys on removal had all the macroscopic appearances of the large white kidney, and Professor Scott has kindly furnished me with the following report on the microscopical appearances:—"Although the interstitial connective tissue is decidedly increased, there is less than in the other case, and the inflammatory granuloma is not well marked. About one-third of each Malpighian corpuscle shows amyloid degeneration. Some of the tubes are dilated, but others are packed with epithelial cells in a condition somewhat like cloudy swelling; in other tubes there is slight fatty degeneration."

The resemblance in the clinical symptoms, and indeed in the macroscopic appearance of the kidneys, in these two cases was very striking, and had investigation not proceeded further the two cases would have been considered similar in every respect, except that in the first case there was a definite and carefully observed attack of acute nephritis, subsequently followed by symptoms pointing to more chronic mischief; while in the second case there was nothing in the history to lead one to suspect that the disease in the kidneys was ever acute, but rather that it was chronic from the beginning. Microscopic investigation, however, revealed the fact that in the first case there was merely evidence of a chronic inflammation of the kidneys, but in the second case there was, in addition to some increase in the interstitial connective tissue,
commencing amyloid degeneration in the Malpighian corpuscles.

I am fully aware that these two cases do not present any features that can be regarded as in any way out of the common, but they exemplify two of the recognised forms of chronic nephritis—a subject which I wish to bring very briefly before this Section of the Academy.

There is, I think, considerable confusion existing as to the exact condition which is described under the term chronic parenchymatous nephritis. If we look at the aetiology of this subject as given by different writers we will be struck by the varied opinions that are held as to the causation of this form of chronic nephritis. While the majority hold the view that the condition results in most cases from a previous attack of acute nephritis, but is sometimes chronic from the beginning, Rose Bradford maintains that "in a small proportion of cases the symptoms of acute Bright's disease merge into those of the chronic disorder; but it is probable that the great majority of cases of chronic nephritis are really chronic from the beginning, and not the direct sequel of the acute."

I am inclined to hold the view that the disease known as chronic parenchymatous nephritis—the large white kidney of nephritis—is always the result of a previous acute attack; and that these cases described as chronic from the beginning are really examples of some other affection—most commonly amyloid degeneration.

Tuberculosis, chronic suppuration, and syphilis are mentioned among the recognised causes of chronic parenchymatous nephritis; but these are the very causes that are prone to produce amyloid disease, and that when, as is often the case, some interstitial changes are found in the kidneys, these changes are not primary, but are secondary to the amyloid degeneration.

It is a well-recognised fact that in many instances th
symptoms of chronic nephritis do not supervene immediately after the acute symptoms have subsided, but that often a very considerable interval intervenes, when the patient is apparently in good health, before any symptoms make their appearance. This fact may, I think, be one reason why some cases are regarded as chronic from the beginning, the acute attack that occurred some time previously having been overlooked. If this view of the aetiology of chronic nephritis be correct the classification of diseases of the kidney associated with albuminuria is somewhat simplified, and the classification I would favour would be briefly as follows:—

1. Nephritis, acute and chronic—the chronic representing the chronic parenchymatous nephritis, or better described, as Dickenson suggests, “the large white kidney of nephritis.” The small white kidney is generally considered to be a later state of this form, and I suppose that in some individuals the renal epithelium is not so rapidly and completely destroyed, and that sufficient is left to carry on life until the increasing fibrosis causes shrinking of the kidney substance; but I should imagine that the condition is very rare.

2. Granular kidney, essentially chronic from the beginning, and resulting most frequently from the toxic action of alcohol, gout or lead.

3. The kidney of amyloid disease.

Dr. McWeeney said, with regard to the microscopic specimens, the impression he got from those of the second case was that it was far from certain whether it was really amyloid degeneration or hyaline. The section showed hyaline change of portion of the glomeruli, but there was no differential staining to show whether there was also amyloid change. The question might have been answered by seeing whether the middle coat of some of the smaller arteries was affected in the same way, but he had failed to see this change. Therefore, he was inclined to
By Dr. George Peacocke. 29

think it was hyaline degeneration. The other section also showed hyaline changes in the glomeruli, but not to the same extent. A pathologist would diagnose the second case as early chronic interstitial nephritis.

Dr. Travers Smith said that his experience went to show that chronic parenchymatous nephritis was insidious in onset. The history of the onset was often misleading. One saw a good many cases of acute nephritis occurring during scarlatina: he had never seen these cases coming back suffering from chronic parenchymatous nephritis, and he did not think it could be common. One of Dr. Peacocke’s cases exemplified the difficulty of diagnosing a mild acute case from chronic parenchymatous nephritis, and this was a serious question from a prognostic point of view. In both you might have extended dropsy, the quantity of urine diminished, hyaline and epithelial casts, blood—all this made it very difficult to come to a diagnosis, particularly in those cases where there was no antecedent cause, such as scarlatina.

Dr. Kirkpatrick said that in examining microscopic sections of kidney disease it was very important to examine all the fields carefully, and to make large sections. When large portions were examined, although they differed in one part from another, yet many of the kidneys had various points in common. A classification thus became possible; but he thought that any classification based on histology alone was unsatisfactory. All of these diseases were more or less of the nature of inflammations, and the effect of the inflammation was evident in one in the parenchyma, in another in the interstitial substance. There were many cases where the differences were so pronounced that one was justified in putting them into different classes; but, when one examined further, the changes were completely progressive, and from the most typical acute nephritis to the most well-marked chronic case there was no distinct breach of continuity on microscopic examination.

Professor White said that he had had an opportunity of applying the special tests for amyloid substance to the sections, and found that a small portion was really amyloid, though the major portion was hyaline.

Dr. Drury said he did not know that he could agree with the statement that many cases of chronic parenchymatous
nephritis resulted from acute. He had been for a long time trying to find bona fide acute cases which turned into chronic, but had never found them. He thought that many of the cases of chronic nephritis, secondary to acute, were really chronic cases in which acute exacerbations had come on during already existing disease. One had to know the history beforehand. He thought that Dr. Peacocke was inclined to lay too much stress on amyloid change as being the primary cause of many chronic tubular cases.

Sir J. W. Moore quite agreed that chronic parenchymatous nephritis seldom followed on scarlatinal nephritis, which was usually succeeded by complete recovery. With regard to causes, he did not hesitate to assign alcohol as of prime importance, though various other causes co-operated—as cold and damp. He also thought the mode of death was important in these cases. Toxaemia was comparatively infrequent in very dropsical cases, whereas in the other cases it was very likely to cause death. The heart was really the crucial point in the management of nephritis, for as long as it could compensate for the renal insufficiency there was some chance of the patient getting on.

Dr. Peacocke, in his reply, said that the cause of death in both cases was nothing very definite. Asthenia was the best term to use for it. In these acute cases which developed into chronic there was often an interval of some months before the latter symptoms came on. He believed that amyloid change did occur even when there was not much suppuration. There was no doubt that very often in those cases which were described as chronic parenchymatous nephritis and large white kidney there was marked amyloid change.
On December 16th, 1898, I brought under the notice of the Medical Section of the Royal Academy of Medicine in Ireland a case of innominate aneurysm which had become entirely quiescent by prolonged rest, sparing diet, and the administration of iodide of potassium internally.

The patient, who was then sixty-five years of age, presented the physical signs of an innominate aneurysm in June of 1897. He was confined to bed for something over two months when the symptoms and physical signs having largely subsided he insisted on going to the South of France. There he continued to carry out the principles of treatment already enumerated, and at the close of the year 1897 he was entirely free from all symptoms. I examined him carefully 18 months after the onset, and unless for a slight prominence and diminished resonance where the tumour had presented, there were absolutely no other physical signs of an aneurysm to be found. In subsequent years he spent the greater part of his time at Continental health resorts, but I saw him during his periodic autumnal visits to Dublin. He grew strong, led an active outdoor life, and never complained of illness. In the spring of 1904 he was at Wiesbaden. On Sunday, April 24th, he was at a dinner party, and enjoyed himself thoroughly; on the following Wednesday he remained indoors, as he felt out of sorts; that night he suddenly sat up in bed, blood began to pour from his mouth, and in a few moments he was dead.

Dr. Altdorfer kindly wrote to me as follows:—"At the post-mortem examination it was found that the aneurysm of the innominate, which you diagnosed, had become consoli-
dated and did not give any trouble, but that there existed a
second—much larger—aneurysm of the thoracic aorta, which
had perforated into the oesophagus close to the stomach, so
situated that it probably could not be diagnosed at all. It
is most astonishing that, according to all accounts, the patient
should have felt so well up till a few days before his death."

Drs. Stein and Altdorfer made the following report:—

"Protocol of the post-mortem examination."

"On lifting the sternum, one notes on the inner side of the
manubrium sterni a greyish red tumour of the size of an apple
attached to the inside surface of the bone, which has destroyed
the latter to more than half of its depth.

"On loosening the sternum completely, the surface of the
above-mentioned tumour bursts, and one notes that there is a
cavity in the interior filling up partially with coagula of blood.

"Both cavities of the pleura contain a few drachms of a clear
amber yellow fluid.

"The apex of the left lung is slightly fixed by adhesions.

"The stomach is extremely distented and dark-coloured.

"The organs of the chest cavity are taken out altogether.

"The cavity of the pericardium contains about two wine-
glassfuls of a clear amber yellow fluid.

"The surface of the heart is covered with plenty of fat. The
heart muscle is in a state of fatty degeneration. The aortic
valve is incompetent and rigid. The walls of the aorta are
covered with many hard plates of different sizes.

"The cavity of the above-mentioned tumour under the upper
day of the sternum represents a dilatation of the aneurysm. The
wall of this dilatation measures about one inch in thickness.

"There is a second dilatation on the arcus of the aorta
expanding into the mediastinum posterius. The cavity of this
dilatation is of the size of the head of a new born child; the
walls are very thin. A part of the wall is fixed by adhesions to
the anterior surface of the spine, and bursts while loosening it.

"The anterior surface of the oesophagus is closely attached
to the wall of the dilated aorta. The interior surface of the
cavity is covered with plenty of the above-mentioned hard
plates. On the back of the cavity is a small spot where the
By Dr. James Craig.

wall is completely destroyed; there is an open passage to the interior of the oesophagus.

"The stomach and the upper parts of the small intestine are filled with dark-coloured blood.

"Liver, spleen, kidneys, are without particular changes.

"The lungs partially contain air; the other parts and the bronchi are filled with blood and coagula.

"Diagnosis:—Aneurysm of the anonyma; aneurysm of the arcus aorta. Spontaneous perforation into the oesophagus; myocarditis; arteriosclerosis. Death by bleeding and suffocation.

"Wiesbaden, the 28th of April, 1904.

"Dr. Med. Albert E. Stein.

"M. Altdorfer, Med. Dr."

I am sure that my hearers will agree with me in considering the case to be one of unusual interest.

The consolidation of the innominate aneurysm was in itself an interesting fact, but the sudden death of the patient from the rupture of another and much larger aneurysm, seven years after the onset of the one which became quiescent, is worthy of note.

There is no evidence at what period the fatal aneurysm began to develop, and while it may be possible that it existed at a date prior to the appearance of the one on the innominate artery, it is much more likely that its onset was of a subsequent date. Owing to the absence of symptoms until the day of death it might be assumed that the formation of the aortic aneurysm had been a very recent occurrence, but its adhesion to the spine and its adhesion to and erosion of the oesophagus must have been the result of prolonged pressure. That such pressure should exist without the production of pain is difficult of conception, and can be explained only by the yielding nature of the structures in front of this portion of the aorta.

Dr. Matson said the perforation into the oesophagus was interesting, and out of sixty-eight cases reported by one observer
only one had terminated in that way. He had had a case in which perforation had occurred into the oesophagus, but in that case there were all the usual symptoms, and there was something to go on in making a diagnosis. What was the cause of the aneurysm in Dr. Craig's case, as his patient seemed to have been a healthy liver and to have none of the usual specific causes?

Dr. T. G. Moorhead said the case was very interesting, particularly from the fact that it pointed out that aneurysm, except in cases where there was trauma or some obvious cause, was a constitutional and not a local disease. The case also illustrated the fact that aneurysm had a tendency to recur, and what he might call the constitutional diathesis to aneurysmal dilatation.

Dr. W. J. Thompson said that about six years ago he showed a specimen before the Pathological Section of a case of a labouring man in middle life. He was very steady, and had never complained of pain or any trouble. While attending to his business he suddenly fell down and blood came from his mouth. On examination life was found to be extinct. The post-mortem showed the presence of an aneurysm, the size of a walnut, situated in the aorta about two inches above the cardiac end of the stomach. It had perforated into the oesophagus. There was no atheroma or disease of the heart.

Dr. Craig said that, with regard to the cause of the aneurysm, the patient was gouty, and attributed the start of the aneurysm to a strain received in lifting a press. The chief point of interest was that this enormous aneurysm could be lying in the thorax, and the patient be apparently perfectly well.
PNEUMONIA IN PREGNANCY.

By H. C. DRURY, M.D., F.R.C.P.I.;
Physician to Sir Patrick Dun's Hospital, Dublin.

[Read in the Section of Medicine, March 3, 1905.]

Pneumonia in pregnancy is stated to be rare. Acute lobar pneumonia "is one of the most widely extended as well as one of the most fatal diseases" (West). By far the largest proportion of cases occur within the ages which coincide with the child-bearing period of life. But although men are attacked more frequently than women in the proportion of two or three to one, it is sufficiently common in women to make it surprising to find that pneumonia as a complication of pregnancy is rare. Though I have met with a very large number of cases of pneumonia, I may consider myself fortunate in having seen three cases occurring in pregnant women. It is difficult to find records of such cases, and references by various writers on this subject are very vague and general. Grisolle collected fifteen cases (Archiv. gen. de Med., vol. xiii., p. 291), which is the largest number I have been able to find.

In such a dangerous disease as pneumonia, which is said to account for 12.7 per cent. of all the deaths from purely medical diseases (West), one would expect that when it attacked a pregnant woman the case was well-nigh hopeless. This is, indeed, the view mostly held by writers, though they, as a rule, give but vague and indefinite reasons for their faith. Moore ("Ency. Med.," vol. ix., p. 446) considers that one reason for the greater mortality of pneumonia among women than in men is that pregnancy adds immensely to its danger. The
statistics of St. Bartholomew's Hospital, however, throw
doubt on this supposed higher relative mortality in
women. West's statistics show a percentage of 23.5 for
men and 22.0 for women, and the statistics of St.
Thomas’s Hospital show 21.4 for men, and 16.3 for
women. Grisolle's fifteen cases would tend to support
the idea that pneumonia is very fatal in pregnancy, for
eleven of those fifteen cases died; but West, who seems
to be little influenced by general statements, preferring
to depend on his own observation and accurate statistics,
says: “Pneumonia but rarely arises in the course of
pregnancy, but when it does it usually causes abortion,
and the child is lost. The mother, however, frequently
escapes.” (P. 298.)

Pneumonia is just the class of febrile disease in which
we would expect premature expulsion of the fœtus, for
here we have, as a rule, a high range of temperature
which is rapidly attained, and may as rapidly fall;
severe constitutional disturbance and great respiratory
embarrassment, with probable cyanosis. Any one of
these conditions we know is liable to bring about the
death or premature expulsion of the fœtus, but when
combined they are almost certain to do so.

As in other febrile states, labour is usually rapid and
easy; in fact, it is recognised by midwifery authorities
that in these conditions "precipitate labour often occurs."
It is necessary for the patient that there should not be
undue delay, and if there is marked dyspnœa, cyanosis,
or symptoms of a failing heart, steps must be taken to
aid and hasten the labour. The relief of abdominal
pressure and the haemorrhage consequent on labour
appear to be beneficial in easing the respiration and re-
lieving the engorgement of the right side of the heart, so
that when labour has been safely accomplished, the
patient is considerably relieved, and sleep—the thing most frequently wanting, the most difficult to obtain, and the most beneficial when it occurs—is obtained. The puerperal state should be managed precisely as in any normal case, and there is no increased risk of sepsis. The pneumonic condition must be treated as though pregnancy was not present. Stimulants will probably be required, but should be withheld if possible till the time of labour. At this time the hypodermic injection of strychnin, and the administration of digitalis or digitalin, may also be required if there are signs of heart failure, and oxygen may possibly be of some slight value when there is marked cyanosis. If there is engorgement of the right side of the heart, venesection to 20 or 30 ounces gives some relief.

The cases I have met with are briefly as follow:

Case I.—J. J., aged twenty-two, primipara, in seventh month of pregnancy, came under observation on the sixth day of disease, and was found to have pneumonia of the whole upper lobe of the right lung. She was an anemic girl, and appeared desperately ill, with orthopnea and an expression of intense anxiety and suffering, but no cyanosis; the pulse 128, temperature 103.4° F. and respiration 28 in the morning, and 40 in the evening. During the night of the seventh day there was an attempt at—or pseudo—crisis, the temperature falling 3.4°, but she was found to have developed pneumonia of the left upper lobe. The next evening the temperature had reached 103.6°, and premature labour came on during the night; it was easy, rapid, and normal; the child was dead, but apparently quite recently so. The morning after labour—eighth day—the temperature fell to 98.4°, but soon rose again, and came finally down to normal on the fourteenth day. Convalescence was now rapid and uninterrupted. After delivery, though still suffering from active pneumonia of the left apex and unresolved pneumonia of the right apex, she expressed herself as "feeling grand." Her pulse never rose above 102, her breathing was easy and not distressed, and she slept most of the day and night.
Case II.—L. O'C., 3-para, in seventh month, aged thirty-two; labour in both previous confinements said to be very short and easy. She was admitted to the hospital on the fourth day of illness. There was then well-marked pneumonia of right base, and fine crepitation at left base; temperature 103°, pulse 120, respiration 24. She was very sick, but did not appear in a dangerous condition. By the sixth day there was well-marked pneumonia of left base, and that night there was a pseudo-crisis, the temperature falling to 98.2°, but other symptoms not abating. On the night of the seventh day she was confined. The labour was easy and very rapid; there was practically no pain. The child was alive, obviously premature, but only lived eleven hours. Patient appeared very easy when seen next morning, and she slept plentifully. Genuine crisis occurred on the night of the eighth day—i.e., seven days after the pneumonia attacked the left base. She made a rapid and uninterrupted recovery, and was up for an hour on the fifteenth day.

Case III.—E. N., aged twenty-eight, 6-para, eight months pregnant. Admitted on fifth day of illness, and found to be suffering from pneumonia of lower lobe of right lung. Condition good; temperature moderate, pulse quiet and of good quality; respirations hurried, but not distressing. These conditions were maintained, and she slept fairly well. Movements of the child were felt on the seventh day. Early on the morning of the eighth day labour came on; it was easy, brief, and quite normal. The child was premature, and just alive when born, but died immediately after. When seen four hours after, the patient was comfortable and easy. Next morning she was noticed to speak rather strangely about her home, and warning instructions were given to watch her closely. Very soon she became wildly delirious, and remained so for six days. Whether this was ordinary febrile or post-febrile delirium, or mania of the puerperal type, it is difficult to say. The temperature, which had rapidly fallen to normal on the ninth day, ran up on the three successive evenings, falling each morning to normal or sub-normal, but from the thirteenth day it remained normal. This latter febrile condition was evidently in some way due to the disturbance of the delirium, as the pneumonic lung began at once and continued to resolve after the ninth day, and the uterine
conditions were quite satisfactory. After the week of delirium she seemed quite well; after another week she was allowed up, and left perfectly well.

These three cases do not give one sufficient grounds for drawing important conclusions: the fallacy of doing so is seen in the fact that all three were in the seventh or eighth month of pregnancy; yet no one would surely suppose that this was the likely period for a woman to be attacked. We have seen inferences drawn, however, from just as absurd bases. Yet there are one or two points illustrated which tend to bear out what I said in introduction. First, it is not necessarily such a deadly complication as some writers would have us believe. Two were cases of double pneumonia—a sufficiently grave condition in itself—but one of these was, in addition, double apical pneumonia. Yet all three, though profoundly ill, never appeared in immediate danger, and their recovery bears out West's remark that "the mother frequently escapes."

In all, premature delivery occurred, which appears to be the usual course of events. Delivery having been safely accomplished, the patient's condition appeared to be materially improved, all the symptoms, temperature, pulse, and respiration being lower. Delivery was rapid and easy in each case, which appears, for some reason, to be the rule in labour during febrile conditions, perhaps due to the softening and relaxation of the genital passages as a result of the high temperature. There was no additional trouble in the management of the puerperal state, the lochia were normal, and had no special tendency to become septic: the breasts gave no trouble, as the secretion was to a large extent checked by the fever.

The three children succumbed, but, though premature, they were alive up to the time of birth, and if they had
been nearer maturity might have survived, one having lived eleven hours.

Dr. Ninian Falkiner said he considered the subject a most important one, and congratulated Dr. Drury on his successful treatment. He read the report of a case of his own, which appeared in the British Medical Journal of March 2nd, 1899. He also gave some statistics of the mortality in those cases. In England in 1901 the cases of death from pneumonia in pregnancy or childbearing showed a percentage of 3.42 of all the deaths of women associated with these conditions. In 1902 the percentage was 6.46, and in Ireland in 1902 it was 1.52 per cent. of all deaths definitely returned as either caused by or associated with pregnancy or childbearing.

Dr. Day said that, with reference to the question of pneumonia in pregnancy, his experience was that women who were young did not die. He had never had to use forceps in a case with fever. The child's chances of survival were very slight. Cases of abortion gave more trouble or cases in which the foetus was not viable. He doubted whether a woman who was pregnant was less liable to get pneumonia than a non-pregnant woman. There was no reason why such patients should get cyanosis as long as one lung was free. What one had to treat was the fever and want of sleep—in other words, the nervous disturbance. There was also a certain amount of hypertrophy of the heart, and it was the heart which usually failed. In these cases no internal examination should be done, and the case should not be interfered with. One thing which made pneumonia peculiarly liable to cause abortion was the high temperature. He thought it was important to have got these three cases together as emphasising the point that pneumonia was not such a dangerous complication of pregnancy as was usually supposed.

Dr. Neill said that delivery in these cases was usually very easy owing to the prematurity of the foetus. He had seen also several cases in the last stages of phthisis in which the delivery was perfectly easy. He had seen a case of placenta praevia occurring in a patient who had advanced phthisis. She got over it well, and had a normal child afterwards.

Dr. Moorhead said he had had one case of pneumonia in pregnancy. The patient was a primipara, aged thirty-seven,
and about four and a half or five months pregnant. The whole of the right lung was involved. Convalescence was prolonged, and there was cyanosis from an early period and all through the fever. A perfectly healthy child was born at full term.

Dr. Drury thought that gynaecologists should pay more attention to the statistics of these cases. The tendency to abortion was not so great when the fever occurred at an early term, but in most cases, especially in the later periods, the foetus was usually not retained.
SOME MISCELLANEOUS CLINICAL EXPERIENCES.

By R. TRAVERS SMITH, M.D., F.R.C.P.I.;
Visiting Physician to the Richmond, Whitworth, and Hardwicke Hospitals.

[Read in the Section of Medicine, March 31, 1905.]

ALCOHOLIC CIRRHOSIS OF THE LIVER.

Case I.—T. J., a widow, aged thirty-eight, of intemperate habits, was admitted to the Whitworth Hospital in October, 1903, where she remained under my care till her death in November, 1904. She complained on admission of swelling of her feet and abdomen, and of pain in her right hypochondrium. She had previously been subject to bronchitis. She had had four children, one still-born, the other three died in early infancy.

Patient was well nourished, but rather pale. Conjunctivae of a yellowish brown hue. The abdomen was distinctly enlarged, the lateral superficial veins evident; it was very easily palpated, as the muscles were weak and the recti were far apart. The liver was found to be uniformly enlarged, extending down to the umbilicus, abnormally tough in consistence, the anterior edge feeling very sharp. The spleen felt double the normal size at least, its rounded border contrasting markedly with the sharp edge of the liver, with which it was in contact. The intestines were flatulent. There was no ascites present. The urine was normal, and contained no bile pigments. A blood examination showed moderate anemia, but no other abnormality. The lungs were slightly emphysematous. The apex of the heart was displaced a little outwards. Over all the precordial region a systolic murmur could be heard, its point of maximum intensity obviously being the second left intercostal space, half an inch from the edge of the sternum. This murmur was not affected by respiration; it became slightly more distinct on exertion; it could be traced only a short distance from the apex into the axilla; and it retained these characters till death. The feet were slightly oedematous.

The diagnoses entertained were:

(a) Passive congestion of liver and spleen, the result of failing right ventricle. This was excluded by the absence of any
considerable enlargement of the heart, a normal pulse, and absence of other signs of passive venous congestion.

(b) Spleno-medullary leukaemia or Hodgkin's disease, excluded by the blood examination and the absence of enlarged glands.

c) Biliary cirrhosis, excluded by the absence of jaundice or of its history.

d) Malignant disease, excluded by the regularity of the enlarged liver and spleen, and the want of any evidence of a primary form. The diagnosis finally arrived at was "hypertrophic stage of alcoholic cirrhosis."

During the patient's stay in hospital, on three or four occasions subcutaneous and sub-conjunctival haemorrhages occurred, apparently spontaneously. Thrombosis supervened in the left femoral vein. Attacks of vomiting were occasionally troublesome. The liver became, as time went on, slightly smaller in size and less smooth on the surface. Severe pain in the right shoulders was a frequent complaint. Three weeks before death I had carefully examined the patient's abdomen one morning, and had felt the large, firm spleen as usual. Within an hour afterwards violent hematemesis occurred. On then examining the abdomen the spleen could hardly be palpated, so much reduced in size and softened had it become. The liver, I am inclined to think, was also reduced in size, but this point was doubtful at the time. During the next few days the spleen gradually regained its usual large size. This diminution in the size of the spleen after hematemesis can readily be explained on the supposition that the bleeding occurred from some of the veins along the greater curvature of the stomach which are tributaries of the splenic vein. Hematemesis recurred several times, and was the immediate cause of death. Only in the last two weeks of life as a terminal condition did ascites develop.

Post-mortem, made six hours after death:—All parts very anaemic. Abdomen contained a lot of clear fluid. Liver weighed 1,030 grammes, typically hob-nailed; adherent to diaphragm. Spleen enlarged, 390 grammes; congested, but rather soft. A single red infarct in its anterior border. A small accessory spleen. The edge of the great omentum was adherent to brim and contents of pelvis, the possible cause of thrombosis of left femoral vein. The stomach contained blood, but no point in stomach or oesophagus could be located as its source, though death resulted
from hæmorrhage. The kidneys showed signs of early chronic interstitial nephritis.

The surface of the heart showed numerous "milk spots." The musculature was in a state of fatty degeneration, the musculi papillares of the left ventricle showing fatty striation. The left ventricle was somewhat hypertrophied and dilated. The cusps of the mitral valve were thickened and shrunken at their free edges, and their chordæ tendineæ were shortened; mitral orifice was dilated, admitting three fingers with ease. The right side of the heart was normal in size, as were its valves. The aortic valves were normal; lungs, emphysematous and oedematous.

The points of interest in the case to me were:

The spontaneous hæmorrhages, as in hypertrophic cirrhosis.

The reduction in the size of the spleen after hæmatemesis, proving the congestive origin of its enlargement.

The very late appearance of ascites.

The opportunity of verifying post mortem that mitral regurgitation can cause a murmur, with its point of maximum intensity at the tip of the left auricle—i.e., in second left intercostal space, half an inch from sternal edge.

**ALCOHOLIC CIRRHOSIS OF THE LIVER.**

**Case II.**—J. D., aged forty, admitted to Whitworth Hospital on December 17, 1904, where she remained till her death, six weeks later. A month before admission she complained of vomiting, swelling of the abdomen, and cough. A fortnight later severe diarrhœa came on. On admission the abdomen was much enlarged from flatulent distention, a circumstance which prevented the accurate mapping out of the liver and spleen. The lungs showed evidence of slight chronic bronchitis. The bowels acted six to ten times per diem. The skin and conjunctivæ were somewhat discoloured. Urine contained a slight trace of albumen. In the course of a fortnight some ascites could be detected, and coincidently the diarrhœa ceased. Three weeks later it became necessary to tap the abdomen, and 105 ounces were drawn off. The cells in the fluid were mostly desquamated endothelium. A week later tapping again became necessary;
then vomiting became severe, and death ensued from exhaustion and heart failure.

Post-mortem.—A small, typically hob-nailed liver was found and an enlarged congested spleen. The stomach and transverse colon were densely adherent to the gall-bladder, the walls of which were thickened.

The points of interest in this case were:—The marked flatulence, the late appearance of ascites, and the course of the temperature, which varied between sub-normal and 103° F. in a most irregular fashion. This temperature, the distended intestines which marked the real size of the liver and spleen, the violent diarrhoea, at first led to the erroneous diagnosis of some form of ulcerative enteritis, probably tubercular.

ALCOHOLIC CIRRHOSIS OF THE LIVER.

Case III.—A country girl, aged thirteen, when admitted to hospital, was found to be suffering from ascites, enlarged superficial abdominal veins, uniformly enlarged liver and spleen. The diagnosis of alcoholic cirrhosis of the liver was arrived at, but on questioning the patient as to what stimulants she used she declared herself an absolute teetotaller. On further investigation the fact was ascertained that for two years before admission she had almost constantly been taking a medicine of herbs concocted for her by her mother. What the indications for this mixture were I could never precisely learn, but its composition was a decoction of dock leaves, with whisky generously added. This patient died in about a month from admission. An autopsy revealed a hob-nailed liver with its usual train of morbid anatomical findings.

BILIARY CIRRHOSIS OF THE LIVER.

Case IV.—R. W., a farmer, aged twenty-one, previously healthy, and always temperate, in July, 1904, first noticed his urine becoming very dark in colour. At about the same time he noticed his eyes to be of a yellow colour, his skin yellow and itchy. After his skin got yellow he suffered from occasional attacks of vomiting, acid eructations, and a slight dull aching in the right hypochondrium. He was admitted to hospital four months later, in November, 1904. He was then found to be
well nourished and muscular. Moderate jaundice was present. The liver was thought to be enlarged on percussion, yet could not be palpated. The spleen was slightly enlarged, but a muscular abdominal wall prevented it from being felt distinctly. Pulse was 60; temperature subnormal. Urine dark and containing a quantity of bile pigments. The stools were acholic, abundant and clay-coloured. The bowels acted regularly. Blood was normal. By the end of December patient left hospital of his own request, between five and six months after the onset of jaundice. By that time the liver could be made out to be distinctly and uniformly enlarged by palpation. The spleen also could readily be felt enlarged. There was no ascites. The jaundice was as intense as on admission. The stools had, as a rule, remained acholic; occasionally they contained some bile colouring.

I made the diagnosis of biliary cirrhosis of the liver on the following data:—Jaundice persisting nearly six months, a gradual and uniform enlargement of the liver and spleen. Though such a condition of the stools as in this case more frequently indicates obstruction of the main bile duct than obstruction of a multitude of smaller bile ducts as from biliary cirrhosis, yet others have found acholic stools in association with hypertrophic cirrhosis. This diagnosis was greatly strengthened by the evident increase of the size of the spleen—a most important point.

ABDOMINAL ANEURYSM.

Case V.—M. H., aged fifty, by occupation a butler, was admitted to the Whitworth Hospital complaining of attacks of violent pain in the left lumbar region extending into the left groin. He stated that, with the exception of constipation, he had always enjoyed good health till about three months previously. From that date he began to experience some pain in the left lumbar region, tending to radiate towards the middle line in front, but as it was not severe he did not seek medical aid. Four days before admission he got the first attack of violent pain which made him feel extremely ill, and incapacitated him from all work. The attacks repeated themselves several times before admission; on each occasion the pain radiated to the groin, and was most intense in that situation. On admission the patient was observed to be pallid, and wore an expression indicative of suffering. On examining the abdomen by palpation a tumour
was detected in the left iliac fossa. Tracing it inwards from this point it extended almost to the middle line, whilst in an upward direction it extended under the arch of the ribs, but receded from the middle line. The right border of the tumour was well defined, especially below, where it felt firm and almost nodular. The left border of the mass could not be limited by palpation. Doubtful fluctuation was detected, but no pulsation whatever. On percussion the tumour was absolutely dull, its area fusing with those of the left kidney and spleen. Dulness was also present at the base of the left lung behind, where the breath sounds were diminished. These signs gave rise to the suspicion that the tumour, whatever its nature might be, was pressing the left cupola of the diaphragm upwards. The left loin did not visibly bulge, though the skin there was oedematous. The patient was unaware of the presence of the tumour, so could offer no clue to the duration of its existence. No signs of aortic aneurysm, either thoracic or abdominal, were detected, despite careful examinations with that object in view. Blood films were made which negatived a suspicion of spleno-medullary leukaemia. The urine was normal.

The diagnosis considered most probable was renal calculus leading to hydronephrosis of the left kidney, although the urine contained neither albumen, blood nor pus. The patient lived for seven days in hospital, each evening getting a violent paroxysm of pain relieved only by morphin. After each attack of pain it was noted that the swelling had extended and was tenser than before. Under these circumstances I advised the patient to submit to an exploratory operation. This he refused to do. He died a few hours later, just as the proposed operation would have been undertaken had he submitted.

Post-mortem.—An aneurysm about the size of a small fist, constricted in its middle by the aortic aperture in the diaphragm, was found. It had ruptured into the abdomen extra-peritoneally. An enormous amount of blood clot was found stripping the peritoneum from the post-abdominal wall down to the left iliac fossa, from the under surface of the diaphragm, and even from the left anterior abdominal wall. The left kidney was embedded in the clot, otherwise it was normal. The blood had not penetrated between the spleen and its peritoneal capsule. Nothing else abnormal was found of any note.
The interest of this case, apart from the interest always attaching to an autopsical correction of a clinical diagnosis, lies in the fact that a man could survive for eleven days with a ruptured aneurysm of the abdominal aorta, and for part of this period be able to get about.

A CASE OF PAROXYSMAL TACHYCARDIA.

Case VI.—P. R., a labourer, aged sixty, was admitted to the Whitworth Hospital in December, 1904. He stated that four years previously he began to suffer from attacks of palpitation of his heart. They occurred once a month at first, lasting about two hours, but gradually became more frequent and of longer duration. These longer attacks were often interspersed with ones of momentary length. Patient stated that the attacks always began with a burning sensation in the neck, which rapidly travelled to his heart, when instantly his heart began to beat violently and rapidly. He was conscious of the great rapidity. The attacks were always accompanied by lightness in the head. Having had good results in another case of paroxysmal tachycardia by administering bromide of potassium, I at once put this man upon the drug. During his stay in hospital on this occasion the attacks were all short, and gradually grew less frequent, finally disappearing. How much of his relief was due to the bromides and how much to the rest and good hygiene it is difficult to estimate. He left hospital without giving me the opportunity of personally observing an attack. The resident staff on several occasions were present during attacks, and counted the heart beats at something over 200. As a rule, the attacks were so short that they were over before anyone could be summoned to his bedside.

The patient returned to hospital early in February of this year (1905), and stated that his heart was as bad as ever again, the attacks having again become longer and more frequent. Two days after admission, on visiting him one morning at 11 a.m., I made the following note:—Patient states that yesterday afternoon he had several short attacks of palpitation. At 8 p.m. the attacks became continuous, lasted all through the night, and only allowed one hour's sleep. He complains of pain below the left nipple and a burning sensation behind the sternum. He says
his head is light, but he is able to read a book. He looks calm, a little paler than usual, but appears in no distress. Extremities are warm and of good colour. On looking across his chest, uncountably rapid vibratory movements are seen of the precordial and epigastric areas. The veins of the neck are not distended, and do not fill from below. On counting the rapidity of the heart beats with a stethoscope they are 212 to the minute. Only the first sound can be heard at the apex, very short, yet distinct. At the base a very feeble second sound can be heard after the first sound. That the heart is really beating at this enormous rate is proved by the facts that the carotid pulse can be counted at 212, and precordial pulsation (more difficult to count) at something over 200. In the radial arteries only 86 distinct pulse beats can be counted per minute; they vary in force more than in rhythm. It is a matter of doubt whether feeble, rapid, indefinite beats may not be felt between the larger ones. The pulse in the brachial and femoral arteries is of the same character as that of the radials. Whilst percussing the heart to try to map out the area of deep cardiac dulness the patient said—"You will find my heart slower now." On immediately counting with the stethoscope the heart rate was found to be 96, and a minute or so later 84. The storm had suddenly passed, the rapid and violent heart's action subsiding in the usual manner by a sensation travelling from the nipple region to the neck. It is my belief that the heart was somewhat dilated during this attack. The attack lasted 15 hours. The patient was now given bromides again. For the next few days some short paroxysms occurred, but for the last nine days none have appeared. He is still in hospital.

Between the attacks the heart's rapidity is usually about 80. The heart cannot be demonstrated to be enlarged. The impulse cannot be felt. The sounds, though clear, are feeble, even allowing for a thick chest wall, and a prolonged rest in bed. The arteries are sclerotic, but not markedly so. The urine is normal.

In my opinion the paroxysmal tachycardia in this case is not purely of nervous origin, but is associated with some degenerative change in the myocardium.
ON SOME COLOUR-TESTS FOR THE CHIEF NITROGENOUS CONSTITUENTS OF URINE.

By WALTER G. SMITH, M.D., F.R.C.P.I.;
Physician-in-Ordinary to His Excellency the Lord Lieutenant of Ireland;
Physician to Sir Patrick Dun's Hospital.

[Read in the Section of Medicine, March 31, 1905.]

The chief object of this note is to demonstrate to the Medical Section an interesting colour test for urea which has lately been discovered by Mr. Fenton, and, in connection with it, I propose also to show some of the older and better known colour tests for the other nitrogenous constituents of urine.

The nitrogenous bodies in urine which respond to colour tests are three in number. 1

1. Uric acid $C_5H_4N_4O_3$.

Uric acid contains 33.3 per cent. of nitrogen, and its average daily excretion is from 0.5 to 1 grm. Its origin is partly exogenous (food) and partly endogenous (tissue metabolism). Compared with urea, its amount in the urine is less influenced by the proteins of the food.

The murexide test is so well known and so justly esteemed that it is unnecessary to dwell upon it. Suffice it to say that the essential factor in its development is the presence of a body termed alloxantin ($C_5H_4N_4O_2$) which is formed, along with alloxan, when nitric acid acts upon uric acid.

If a little alloxantin be smeared upon a piece of glass

1 Hippuric acid is excreted in appreciable amount, 0.1 to 1 grm. daily, but there is no test for it directly applicable to urine.
or cardboard, gently warmed, and held over a test-tube containing some liq. ammoniæ, the fine red colour of murexide (ammonium purpurate) is at once produced. \([\text{Experiment shown.}]\)

But the murexide test requires a little time and care to carry out successfully, and involves evaporation to dryness before adding ammonia.

For uric acid in solution a simple and convenient test was indicated by Offer in 1894 (Centralbl. f. Physiologie, 1894, Bd. viii.). It consists in adding a few drops of the yellow solution of phospho-molybdic acid \((\text{H}_3\text{PO}_4.11\text{MoO}_3.12\text{H}_2\text{O})\) to the urine made strongly alkaline with liq. potassæ (vel sodæ). A rich Prussian blue is developed. \([\text{Experiment shown.}]\)

Red gravel, or a fragment of a uratic calculus, or of a gouty tophus, is likewise easily identified by dissolving in liq. potassæ, and adding the phospho-molybdic acid. The blue colour is the result of a reduction process. Neither urea, kreatinin, nor grape sugar affects it; but it reacts with levulose, albumin, and alkaloids.²

2. Kreatinin, \(\text{C}_4\text{H}_7\text{N}_3\text{O}\) contains 37.1 per cent. of nitrogen; daily excretion \(\frac{1}{2}\) to 1 grm. It is chiefly exogenous in origin, derived from the kreatin of meat, but is, in part, endogenous, due to metabolism of muscular tissue. It is not known how much of the ingested kreatin is excreted unaltered, or as kreatinin, or as urea. The best mode of demonstrating it in urine is by Weyl’s test.

Add a few drops of a weak, freshly-prepared solution of sodium nitroprusside \((\text{Na}_2\text{Fe(CN)}_5(\text{NO})_2\text{H}_2\text{O})\) and then, cautiously, a little liq. potassæ (v. sodæ). A red colour is produced. \([\text{Experiment shown.}]\)

²This test was accidentally re-discovered some time ago in the Chemical Laboratory, T.C.D., by a medical student, Mr. Henley, now in the I.M.S.
The Chief Nitrogenous Constituents of Urine.

Acetone \( (C_3H_6O) \), which is sometimes present pathologically in considerable amount, reacts similarly with liq. potassae.

If liq. ammonia be substituted for the liq. potassae, acetone develops a rich purple colour; kreatinin a clear red.

3. Urea \( (CH_4N_2O) \) contains 46 per cent. of nitrogen.

Its origin is mainly endogenous, and its daily excretion amounts to 30 to 40 gms.

Kossel and Dakin have recently made the striking discovery of an enzyme, termed arginase, which acts upon arginin, one of the simpler cleavage-products of proteids.

Arginase is found in the intestinal mucous membrane, but more especially in the liver, and it splits arginin almost quantitatively into urea and ornithin \( (\text{Zeitschr. f. physiol. Chem.}, \text{Bd. XLI.}, 321) \).

Hitherto the usual mode of identifying urea was by microscopical examination of some of its crystalline salts, either the nitrate or oxalate. There was no satisfactory colour test for it.

In 1903, however, Mr. Henry Fenton \( (\text{Journ. Chem. Soc.}, \text{Feb., 1903}) \) discovered an interesting colour test, which I shall presently demonstrate.

The essential reagent is a compound termed methyl-furil \( (C_{11}H_8O_4) \), a derivative of furfural. This compound occurs in yellow, lustrous needles, and I am much indebted to Dr. Emerson Reynolds for a specimen of it.

The test will not answer directly with urine until it is evaporated to dryness or nearly so. If a fragment of urea or of the dried urine-residue be ground up with a trace of methyl-furil, and a few drops of strong HCl be added, a rich blue colour is developed. \([E \text{xperiment shown.}]\)

If the substance to be examined is in solution it may
be mixed with an alcoholic solution of the reagent, and evaporated to dryness on the water-bath.

0.1 mgm. of urea yields a strongly-marked colour, and, with care, it is easy to detect 0.01 mgm., or even less.

Thio-urea gives a slight greenish colour. The blue colour is characteristic of urea (carbamide) and of mono-substituted carbamides (alkyl group). Acidyl-carbamides do not give the reaction.

With primary amines (e.g., aniline), in acetic acid solution, the test produces a brilliant green, without the aid of a condensing agent—e.g., HCl₂ or POCl₃.
THE *BACILLUS COLI COMMUNIS* AS A CAUSE OF SEPTICÆMIA.

**By T. GILLMAN MOORHEAD, M.D., M.R.C.P.I.**

Physician to the Royal City of Dublin Hospital.

[Read in the Section of Medicine, May 19, 1905.]

Since the first description by Escherich in 1884 of the *Bacillus coli communis* as a normal inhabitant of the human alimentary canal, a considerable change has taken place in the views currently held with regard to the rôle that that organism is supposed to play in human and animal pathology.

Escherich himself, although he had shown that the bacillus could produce a fatal toxæmia in animals, was of the opinion that it was quite harmless to man, and that its importance principally depended on its ability to further the ultimate processes of digestion within the alimentary canal. Before long, however, evidence began to accumulate to show that the organism was widely disseminated throughout the animal kingdom and had pathogenic properties, and in 1887 Javel, of Berne, reported a case of infection of a goitre wound by the bacillus. Four years later Wurtz was able to prove the occasional causal agency of the organism in the following extensive list of pathological conditions:—(1) Cholera nostras, dysentery, membranous colitis, and other intestinal lesions; (2) peritonitis; (3) angiocholitis and cholangitis; (4) cystitis; (5) various pulmonary diseases, including pleurisy and broncho-pneumonia; (6) endocarditis; (7) meningitis; (8) arthritis. This list can now be still further extended by the addition of pancreatitis, mastitis, otitis media, conjunctivitis and many other conditions, but it is unnecessary to dwell upon these facts, inasmuch as surgeons and bacteriologists have long
since learned the pyogenic properties of the bacillus, and are accustomed to suspect its presence more especially in abscesses formed in the neighbourhood of the intestinal canal, while it is also pretty generally admitted that metastatic abscesses may owe their origin in some cases to its action.

In these cases in which the organism acts as a pus producer, some at least of the symptoms from which the patient suffers must be regarded as being of a sapraemic nature, due to the absorption of the colon bacillus toxins, for that it is capable of producing powerfully poisonous substances has been shown by Sidney Martin, Alessandro Carega, and others. Whether, however, the organism can ever assume a septic or septicæmic role, as distinct from a pyogenic one, and produce an actual septicæmia, is a point which has not yet been definitely determined, and is the one which forms the object of this paper.

In reviewing the facts that bear upon the question, as far as the human subject is concerned, the evidence in favour of the existence of a \textit{B. coli} septicæmia may be divided into (1) reports of cases presenting during life septicæmic symptoms, and in which the \textit{B. coli} was found in the peripheral blood during life; and (2) reports of similar cases in which the \textit{B. coli} has been found in the blood and widely disseminated throughout the viscera after death.

If, under the first heading, a large number of cases could be cited, and the bacillus could be proved in such cases to be virulent by animal experiments, the necessary proof would be at once complete. At present, however, there are but few records of cases in which the \textit{B. coli} has been found in the blood during life, for after a fairly complete search through the literature I have only been able to find the following well-authenticated reports:—(1) A case reported by Henschen\textsuperscript{9} in 1901, in which death resulted from septic ulceration of the aortic valves. During life the colon bacillus was isolated in pure culture from the blood in the early stages of the disease.
Bacillus Coli Communis as a cause of Septicæmia.

while later on streptococci also appeared; (2) Siredey reported in 1895 a case of general infection with the *B. coli* during an attack of influenza, in which the colon bacillus was obtained from the spleen during life; (3) Kowalewski and Moro reported in 1901 two fatal cases in children in which the *B. coli* was obtained from the blood during life, while colitis was found *post mortem*; (4) Sittmar and Barlov recorded in 1894 a case of general *B. coli* septicæmia following a local infection of the urethra. The *B. coli* was found in the blood during life.

The very small number of the above cases of course detracts from their value, but, in considering them, it must be remembered that it is only in rare cases that the colon bacillus would be sought for during life, and that in many instances the finding of a bacillus, instead of a coccus, in an obviously septicæmic case, would lead the observer, owing to the prevalent views with regard to the etiology of all septicæmic conditions, to believe that he was dealing with an external infection.

The next evidence that must be dealt with consists of the bacteriological findings at autopsies, and here we are met with some surprising results and with varied interpretations of them.

A review of the observations published during the last 15 years renders it at once evident that the colon bacillus is frequently found in the abdominal organs *post mortem*, and also, but less frequently, in the cardiac blood, and, according to the opinions held with regard to the significance of this fact, bacteriologists may be divided into the following groups:—

(1) Those who regard the presence of the bacilli as due to a *post-mortem* invasion of the tissues through the intestinal walls; (2) those who believe that the organisms first gain entrance to the body during the so-called agonal period which immediately precedes death; and (3) those who believe that the *B. coli* may invade the body during life, either through diseased or healthy intestines, and either itself produce a
By Dr. T. Gillman Moorhead.

septicaemia, modify the course of a previously existing septicaemia, or prepare the way for a septicaemia to be caused by some other organism.

Before the above-mentioned post-mortem evidence can be criticised, however, it is necessary to consider one or two general bacteriological questions, and, in the first place, we must know what reliance can be placed, from a diagnostic point of view, on the finding of any particular organism in the organs and blood post mortem. In the early days of bacteriology it was generally believed that the finding of an organism in the blood and organs, within a few hours after death, constituted valid evidence that the bacterium in question had taken a share in the production of symptoms during life, provided that it proved toxic when injected into animals. This early view has, of course, been modified, and has lately received some considerable shocks, for it has been conclusively proved by Ford that bacteria can be cultivated almost invariably from the healthy organs of animals, even when the organs are removed immediately after death; while further, Gradwohl and others have shown that organisms may rapidly wander post mortem from adjacent viscera into the cardiac blood. When, however, Ford's results are examined into, it is found that he has only dealt with the abdominal viscera, and that it was only after the lapse of many days in culture media that growths were obtained from these—this latter fact suggesting that the bacteria in the viscera were few in number, and, possibly at first, in an enfeebled state produced by the activities of the healthy tissue cells. Again, in his experiments on human organs removed at autopsy, it is to be noted that only the abdominal viscera were examined, and, still more important, that in the majority of cases the same organism was not obtained from each viscus, and that, in many cases, mixed cultures were obtained from individual viscera. In consequence Ford's valuable conclusions cannot
upset a *post-mortem* bacteriological diagnosis when the same organism is found in pure cultures, widely disseminated throughout the body, and when it develops rapidly and abundantly in media, always provided, of course, that the autopsy is made soon after death, and that the organism found proves toxic to and is recoverable from animals. Gradwohl's conclusions, though contradictory to those of some authorities, are sufficiently definite to be accepted. His main statement is that not only may organisms wander rapidly after death from diseased organs into the cardiac blood, but also that the bacteria commonly present in healthy organs may rapidly escape through their walls into the blood vessels, and quickly reach the heart. The acceptance of this statement in its entirety does not, however, invalidate what has been already said—namely, that the wide distribution of an organism in pure culture throughout the body, especially in a case which presented septicaemic symptoms during life, may be relied on as making a bacteriological diagnosis.

To return now to the *Bacillus coli*. I see no reason why that organism should not be regarded as the cause of septicaemia, if it be found in pure culture widely disseminated throughout the body *post mortem*, and if it be proved virulent by animal experiments. Those who deny such a possibility, owing to the frequent occurrence of the *B. coli* in the organs *post mortem*, in cases where it has evidently not been present in the blood during life, would be compelled logically to deny also the existence of a streptococcal or staphylococcal septicaemia, for a glance at the results of Ford, Gradwohl, J. O. Symes and others show that these cocci are often to be found scattered in viscera *post mortem*.

Moreover, when the *B. coli* has been found in pure culture *post mortem*, in cases where there was no suspicion of any septicaemic state, it has almost invariably been found only in the abdominal viscera when the autopsy has been performed.
soon after death, while in autopsies deferred for twenty hours or more it has also been found in the cardiac blood. While, therefore, it must be admitted that the *B. coli* can pass rapidly through the intestinal wall after death, the fact must not be regarded as negating the possibility of a colon septicæmia.

As regards the occurrence of an agonal inwandering of bacilli through the intestinal wall, opinions are numerous, but evidence is scanty, and what there is appears to show that such an invasion occurs only to a limited extent, and that the organism in such cases does not pass further than the abdominal organs. Birch-Hirschfield" admits its occurrence, but lays much more stress upon the *post-mortem* invasion, which, he states, frequently occurs. Even if its occurrence be admitted, however, it does not negative the possibility of a septicæmia.

To sum up now the evidence that does exist in favour of the existence of a *B. coli* septicæmia, we have the following facts:

(1) Its known power of producing a septicæmia in animals, and in connection with this fact it may be stated that Lésage and Macaigne 17 have shown that the organism, isolated from the intestines in cases of diarrhœa, is much more toxic than that obtained from the healthy alimentary canal. They, in fact, believe that the bacillus of the normal intestines can only exert a local pyogenic toxicity, while that from diseased intestines has septicæmic powers. It may also be stated that Ferranini 18 has shown that the *B. coli* exerts a much more powerful effect on ill-fed animals than on well-nourished ones: that, in fact, its toxicity increases with the diminution in resisting power of its host.

(2) The cases already referred to in which the *B. coli* was found in the blood during life.

(3) Cases of general sepsis reported by Schenck,19 Gebhard,20 Kerr,21 Eisenhardt,22 and others, in which the *B. coli* was
found universally throughout the body in pure culture after death, and along with these may be mentioned Kamur's work on Winckel's disease.

(4) The occurrence under natural conditions of infectious diseases in the lower animals apparently caused by the colon bacillus.

The power of the *B. coli* in modifying the course of, or preparing the way for, other forms of septicæmia is indicated by the following facts:—(1) Motta Coco, experimenting on animals, found that the *B. coli* assumed very virulent properties in company with streptococci; (2) Widal and Besançon working with the streptococci of the mouth, ascertained that many which were harmless when injected alone into animals became virulent when mixed with the colon bacillus; (3) Blasi and Russo-Travali reported in 1896 a case of mixed infection with the *B. coli* and diphtheria bacillus, and found experimentally that a mixture of the two bacilli was much more poisonous than either organism singly. Schenck also reported in 1898 a case of combined coli and streptococcal infection, and makes the statement that the *B. coli* is always very virulent when combined with streptococci; while, as showing that the opinion that the *B. coli* might combine with other organisms to produce a septicæmia has been long held, it may be added that in 1896 Sims Woodhead said that he believed many so-called streptococcal infections to be really secondary to colon infections.

From the above reasoning I think it may be concluded that the existence of a *B. coli* septicæmia is probable, and at any rate is certainly not disproved. Possibly its existence accounts in some cases for the inefficacy of anti-streptococcic serum. The following case is, I believe, an example of it, and, as it is of a rather unusual character, the report of it is given in some detail.
By Dr. T. Gillman Moorhead.

W. M., aged thirty-seven years, married, by occupation a wirer, engaged in setting up telegraph wires, was admitted to hospital on 22nd October, 1904, complaining of pains in his head, back, and limbs.

He stated on admission that he had felt ill for the last three weeks, and had been compelled to give up work for six days before admission on account of the pains in his bones. He had had no diarrhea nor vomiting, but suffered slightly from pain in the stomach during the first few days of his sickness. On the evening before admission he had a severe shivering fit, followed by profuse perspiration. He had had no cough nor soreness of throat.

Previous History.—The patient had lived all his life in Ireland, and had been at the same work for several years. The only other sickness that he remembered having was a mild attack of rheumatic fever six years previously, for which he had gone to hospital. He had never been a heavy drinker, and he denied having had any form of venereal disease. He had been married for the last twelve years.

Family History.—Father and mother were both dead, but the cause of death was unknown. There were two brothers and one sister, all of whom were alive and healthy. His wife was alive and well in every way. She stated that she had had five children, all of whom were healthy. The last child was born twelve months previously. She had not had any miscarriages.

Condition on Admission.—Patient was a strong-looking, well-built man, in height 5 feet 9½ inches, weight 9 stone 2½ lbs. His expression was rather dull, and a few drops of sweat were present on the forehead. The tongue was lightly coated all over with a thin whitish fur; teeth good; throat healthy; no enlarged glands to be felt in the neck or elsewhere. The thoracic organs appeared quite healthy, except that the first sound of the heart was rather feeble; pulse 64 to the minute, regular, but very compressible; respirations regular, 18 to the minute. The abdominal organs all appeared healthy, and although the patient complained of a slight feeling of soreness there was no tenderness on pressure over the abdomen. Two or three slightly raised papules were present on the abdominal wall, resembling the rose spots of enteric fever, but of a darker colour. There was slight subsultus tendinum, but otherwise the nervous and muscular system was quite normal.
Urine acid, sp. gr. 1010, no sugar, no albumen, quantity in first 24 hours = 52 ounces. Temperature 100° F.

Subsequent History.—The patient passed a good night, and on the next morning his temperature was normal, and he expressed himself as feeling quite well except for slight pain in the knee-joints. A blood-examination on this day gave the following results:

Red blood corpuscles . . . 3,300,000 per c.m.
White blood corpuscles . . . 30,000 " "

Differential count of white cells:
Neutrophiles . . . . . . 78 per cent.
Eosinophiles . . . . . . 2 " "
Lymphocytes . . . . . . 16 " "
Hyaline cells . . . . . . 4 " "

A fluid motion was passed on this day, and contained traces of blood, which, however, from its appearance, was evidently derived from some small internal hemorrhoids. A trace of blood was also found on one or two subsequent occasions, but was always of the same character. During the afternoon the patient began to complain of headache, drowsiness, and pains all over his body; and at 4 p.m. he had a severe rigor, followed by a rise of temperature up to 105.2° F. The temperature remained high for a couple of hours, and then dropped suddenly to normal. During the period of pyrexia there was great thirst, and the skin was hot and dry, and became covered all over with a raised blotchy red eruption. Then, as the temperature fell, the rash disappeared and there was profuse perspiration.

The patient again passed a good night, and felt quite well the next morning, but again during the afternoon had a rigor with a rise of temperature up to 104° F., and symptoms similar to those of the previous day. The temperature again fell rapidly, accompanied by a general amelioration of discomfort. These two days proved to be only the precursors of similar ones, for during the next ten weeks, or, more exactly, 73 days, this type of pyrexia, accompanied by an eruption and joint pains, and followed by profuse perspiration, was repeated daily. The exact hour at which the rigor and rise of temperature took place tended, however, to vary slightly from day to day.

During the first week a Widal test was carried out daily, but always gave a negative result, and on several subsequent occasions
it also proved negative. The agglutination test with two varieties of the paracolon bacillus also gave a negative result. On the eighth day of the patient’s stay in hospital some blood was withdrawn from the median basilic vein and was plated out on agar. One colony of staphylococcus aureus developed on the second plate, and was accordingly judged to be a skin infection. On six other occasions an attempt was made to grow organisms from the blood, and the media were varied in the hopes of obtaining a successful result. Thus large quantities of broth were twice used in order to neutralise any possible inhibitory action of the blood. Both, however, remained sterile. In a similar manner milk was used, half an ounce of blood being added to 200 c.c.s. of milk. In the first experiment a bacillus developed after five days, and caused curdling, but, owing to an accident to the culture, the organism could not be identified, and subsequent attempts with the same medium failed to produce any growth. On one occasion 5 c.c.s. of blood from a vein in the arm was injected intraperitoneally into a guinea-pig, but without effect.

During the entire period of pyrexia frequent enumerations of the white cells were made, and showed that the leucocytosis present at first was tending to diminish. Thus, on October 5th, the number was 26,250 per c.m.; on Oct. 20, 20,100 per c.m.; on Nov. 5, 17,500 per c.m.; while a few days later the number was only 12,500 per c.m., and they remained about this level until the termination of the case. Differential counts showed about the same relative numbers as were present at the time of admission. No myelocytes or other abnormal cells were at any time detected. The number of erythrocytes remained at about 3,000,000 per c.m. till towards the end of the case, when the number sank rapidly to a little above 2,100,000.

The urine throughout the febrile period remained normal in quantity and free from abnormal constituents; the bowels were usually constipated, requiring enemata or mild purgatives to relieve them, but the motions were normal in appearance and after the first there was no abdominal pain. The spleen was normal in size throughout.

The patient himself felt quite well throughout the earlier hours of each day, except for occasional joint pains, and for a little oedema about the ankles, which caused some discomfit. The
eruption, already noted as appearing during the first pyrexial period, was very variable in its occurrence and in its duration. At one time it remained persistent all over the body for several days, and then quite suddenly disappeared. It was at first of a purely papulo-erythematous type, but later on became petechial. The lungs remained healthy throughout, and the heart sounds normal; the pulse was feeble, but remained slow, and even when the temperature was at its highest point seldom exceeded 80 to the minute. During the first couple of weeks the patient was kept in bed and on a strict fever diet, but later on he was allowed to get up and even occasionally to go out, and his diet was largely increased. His appetite remained good, and he increased in weight from 9 stone 2½ lbs. on admission to 9 stone 9 lbs. on December 2. Various drugs were administered, but all without any apparent effect on the symptoms. Thus at different times the patient was placed on quinine, salicylates, potassium iodide, and mercury. The question of using anti-streptococcic serum was considered on more than one occasion, but owing to the negative results of the blood examinations and the absence of any apparent focus from which streptococci could enter the system it was not used. On December 6, the 76th day in hospital, the symptoms began to change. The patient complained of severe headache, and vomited once without apparent cause. The headache was relieved at first by treatment, but returned, and became very severe, persisting on and off for the next couple of weeks. On December 8, the temperature only rose to 100° F., and then, after a few days of mild pyrexia, became normal, and remained so till three or four days before death, when it sank to 95.4° F. On December 12, a well-marked Kernig's sign was first observed on both sides, and on the succeeding days the knee-jerks became increased, and Babinski's sign appeared. Some slight spasm of the muscles of the arms was also noted. The pupils became very sluggish in their reaction to light, but an ophthalmoscopic examination by Mr. Benson revealed nothing abnormal with the fundus. On December 13, a lumbar puncture was performed, but no fluid was obtained. On this day the patient was semi-comatose, and he remained so, with slight intermissions, until the onset of complete coma about a month later. During this month one curious symptom was present, consisting of apparently intense itching, as the patient
used to scratch himself automatically all day, and, in spite of his hands being tied and of careful watching, he eventually succeeded in producing ulcers in one or two places, which became infected with streptococci and staphylococci, and tended to spread. Loss of the power of swallowing and relaxation of the sphincters finally appeared, and death took place on January 25, 126 days after admission. For two days before death the pulse rate was only 34 to the minute, and the respirations from 6 to 10.

Post-mortem.—This was made six hours after death. The pleura and lungs were normal; the pericardium was bound by light, and apparently rather recent, adhesions to the whole surface of the heart; the cardiac chambers and valves and the large arteries were normal. The intestines, on opening the abdomen, were seen to be congested, and a few subperitoneal petechiae were observed; the peritoneum on the posterior abdominal wall was thickened, and the retroperitoneal and mesenteric lymph glands were somewhat enlarged. The inner surface of the intestine, especially the upper part of the jejunum, showed intense congestion and catarrhal inflammation, while in the ileum the Peyer’s patches were reddened and a little raised above the surface; microscopically the patches showed cellular hyperplasia and hyperemia, but no necrosis. Within the stomach several scattered submucous hemorrhages were present, but without ulceration; the large intestine was healthy. The mesenteric glands showed some increase of connective tissue, and also large patches of hyaline material. The spleen was normal in size and appearance, both macroscopically and microscopically. The liver and kidneys showed cloudy swelling, but were otherwise normal; suprarenals and accessory glands normal. On removing the calvarium, the convolutions of the brain appeared flattened and unusually dry, and the cerebral veins were dilated; the meninges were quite healthy, and microscopically showed no changes. Both lateral ventricles and the third ventricles were greatly distended with clear healthy-looking cerebro-spinal fluid. The ependyma appeared normal microscopically. The meninges of the spinal cord were congested, and there was an increase of subarachnoid spinal fluid which was slightly turbid in appearance. The spinal cord itself microscopically was normal.

Bacteriological Examination.—The following cultures were
made, the greatest possible care being taken to observe all sterile precautions:—

(1) About 5 c.cs. of cerebro-spinal fluid from the left lateral ventricle of the brain were received into 250 c.cs. of broth.
(2) Agar plates were prepared from the fluid in the right lateral ventricle of the brain.
(3) Streaks of fluid from the spinal canal were made on cold agar plates, and on serum tubes.
(4) Agar plates were made from the spleen pulp, and also streaks of the spleen pulp on agar.
(5) Five c.cs. of blood from the right auricle were received into 500 c.cs. of glucose broth.

In addition to the above cultures, about 5 c.cs. of the cerebro-spinal fluid were injected into the peritoneal cavity of a full-grown rabbit. This caused some rise of temperatures and sickness for a couple of days, but the animal eventually recovered.

The above described cultures were incubated for 24 hours, and were then examined. The agar plates from the cerebro-spinal fluid and those from the spleen pulp gave a pure culture of the B. coli communis. The growth of cerebro-spinal fluid on broth, and of the heart blood on glucose broth, both contained a bacillus which was ultimately identified as the colon bacillus; and also another organism of a diplococcoid form which will be referred to immediately. The streaks made from the spleen pulp on agar and also the cultures from the fluid in the spinal canal contained also the colon bacillus and a diplococcus.

The colon bacillus from each of the above-described sources was found to be freely motile, to ferment glucose and lactose, to produce gas and indol, to curdle milk, and to give the neutral red reaction. It was also found that about .25 of a cc. of its culture in broth proved fatal to full-grown guinea-pigs in about 18 hours, when injected either intraperitoneally or subcutaneously, and that the organisms could be obtained again in a virulent state from the blood and viscera. The attempt to isolate the diplococcus, already referred to, proved a difficult matter, for it appeared only capable at first of growing in a state of symbosis with the colon bacillus on agar, gelatine, blood serum, and alkaline serum. An emulsion of blood serum, in which both the coccus and the colon bacillus were growing, was even injected into a guinea-pig, but only the colon bacillus could be obtained
again from the blood and viscera. Finally, plates of Conradi-Drigalski medium were used, and on this some very small, almost microscopical, colonies developed in addition to the colon colonies, and proved to be the pure culture sought. The organism then turned out, as was suspected, to be the pneumococcus, although rather atypical in some of its characteristics. It did not prove fatal to either guinea-pigs or mice, when injected in large quantities subcutaneously and intraperitoneally, nor did it produce pneumonia when injected directly into a mouse's lung.

Sections of most of the tissues were stained to show bacteria, and gave the following results:—In the intestinal wall, and invading the agminated follicles, numerous organisms were found, many of which were bacilli, and did not stain by Gram; some Gram-staining bacilli and numerous cocci were also found. In the liver, kidney, suprarenals, and spleen numerous non-Gram-staining bacilli were found. No organisms were found in the heart wall. In the meninges of the brain and just beneath the pia mater numerous non-Gram-staining bacilli were found, and also in a few sections some diplococci resembling those found in the cultures. No organisms were obtained in sections of the ependyma.

The post-mortem findings in this case are, I think, quite sufficient to justify the conclusion that the B. coli was the principal factor at work in producing the symptoms. It is hard to believe that it could be found so universally in the body, in so virulent a condition, and so far from the intestinal tract as the cerebro-spinal fluid of the lateral ventricles if it had entered post mortem or even during the prolonged agonal period. It may be, I think, admitted that the non-Gram-staining bacillus found in the sections was the colon bacillus. The fact that so many different varieties of organisms were found in the intestinal sections also supports the conclusion that this case was one of a true colon infection, for if the B. coli entered the vessels post mortem it is hard to see why the other bacilli were prevented from doing so. The mere motility of the colon bacillus could not explain such an occurrence as Gradwohl's investigations show.
Bacillus Coli Communis as a cause of Septicaemia.

The part played by the pneumococcal organism it is impossible to state, but the fact that it had lost all toxic properties seems to show that its influence was quite a secondary one, and the difficulty of obtaining a growth apart from the colon bacillus points in the same direction. Most probably, however, it aided in increasing the virulence of the other organism. If the case had been primarily a pneumococcal infection one would expect more marked local phenomena, and, in accordance with the results of Widal and Besançon, one would expect the virulence of the pneumococcus to be increased rather than diminished. It has been too long believed that the colon bacillus is capable of overgrowing other organisms, but that opinion is beginning to alter is borne out by some of the statements in the recent Erasmus Wilson Lectures of Dudgeon and Sargent. Again, the local intestinal lesion that was present, and of which the early abdominal pain may have been a symptom, and the enlarged mesenteric glands, point much more strongly to the colon bacillus than to the pneumococcus. Some experiments are at present being carried out to see if the pneumococcus obtained can heighten the virulence of other strains of the colon bacillus.

It is of course unfortunate that no growth could be obtained from the blood during life in this case, but the absence of such growths does not prove the non-existence of organisms in the peripheral blood, as it is a well-known fact that the results of blood cultures during life are very uncertain. Frequently a growth may be obtained on one day and none at all on the next, or organisms may even be found in stained preparations of blood which has failed to develop anything when grown in the ordinary nutrient media.

My thanks are due to Professor A. C. O'Sullivan and Dr. J. T. Wigham for aid and suggestions in carrying out the bacteriological investigation of this case. The last-named was also good enough to examine the spinal cord for me.
REFERENCES.

2 Wurtz. Archiv. de Médecine Expérimentale. 1893.

Jensen. B. coli als Krankheitserreger bei Tieren.
5 and 6 Ergebnisse der Path. und Path. Anat. 1897.
7 Sidney Martin. Lancet. 1898.
8 Alessandro Carega. "Ueber die aktiven Substanzen des B. Coli."

Centralbl. für Bakt. August, 1903.
9 Quoted by König, H. Histolog. Untersuchungen in Endocarditis.
16 Birch-Hirschfield. "Ueber das Eindringen von Darmbakterien, etc."

27 Blasi and Rasso-Travali. Annales de l'Institut Pasteur. 1896.
28 Schenck. Loc. cit.
ABSTRACTS.

SECTION OF MEDICINE.

Friday, November 11, 1904.

The President, Dr. W. J. Smyly, in the Chair.

Sporadic Cretinism.

Dr. Kirkpatrick showed a child, aged four, which at the present time weighs 15 lbs. Two years ago the child was under observation, and then weighed 13 lbs. The child is greatly emaciated and shows little or no signs of intelligence, and has never made any attempt to speak. The skin is clear and soft, and the hair well grown, the characteristic cretinoid aspect being completely absent. There is no history of congenital syphilis, nor are there any well-marked signs of this condition, and though there are some signs of previously existing rickets, there was no evidence of the acute stage of this disease while the child was under observation. No trace of the thyroid gland can be detected by palpation in the neck, and during the last fortnight, while the child has been under thyroid treatment, he has shown some signs of improvement. Dr. Kirkpatrick was inclined to look on the case as a rather anomalous one of sporadic cretinism.

Dr. Walter Smith asked was the possibility of rickets with hydrocephalus considered as a diagnosis, as the symptoms would fit in with that?

Dr. Langford Symes said that one or two possibilities occurred to him. First, rickets. The head was typically rachitic, with large fontanelles and extreme bossing. There was also some beading of the ribs, though there was a lack of the enormous beading one would expect. Second, there was a possibility of some congenital syphilis. The child’s teeth were strongly notched. The abdomen was peculiar, being a very large, protuberant one, and the intestines were apparently a good deal matted. The child was also suffering from great marasmus. There was serious mental defect, and probably some hydrocephalus.

Colonel McNeece asked was there any history of consanguinity? He related a case of two first cousins having married, the parents of one being also first cousins. The first child presented exactly the same appearance as Dr. Kirkpatrick’s patient.
Twins were afterwards born, one like the first child, the other healthy. There was no history of syphilis.

Dr. Kirkpatrick, in reply, said that ten days ago the child was put on thyroid extract, and during that period the child had shown signs of improvement. It had shown more intelligence, took more interest in its surroundings, and was more anxious for food.

Dr. Travers Smith exhibited the four following patients:

(a) A girl, aged ten, with a distinct systolic thrill, felt all over the precordial area, most marked in the region of the pulmonary artery. The thrill corresponded to an exceedingly loud, rough murmur, most audible in the pulmonary area. The right ventricle was slightly enlarged. A diagnosis of some congenital defect of the pulmonary artery leading to slight stenosis was made.

(b) A middle-aged woman with complete loss of perception of tactile, thermal, and painful sensation in her entire skin. The mucous membranes were not affected. The conditions had existed for a week or more. The diagnosis of hysteria was made.

(c) A man aged thirty-five, who a year previously suffered from left-sided hemiplegia and hemianesthesia. Thrombosis of the branch of the middle cerebral artery supplying the right internal capsule was diagnosed, and though a history of syphilis was denied, this was considered the most likely cause. Now he complained of typical lightning pains, Argyll-Robertson pupils had developed, and there was impaired sensation in the feet. The knee-jerk on the left side was still greatly exaggerated, and Babinski's sign was present, whilst the right knee-jerk was almost completely lost. Ataxia was doubtful, even with severe tests. Tabes dorsalis was now diagnosed, thereby confirming the suspicion of syphilis.

(d) An elderly woman, with hemiathetosis affecting the leg, arm, lower part of face, and tongue on the right side.

Caisson Disease.

Dr. J. B. Coleman made a communication on the subject of caisson disease, as experienced by workers engaged in boring a tunnel for five miles beneath the bed of Lake Erie from and after the year 1897. The symptoms came on after rapid decompression of one to three minutes. From ten minutes to six hours after reaching normal pressure the patient suffered excruciating pains,
usually in his knees, sometimes in shoulders and elbows, rarely in his hips; maniacal attacks supervened occasionally; retention of urine was common, and in the severe attacks paralysis of the sphincter ani. Paraplegia occurred and lasted for a few hours to months, and in severe cases persisted with the symptoms of spastic paraplegia; transient facial paralysis was seen; anaesthesia extending up to the ribs was common; an intense feeling of pruritus ("the itch") might occur without pains; bleeding from nose and ears; vertigo, dimness of vision, headache, vomiting, and muscular cramps were other symptoms noted. Urgent attacks of dyspnœa ("the chokes") were frequent, during which the patient felt his lungs over-distended; then a cough was set up and large quantities of gas, seemingly from the stomach, issued from the mouth. Relief from the joint pains and from retention of urine was obtained by a very hot bath. After work the men felt "wornied and bruised," and on returning to the compressed air they became exhilarated and relieved. Except in the most severe attacks the symptoms always abated on undergoing recompression. The highest pressure was 46 lbs. to the square inch.

Dr. G. Peacocke described two cases of Hodgkin's Disease (see page 1, ante).

Friday, December 9, 1904.

Dr. Hawtrey Benson in the Chair.

Living Exhibits.

Dr. Coleman exhibited a case of (a) Congenital Bulbar Paralysis, and a case of (b) Pityriasis Rubra.

Dr. T. P. C. Kirkpatrick showed a case of Ichthyosis Simplex.

Dr. F. C. Purser showed a Peculiar Deformity of the Spine, with nervous symptoms—a girl, aged eight, who was born with spina bifida. Marked deformity of the spine remained, causing muscular wasting, anaesthesia, lost knee-jerk, and trophic sores on the left leg.

Dr. O'Carroll reviewed three cases of Syringomyelia (see page 6, ante).
Abstracts.

Friday, January 20, 1905.

SIR J. W. MOORE in the Chair.

DR. PARSONS read notes on three cases of Paratyphoid Fever (see page 16, ante).

DR. PEACOCKE described the clinical history of Two Cases of Large White Kidney (see page 25, ante).

Friday, March 3, 1905.

SIR J. W. MOORE in the Chair.

Exhibits.

DR. W. J. THOMPSON—A case of Alopecia affecting the entire scalp of a boy.

A Case of Hysterical Chorea.

DR. JAMES CRAIG exhibited a case of hysterical chorea in a girl twenty-two years of age. There is no neuropathic family history obtainable. She was in good health, and was occupied as a laundress until two years ago, when she found herself unable to write on account of a shaking in her right hand. The shaking then extended to her head. At the present time she has a rhythmical rotary tremor of the head—coarse in character, particularly when she is under observation, but at times much finer, and frequently not to be noticed at all. There are no contractions seen in the facial muscles. There is a fine tremor occasionally present in the hands, but arhythmic contractions, incoördination or weakness of muscles are not observed. There is an absence of any of the other manifestations of hysteria, nor is there any evidence of any organic disease of the nervous system. Rhythmic hysterical spasm is the better name for the condition, as the term chorea is misleading.

DR. CRAIG read a paper entitled "Sequel to a case of Innominate Aneurysm, reported in 1898" (see page 31, ante).

DR. DRURY read a paper on Three Cases of Pneumonia in Pregnancy (see page 35, ante).
Section of Medicine.

Friday, March 31, 1905.

Dr. H. C. DRURY in the Chair.

Dr. BEWLEY showed a case of Syringomyelia, and read the following remarks thereon:—

Syringomyelia belongs to the rarer class of diseases of the spinal cord, and therefore it seems that well-marked cases which are met with from time to time should be exhibited and recorded.

The symptoms of this condition are most often noticed about the time of puberty, or rather later. They may be divided into several groups:—(1) The peculiar disturbance of sensations, called by Charcot "dissociated anaesthesia"—i.e., diminution or loss of sensation of pain and of heat or cold, while ordinary sensation is fairly well preserved. (2) Trophic changes in the parts affected, leading to various phenomena, among which is frequently found curvature of the spine. (3) Muscular atrophy. (4) Alteration of the spinal reflexes. (5) Paralysis of the sympathetic nerve in the neck. (6) Affections of the cranial nerves. These symptoms are usually more marked on one side than on the other.

The case which I have to exhibit to you presents several of these symptoms in a typical form.

G. H., aged twenty-two, of healthy parentage as far as I can ascertain, noticed about the year 1901, when she was sixteen years of age, that the fingers of her right hand were inclined to become inflamed from time to time. Some of these inflamations were severe and suppurative in type, and the bones of the ungual phalanges were affected. Some time afterwards she noticed that when she put her hands into hot water she felt the heat much more with the left than with the right one. About 1903 she found she became very tired on trying to walk any distance, and she was told her back was crooked. About a year ago she burned her right forearm by holding it too near the fire; she did not perceive the heat, but the result was an ulcerated surface, which has now healed, leaving a scar. In December, 1904, one of her fingers became very inflamed, and she was admitted into the Adelaide Hospital, under Mr. Gordon, who recognised the condition as syringomyelia, and had the case transferred to me.
Her present condition is as follows:—

1. Sensation.—Ordinary sensation is somewhat dulled on the right arm, but the deficiency is slight; elsewhere sensation is equal on both sides. Sensation of pain is very deficient all over the right side of head and body, right arm, and right thigh, extending in front down to the middle of the thigh, and behind to the gluteal fold. In the trunk and scalp the analgesia extends up to the middle line; in the face it falls short of the middle line by about 1½ inches. In the right arm and hand the analgesia is absolute; a pin can be stuck into her flesh without causing any pain. Elsewhere pain can be felt, but in a lesser degree than on the left side of the body. The mucous membranes seem to be sensitive to pain on both sides. The sensation of heat is diminished over the same area, but as the power of distinguishing heat from cold is nowhere lost, but only dulled, it is difficult to be exact as to the limits of the dulling of thermal sense. In the right arm, heat, though felt as heat, seems incapable of causing painful sensations. The distribution of the analgesia, therefore, is strictly confined to the right side, and involves the portion of the body which receives innervation from the brain and from the spinal cord down to the level of about the 3rd lumbar segment. The thermal insensibility occupies approximately the same area, but I am less sure about its limitations.

2. Trophic Disturbances.—She has had a number of whitlows affecting the fingers of the right hand, as a result of which the ungual phalanages of the ring and little fingers have been lost, while the other fingers are thickened by the inflammation which occurred. These whitlows were all devoid of pain. A month ago she had a sloughing sore on the right wrist, devoid of pain; it has now healed. The left hand has been free from sores.

The spine is curved to the right in the whole dorsal region, and the vertebrae are rotated so that the ribs project posteriorly on the right side.

3. Atrophy.—The arms show no atrophy; the muscles are not weakened on the affected side.

4. The Special Reflexes.—There is increased knee-jerk on the right side. There is no clonus, either ankle or rectal; but on the right side four or five jerks are felt when the attempt is made to get clonus, while on the left there are no such jerks.
Babinski's phenomenon is absent. In the arms there is a triceps jerk on the left, but none on the right, side.

5. The cervical sympathetic is not affected. The pupils are normal. The ophthalmoscopic appearances are normal.

6. The analgesia present in the right side of the head has been mentioned; otherwise the cranial nerves are unaffected.

The chief points of special interest in this case are—
(1) the exclusively unilateral distribution of the phenomena, and (2) their wide extent on the right side of the body. It is common for one side to be involved more than the other, but generally there is some degree of involvement of both sides. It is usual for the symptoms of syringomyelia to be confined to the arms and upper part of the trunk; in this case the analgesia exists over the right half of the head and body, which receives its nerve supply from the brain and spinal cord as low as the 3rd lumbar segment.

Dr. C. M. O'Brien showed five cases of Rodent Ulcer cured by X-rays and Radium.

Dr. R. Travers Smith read a paper entitled "Brief Notes on some Miscellaneous Clinical Experiences" (see page 42, ante).

Dr. Walter G. Smith read a paper entitled "Some Colour Tests for the Chief Nitrogenous Constituents of Urine" (see page 50, ante).

---

Friday, May 19, 1905.

Dr. James Little in the Chair.

Dr. F. J. Dunne exhibited a case of Tuberculosis of Lungs and of Knee treated by Professor Deny's Tuberculin, and read the following remarks:

The patient, a girl, aged thirteen years, was admitted to the Royal City of Dublin Hospital, November 2, 1903, suffering from a tubercular abscess of the front of the knee, probably starting in the prepatellar bursa; it was opened and scraped by Mr. Jameson Johnston.

In January, 1904, when the patient came under the care of Dr. Stoney, there were two long subcutaneous sinuses on the front and inside of the knee; the joint was not involved. The
Abstracts.

whole area was dissected out and healed up, and she was dis-
charged well on January 21, 1904. About a month after leaving
hospital she knocked her knee against the side of the bed, and
it broke out again. She attended the dispensary to have her
knee dressed frequently. In the beginning of August last she
came back to the dispensary as the site of operation had broken
down, and there was a large ulcerated area. She was again
admitted to the Royal City of Dublin Hospital on August 23.
There was great pain on moving the knee, and Dr. Stoney was
of opinion that the lower end of the femur was diseased (dead
bone being felt at the bottom of the wound), and also the joint.
Excision or amputation was discussed, but as she had a very bad
cough and the left lung was found to be solid, nearly from apex
to base, with tubular breathing and crepitation, and the right
lung crepitating for a hand’s breadth below the clavicle, and
as tubercle bacilli were found in her sputum, it was decided that
the disease was too far advanced for operation. She was sent
to the South Dublin Union on September 9, 1904.

On admission to the Children’s Infirmary, South Dublin Union,
there was a large deep ulcer on the anterior and inner surface of
the knee, with considerable swelling and inflammation, both above
and below the joint, and free discharge. The knee was very
painful even when at rest. Temperature, 101°; pulse, 100;
respirations, 30. Patient was coughing a good deal and sweating
profusely at night. The body was thin and wasted. The right
lung was slightly dull on percussion, and crepitating freely for
about three finger breadths below the clavicle. Left lung was
dull on percussion down to 4th rib in front, and about two inches
below inferior angle of scapula behind, and over this area there
were crepitations, tubular breathing, and moist râles. There was
copious expectoration, and it was loaded with tubercle bacilli
and cocci. The knee was poulticed for a few days, and then
treated throughout with iodoform emulsion. The discharge
lessened, and inflammation subsided, but there was very little
attempt at healing made until after the injections were com-
menced. The temperature was markedly hectic in type for
the first four weeks, ranging from 99° in the morning to 101° at
night, and several times touching 102°. After this there was
generally 1° difference between morning and evening. About the
middle of October a slight redness and swelling appeared above the
Section of Medicine.

ankle, and early in November a small patch of ulceration at inner ankle and heel made its appearance, while the whole leg, from knee to ankle, became red and swollen. When the little ulcer commenced to discharge the redness and swelling subsided to some extent.

Present condition of patient:—Neither cough, expectoration, nor night sweats. The right lung has quite cleared, is resonant, and the breath sounds are normal. The left lung is slightly dull on percussion, giving about the same note as a thickened pleura. Breath sounds are good all over, except a small area close to inferior angle of scapula, where the dulness is more marked, and slight tubular breathing and faint crepitation may still be heard. The knee presents a healthy scar, being adherent to the bone for a space of about 1 ½ inches. Movement is perfect, and the patient can run and walk without any trouble.

I commenced the tuberculin injections on November 30. My first injections were made at intervals of four days, the temperature being taken five times daily. The first four injections were practically without visible result, except to steady the temperature, which now frequently touched or went below normal. The cough and night sweats both became less frequent. For nineteen injections the temperature was normal at some period of the twenty-four hours, with the exception of two days, though very frequently the variation in the twenty-four hours was as much as 2 degrees. From the 18th of February to the 23rd of March—the 20th to 25th injections—the temperature rose above normal only on seven days. From March 29th to April 4th I gave the 26th and 27th injections, both of which were followed by a rise to 100°, lasting for only a few hours. Since the 6th of April there has never been a rise of temperature beyond 98°, though I have given one cc. of No. III., which is pure undiluted tuberculin.

The small ulcer which I have mentioned at the inner ankle healed up rapidly, and was quite well by the end of December. The knee healed very rapidly up to about the same time, when there was a small patch, about the size of a sixpence, which kept oozing until the end of January. The girl was kept in bed as little as possible, and when the days were fine I had her carried out to the open air, though she felt the cold pretty acutely at first.

As regards treatment—She has been on an emulsion of cod-liver
Abstracts.

oil and syrup of calcium lactophosphate from the start, and ordinary cough mixtures and atropin and zinc pills up to the time I commenced the injections. Since the 1st of January she has had no medical treatment except the emulsion. The weight at the end of November was 4 stones 8 pounds, and it has gradually increased to 5 stones 11 pounds on May 15th. The sputum in November was crowded with tubercle bacilli and cocci in chains and pairs; in December, few bacilli, numerous cocci; in January, bacilli much more numerous, few cocci; in February, very few bacilli, and since then no tubercle bacilli have been found in repeated examinations.

Effect of 33 tuberculin injections on temperature in case under discussion:—On 11 occasions there was a rise in temperature within 48 hours, and usually within 24 hours; on six occasions 1°, and on five occasions 2°. On 4 occasions there was a fall in temperature—once 1°, twice 2°, and once 3°—within 24 hours. On 18 occasions there was no appreciable change of temperature.

I have given altogether 333 injections in various tubercular cases, and after 72 of these there was a rise in temperature within 48 hours, once 4°, five times 3°, eight times 2°, and 56 times of 1°.

There was a fall of temperature after 50 of the injections, four times of 3°, six times 2°, and 40 times of 1°. After the remaining 211 injections there was a rise or fall of temperature of less than 1°.

I have made all my injections into the muscles of the back, a little below and between the scapulae. In no single instance has a skin abscess or sore of any description arisen. The only local effect was some slight redness and thickening at the site of puncture.

Tuberculin is a liquid obtained by filtering through porcelain a culture of the bacillus of Koch developed in broth (bouillon de viande), with the addition of glycerine and peptone after the usual proportions.

It is an absolutely limpid liquid, deprived of all living germs, but charged with the products of the secretion of the microbe.
It exists in the following strengths:—

T. III. tuberculin, pure, non-diluted.
T. II. " " diluted $\frac{1}{10}$.
T. I. " " " $\frac{1}{100}$.
T. O. " " " $\frac{1}{1000}$.
T. O. $\frac{1}{100}$ " " $\frac{1}{10000}$.
T. O. $\frac{1}{1000}$ " " $\frac{1}{100000}$.
T. O. $\frac{1}{10000}$ " " $\frac{1}{1000000}$.

This graduation in strength enables the operator to very gradually increase the dose according to the amount of reaction exhibited by the patient.

I feel that this record of an interesting case, and of a means of treating tuberculosis new to medical science in Ireland, would be incomplete if I failed to express my gratitude to Professor J. Denys, of Louvain, who not only placed his tuberculin at my disposal, but also has at all times most promptly replied to my questions, explained my difficulties, and given me his advice about my various cases.

I am also indebted to Dr. R. Atkinson Stoney for his notes on this case.

DR. T. GILLMAN MOORHEAD read a paper on the Bacillus Coli Communis as a Cause of Septicæmia (see page 54, ante.)
SECTION OF SURGERY.

INEFFICIENT OPERATIONS FOR GASTRIC ULCER.

By A. B. MITCHELL, M.B., F.R.C.S.;
Surgeon, Royal Victoria Hospital, Belfast.

[Read in the Section of Surgery, November 4, 1904.]

The past four or five years have witnessed a very striking change in the attitude of the profession with regard to the treatment of gastric ulcer. Physicians and surgeons are alike agreed that these cases are essentially medical in the first instance, but very few medical men would now allow a case of gastric ulcer to suffer year after year as a chronic invalid without at least raising the question of surgical interference. It would, therefore, appear highly desirable that we, as surgeons, should endeavour to arrive at some definite conclusion as to the nature of the operation likely to result in permanent relief. This can, I think, only be effected by a faithful record of those operations which have failed in their object.

I know no disappointment more bitter than to have a patient come back some months after a serious operation and volunteer the statement that relief was only temporary, and that the discomfort is as bad as ever.

This is a very serious matter for the patient, the family physician, and the surgeon. The physician who, after consultation with us, has taken the responsibility
of recommending his patient to submit to operation is very naturally aggrieved at the ultimate result, and is apt to be somewhat distrustful of our prognosis on future occasions.

In May, 1901, I read a brief paper before this Section, in which I spoke enthusiastically on the subject of operation for gastric ulcer, and in November, 1902, I brought a series of eighteen cases before the notice of the Ulster Medical Society. My object to-night is to give an honest record of my failures, in the hope that other surgeons may obtain guidance from my experience. I may here note that the word failure does not refer to mortality. In all the cases I have here to relate the patients recovered from the operation and obtained temporary relief, but, unfortunately, it was only temporary.

Excision of Ulcer. Relapse after one year.

Case I.—Miss S., aged twenty-four years, has suffered from seven to eight years from very severe pain after food. She had been treated by most skilful physicians. On two different occasions she had submitted to eight weeks’ rest in bed, rigid dieting, and even rectal feeding, but without avail. On 1st February, 1902, I operated in a Nursing Home in presence of Sir Wm. Whitla. A small ulcer, about the size of a sixpence, was found on the anterior surface of the stomach, and was freely excised. No other ulcer could be found. She made a good recovery. Two months later she wrote that she was very well, and could take ordinary food with comfort. In January, 1903, she reported herself as being in good health, but the following April she came to me complaining of a relapse of her old symptoms. She saw me two or three times, and got some relief from diet and drugs, but her condition was not satisfactory.

This case I had published as a success.¹ A record of its failure seems to be of importance in view of the fact that many eminent surgeons have recommended excision as the operation of choice where it can be easily carried
out—e.g., Mr. Mansell Moullin in an interesting article in the *B. M. J.*, October 17th, 1903, says:

"The ideal mode of treatment is by excision." . . . The scar left is a simple linear one, not likely to contract or lead to the development of adhesions."

He then refers to a series of eight cases, all of which he reports did well. The subsequent history of these cases would be interesting. As the result of my own experience, and for the reasons which I shall shortly submit, I no longer regard excision as an efficient operation.


Case II.—Miss M., aged thirty-two years, had suffered from typical symptoms of gastric ulcer for about twelve years. She had been submitted to the most careful and thorough medical treatment, but without relief.

In October, 1902, she entered a Nursing Home, and on operation I found a large ulcer on the anterior surface extending across lesser curvature to the posterior surface, with numerous adhesions. This ulcer was simply infolded by a long line of continuous suture, which extended about four-fifths round the stomach, thus securing complete rest to the ulcerated area, as suggested by me in my paper before this Section in 1901. The immediate result was all that could be desired—within ten days she could eat all kinds of food with perfect comfort. She remained in excellent health for sixteen months, with the exception of chronic constipation, to which she had always been a victim, and which was not in any way affected by the operation. Her former symptoms then began to come back, and she gradually got worse, till she was as bad as ever. She told me she had boasted so much about her cure after the operation that she was ashamed to let anyone know how bad she was. Medical treatment was again tried and failed. She herself asked whether nothing more could be done. I suggested gastro-jejunostomy; she at once consented, and again entered the Nursing Home on July 7, 1904, one year and ten months after the first operation.
Inefficient Operations for Gastric Ulcer.

I did a posterior gastro-jejunostomy, at the same time producing an artificial narrowing of the pylorus by the insertion of a few Lembert sutures. She made a rapid recovery, and left the Home within three weeks feeling quite well. She now reports herself in perfect health, stating that her digestion is normal, and she has no further need for aperient medicine.

I have no doubt the result will be permanent. At this operation a circular scar could be seen surrounding the stomach where the line of continuous sutures had been inserted, and a few slight adhesions were present. When the stomach was opened the portion originally infolded, consisting of indurated cicatricial tissue, could not be felt by the finger introduced into the cavity. The mucous membrane was quite smooth and velvety on the side of the operation. A fresh ulcer had, however, formed higher up on the posterior surface.

This case was also published by me as a success.¹ I have here to withdraw my former recommendation, and acknowledge that "simple infolding of the ulcer" is an inefficient operation.


Case III.—Miss M·D., aged forty-one years, had suffered from the stomach for twenty-six years. In March, 1902, I operated and found a well-marked hour-glass stomach. Gastro-gastrostomy was performed. Relief was immediate and apparently complete. For more than a year she enjoyed excellent health, then she began to complain of the old pain, and her condition gradually got worse. In April, 1903, she came to town to see me; I was from home. While in her sister's house she suddenly perforated. She was operated on, and the perforation sutured by Dr. John Campbell. She made a good recovery, but soon after getting up began to suffer from pain and vomiting. We tried rest and medicine, but without benefit, and when she ventured the remark that she simply could not
continue to live in her present state I, with considerable diffidence, put before her the possible benefits of gastro-jejunostomy. She decided to have this done. Accordingly, she entered a Nursing Home, and on 30th November, 1903, I performed the posterior operation. Very great difficulty was experienced owing to extensive adhesions—the anterior surface of the stomach being firmly fixed over a large area. She suffered for some time from regurgitant vomiting, but this eventually completely disappeared, and she is now well, having successfully braved her third and, I trust, last abdominal section in twenty months.


Case IV.—Miss M'K., aged forty-eight, was operated on for old-standing stomach trouble, characterised by persistent vomiting and pain, by Dr. John Tennant and myself in the Ulster Hospital, on 11th July, 1902. Pyloric obstruction, due to cicatricial contraction, with some silvery adhesions, was found to be the cause of her symptoms. The first portion of the duodenum was long, and came freely into the operation wound. A gastro-duodenostomy was performed. She made a rapid recovery, got complete relief from all discomfort, and left hospital one month after operation feeling in excellent health, and having gained 1 stone 5 lbs. in weight. Four months later she reported herself as being quite well, and her weight, which previous to operation was only 4 stones 1 lb., had increased to 6 stones 10 lbs., a total gain of 2 stones 9 lbs. This patient remained in good health for over eighteen months. She then came to me stating that the old pain and vomiting had returned. She was submitted to the most careful medical treatment, but steadily got worse. She vomited everything, and constipation was very troublesome. I finally recommended her to submit to another operation. On October 12th, 1904, I performed a posterior gastro-jejunostomy, and she is now able to take all sorts of food without discomfort, and, as usually happens after this operation, the constipation has been relieved.

The cause of relapse in this case was the formation of adhesions round the gastro-duodenal opening and a considerable degree of gastric ptosis, which prevented the
artificial opening acting as an efficient drain to the stomach. This case was recorded as a success, but here again I have to venture the opinion that the old operation of gastro-duodenostomy is not an efficient one. I do not, of course, include Finney’s method of gastro-duodenostomy, of which I have no personal experience.

The question naturally arises—Is there any satisfactory reason why these operations, which at first seemed to produce such brilliant results, should ultimately have to be condemned as failures? The answer is “Yes.”

In the first three cases no attempt was made to interfere with the action of the pyloric sphincter, which, continuing to act, prevented complete rest, and caused the retention in the stomach of certain irritating substances.

In the fourth case the artificial opening being made close to the original ulceration, and being limited in size by the site selected, became obstructed by surrounding adhesions. It would, therefore, appear that those surgeons are correct who take the view that the most efficient surgical procedure for the relief of symptoms dependent on gastric ulcer or its sequelae is to be found in a properly performed gastro-jejunostomy, or possibly in cases of ulceration at some distance from the pylorus. The modern method of gastro-duodenostomy suggested by Finney, which is really a modification of pyloroplasty, may be found satisfactory.

The advantages of such a procedure are that we secure:—

1. Prolonged rest to the stomach.
2. Efficient gastric drainage, thus getting rid of the evil effects of excess of acid and other irritative substances. It thus deals not only with the actual ulcer, but with what I may call the “ulcerative habit” which these patients have acquired.
3. We provide a cure for multiple ulcers, should more than one be present at the time of operation.

4. Chronic constipation, possibly one of the causes of the relapse of ulcers which have undergone cure by medical treatment or otherwise is almost invariably cured.

How this cure of constipation is effected may be open to question. I think, however, the two chief elements are:—

1. That fluids are no longer absorbed in large quantity from the stomach, but find their way freely into the intestine and act as an intestinal flush.

2. That the digestive process, which now takes place in the bowel instead of in the stomach, acts as an intestinal stimulant and aperient.

In future, therefore, it is my intention, when called on to operate for gastric ulcer, to go straight for a gastro-jejunostomy. Should the case be one of hour-glass stomach I shall first do a gastroplasty to relieve the constriction, to be immediately followed by Finney's gastro-duodenostomy or Hacher's posterior operation, should the patient's condition admit of the double operation.

It is not the object of this paper to discuss the merits of the various methods of gastro-jejunostomy. Suffice it to say that my own practice favours the posterior method, according to the modern plan of making the junction as close to the origin of the jejunum as can be conveniently done or as will admit of efficient drainage.

Gastro-jejunostomy, whilst it is the operation of election—an operation which Mr. Moynihan says "is suitable to every case, and always efficient"—is, in the experience of almost every operator, liable to be followed occasionally by very troublesome regurgitant vomiting. I am
aware that modern technique has greatly reduced this risk, but as it is a possible complication the best and most efficient method of dealing with it is a matter of importance. Here again I regret to have to record a most disappointing result.

Case V.—Mrs. D., aged thirty-eight, who had suffered from symptoms of gastric ulcer for eight years, was operated on by me on May 3rd, 1904. A posterior gastro-jejunostomy was done, but the jejunal loop was left about eight inches long. Regurgitant vomiting set in on the third day, and, though not very severe, persisted in spite of all treatment, including lavage. She declined further interference, and left hospital five weeks after operation, able to get about, but still vomiting at frequent intervals. She came to me at the end of September, four months after operation, looking very thin and weak, and stated that she was getting steadily worse. She had no pain, but severe nausea and vomiting occurring, sometimes daily; at other times at intervals of two or three days. She again went into hospital, where I opened the abdomen. Prof. Sinclair kindly assisted me, and gave me the benefit of his advice. It was decided to perform a lateral anastomosis between the ascending and descending loops of jejunum; this was done at a point about four inches from the junction with the stomach. This procedure presented no special difficulty; an artificial opening, two inches in length, was made, and the patient rallied well, but the operation did not afford the slightest relief to the vomiting, which continued till her death, ten days later. There was no trace of peritonitis or other complications, death being due to exhaustion.

I submit for your consideration the specimen removed from the patient, which shows both the anastomotic openings to be widely patent. The old gastric ulcer is nowhere to be seen.

Bearing on this subject, the following quotation from Cheyne and Burchard's "Surgical Treatment" is of interest: "It has been suggested that the Y operation should be employed for these cases, but this operation had better
be the original proceeding if it is to be performed at all. In the cases of which we are speaking it is more troublesome than the lateral anastomosis, and is not really more effectual.

This expression, coming from so high an authority, had great weight with me — my own inclination being in favour of the $Y$ operation.

About two weeks later I performed an anterior gastro-enterostomy on a young girl. Her stomach was extensively diseased, and so bound down by adhesions that a posterior junction could not be effected.

She was too weak to bear the $Y$ operation, so I had to content myself with fixing the jejunum to the stomach wall for about an inch on each side of the anastomosis. Regurgitant vomiting was very severe and persistent. On the 10th day I again operated. I divided the bowel immediately above the junction with the stomach, closed the distal end, making a blind cul de sac. The upper or duodenal end was then implanted into the descending loop of jejunum about three inches below the stomach. Relief was immediate and complete. She never vomited again, and made a rapid recovery.

This method is, therefore, in my opinion, preferable to the lateral anastomosis in such cases. If the junction can be successfully carried out it can hardly fail to be efficient.

Now, in submitting these few cases for the consideration of this Section I am quite aware that the failures I have related may have been due to some defect on the part of the operator, and we all know what a slight error can mar an abdominal operation, for here, indeed, it may be said our patient's life "hangs on a thread:" but I shall be fully satisfied if this record has the effect of
saving any other surgeon from the mortification which I have experienced.

1 Transactions Ulster Medical Society, 1902-3.

Sir Thomas Myles stated that his experience of gastro-jejunostomy for gastric ulcer was small, but he had performed the operation for other conditions, such as pyloric obstruction and malignant disease. He thought it would be a matter of considerable interest in cases where gastro-jejunostomy had been performed for the relief of gastric ulceration with hyperchlorhydria to ascertain the condition of digestion afterwards. For regurgitant vomiting he found relief was usually afforded by propping the patient up in bed. He had never found it necessary to perform a second operation after gastro-jejunostomy, and had operated in sixty-one cases without a death. In order to prevent a spur forming at the site of the gastro-intestinal junction he usually sutured the jejunum along nearly the entire length of the posterior wall of the stomach.

Mr. Mitchell, in reply, stated that experience had led him to prefer gastro-jejunostomy to other operations in cases of gastric ulcer. With regard to regurgitant vomiting, he had observed that when gastro-jejunostomy was done for pyloric obstruction there was never any trouble in this respect, and latterly, when performing gastro-jejunostomy he had been in the habit of puckering in the pylorus with three or four sutures, thus doing away with the action of the pyloric sphincter, and these cases were amongst the most satisfactory which he had had.
THE HEALING OF WOUNDS: ASEPTIC VERSUS ANTISEPTIC METHODS.

By WILLIAM S. HAUGHTON, M.D.;
Surgeon to Steevens' Hospital.

[Read in the Section of Surgery, November 4, 1904.]

The healing of wounds, whether inflicted accidentally or designed by the surgeon, is a subject of vital importance to us all, and one which engages the earnest attention of every member of the medical profession.

Every thinking surgeon must regard the name of Lister with feelings of unbounded admiration and respect, for to him is due the greatest discovery of the century, if greatness be measured by the amount of relief from pain, suffering, and death which he has conferred on mankind. And, further, by the enormous extension of possible and successful fields of surgery which he has opened up, with corresponding benefits to our patients. It is, therefore, with the greatest satisfaction that one seeks to realise more fully the first principles for success laid down by Lister by making a study of the habits of pathogenic germs, and how best they may be defeated.

For this reason it is necessary to examine the most recent methods advocated for the healing of wounds, and to compare them with older ones, with a view of adopting only those which the tests of practical experience prove to be the best.

With this object in view, during the past six years I have made a very careful comparison of the relative merits of various forms of antiseptics in the healing of wounds and disinfection of skin, and compared these
results with those which may be obtained by a more purely aseptic technique.

From the results of these investigations in my own surgical practice I am convinced there is a very considerable difference in the behaviour of wounds according to the technique employed in their treatment.

Considerable attention has been directed to wound treatment during the past eighteen months by British surgeons, and none too soon, seeing that our Continental brethren are leading the way in this as in many other matters, and so much confusion seems to exist in the use of the terms antiseptic and asepsis, and also in their various applications, whether separately or combined, that it becomes necessary to define what I mean by the terms in their more modern and restricted acceptation.

Of course all surgery is antiseptic in the sense that it takes preventive measures against blood-poisoning due to pathogenic germs. Further, all surgery aims at being aseptic when we operate through unbroken skin in a region believed to be not previously infected. But the methods adopted to attain these ends vary considerably.

Antiseptics, as generally understood, are chemical poisons introduced into a wound or applied to the skin or other things for the purpose of killing or rendering innocuous those pathogenic germs believed to be introduced into the wound, or which exist on the skin or elsewhere.

Aseptic technique, on the other hand, is generally interpreted as meaning, in the first place, the boiling or steaming of everything which can be so treated for sterilisation purposes, and, for the remainder, a scrupulous attention to mechanical cleanliness in its most exact and exacting sense.

There is really no antagonism in the principles underlying these two methods when properly used—namely, to
reinforce each other where circumstances require. But there is considerable antagonism between the so-called "Antiseptic" and "Aseptic Schools" at the present moment. This arises from the different value which each "school" sets on the power of chemical poisons, as used in surgery, and of simple mechanical cleanliness, respectively.

In my opinion the distinguishing features of the aseptic surgeon are:

1. His appreciation of the meaning of real cleanliness in the surroundings of an operation;

2. His capacity for thorough cleansing of himself and his operation environment, recognising that the weakest link in the chain of asepsis is the skin, which requires something more than simple cleanliness, and tolerates something less than immersion in strong antiseptic lotions of standard bactericidal strength for the prescribed germicidal periods;

3. (This is, perhaps, the most important, and certainly is the most difficult.) His ability to maintain unbroken the aseptic chain by never allowing himself to touch "dirt," even under the trying strain of mental and physical exertion, during a prolonged operation; and

4. His "abstinence" (to use Professor Kocher's own word) from touching sepsis during the intervals between operations.

One would expect from purely theoretical considerations that the average results of these two methods would differ somewhat widely; and this is fully borne out by the practical experience of those who have given them a fair trial.

In my own experience a purely antiseptic treatment of operation wounds is usually followed by some pain, some inflammation, and a tendency to slow healing. There is
usually no rise of temperature, but there are occasional stitch abscesses.

But purely aseptic technique is generally followed by no pain, no inflammation, more rapid and more firm healing with a marked diminution of fibrous tissue in the scar, and no rise of temperature, while the occurrence of stitch abscesses reaches a vanishing point.

Of course it is folly to imagine there is only one way of doing anything. There may even be several good ways, suited to the individual peculiarities and habits of each surgeon. But I maintain it is our duty to seek for and abscesses reaches a vanishing point.

Many, perhaps most, careful surgeons employ a combination of these two methods, and obtain excellent results. But how far this success is due to the merits of one or other system can never be determined exactly, in the physical sense, so long as they are confused by combination.

It will be my endeavour to show that as one comes to rely less on the help of antiseptic poisons, and more on ordinary mechanical cleanliness in its exact and laborious sense, the more gratifying will be the results to patient and surgeon.

Now let us consider the channels through which infection of an operation wound may take place, and which indeed have been proved, both bacteriologically and clinically, to be sources of real danger.

There is no finer classification of this part of my subject than Professor Kocher's—that great master of aseptic technique—repeated visits to whose Klinik have taught me the value of thoroughness in every detail pertaining to operative work. His classification is:

Infection of a wound with pathogenic germs may take place from—(1) air; (2) contact; (3) inoculation;
implantation; (5) necrosis ("wound necrosis"); (6) incubation.

1. Air-borne Germs come from dust: dried material from mouth, nose, and clothes of everyone around operation, including patient himself.

These are obviously diminished by ordinary cleanliness of the parts mentioned, especially the nose and mouth of patient—a matter frequently overlooked. The operator and assistant should also be very particular in their own cases, and avoid all unnecessary talking over a wound. The amount of infective material projected from a mouth containing a single carious tooth during ordinary conversation is astonishing, and capable of infecting a sterile nutrient plate several yards away from the speaker (Flügge).

Spectators should, as much as possible, be covered in sterilised, or at least clean, overalls.

The theatre walls, ceiling, and floor should be as smooth as possible, the walls and ceiling preferably white, to show up dirt and reflect light. Ordinary white enamel paint probably yields the best surface, with the exception of glass.

There should be a very liberal proportion of window space, a large top light, one-third or one-half of ceiling area, fitted with outside blinds to stop direct sunshine when necessary. Windows also should be on two or three sides of room. This arrangement avoids shadows when stooping over wound or assistants stand around.

In our theatre in Steevens' Hospital we have the entire place hosed down regularly, and, while the walls are still wet, formalin tabloids are burnt, with the object of getting the vapour into solution on the walls.

It is needless to add that the furniture of the operating room should be as simple as possible—with every part
accessible for cleaning—and that there should be no "dust traps" in the shape of projections from the walls or hanging arrangements from the ceiling.

It is, further, a great advantage to keep the floor of the theatre wet all through an operation, as this prevents dust from one very undesirable source from rising at all. Masks for the face and caps are worthy of consideration.

Face masks undoubtedly absorb many germs projected from the mouth and nose of the operator and assistants over the wound. But if not changed occasionally may, like cotton gloves, become a source of danger.

Caps are also undoubtedly good—preventing germs from the head, especially when your assistant knocks his head against yours, or drops of sweat from falling into wound. In all prolonged operations it is my practice to wear a small towel twisted into the shape of a cap and secured by a forceps.

2. Contact Infection from germs introduced by anything which may touch the wound, such as instruments, dressings, lotions, parts of the patient's or surgeon's body, or objects which may be touched by any of these things—i.e., the operating and instrument tables, bowls, and trays—immediately around the operation.

Instruments, dressings, and lotions are perfectly sterilised by boiling for 20 minutes—the instruments being boiled in 1 per cent. soda solution to prevent rusting. Or the instruments and dressings may be perfectly sterilised by circulating high-pressure steam for 15 minutes at 125° C. (Tavel).

The surgeon, assistants, and nurses should wear a sterilised overall, reaching from neck to feet, with long sleeves buttoning round wrist.

My friend Major Holt, of the R. A. M. C., has suggested an extra sleeve, fastening with tape above the elbow, and
buttoning round wrist, which I have adopted, as it can be readily replaced by another if accidentally soiled. This covers the forearm completely—a part seldom effectively disinfected, and one which frequently comes in contact with the wound in making deep manual exploration during a laparotomy.

The patient's body and operating table should be entirely covered with sterilised sheets (two or three layers thick), arranged so as to leave only the smallest necessary aperture through which the operation may be performed with ease. These sheets are held in position by clip forceps, and a smaller sheet, with a hole corresponding to operation wound, is further applied over all.

I have used these Kocher's sheets for more than four years, and the comfort of them is enormous, preventing contact infection, and permitting wipes and instruments to be laid down at will anywhere, on patient or table, with safety. They save a great deal of mental anxiety in trying to remember the right place to lay down each instrument. They are vastly superior to towels, so generally used, which are too small to be efficient, and, further, slip about and open up chinks, which expose unsterilised areas.

All the instrument tables around operation should be similarly covered with sterilised sheets, three to four ply thick, and all bowls, trays, bottles, and nail brushes thereon boiled for twenty minutes beforehand and placed there with long sterilised tongs.

The instruments are best used dry, and sterilised in trays with perforated bottoms, which trays are lifted bodily out of boiler with special sterilised lifter, and placed, still containing the instruments, on these sterilised table-cloths. Thus they are never handled till the surgeon uses them. When lotions are necessary the best to use
The Healing of Wounds.

is sterilised normal saline at body temperature. This is specially the case in laparotomies. The anaesthetic is best administered under a sort of tent, made by prolonging a sterilised sheet over patient's face and pinned to anaesthetist's shoulder. Other advantages of this manoeuvre will occur to the thoughtful surgeon!

When the operation area approaches mouth and nose of patient, as in neck and chest operations, it is best to use a wire frame, over which is stretched a sterilised sheet (which I will demonstrate later by lantern slides).

The question of operation nurses is a very serious one. The theatre sister should be permanent as far as possible, and have under her at least one other nurse whose duties lie solely in the theatre.

For success it is absolutely necessary to define and separate the duties of these two nurses—one of whom is called the aseptic nurse and the other the septic nurse.

The aseptic nurse prepares her hands exactly like the surgeon and assistants, and handles only those objects in the sterilised area, which may be termed the inner circle—i.e., instruments, bowls, lotions, ligatures, &c. She alone may hand or clean these things.

The duties of the septic nurse lie in the outer circle, and include the handling of everything outside the sterilised area which may be required. She boils any fresh instrument wanted, or any one in use which may become soiled, and returns it to the sterilised area with a long sterilised tongs. The same applies to fresh supplies of wipes or lotion.

The drill of this system very soon becomes efficient, as the duties of the nurses are distinct, and not allowed to overlap.

3. **Inoculation infection**, which is conveyed to the wound by germs on or in the skin. It has two sources—
A. skin of patient; B. skin of hands of surgeon, assistants, and nurses.

This focuses our attention on the weakest link of the aseptic chain—namely, disinfection of the skin.

The subject is full of interest and worthy of exhaustive experimental study. It is impossible, in the scope of my paper to-night, to detail my own experiments, or those of other workers, which I hope to do on another occasion. I can merely outline the results of my investigations, which corroborated those of other workers.

Sterilisation of the skin is impossible, according to the most accurate and consistent observers. It may be temporarily disinfected just before operation, but its surface does not very long remain so. Whether this be due to the sweat floating up germs from deeper layers, or that the epidermal scales become macerated from contact with blood and lotions, and so more easily detached with the germs they contain, matters little. The real question is what method will make skin safest and preserve it so for the longest time?

Kocher, Ahlfeld, Mikulicz, Fürbringer, Leedham-Green, and others, agree that soap and hot water, followed by alcohol, furnish the best results.

Mikulicz combines the method and shortens the time by a spirit soap.

Fürbringer uses the hot-water-alcohol method, and adds an aqueous solution of sublimate.

Leedham-Green, after very exhaustive work, is satisfied that the hot-water-alcohol method is best of all, after trying every kind of antiseptic lotion usually advocated.

Kocher and Ahlfeld are agreed also on this point, as the method yields the most consistent results.

My own experiments from time to time corroborate these views, and the most recent of them, for which I am deeply
indebted to Dr. Earl, and for the great assistance and advice he has given me in the matter, prove, so far as we have gone, that the alcohol method is equal to, if not better than, antiseptic methods. Experiments, however, must be conducted in a larger series before speaking conclusively on the matter.

The method of preparing the hands is as follows:—

After cleaning and trimming the nails very short, the skin is scrubbed thoroughly for 15 to 20 minutes in a stream of very hot water, with plain soap and a nail brush, all over hands and forearms. The nail brush must be boiled for 20 minutes before and after use, and stored in a boiled tray, containing 1-1000 sublimate solution. All soap is rinsed off in a stream of water, and, without drying, the hands are carefully rubbed all over with dilute alcohol (Kocher approves 85 per cent., Leedham-Green 70 per cent. alcohol) from 2 to 5 minutes.

The advantages of this method are:—

(a.) Prolonged washing removes an enormous amount of dirt containing germs.

(b.) The dilute alcohol (70 to 85 per cent.) dries and hardens the epidermal scales, and thus prevents the detachment of germs for a considerable time. This hardening effect of alcohol locking up the germs was first pointed out to me by the present Master of the Rotunda, Dr. Tweedy, while my colleague at Steevens' Hospital, to whom I am also much indebted for many suggestions and much assistance in working out the details of the aseptic method.

Kocher, Ahlfeld, and most workers are agreed on this point, and do not attribute the success of this method to any germicidal property of the alcohol (though dilute alcohol undoubtedly possesses this property also). The
most rigorous antiseptic methods approach, but do not surpass, the hot-water-alcohol method of Ahlfeld.

Dr. Earl’s experiments in my own technique confirm this view, as tested on three different pairs of hands prepared by different methods. We got complete disinfection with soap, water, and sublimate (1-1000), and also with soap, water, and alcohol. But in one test with sublimate (1-1000) on my own hands a non-pathogenic spore-forming bacillus was isolated under the nails. Plain soap and water, for 20 minutes, in another test, not followed by alcohol or any antiseptic, produced Staphylococcus pyogenes albus from the hands, and a non-pathogenic spore-bacillus from nails.

Dr. Earl’s further tests of my technique proved boiled rubber gloves, saline lotion, catgut and silk ligatures and dressings to be absolutely sterilised, also the inside of patient’s wound and skin margin. But in the case of patient’s skin—a knee case—a non-pathogenic coccus was isolated. This patient’s skin was prepared by an elaborate antiseptic method.

Grouping the results of all careful workers it seems established that certain sterilisation of skin is not possible, and when obtained is not permanent.

Further, the use of strong antiseptic lotions on hands engaged in daily operations sooner or later will crack and fissure the epidermis. This produces recesses in the skin, which harbour dirt and germs, and render its disinfection still more difficult.

These considerations seem to me a very strong argument for the use of a non-permeable, non-punctured, sterilisable glove—which conditions are all satisfied by rubber gloves.

Sterilised cotton gloves were first advocated, and are still used by many leading surgeons, whose excellent
results justify the means, notably Kocher. But they appear only to be justified where purely aseptic cases are handled, and where "abstinence" from touching sepsis is rigorously practised between operations.

Further, they must be changed frequently, as cultures of *Staphylococcus pyogenes albus* and other germs frequently appear in gloves worn more than half an hour. The personal factor of each surgeon still further affects this point, the rate of sweating being very important.

Rubber gloves, on the other hand, need never be changed, even during the longest operation, if not punctured or torn—which accidents become much less frequent with practice; and air infection of the gloves is met by frequent washing in sublimate (1:1000), followed by normal saline, to prevent access of any antiseptic to the wound.

The objections to rubber gloves are:—1. Loss of tactile sense; 2. danger of puncture; 3. expense. Practice will soon convince anyone that the loss of tactile sense is more imaginary than real, and in the two and a half years I have used them I have only once or twice, in early days, felt inclined to remove them for this reason. Their puncture by a needle or instrument is always felt at once. The instrument or needle is at once discarded for re-sterilisation, the puncture treated with strong sublimate, and a fresh rubber finger-stall applied, or an entirely new glove substituted.

Their expense in private work is trifling compared with results obtained. In hospital work care saves many accidents, and small holes can be very well mended after the method of a puncture in a bicycle tyre. Altogether, they satisfy almost ideal conditions, as regards sterilisation and protection of patient and surgeon mutually. They must be used absolutely wet, or quite dry. In the wet
method they are sterilised by boiling for 5 to 20 minutes. In the dry by superheated steam, like dressings. This latter method I have frequently used and find it perfectly satisfactory.

It seems to me worthy of attention as to whether we may prolong the period of safe disinfection of the skin by using dry gloves and avoiding maceration of epidermis following on the soap, hot-water-alcohol method of disinfection, which locks up germs from free dissipation.

Professor Kocher suggests that the highest virtue in a surgeon is "abstinence" from touching sepsis between operations. It is good to disinfect hands which have touched sepsis; it is better never to touch sepsis at all.

This requires constant watchfulness on the part of the surgeon, and the condition can be very fully realised by using rubber gloves in all septic dressings or examination of septic and mucous surfaces.

Kocher remarks on this point:—"It is certain that he who cannot conform to "abstinence" as above defined, can never know the ideal meaning of a purely aseptic treatment of wounds. He remains condemned to the use of antiseptics."

Apart from the protection to the patient provided by rubber gloves, it is, perhaps, needless to remind you of the personal immunity from sepsis which they materially increase for the surgeon.

In one very faulty point of technique rubber gloves score off all other methods—namely, the tying of ligatures towards the middle or end of an operation—more especially stout ligatures. Here very considerable force is used, pressing the ligature, usually silk, deeply into skin of operator's fingers, and obtaining while there a full dose of whatever germs his skin is capable of providing, which
are further implanted in wound. This is believed to be the true source of the trouble which has led to many unfair attacks made on silk as a suture material. Rubber gloves of course furnish absolute protection from this source.

The skin of the patient is quite another matter, and cannot be covered with rubber gloves, as its exposure is necessary. We should, however, cover it up as much as possible, and, further, prepare it very carefully, exactly like the skin of operator’s hands, only this should be done twelve hours before operation and again just before. In the interval it is best covered with an impermeable sterilised dressing to maintain the first cleansing from air and contact infection. The question of leaving an antiseptic like carbolic, 1-40 or weaker, on the skin overnight is one which requires qualification. If the skin of patient is rough, and obviously difficult to clean, we had better use strong antiseptics in cleansing, and leave one on for twelve hours to assist the process. But this method always exposes the patient to the risks of a chemical dermatitis, which nearly always becomes septic by infection from skin-germs. And many times local sepsis of the wound arises from this source.

Pimples, acne spots, sinuses, or ulcers near an operation wound enormously increase the risks of sepsis, and require very special treatment, the best being the application of the thermo-cautery.

4. Implantation Infection. This arises from germs included in the ligature or suture material, implanted permanently or temporarily in the wound. Its avoidance is very simple if the following technique be adopted, always using rubber gloves:—

(a.) Use very fine silk, No. 1 or No. 2. Fine silk is more easily sterilised by boiling, and permeated better by
By Mr. W. S. Haughton.

antiseptics. It also leaves less foreign bodies in wound. If required strong, simply multiply the strands to required strength. I have more than a dozen times ligatured the femoral and axillary arteries with two to four strands of No. 1 silk without trouble.

Kocher's preparation is:—1. 12 hours in ether; 2. 12 hours in alcohol; 3. boil 10 minutes in 1-1000 sublimate; 4. wind on reels with clean or gloved hands; 5. boil for 10 minutes again in 1-1000 sublimate just before operation; 6. use direct from sublimate.

I have used this silk exclusively for nearly three years with perfect results. Sometimes, in very extensive operations, such as Halsted's full operation for breast cancer, I have buried 150 ligatures of this silk and have never seen any trouble arise.

The boiling on two occasions insures complete sterilisation, which is maintained by the sublimate. Further, the inclusion of a small quantity of active germicide effectively deals with germs accidentally lodged in the silk, which otherwise would use it as a modus for development before attacking the wound.

Silk is only used in aseptic cases, (b.) catgut in septic or aseptic cases. For four years I have used the formalin method of preparation devised by Dr. Roy Dobbin while House Surgeon at Steevens' Hospital. I have tested it both bacteriologically and clinically in some hundreds of operations, and have never had any trouble from this source.

The tested strands of No. 0 gut are fixed in 4 per cent. formalin, washed and boiled for 5 to 10 minutes, then stored in glycerine and alcoholic biniodide of mercury (1-1000). For tensile strength and pliability I find it the best of all methods, and it is always perfectly sterile.

(c.) For skin suture I use worm gut only, as it is com-
pletely sterilised by boiling, and cannot in single threads introduce germs from skin by capillarity.

5. Necrosis Infection.—Under this heading Kocher includes mechanical, thermal, and chemical injury of tissues due to faulty technique, which reduce the vitality of the tissues, and render them an easier prey to pathogenic germs which may be introduced.

Mechanical injury of a wound is due to splitting, tearing, and crushing of tissues. A sharp scalpel and fine-pointed haemostatic forceps of Kocher's own pattern obviate much of this.

Thermal injury is due to having air in theatre too cold, or scalding tissues by too hot lotions. The first is avoided by operating at a temperature of 70° to 80° F. The second by using a thermometer in the "normal saline" lotion.

Drying of the wound (Walthard) is also injurious, and may be avoided by covering with compresses wet with normal saline lotion—specially important in laparotomies.

Chemical injury is undesirable and preventible. It causes tissue-necrosis, which favours infective necrosis in the presence of germs.

In other words, antiseptic poisons should never be introduced into a wound at all, as they do more harm than good by lowering vitality of tissue, thus reducing its resisting power to germs, and that, too, in a more rapid ratio than the vitality of the germ is reduced by the germicide under "operation conditions."

I have frequently proved the same in accidental and septic wounds, which, as a rule, heal much more quickly when cleansed with normal saline. I find from other quarters this experience is not singular.

6. Incubation Infection.—This is due to providing any germs introduced with a nutrient medium accumulated in
the wound, such as blood or serum, which forms a sort of foreign body in the wound, in which there is no healthy circulation, and which keeps the two surfaces of the wound from accurate contact, and consequently delays union.

This is avoided by accurate hæmostasis, accurate approximation of wound surfaces, and, when this is impossible, by the use of glass drainage tubes and secondary sutures.

Accurate hæmostasis can only be obtained by the application of a pressure forceps to every bleeding point, picking up a vessel alone and not lumps of tissue. This is best accomplished by Kocher's own pressure forceps, now familiar to everyone. It is advantageous to postpone ligature to end of operation, as then many vessels will be stopped by simple pressure or torsion. For this purpose a considerable supply of these forceps is desirable. I personally never feel comfortable now starting a big operation with large section of muscle surface, such as Halsted's excision of breast, with less than fifty to seventy of these forceps, and I frequently have them all applied on two or three distinct occasions in such an operation, thus greatly reducing shock from hæmorrhage and seeing one's way much better, besides subsequent advantages of checking collection of blood and serum.

Professor Kocher starts work every day with a tray containing one hundred to two hundred of the forceps available. Halsted, I am told, occasionally uses a still larger number.

Finally, accurate apposition of wound surfaces is absolutely essential for success, and can only be acquired by long practice. When this is impossible it is best to use glass drainage tubes and secondary sutures. The wound is best dressed in a typical case with xeroform gauze strips.
The Healing of Wounds.

covered by sterilised butter muslin and collodion, into which is rubbed sterilised subnitrate of bismuth while drying. This forms an impermeable and waterproof scab, or wound splint, under which primary union proceeds most satisfactorily. Further, this dressing, especially in movable skin, like the abdominal wall, provides a sort of splint which affords great support and fixation, so necessary for repair of a wounded tissue.

Such are the details of the technique which I have gradually pieced together during the last five or six years, and which I have practised in their entirety as far as possible during the past three years with increasingly satisfactory results. The method is based on my personal observations during repeated visits to Kocher's Klinik, where the aseptic system is more thoroughly carried out than in any other place with which I am familiar.

In my earlier days of antiseptic zeal I obtained good results—to me surprisingly good results—but they were expensive, both in the bill for drugs and epidermis of hands, which suffered a good deal in the struggle. There was a certain set of characters about these wounds which I recognise the meaning of more clearly now. In my practice of this antiseptic method, primary union (so-called) was the rule, and there was no fever, and little, if any, pain. But sometimes the skin edges of the wound and suture holes were red and a little swollen, and if sutures were removed under seven to ten days there was a tendency towards delayed union. Stitch abscesses were very rare, but did occur. No serious trouble, however, followed these conditions in any of my cases.

When I adopted more purely aseptic technique the above-mentioned characters disappeared. Primary union without fever was still the rule. But an entire absence of pain, even in the most extensive operations, became the
rule also. Redness became still more rare, as did also stitch abscess.

But, apart from the disappearance of pain, what struck me as most remarkable was the rapidity of union, its firmness, and the small amount of fibrous tissue developed in a scar. It came more to resemble what has been described as "primary adhesion" of tissue to like tissue, and forms the ideal method of healing wounds according to our present knowledge.

It was with feelings of doubt I began to apply aseptic technique to accidental and septic wounds, but my experience now in the accident room and other septic cases convinces me that even such wounds will heal more rapidly with douching of warm normal saline, provided real mechanical cleanliness is insisted on in and around the wound. This experience is corroborated by other workers who have given the method a fair trial amongst septic cases, of which I could recite many instances which have come under my notice here and abroad.

Many papers have appeared on the subject of my communication to-night, and much discussion has taken place thereon lately in England, and it is interesting to read the difficulties which have been raised. Mr. Watson Cheyne draws a harrowing picture of the state to which the surgical nurse is reduced while trying to do two things at the same time. All his objections to the aseptic method can be at once simplified and dismissed by making two nurses do the work, and defining their separate duties as I have described.

In conclusion, I must record here my sincere thanks to the resident and nursing staffs of my hospital for their loyalty and diligence in carrying out all the details of this system, any success which may have attended it being chiefly dependent on their intelligence and zealous
work. On my house surgeons fell the chief responsibility of superintending these arrangements in my absence, and I feel my special thanks are due to them, amongst whom I would mention Dr. Roy Dobbin, Dr. Robert Halahan, and Dr. Fred. Blackley.

Mr. Lentaigne said that, as to the definition of aseptic and antiseptic surgery, he understood aseptic surgery to mean where nothing went into the wound in the shape of an antiseptic, chemical or otherwise, and antiseptic surgery, where septic processes in wounds had to be fought by the aid of chemical antiseptics. He considered it a matter of impossibility in general surgical practice to avoid occasional contact with sepsis, and accordingly he did not consider one could rely solely on aseptic measures.

Mr. Tobin thought the ideal condition of things would be that in which all surgeons used the same methods. It would be well, too, if surgical nurses were all trained on similar lines. He did not think the danger likely to follow from puncture of rubber gloves during an operation very great.

Professor Bennett agreed with Mr. Haughton as to the advantage of using alcohol as a final disinfectant in cleansing one's hands and the surroundings of a wound. He had found, however, that alcohol applied to the skin of his arms produced severe urticaria; he had found no unpleasant consequences, however, from employing it for his hands.

In reply, Mr. Haughton thought that abstinence from sepsis should be the constant aim of the aseptic surgeon, and the best way to do this was by wearing rubber gloves when dealing with septic cases of all kinds. In the treatment of actually septic wounds he thought weak antiseptics were better than those of greater strength, as the latter acted injuriously upon the tissues. He considered alcohol at a strength of 70 per cent. was better than full strength when employed for the hands. He thought silk was an absolutely satisfactory ligature material for burying in wounds, if No. 1 were used.

[At the conclusion of his paper Mr. Haughton showed a series of lantern slides illustrating various points in aseptic technique.]
NEW METHODS FOR THE PERFORMANCE OF HERNIOTOMY FOR INGUINAL AND FEMORAL HERNIÆ.

By EDWARD H. TAYLOR, M.D., B.Ch., F.R.C.S.;
Deputy Professor of Surgery in Trinity College;
Surgeon to Sir Patrick Dun's Hospital.

[Read in the Section of Surgery, December 2, 1904.]

Since the advent of the antiseptic era in surgery the subject of the radical cure of abdominal herniae, but more especially of the inguinal and femoral varieties, has proved one of great interest, and has taxed the ingenuity of surgeons generally. Although many different procedures have been introduced, few have stood the trying test of time, for while none have proved absolutely perfect many have fallen far short of the hopes which were entertained of them by their promoters, and others have been found to possess serious imperfections. In introducing still another mode of procedure I feel I ought to offer some sort of an apology, but the operations which I am about to describe have fulfilled my expectations so well that I thought they would prove of interest to this Section of the Royal Academy of Medicine.

The aims of herniotomy, whether it be performed for inguinal or femoral rupture, are in the main two—1. To dissect out the sac, its contents of course having been reduced, and occlude it at its neck—i.e., where it becomes continuous with the peritoneum of the abdominal parietes. The occluded neck is then allowed either to drop back towards the abdominal cavity or is fixed to the abdominal parietes by some of the methods in vogue, such as that of Kocher or Macewen. 2. To occlude or narrow
as far as possible the passage by which the hernia left the abdomen.

[At this stage of the communication the anatomy of the inguinal canal was explained by means of a number of lantern slides.]

Stated briefly, the weakness of the abdominal wall in the inguinal region may be attributed—

1. To the presence of the interstitial space occupied by the spermatic cord in the male, the round ligament in the female—the inguinal canal.

2. To the muscular deficiency in this region due to the failure on the part of the internal oblique and transversalis muscles to reach the inner or pubic half of Poupart's ligament. At this level the aponeurosis of the external oblique is merely reinforced behind by the transversalis fascia without the intervention of the other flat muscles.

For some years the operation which I have almost invariably practised in the adult for the radical cure of inguinal hernia is that associated with the name of Bassini, with, perhaps, a few slight modifications. The operation which I am about to describe resembles Bassini's in one respect—viz., in that its object is to bring down the lower borders of the internal oblique and transversalis muscles to Poupart's ligament—but it differs from Bassini's operation in that the external oblique is not divided, and the inguinal passage is exposed and dealt with from behind.

Operation.—Omitting all reference to preparatory treatment and cleansing of the patient's skin, I shall now briefly describe my method of procedure. The patient having been put into the Trendelenburg position, the operator, standing on the side opposite to that upon which the hernia exists, makes a vertical incision about four inches in length over the lower part of the rectus muscle
at the junction of its outer and middle thirds. The sheath of the muscle is divided, and the outer border of the latter is defined and drawn inwards by a broad abdominal retractor. The transversalis fascia is then carefully divided, the deep epigastric vessels being in the meantime protected from injury. The operator now pushes back the parietal peritoneum outside the deep epigastric vessels and directs his fingers towards the internal abdominal ring. This step is greatly facilitated by strongly retracting the outer edge of the abdominal wound. By gentle traction the neck of the sac is drawn up out of the inguinal canal and opened between haemostatic forceps. Assuming now that the sac is empty the right or left index finger is inserted into its interior and its isolation is effected. This is most readily accomplished by the aid of a sterilised muslin wipe, the laxity of the surrounding tissue enabling one to sponge away the structures constituting the spermatic cord. The sac is drawn out through the abdominal wound, and is removed after a ligature has been applied to its neck, or the sac may be cut away and the peritoneal aperture closed by a continuous suture. The inguinal canal is now identified and readily explored with the finger, being large and patulous in large herniae of long standing. By means of a curved needle, with a slot at one side leading to the eye, a series of sutures is passed connecting the conjoined tendon and the transversalis muscle above with Poupart's ligament below. As these sutures are being passed the structures of the spermatic cord are displaced outwards. The retraction of the outer margin of the abdominal wound relaxes the conjoined tendon very fully, and facilitates the apposition with Poupart's ligament. The ends of the sutures having been cut short, the operator examines the internal abdominal ring, and if
it appears unduly patulous an extra suture is introduced as before, but preferably at the outer side of the spermatic cord. The retractors are now withdrawn, and the rectus muscle is allowed to resume its natural position, but, in addition, I connect its outer border by means of a few sutures with the deep aspect of the transversalis tendon. The margins of the divided rectus sheath in front are brought into apposition and sutured, and, finally, the skin wound is closed by a continuous subcuticular suture of silkworm gut.

So far I have alluded only to the inguinal variety of hernia, but a similar form of procedure may be carried out for the radical cure of femoral hernia. Through the same form of abdominal incision the upper or abdominal aspect of the femoral canal is exposed. By gentle traction the hernial sac, emptied of its contents, is drawn up somewhat, opened, and separated from its surroundings. It is then closed at its neck, and the fundus removed as before. Two or three sutures of silk are now passed by means of a special needle which I have devised for this purpose, through Poupart’s ligament in front and Cooper’s ligament behind, and the femoral ring is closed. In some of my cases, for greater security, I have passed these sutures through holes drilled in the pubic bone. An ordinary bone awl answers well for this purpose. I am aware that a method for dealing with femoral hernia somewhat similar to the above has been described by Mr. Mayo Robson (“Year-book of Treatment, 1904”), but it differs from mine in certain important respects, into which I need not now enter.

In conclusion, I have only to indicate the points in favour of the operations which I have just described.

1. In approaching the inguinal or femoral hernial aperture on its abdominal aspect the method of procedure
just described does not entail any division of muscle, nor the sacrifice of any nerve or blood vessel of importance.

2. It affords good access to the inguinal and femoral rings. These can be closed as I have described in a very secure manner, and, in the case of an inguinal hernia, the abdominal wall is effectively strengthened at a place where it is naturally weak and yielding.

3. This method of dealing with inguinal herniae appears to me particularly well adapted to those which are of long standing—viz., those in which the inguinal rings are dilated and have closely approximated to each other. At first my idea was to employ this operation only for such cases, but, with increasing experience, I am inclined to give it a wider application.

4. It is singularly well adapted to femoral herniae. Indeed the method of closing the femoral ring which I have described is, in my opinion, as near the ideal as possible.

Against these methods of performing herniotomy, but more especially in connection with inguinal hernia, the objection may be raised that they are somewhat difficult. Whatever difficulty there is is met with in passing the sutures through the conjoined tendon and Poupart's ligament, but this may be largely minimised by seizing the latter with a vulsellum forceps and making slight upward traction as the suture needle is passed beneath it. Much of the ease with which the operation may be performed depends upon thorough relaxation of the abdominal muscles under the anaesthetic. It is easier also in a spare patient than in one whose abdominal wall is very thick and loaded with fat.

Mr. Jameson Johnston expressed himself in favour of Bassini's operation, as he considered it the simplest, most scientific, and
the easiest. He regarded the objection to dividing the aponeurosis of the external oblique in this operation as more or less theoretical. Mr. Taylor's operation would probably be a good one in old herniae, in which the internal abdominal ring approached the middle line.

Mr. T. E. Gordon considered there was no one method of operation for hernia. Mr. Taylor's method was not suitable in the case of a congenital hernia in a young child. He believed that many of the operations practised were hopeful as regarded ultimate success. The essential thing in these operations was asepsis.

Sir Thomas Myles referred to the valve-like nature of the inguinal canal. So long as its anterior and posterior walls remained in contact and the muscles are sufficiently tense a hernia was unable to push these walls apart. He thought there was a hereditary predisposition to hernia. The valve-like closure of the canal, due to muscular action, was essential, and that being so, no amount of suturing of the conjoined tendon to Poupart's ligament would provide an obstacle to recurrence, because sufficient space had to be left to permit the cord to pass.

Mr. Blayney was of opinion that the weakness of the abdominal wall in the inguinal region was for a special purpose—viz., to allow the thin fascia transversalis to be pushed against the external oblique when the intra-abdominal pressure was increased.

Mr. Taylor, in reply, stated that he believed his method of operation was best suited for large, well-developed inguinal herniae, in which the inguinal canal had undergone marked changes in size and direction. As bearing upon the success of the procedure he thought it of importance to mention that the sutures in the conjoined tendon should not be tied too tightly, and that the parts should be kept at rest for a sufficient time afterwards to permit of healing taking place. Patients were frequently allowed to get about too soon after herniotomy; absolute rest for at least a fortnight was desirable. As to the mode of development of an inguinal hernia, he thought it probable that there was in many instances a degree of congenital weakness at the internal abdominal ring, in consequence of which the latter went on increasing in size. There was such a thing as a hernial type of abdomen, and it was in such cases that
weakness or dilatation at the internal abdominal ring was most likely to be found. Upon the hernia commencing to develop, the conjoined tendon and the fascia transversalis yielded more and more, and the inguinal passage became progressively larger and less oblique in its direction.
A CASE OF INTESTINAL OBSTRUCTION BY A GALL-STONE.

By T. E. GORDON, M.B., F.R.C.S.;
Surgeon to the Adelaide Hospital.

[Read in the Section of Surgery, December 2, 1904.]

Dr. WRIGHT supplied me with the following notes of this case:

Mrs. F. F., aged seventy-three years; bore seventeen living children, and has been very healthy all her life, with the exception of two or three illnesses connected with child-bearing, one being an attack of abdominal inflammation and another of phlegmasia alba dolens. On the night of February 12th, 1905, she was taken suddenly ill with an extremely acute attack of pain in the hepatic region, accompanied by vomiting; this was relieved by hypodermic injections of morphia. Two days after the patient was distinctly jaundiced, and I had no doubt at the time the pain was due to the passage of a gall-stone. After the acute pain subsided, a good deal of tenderness persisted over the region of the gall-bladder for a month; her temperature was slightly raised, her tongue furred, her bowels constipated, her appetite poor, and, in fact, from February 12th until the middle of April she was not well, suffering from what I looked upon as a condition of slight cholecystitis, brought on by the discharge of the gall-stone. Early in May she seemed to be perfectly well, so far as any liver or stomach trouble was concerned. On the 3rd June she got an attack of phlebitis in one of the superficial veins of her right leg, which completely disappeared before the end of the month, and again she seemed quite well. On Saturday evening, the 6th August, I was sent for and found her suffering from intense pain in the pit of the stomach. This had begun the evening before as a sensation of fulness, which had gradually increased until it became acute pain. Her son, who was a medical man, and staying in the house, had given her twenty-six drops
of Collis Browne's chlorodyne without effect. I gave her \( \frac{1}{4} \) gr. of morphia hypodermically, and ordered poultices to be constantly applied. I also left a dose of 30 minims of nepenthe, to be taken if the pain was not better. I was sent for early the following morning, as she had had a wretched night—sleepless, retching, and in constant pain, which none of the opiates had relieved. I found her looking very anxious; there was no rise of temperature, and no sign of any tenderness or tension in any part of the abdomen, but while I was standing by her bed she said she felt sick, and at once vomited, the discharge being so copious and expelled with so little effort that I felt convinced there was some obstruction of the bowel, and told her son I should like to get a Dublin surgeon to see her with me. I then gave her \( \frac{3}{4} \) gr. of morphia hypodermically, with the result that before I left the house she had dropped into a profound sleep. At three o'clock that day I met Mr. William Taylor in consultation. The patient awoke from the morphia sleep just as we came into the house, so that the last dose had given her six hours uninterrupted rest. She told us she "felt ever so much better, not a bit sick," and she took a little freshly made tea without any return of the vomiting. Mr. Taylor examined her abdomen most carefully, but could detect nothing to indicate where the seat of the mischief was, although it was somewhat distended with flatulence; the walls were soft, and there was no sign of tenderness or tension. We decided there was no indication for immediate operation, but that it was better to wait and see what medical treatment would do for her, as it seemed probable that all her symptoms might be due to the passage of another gall-stone, and that the acute stage of the attack had passed. That night I gave her a five grain dose of calomel, followed by a copious turpentine, soap and water enema in the morning, but without result. On the 8th and 9th she seemed fairly well, although feeling nauseated. There was no vomiting or further increase in the abdominal distention, no tenderness or tension to be noticed anywhere, but there was absolute constipation. On 10th August I got Dr. Lennon to see her in consultation. That morning, before he came, she had brought up without effort some mouthfuls of that inky black vomit one sees discharged from the stomach in bad abdominal cases. He also examined her very carefully, and expressed a hope, as he could not detect anything definite
in the abdomen, that the vomiting and constipation might be caused by faecal accumulation, and suggested copious enemata to be administered by the long tube every three hours. Of these she received four without any result whatever. During that night she several times brought up six or eight ounces of ster-
coraceous vomit, and on the morning of the 11th I felt that if we were not able to relieve her by an operation she would certainly die, and that soon. I therefore telephoned to Dr. Lennon asking him to come and bring Mr. Gordon with him (as Mr. William Taylor was out of town) prepared to open the abdomen at once should we decide that such a course was justifiable. Drs. Lennon and Gordon came at 4 p.m., and the latter will relate the surgical notes of the case. From a medical point of view I may give a brief résumé of her illness as follows:—

In February a distinct attack of biliary colic, followed by an inflammation of the gall-bladder, which only subsided after two months. An interval of apparently perfect health, as far as abdominal symptoms were concerned, from April to the 6th August; then intense pain over the liver, followed by complete intestinal obstruction, but with no fever or sign of tenderness or tension in any part of the abdomen.

When I saw this lady with Dr. Wright and Dr. Lennon it was clear that she had an intestinal obstruction. The abdominal distention, complete constipation, and faeculent vomit were conclusive evidence. The question was whether an operation offered a reasonable chance of recovery. We decided it would, because, although very ill, the patient did not present the aspect of extreme toxæmia.

On opening the abdomen in the epigastrium some peritoneal fluid escaped—sufficient in amount to explain a dulness in the flanks which we had previously noticed. I first examined the gall-bladder region, and found there firm adhesions, and this evidence of long past peritonitis was present in other parts of the abdomen. I specially noticed a strong band which tied down the omentum to the pelvic viscera. Fortunately this band did not add much to the difficulties of the case, for I was able to turn out the omentum sufficiently to bring the transverse
colon and small intestine into view. The colon was contracted, the small intestine moderately dilated, and its walls congested. I drew out the latter, following the direction of most congestion, and within the first foot of the bowel, thus exposed, I came upon an impacted gall-stone. I removed this through an incision, which I then sutured. Before sewing up the abdominal wall I made a knife puncture in another part of the small intestine, which was specially distended, and allowed a quantity of foul intestinal matter to drain away.

I wish to draw special attention to the state of the bowel at the site of the stone. As you look at this stone you will be struck by its small size. There would appear to be no difficulty for such a stone to pass along the small intestine. As a matter of fact, it was firmly impacted, and was causing complete obstruction. We observed two structural alterations in the wall of the gut. First—When the incision was made there was no pouting of the mucous membrane; it was as though the intestine had been crushed by a Doyen's forceps before its division. Second—We found considerable difficulty in suturing; owing to the friable condition of the tissues the thread repeatedly tore out. That small stones may cause intestinal obstruction is well known (vide Treves' "Intestinal Obstruction" and Mayo Robson's "Diseases of the Gall-bladder and Bile-ducts").

After the operation all vomiting ceased (I should have mentioned that we washed out the stomach at the conclusion of the operation), and the patient was able to leave her bed within a fortnight.

The various phenomena of this case do not admit of certain explanation. Three stages in the course of the illness are fairly clearly defined.

First, a stage of onset, lasting from the evening of August 5th to the morning of August 7th, marked by intense pain in the epigastrium, retching and vomiting, without fever or local tenderness.

Second, a stage of quiescence, with absence of vomiting or even feeling of sickness. This continued throughout August 7th, 8th, and 9th, and it was on the first of these three days that the patient was seen by Mr. W. Taylor.
Case of Intestinal Obstruction by a Gall-stone.

Third, a stage with unequivocal signs of intestinal obstruction, especially faecal vomiting. The first sign which marked the onset of this final stage was "that inky black vomit one sees discharged from the stomach in bad abdominal cases" (I quote Dr. Wright). This occurred on the morning of August 10th, and the operation was performed at 6 p.m., August 11th.

The chief interest and all the difficulty belong to the first and second stages. Were the early symptoms due to the passage of the stone from the gall-bladder to the duodenum by way of a fistulous opening? Again, was the stone impacted when Mr. Taylor saw the patient on August 7th, or did complete obstruction only set in with the return of vomiting on the 10th?

In attempting to answer these questions one naturally turns to the history of other cases of gall-stone obstruction. From such a study one learns:

1. That the passage of a large gall-stone into the intestine is usually marked by the signs of a local peritonitis, including tenderness and fever.

2. That the passage may be accomplished without causing any symptoms, or symptoms too slight to raise a suspicion of the event.

3. That it is quite the rule in these cases for an interval to occur of variable duration between the supposed date of escape of the stone and the onset of the intestinal obstruction.

In the present case I think the severe onset pain was due to the escaping stone. The absence of fever and local tenderness is a difficulty, but it is met by the second fact (if it really is such) which I have just alluded to—i.e., that a fistula may form and a stone pass into the intestine without any symptom.

In the quiescent period, in which the patient was seen
by Dr. Wright and Mr. Taylor, I do not think there was any obstruction. If the stone had been impacted as I saw it at the operation it is inconceivable that vomiting and nausea could have so entirely ceased. This is the more unlikely seeing that the calculus occupied, not, as is more usual, the lower part of the ileum, but a position probably very high in the jejunum.

Of the third stage I have nothing special to say. Spasm is obviously an important factor in its production.

The treatment of these cases is, fortunately, easily described. If you can make the diagnosis—operate! If you cannot make the diagnosis—operate!

Mr. W. Taylor stated that he had seen the patient a few days before Mr. Gordon performed the operation, and on the day before he left town for his holiday. She was then under the influence of opium. He thought that at that time the gall-stone made its exit from the bile passages. At the same time, however, he had not overlooked the possibility of intestinal obstruction. He would like to know if any reasonable explanation could be offered why a calculus of this size should become impacted.

Sir Thomas Myles alluded to a case in which he had removed a large impacted gall-stone fifteen years ago. He thought it likely that the impaction in the present case was due to the opium administered, the muscular tissue of the bowel being paralysed. He would advise in such cases that the incision into the intestine be made not directly over the gall-stone, but on the proximal side, as otherwise one cut through infiltrated and devitalised tissues.

Mr. Jameson Johnston inquired as to the amount of distention present and the condition of the bowel at the site of impaction. Mr. Gordon alluded to spasm, but he did not think spasm could exist under the conditions present—viz., inflammatory infiltration and oedema of the bowel wall.

Mr. Blayney thought the impaction of the gall-stone in the present case might be due to its rough exterior, by which the
mucous membrane of the intestine was irritated and abraded, thus permitting micro-organisms to act. This resulted in oedema of the submucous tissue which extended inwards rather than outwards. He believed the impaction was due more to inflammatory oedema than to spasm.

Mr. Gordon, in reply, said he thought some of the suggestions which had been made as to the cause of the impaction were correct, but he himself had none to offer. The abdominal distention was not very great. He made the parietal incision above the umbilicus, because the early pain complained of was referred to the upper part of the abdomen.
CHOLECYSTOTOMY FOR ACUTE CHOLECYSTITIS DURING CONVALESCENCE FROM ENTERIC FEVER.

By R. CHARLES B. MAUNSELL, M.B., F.R.C.S.; Surgeon to Mercer’s Hospital, Dublin.

[Read in the Section of Surgery, January 13, 1905.]

The case which I bring before you this evening is one which occurred in my hospital practice almost five years ago. At the time it was of intense interest to me, as the condition of which it is an example was then almost unrecognized, or at least unreported, in the British Isles. Even yet I may safely class it amongst the rarities in surgical literature, but at the same time I am inclined to believe that this, or closely allied conditions, are not very uncommon during or shortly after enteric fever. As the patient has lately returned, suffering from another complaint, I take this opportunity of showing him to you and of reading his history.

William C., aged eighteen, grocer’s porter, came to the surgical dispensary at Mercer’s Hospital on April 24th, 1900, suffering from a crushed finger. He looked very ill, and stated that he had felt sick for some days before he met with the accident. His tongue was coated, bowels constipated, and temperature 103°, pulse 80. I instructed my resident to admit him to the medical wards, under my colleague, Dr. Ledwich, which was done on April 25th.

The patient passed through a typical attack of enteric fever, the temperature ranging between 102° and 105°, pulse between 80 and 110. Widal’s reaction was positive. The bowels were at first constipated; afterwards typical diarrhoea was present, with haemorrhage on the ninth day, dating from his admission. The temperature and pulse became normal on the fifteenth day.
On May 16th, seven days from the subsidence of fever, solid food was given, and by May 25th he was on a diet of fish, chicken, &c.

On May 27th he vomited, and complained of abdominal pain, so solid food was stopped.

On the morning of May 28th Dr. Ledwich requested me to see the patient in consultation with him.

The condition found was—temperature 98.4°, pulse 90, tongue slightly coated, colour of face dusky, and expression anxious. The patient complained of severe abdominal pain. The abdominal walls were rigid and tender to touch, especially on the right side; the pain, tenderness, and rigidity were all most marked just below the ninth costal cartilage, and an indistinct tumour could be felt between this and the umbilicus. There was no jaundice present. The urine was of sp. gr. 1018, slightly alkaline, and contained a trace of albumen.

We considered that, probably, acute cholecystitis had occurred, and a further consultation was arranged. My colleague had to leave town during the afternoon, so at 6 p.m. I received a message saying that the temperature was 99.4°, pulse 100, and pain severe. At 8 p.m. I again examined the patient and found the rigidity and pain more marked—the temperature 101°, pulse 110, and the colour of the face much more dusky. I determined to explore the region of the gall-bladder without further delay.

Operation.—Ether having been administered an incision was made over the tumour in the direction of the fibres of the external oblique muscle. The fibres of both oblique muscles were then dealt with as in M'Burney's operation for appendicectomy, and the remaining structures divided. A quantity of clear serous fluid was sponged away from the neighbourhood of the gall-bladder, and this organ carefully drawn into the wound. It was about the size of a turkey's egg, very tense and deep purple red in colour.

A sterilised hypodermic needle was passed obliquely through the tense wall, and enough fluid withdrawn to lessen the tension, and also to serve for bacteriological examination. The fundus of the gall-bladder was stitched to the peritoneum and fascia of the wound with fine silk, and the exposed area opened. The fluid which issued was turbid mucus, and later muco-pus. A fine probe was passed to search for any gall-stone which might be present.
By Mr. R. Charles B. Maunsell. 127

in the cystic duct, but this search was not long continued, as it excited free haemorrhage from the inflamed mucous membrane. A rubber tube was fixed in the gall-bladder, and most of the skin wound closed with silkworm gut sutures.

Next morning the temperature was slightly subnormal, and pulse 108.

The after-history of the case was uneventful; bile began to flow from the wound on the fifth day; the wound was completely closed at the end of a month, and the patient left hospital a week later.

No gall-stone was found, although carefully sought for during the week subsequent to operation.

I submitted some of the patient's blood and the fluid aspirated from the gall-bladder to Dr. H. E. Littledale, who was then Assistant to the Professor of Pathology in Dublin University. He very kindly sent me a very full report, a précis of which I give.

The blood showed a positive Widal reaction, with a dilution of 1 in 100.

The fluid was examined microscopically, and the following culture methods employed:—(1) Stab culture in glucose agar; (2) Piorkowsky's urea gelatine plates; (3) glucose litmus agar stab; (4) glucose gelatine stab; (5) agar slope; (6) litmus milk; (7) peptone beef broth.

By all these methods an absolutely pure culture of Eberth's bacillus was obtained.

The patient remained in good health until October 24th, 1904, four years and five months after operation, when he was admitted under my charge with temperature 103°, pulse 120, and severe griping abdominal pains, chiefly at and below the umbilicus.

The scar over the gall-bladder is a very sound one, and shows no tendency to hernial protrusion.

Next morning the temperature was 105°, pulse 104, and the pain and tenderness were referred to the right iliac fossa. When I saw him about two hours later the temperature had fallen to 102°, but the pain was still severe. Within half an hour the temperature again rose to 105°, with a rigor, and the pain and tenderness almost disappeared, but the pulse and aspect of the patient were worse. A diagnosis of gangrenous appendicitis was made, and immediate operation performed. A large club-shaped
appendix was removed, and gauze drainage inserted through a stab wound in the loin above the iliac crest, the anterior incision being completely closed. Convalescence was uneventful, except for an abscess which developed in the anterior wound about ten days after operation. On examination the appendix was found to be tensely filled with pus, and its distal third was gangrenous. I regret to say that it was placed in formalin immediately upon removal, and so no culture of bacteria could be made.

The patient is now, January 13th, 1905, a fine healthy young man, who suffers in no way from his trying experiences.

I have added the account of his second illness as, although I have no actual proof of it, yet it most likely was caused by pathological changes in the lumen of the appendix due to typhoid ulceration, if not to actual persistence of the Eberth's bacillus in this cul-de-sac, as suggested by the cases of long-deferred recrudescence in other regions collected by Keen and Westcott.

In discussing this case we must leave out of consideration other hepatic complications or sequelae of enteric fever and confine ourselves to a few lesions of the gall-bladder. For many years ulceration and perforation of the gall-bladder have been recorded as post-mortem discoveries in enteric fever by Murchison and other writers. Gilbert and Girode in 1890 were the first to demonstrate Eberth's bacillus in cholecystitis occurring during typhoid fever. Dupré, von Dungern, and others have demonstrated it in gall-bladder lesions occurring from six months to fourteen and a half years after the primary infection.

It is important to know whether all cases of infection of the bile passages with this bacillus necessarily lead to serious results. In answer to this, Chiari has examined twenty-two cases of enteric fever post mortem, and has found the bacillus present in the gall-bladder in nineteen cases, without obvious lesion in six cases and only slight
lesions in the remaining thirteen. Councilman regards the gall-bladder as the surest place to obtain a pure culture of this organism. Welch has demonstrated Eberth's bacillus in the gall-bladder of rabbits many months after inoculation. Chiari suggests that the gall-bladder may be the origin of relapses in typhoid fever.

We must, therefore, recognise that in most cases of enteric fever Eberth's bacillus is present in the gall-bladder, but that only in a minority of the cases does it lead to serious results.

Amongst these serious results we may consider the following:

(1) Gall-stone Formation and its various Complications.—Gilbert, Hanot, Fournier, and others have clearly proved that gall-stones are bacillary in origin, and have demonstrated the Bacillus coli, Eberth's bacillus, &c., forming nuclei for these concretions. The stones so formed may lead to a train of symptoms which are too many even to be enumerated in this communication.

(2) Simple Cholecystitis.—This is an extremely common post-mortem finding in enteric fever, and gives rise to no symptoms during life unless complicated by well-marked cholangitis, or by calculi in the ducts.

(3) Hydrops.—In this condition the gall-bladder is distended with a clear or slightly turbid fluid which contains numbers of bacteria.

The symptoms are pain, tenderness, and tumour in the region of the ninth costal cartilage, or somewhere between it and the umbilicus. If this condition is not promptly treated in the majority of cases it soon merges into—

(4) Suppurative Cholecystitis.—Not only is the gall-bladder inflamed, but it is tensely filled with pus, and perhaps its mucous lining is ulcerated. The symptoms—unless
the patient is too dull from toxæmia—are severe pain, rigidity, tumour, elevated temperature, and if the mucous membrane is ulcerated rigors most probably will occur. In a few almost miraculous cases recovery has been recorded after spontaneous discharge of the pus either through the abdominal wall or into adherent intestine, but, as a rule, unless surgical aid is obtained, death will follow from one of the following stages of the infection.

(5) Phlegmonous and Gangrenous Cholecystitis.—These two differ only in the severity of the necrotic process. By phlegmonous cholecystitis is meant that there is marked thickening and pus infiltration of the wall of the gallbladder, with local peritonitis around it. By gangrenous cholecystitis is meant that more or less of the wall has become necrotic in its whole thickness. The local collection of purulent material in many cases determines the death of the patient during a severe fever or whilst he is weak during convalescence. In other cases the peritonitis spreads, or the most exaggerated form of infection may occur, viz. —

(6) Perforation or Rupture of the Gall-bladder.—This, of course, leads to purulent peritonitis and death unless promptly treated in exceptional cases.

It has been suggested that these complications of enteric fever only occur when gall-stones have been formed previous to the advent of Eberth's bacillus. In reply to this I cannot do better than quote a paragraph from Keen and Westcott's valuable monograph:

"A second question requiring answer is whether the typhoid bacilli are capable of producing cholecystitis, empyema of the gall-bladder, and ulceration independently of gall-stones. Of this I think there can be no doubt. Of the 74 cases of biliary infection in our table,
in 18 cases gall-stones were found. In a few others which did not come to post-mortem there may have been also gall-stones present. In 38 cases, however, no gall-stones were present, and of these, 8 were in persons under twenty-five years of age, and 6 under fifteen, at which period of life it is extremely uncommon to find gall-stones. In 34 of the cases reported cholecystitis, empyema, or ulceration were found in the gall-bladder without any gall-stones, and the typhoid bacilli were identified by bacteriological examination in 11 cases."

Time forbids that we should discuss treatment fully, but yet this all-important question must be briefly reviewed.

Keen and Westcott give the details of a case of hydrops in which Mason thrust an aspirating needle through the abdominal wall and removed 55111111 of fluid (which he described as like urine in colour), with good result.

Osler reported a case in which a like treatment was adopted, but cholecystotomy had subsequently to be performed, and the patient, who was suffering from a very severe attack of enteric fever, died. I hardly think anyone skilled in abdominal surgery would now advocate this crude method of aspiration. Undoubtedly the proper treatment in cholecystotomy, under local analgesia if necessary, with subsequent drainage by a tube fixed in the gall-bladder. In phlegmonous or gangrenous cases cholecystotomy with drainage through a tube fixed in the cystic duct should be adopted, or, if the patient is too ill to justify the expenditure of the few extra minutes which this operation entails, the gall-bladder should be freely opened and copious gauze packing placed in the right hypochondriac region around a tube leading to the diseased organ.
Cholecystotomy for Acute Cholecystitis.

In cases which have perforated, other abdominal incisions are necessary to enable the operator to thoroughly cleanse the peritoneal cavity.

In conclusion I will quote a few statistics given by Keen and Westcott to show what a serious condition we are discussing, and how useful surgery has been in its treatment. They collected 74 cases of cholecystitis occurring during or shortly after enteric fever. Of these 30 resulted in perforation. Of the 30 which perforated 26 were not operated upon, and all died. Of the 4 which were operated upon 3 recovered and 1 died twenty-four days subsequent to the operation. It is interesting to note that in the 23 fatal cases gall-stones were found post mortem in 7, and no gall-stones were found in 16. So it is evident that severe and fatal inflammation can occur in gall-bladders which have been healthy previous to the infection with Eberth's bacillus.

Mr. Jameson Johnston related two cases of typhoid fever in which complications had arisen calling for surgical intervention. In one there was necrosis of the laryngeal cartilages, and in the other there was a chronic abscess of bone, and in both typhoid bacilli were found in the pus.

Sir Charles Ball had seen many cases in which disease of bone had occurred many months after typhoid fever. He related a case also in which, during the acute stage of the fever, a large abscess developed in the upper lobe of the lung.

Dr. Finny considered that the question of interest in Mr. Maunsell's case was whether the acute cholecystitis was a complication of the acute fever or a sequela. These complications were to be expected, not in the height of the fever, but just when the patient seemed to be getting better. He did not think typhoid bacilli were the sole cause of the complication in the present case, as they are often found in great numbers in the gall-bladder without any results, locally or otherwise, in the neighbourhood. Mr. Maunsell's patient first came under observation.
on account of a septic finger, and it was just possible that septic organisms were associated subsequently with the typhoid bacilli.

Mr. Maunsell, in reply, stated that in looking up the literature of the subject he had noted instances of complications arising fourteen and a half years after typhoid fever, and in which pure cultures of the typhoid bacillus were obtained. He had thought of the possibility of the crushed finger having had something to do with this case, but on very careful examination no septic organisms could be found.
CONSERVATIVE PERINEAL PROSTATECTOMY.

By C. ARTHUR BALL. M.D., F.R.C.S.;
Assistant Surgeon to Sir Patrick Dun's Hospital.

[Read in the Section of Surgery, January 13, 1905.]

It will no doubt be conceded that the object in all surgical procedures should be to confer the maximum benefit on our patient consistent with the minimum amount of injury to the anatomical structures involved.

The methods of prostatectomy as usually performed by the suprapubic or perineal route cannot be regarded as ideal surgical procedures, for the prostate has to be removed largely by the sense of touch: haemorrhage during the operation is not fully under control; and the amount of injury inflicted on the bladder, urethra, and ejaculatory ducts is frequently considerable.

Although excellent results frequently follow prostatectomy, some of us have seen trouble following the operation, such as incontinence of urine due to injury of the sphincters of the bladder and urethra: septic trouble spreading down the injured ejaculatory ducts to the epididymis and testicle: and persistence of a vesical fistula.

The question we have to decide is—Can the prostate, or rather a sufficient part of the prostate, be removed so as to cure the patient without inflicting permanent injury to important anatomical structures?

Perineal prostatectomy as performed by Dr. Young of the Johns Hopkins Hospital, seems to me to fulfil these conditions more fully than the methods previously advocated. I will, therefore, endeavour to demonstrate
to you to-night the essential features of his operation of conservative prostatectomy.

To remove the prostate by the perineal route some method of traction is necessary to bring the prostate within easy reach. A suprapubic incision has been used to push the prostate down into the perineum; the only advantage this possessed over the suprapubic enucleation was the establishment of perineal drainage. The hooks, as used by Murphy in some cases, gave a good perineal exposure, but in others tore so readily through the prostate that they were of little use. Sym's used an intravesical balloon; this overcame the great objection to perineal prostatectomy, but hardly gave sufficient strength for the necessary traction. Young's prostatic tractor has, he says, transformed the operation of perineal prostatectomy for him. I have tried his instrument several times on the dead subject, and once on the living, and its use affords a view of the prostate such as I had not thought possible. The steps of the operation described by Young are briefly as follow:—

*Position of the Patient.*—The patient is placed in the exaggerated lithotomy position, a grooved sound having previously been passed into the bladder as a guide to subsequent urethrotomy. If the patient is thin, and the prostate apparently close to the perineum, a median incision is made not more than 6 cm. long, the posterior limit being near the anus. The central tendon of the perineum, connecting the bulb in front and the levator ani muscles behind, is exposed.

---

Fig. 1.—Young's prostatic tractor, closed and ready for introduction.
If the patient is a large man, with thick perineal muscles, and the prostate is found on rectal examination to be situated deep, or the peri-rectal fat is abundant, an inverted V-shaped incision is used; the apex of this incision is situated just over the posterior part of the bulb. The two branches are each 5 cm. long, the posterior limits being about midway between the anus and ischial tuberosities.

Exposure of the Membranous Urethra.—After the superficial muscles have been exposed by blunt dissection, the central tendon is caught by a clamp near the bulb and divided. This at once frees the sphincter and

![Fig. 2.—Young's prostatic tractor, open.](image)

the levator ani from their anterior attachment, and exposes the rectum, drawn forward by the recto-urethralis muscle. The recto-urethralis is a short muscle with rather indefinite margins which, as its name implies, joins the rectum with the urethra. It is apparently responsible for the acute anterior flexure of the rectum which lies so close to the apex of the prostate and membranous urethra, and which one finds in rectal examinations. (Mr. F. Wood Jones² has found that the band of musculature which passes from the front of the rectum, to be lost in the tissue behind the membranous urethra, is a remnant of the recto-cloacal communication in the embryo.) In order to reach the membranous urethra and the apex of the prostate it is necessary to
divide this muscle; this exposes the recto-prostatic space described by Proust, who has shown that unless this muscle is divided the operator is apt to tear the rectum, which is drawn forward by it.

Division of this muscle allows the rectum to drop back, and leads at once into the space surrounding the posterior surface of the prostate.

Fig. 3.—A and B are drawn actual size from sections through the prostate of a formalin-hardened subject kindly lent by Professor A. F. Dixon. A. A short distance below the bladder floor. B. Just above the opening of the ejaculatory ducts into the prostatic urethra. The large veins in the capsule of the prostate are well shown, and explain the occurrence of pulmonary embolism after prostatectomy. Note the way the large vessels are confined to the anterior and lateral aspects of the capsule. The prostatic urethra is seen in the centre of each section. In A, note the position of the ejaculatory ducts, lying close to one another, in the middle line near the posterior surface of the capsule. In B, the ducts are seen piercing the veru montanum to open into the urethra.
Urethrotomy and Insertion of Tractor.—After the membranous urethra has been exposed, a retractor is inserted and the apex of the prostate brought into view. The membranous urethra is then opened on the sound, and the edges of the urethral wound caught up by silk sutures or Kocher clamps. A sound of moderate size is then passed through the incision into the prostatic urethra and bladder, and the sphincters dilated by a to-and-fro motion of this instrument. The prostatic tractor, closed, is then passed into the bladder, the edges of the urethral wound being held apart to facilitate its introduction. As soon as the beak is free in the vesical cavity the blades are separated. The instrument is now ready for what-
ever traction may be necessary to draw the prostate well down into the perineal wound.

*Exposure of Prostate and Incision of Capsule.*—Lateral retractors are so placed that with the posterior retractor drawing the rectum backwards, and the prostatic retractor drawing the gland outwards, a splendid exposure of the entire posterior surface of the prostate is obtained. An incision is then made on each side of the median line for almost the entire length of the posterior surface of the pros-

![Image](image.png)

*Fig. 5.*—Showing position of blades in interior of bladder in case of median and bilateral hypertrophy (Young).

tate, and about 1 cm. deep. The two lines are convergent, being about 1.8 cm. behind, and 1.5 cm. apart in front. The bridge of tissue which lies between them contains the ejaculatory ducts, and its preservation is of utmost import ance if the integrity of these structures is to be left uninjured. It is for this purpose that the initial capsular incision is made 1 cm. deep on each side, as these define at once, and correctly, the width of the "ejaculatory bridge," and prevent its being torn, as might happen it
we depend on blunt dissection. Another advantage is that these incisions bring us at once to the side of the urethra where the internal enucleation (urethra from inner surface of lobe) can be easily accomplished later on.

*Enucleation of Lateral Lobes.*—We are now ready to
begin the external enucleation, the separation of the capsule from the lateral lobes, which is best done with the blunt dissector. Capsules are of varying thickness, and contain several layers of cleavage. It is important to start the separation in the right layer, not too deep, as you may be led into the substance of the lobe, and not so superficially as to be outside of most of the capsule. After the stripping-up process has been started correctly, it is easily continued by blunt dissectors until first the lateral and then the anterior surface of the lateral lobes have been freed from the capsule. The internal enucleation should be taken up after the external, as it is a much more delicate procedure, and often requires considerable care to prevent tearing into the urethra. As remarked above, the primary incision is made with the scalpel until the level of the urethra is reached, after which the blunt dissector is used. During this procedure the shaft of the prostatic tractor is grasped firmly in the operator's left hand, and serves not only to draw the prostate so well down into the skin wound that every procedure is done in plain view, but to steady the prostate and mark out the course of the urethra so that it can be avoided. When the enucleation of a lateral lobe has progressed fairly well on each side, it is advantageous to have traction made on the lobe itself in order to facilitate the separation of the deeper portion. The special forceps designed by Dr. Young is here of great assistance, as with it great traction can be applied without tearing the lobe, thus facilitating the deeper enucleation. Nearly all the enucleation is done with the blunt dissector, but when the intra-vesical portion of the lateral lobe is reached the finger should be used to avoid tearing through the thin mucous membrane covering it. The intra-vesical blade of the prostatic tractor, which can be
distinctly palpated through the mucous membrane by the enucleating finger, serves to direct the separation of the deeper portion, and warns against tearing into the bladder. It also shows when some of the lobe has been left behind.

*Enucleation of the Middle Lobe.*—After the lateral lobes have been shelled out, attention should be directed to the median portion of the prostate. When there is more or less hypertrophy of the prespermatic group of glands the mass may be easily seen or felt by the finger in one of the intra-capsular cavities. The median enlargement is generally more or less definitely attached to one or both of the lateral lobes, and is often already so loosened from its encapsulations that there is no diffic-

![Fig. 7.—Young's lobe forceps.](image1)

![Fig. 8.—Schematic cross-section after enucleation of lateral lobes showing ducts and median bridge of tissues. Instrument in urethra.—(Young.)](image2)
cult in shelling it out through one of the lateral cavities without disturbing the integrity of the ejaculatory ducts and prostatic tissue immediately surrounding them. The tractor can be used to depress the middle lobe and draw it down, so that, pushing against it with the index finger of the left hand, which has been inserted in the right intra-capsular cavity, it can be grasped with the forceps and rapidly enucleated. By this technique Dr. Young has also been able to remove median lobes of considerable size without destroying the ejaculatory ducts.

Drainage.—Before withdrawing the tractor, a careful examination should be made by inserting the finger into both lateral cavities and palpating the blades through the vesical mucosa, in order to determine that no important glandular mass has been left behind. Abundant vesical drainage should be provided, as a small tube may easily become plugged by blood clots, and give great annoyance afterwards. Dr. Young now uses two tubes—one a small catheter, the other an ordinary tube about 1 cm. in diameter. These are fastened together by ligatures at four places, about one inch apart, and are prepared before the operation, so that as soon as the tractor is withdrawn they can be inserted into the urethra and bladder. The catheter is immediately connected with a tank of normal salt solution, and the bladder thoroughly washed clean of blood. After the tubes have been properly adjusted they are tied by a heavy silk suture to the skin at the upper angle of the wound. The lateral prostatic cavities are then firmly packed with gauze, and additional packing is placed in the retro-prostatic space.

The wound is closed by deep catgut sutures, and if the inverted V-incision is used one side is completely closed; the other is left open at the upper part for the tube and gauze.
After-treatment.—Continuous irrigation of the bladder is kept up if the patient has cystitis (and most have), often for a week, with great benefit to the vesical inflammation.

A submammary infusion of 1,000 c.c. salt solution is given either on the operating table or after the return to bed. This is considered so valuable, both as a preventive to shock and anuria, and as a cure for post-operative thirst, that it is never omitted.

The patient is generally kept in bed a week, when the drainage-tubes are withdrawn. The gauze drains are loosened on the second day and gradually withdrawn, all of the original being out by the sixth day. On the ninth day, after passing a sound, a small catheter is inserted through the meatus into the bladder, and held in place by straps. Permanent drainage is thus maintained for five days, the bladder being irrigated twice daily with boric solution. When the catheter is withdrawn, a good portion of the urine will be found to come through the perineum, but in a short time almost all will come through the meatus, though it is sometimes five or six weeks before the minute perineal fistula closes.

Dr. Young kindly informs me (Jan. 3rd, 1905) that he has now performed his operation of perineal prostatectomy on some 75 cases, and he reports as follows:—

"There have been no deaths attributable to the operation. Three patients have died since the operation—one, eighty-five years of age, five weeks after of other causes: one two weeks after of pulmonary embolism; and the third, who was in a chronic condition of uremia and had received little benefit from prolonged catheter treatment, two weeks after the operation, and the autopsy showed greatly dilated ureters and kidneys, so that nothing could have saved him."
"The results have been splendid, full power of micturition being established and perfect continence given. In only two or three cases has a perineal fistula persisted, and these from lack of after-treatment.

"I am glad to say that the results as to the preservation of sexual power have been fully borne out, the great majority of those who had unimpaired sexual power before the operation being preserved in the manly vigour after operation."

The following is my personal experience of perineal prostatectomy: —

Case I.—Patient, a man, aged sixty-four, was admitted to Sir Patrick Dun's Hospital, July 19th, 1904, complaining of frequent passage of water, stating that for the last two years he had passed water every quarter of an hour day and night, except one night fourteen months ago, when he drank too much, with the result that he was three weeks in hospital in the country. This was the only occasion on which he had retention of urine. Asking him if he got up every quarter of an hour at night to pass water, he replied, "If I did I would be dead long ago."

On Examination.—He was a fine, strong-looking old man. His urine contained some pus, but the cystitis present was only slight. He passed water frequently, about one ounce at a time; residual urine, half an ounce. He had no difficulty in passing water, and washing out the bladder it was impossible to get him to retain two ounces of fluid, thus giving the idea that he had a contracted bladder, a very unusual symptom of prostatic enlargement. His urethra did not admit the passage of the cystoscope and with the small capacity of the bladder it would have been risky. Per rectum the prostate was considerably enlarged, hard, and not tender to the touch.

I decided to perform perineal prostatectomy, and having just heard of Dr. Young's operation thought it would be a nice thing to leave the urethra and ducts intact, but unfortunately I had not full particulars of the operation.

On July 25th, using a sound to depress the prostate, and making bilateral capsular incisions, I enucleated the lateral lobes, with the aid of Murphy's hooks. Before proceeding very far
Conservative Perineal Prostatectomy.

I found the sound exposed in the wound, and that the urethra had to a large extent come away with the prostate, and the torn ends of the ejaculatory ducts were seen in the wound. A drainage-tube was placed in the bladder, and the wound partially closed, the cavity round the tube being plugged with gauze.

The patient was very well for the first few days following the operation, the drainage-tube being removed on the fourth day.

Nine days after the operation the right epididymis was swollen and very painful, and the patient's temperature began to rise; this trouble made two or three attempts to subside, but gradually increased, and one week later the right testicle was greatly swollen and the skin adherent over it, and as the fever had increased, an incision was made into the swelling; very little pus escaped and the testicle seemed necrotic.

During this time the perineal fistula was rapidly closing, and fifteen days after the operation urine was passed per urethra for the first time. As the bladder became more distended, a certain amount of hemorrhage into it took place; this recurred on several occasions, being apparently due to the stretching of some granulating surface. It was finally stopped by injecting the bladder with a weak adrenalin solution.

Four weeks after the operation the left epididymis became affected; this, however, subsided after some days. The right testicle continued to suppurate, and finally the greater part came away as a slough. This complication, due to sepsis spreading down the injured ducts, reduced the patient's strength to a marked degree, and for a time I was very uneasy about him. After the separation of the slough, he began to improve steadily, and when he left hospital he was passing water naturally every two hours during the day, and at night he sometimes slept for four hours at a time.

Septic trouble spreading down the injured ejaculatory ducts seems to be a fairly common complication. Petit reports that 12 cases in 30 suffered from epididymitis after perineal prostatectomy.

The fact that I nearly lost this patient owing to this complication has made me determined in future cases to respect the ducts and avoid their wilful injury.
Case II. was that of a patient aged sixty-three, who was admitted to Sir Patrick Dun’s Hospital on December 5th, 1904, suffering from a large inguinal hernia, which had been strangulated for twenty-four hours. I was sent for to operate. The hernial sac contained the cecum and vermiform appendix, and it was necessary to slit up the external and internal oblique muscles in order to reduce the mass. He made a good recovery. The only trouble he had was from his bladder; he passed water very frequently and complained of pain towards the end of micturition. The urine contained an enormous quantity of pus. A gum-elastic catheter was passed five days after the operation, and about one ounce of very foul-smelling pus withdrawn. His bladder was washed out daily with boric lotion, which, with urotropin internally, slightly improved the cystitis. He stated that his bladder trouble began six years ago with retention of urine. For the last four years, acting on the advice of his medical attendant, he has passed a catheter on himself every evening. For the past six months he has had severe pain towards the end of micturition, most severe after the passage of the catheter, and has had to pass water almost every hour.

On rectal examination the prostate was considerably enlarged and tender, and came unusually low down, so that, in spite of its enlargement, the seminal vesicles could be easily felt.

Although old and feeble-looking, he made such a good recovery from the strangulated hernia that I thought his kidneys were not at any rate seriously affected; and as he was anxious to have something done to relieve his prostatic trouble, I performed a perineal prostatectomy on December 19th, a fortnight after the hernia operation.

The operation was carried out in the manner I have just described. By means of the tractor the prostate was so well exposed that the use of retractors was unnecessary. The right lateral lobe was much larger than the left and was enucleated first. Both lateral lobes were easily removed without injuring the ejaculatory ducts or the mucous membrane of the bladder, but I, to some extent, injured the prostatic urethra in one place by what was probably the unnecessary use of a scissors. A small median enlargement was then enucleated through the left lateral cavity. It was unfortunately now found that the bladder contained calculi. Three in all, polished and faceted, were
Conservative Perineal Prostatectomy.

removed; the two larger were one inch in diameter, and their removal was difficult, and spoiled the carefully preserved urethra.

Had I examined the bladder with the cystoscope, or even with a metal instrument before the operation, I would, I think, not have attempted conservative prostatectomy, but would rather have done a median lithotomy, and enucleated the prostate through the prostatic urethra. But owing to the recent hernia operation, and the irritable condition of the patient, I was anxious to avoid examining him more than was absolutely necessary. The slight loss of blood during the operation was very striking, practically the only haemorrhage taking place during the extraction of the calculi.

The patient stood the operation well; continuous irrigation of the bladder was kept up for six days, when the tubes were removed.

His progress continued satisfactory, and he was up for the first time two weeks after the operation; three days later vomiting and fever set in; the wound presented a healthy, granulating appearance, and nothing was found to account for his symptoms, which, after four days, seemed to be subsiding, his temperature having fallen to 99°; it rose again the same evening to 100°; the next morning it reached 103°, the pulse 120. A careful examination of the wound and bladder again revealed nothing to account for his condition. Urine apparently free from pus flowed through the fistula; the temperature and pulse increased, and he died the following morning, twenty-three days after the operation, his temperature just before death being 105.8°. Unfortunately, no autopsy was possible.

The sudden onset of secondary septic trouble so long after the operation, and with the satisfactory condition of the bladder and wound, must be considered an unusual and unfortunate occurrence.

These two cases have impressed me with the fact that the operation of conservative prostatectomy is not more difficult than the ordinary perineal operation, and with practice should be done almost as quickly.

Now that prostatectomy is being done in younger men, the fact that the powers of procreation have not
been destroyed may often mean a great deal to the patient and his next-of-kin.

In conclusion I wish to thank Dr. Young for furnishing me with particulars of his work, and for the many things I learnt from him while at the Johns Hopkins Hospital; also to Mr. E. H. Taylor for placing the above two cases at my disposal, and for very valuable assistance and advice during the operations.

Since writing the above paper I have had an opportunity of performing perineal prostatectomy on a patient aged seventy-five. He is now, four weeks after operation, passing water naturally at three hour intervals.

1 Young. Journal of the American Medical Association, October 24, 1903. Monatsberichte für Urologie. Bd. IX. Heft 5, b. 6, 1904.

Mr. Jameson Johnston stated that he had always performed the suprapubic operation, and still thought it the right one. He did not think there were such things as lobes in enlarged prostate; the prostate was simply adenomatous, and there was no definite lobar arrangement. The ingenious idea of Lane's operation was to be admired—viz., the preservation of the ejaculatory ducts.

Mr. Edward H. Taylor stated that he had had the privilege of being present at Mr. Ball's prostatectomy operations. He had previously no experience of the perineal method, but had performed the suprapubic operation, and was much impressed in its favour. It was not very difficult, but left much to be desired, as one had to work largely by the sense of touch. He was impressed by the ease with which the prostate could be exposed by means of a curved incision in front of the anus, the facility
Conservative Perineal Prostatectomy.

with which the capsule could be peeled off the lateral lobes, and the large veins of the vesico-prostatic plexus avoided. Against the operation was the fact that it entailed an extensive dissection and division of the compressor urethrae muscle. There was considerable risk therefore of injuring the sphincteric apparatus of the bladder, and of subsequent incontinence of urine. There was much less risk of incontinence with the suprapubic operation.

Mr. Maunsell thought it was a mistake to say that all cases should be done either by the suprapubic or by the perineal operation. His experience was only with the former, because in his cases the prostatic enlargement was very marked. He would not think of removing a large adenomatous prostate through the perineum, and would employ the perineal operation only for prostates of small size. The speed with which it could be performed was in favour of suprapubic prostatectomy, and this was a point of importance with elderly patients.

Mr. Lentaigne said he had often thought that a better method than the suprapubic in prostatectomy could be devised, but he was not yet converted to the perineal route; it was much more likely to lead to septic troubles than the suprapubic, as there was much more division of tissues.

Sir Thomas Myles said he did not quite understand what became of the mucous membrane of the bladder which covered the upward bulging portions of the prostate. Was it torn in parts or was it removed en bloc? Was the ideal method to leave an intact urethra and an intact bladder? He described the method by which he had carried out the perineal operation. In his opinion, if the bladder mucous membrane covering the prostate had to be divided at all it had better be cleanly removed, otherwise a ragged surface was left and pieces of the mucous membrane might subsequently obstruct the urethra.

Mr. Stoney stated that, viewing prostatectomy from the anatomical standpoint, the best method was that which aimed at removing the prostate from within the bladder. In the perineal operation the prostatic capsule was divided and the pelvic cellular tissue was exposed to septic influences.

Mr. Ball, in reply, said he had not intended to deal with the question as to which of the two operations of prostatectomy was the better. The mortality after the perineal operation was less
than half that following the suprapubic, which fact alone would tend to make one employ the perineal route. Although in many cases suprapubic prostatectomy could be done with rapidity, yet this was by no means always the case. Also, he thought drainage was better by the perineal than by the suprapubic route. In conservative perineal prostatectomy the mucous membrane of the bladder over the prostatic lobes was supposed to be left intact.
THE APPLICATION OF PLASTER OF PARIS FACILITATED BY A NEW APPARATUS.

By W. I. de COURCY WHEELER, M.D.;
Surgeon to Mercer's Hospital;
Demonstrator of Anatomy, Trinity College, Dublin.

[Read in the Section of Surgery, February 24, 1905.]

The application of plaster of Paris to recent fractures has gained favour, not only on the Continent, but also in London, and to a lesser extent in Dublin, within recent years; however, whether plaster be applied immediately, or at any time subsequent to the injury before union has taken place, many difficulties arise in its application which are especially noticeable when it is intended to secure the entire limb and pelvis in a plaster casing when the femur is broken. For instance, it is impossible by the existing methods to fix the leg and pelvis in plaster of Paris with any certainty that the deformity has been accurately corrected, or that sufficient extension has been maintained to reduce permanent shortening to a minimum. During the operation two or three assistants were always essential, and when the pelvis was reached, the greatest awkwardness accompanied the hoisting of the patient off the bed or table at every roll of the bandage. With these manipulations it was unlikely by the time the plaster was completely on that the fragments had remained in proper apposition.

Various appliances have been thought of to overcome the difficulties enumerated above. The principles of the machine about to be described were suggested to my mind by a device described by Agnew twenty years ago, and
also by the knowledge that various surgeons, notably Kocher, of Berne, and Doyen, of Paris, have appliances to aid them in the accurate setting of fractured femurs, and their results show many cases where no permanent shortening has existed. Even Agnew, by the device just alluded to, treated fractured femur by the primary plaster method, with results very rarely surpassed at the present day. His apparatus consisted simply in an upright bar well padded and fastened to the end of a table to receive the perineum. A horizontal bar attached to the upper end of this extended backwards parallel to the patient; the latter was slung by a towel to this, so as to raise him slightly from the table, and thus to give easy access to the pelvis, which was encased in plaster of Paris, together with the entire limb on the fractured side. This machine, simple and easily extemporised, did not overcome the disadvantages of having to rely on an assistant to keep up extension during the application and subsequent drying of the plaster. Such extension is likely to be very inaccurate, and there is no guarantee that it is not relaxed at intervals, or that the two legs are ever exactly the same length. An error in this respect is rendered all the more likely by the fact that relative abduction and adduction of the two legs can usually only be roughly estimated. Indeed, obliquity of the pelvis is a factor likely in these cases to cause considerable error in estimating length.

A modification of Doyen's apparatus I have seen used allows free access to the limbs, but not to the pelvis. It registers abduction but does not provide for extension, and therefore fails to supply two of the most necessary requirements for the treatment of these injuries. Kocher's apparatus I have not had an opportunity of seeing in use. It is very perfect, but an expensive and
somewhat complicated machine, fitted with a system of dynamometers, which ensure the greatest accuracy. It is necessary before describing the application of the plaster to call special attention to an elementary point, but one often disregarded—i.e., that it is well worth while to make sure that the plaster itself is thoroughly reliable in all respects, because after an extensive application, and where much trouble has been taken to accurately reduce the fractured limb, it is most disappointing if the dressing is found defective, either failing to set or else cracking in the places where strength is most required. As an example of the latter, the groin may be mentioned, and it is wise to apply strips of aluminium or lead or bits of sticks through the plaster in this region to give it strength.

The apparatus (Figs. 1, 2, and 3) is very simple and
inexpensive. It consists essentially of two iron gates (E) about four and a half feet long, swung on an upright (B), which latter is surmounted by a padded crutch (A), so shaped that a small portion of the padded surface is horizontal (X), and will afterwards be seen to support the coccyx and lower part of the sacrum when plaster is being applied to the pelvis (Fig. 3). The crutch and upright on which it sits are supported by a heavy stand (C) with two diverging legs on each side drilled with holes, so that if required it can be screwed permanently to the ground: this, however, is not necessary. At the distal end of the gates can be seen a roller (D) over which the bandage is placed while extension is being applied. The roller is square on section in order to obtain a firm hold on the bandage, and also because, when sufficient extension is obtained, by gently pushing the handle inwards the square bar fits into a hole of the same shape and diameter, and so locks. By this means, every quarter-turn the handle can be fixed, a simple substitute for a ratchet.

Now comes an important part of the machine, a bar (F) fixed to the stand and running at right angles to it between the two gates. To the end of this bar a graduated curved iron it attached (G), so that the gates can be diverged from the middle line each to a measured amount, and so the limbs abducted to the same extent. By this method there can be no obliquity of the pelvis.

To make the apparatus complete two Salter's sportsmen's balances are necessary, registering at least fifty pounds, so that by attaching these to the rollers the extension can be accurately measured, and the same amount applied to both sides if required (H.).

I will now briefly describe the setting of a fractured femur on the new apparatus either immediately after the
injury or at any time subsequently before union has taken place, according to the indications present. The patient being anaesthetised, the apparatus is brought to the end of the table, which is raised or lowered until the horizontal part of the crutch is level with the surface of the table (Fig. 1). If the table cannot be raised or lowered, it would be better to have the crutch fitted like a bicycle saddle in order to be able to make it any required height.

A clove hitch is applied to the ankle, the latter having been first well padded with wool, or, better, a skein of knitting wool is applied to the foot (figure 8 fashion) and fixed by a bandage, as recommended by Watson Cheyne. Either of these will do for extension purposes. A leather anklet with stirrups would serve the same object.

The patient is now brought to the end of the table astride the crutch (Fig. 2), the surgeon taking charge of the injured limb. The skein of wool, or whatever is used, is attached by a bandage to the iron roller, the graduated balances intervening. Both legs having been thus
fastened, the gates are opened to the same measured extent: this will vary in different cases. The limbs can now be accurately measured and extension put on by means of the rollers until both are equal: they are then securely held by the apparatus in this position. The plaster is rapidly rolled on, there being free access to the leg, until the pelvis is reached. The table is now lowered two or three inches, the coccyx and last bit of the sacrum are supported on the crutch, the back becomes arched, and a space remains between the table and the greater bulk of the pelvis, round which the roller bandage can be continued without any assistance, and little disturbance of the patient (Fig. 3). It is recommended not only to encase the injured limb and the pelvis, but also to continue the plaster down the sound limb as far as the knee, and thus secure the immobility of the pelvis, and provide counter-extension at the perineum.

It is wonderful to see how well patients can walk with crutches, and even sit down after a fashion, with one and
a half legs in plaster together with the entire pelvis, and it is safe to allow them to do this in many cases four or five days after the accident, provided the plaster be firm, all the weight be kept on the sound leg, and there be a bandage round the neck supporting the injured limb.

It must not be thought that this device is only of use to those who apply plaster as a primary treatment; it is hoped that it may also be of service to everyone who uses plaster of Paris at any period during the treatment of fractured thigh bones, and also during certain operations on the bones where extension is required and a plaster dressing used.

I have treated five or six cases in this manner with success in every instance: the plaster was rapidly applied, scarcely any assistance was necessary, and the patients expressed themselves free from pain and more comfortable than when a M'Intyre's splint or weight and pulley was used. An oblique fracture in an adult near the middle of the shaft is the most suitable case. In a great number there should be no permanent shortening in fractured femur, and there have been many such results in this country and abroad.

I am indebted to Dr. Haughton's paper read before the Academy two years ago for much information on this subject; and to Dr. Harnett, one of the house surgeons at St. Thomas's Hospital, London, for showing me many cases of fractured femur treated there by the primary plaster method.

One of the residents in Mercer's Hospital (Mr. Smith) was kind enough to act as model while photographs were being taken from which the accompanying illustrations have been made. Finally, it is quite obvious that some cases of fractured femur could not be treated by the method described, with the apparatus in its present form,
but a few alterations which have been suggested to me would make it, I think, suitable for almost every case.

Mr. E. H. Bennett was of opinion that the apparatus was probably useful in general hospital work, but could hardly be used by country practitioners.

Mr. Tobin thought that Mr. Wheeler’s method would be useful in the treatment of fractures of the femur in field service.

Mr. W. S. Haughton considered the device both simple and efficient. It saved labour, made the subsequent nursing simple, and saved the surgeon paying numerous visits.

Mr. Wheeler, in reply, stated that the apparatus was very portable, was by no means difficult to make, and cost about two pounds.
FRACTURE OF THE TIBIA.

BY EDWARD H. BENNETT, M.D., F.R.C.S.;
Surgeon to Sir Patrick Dun's Hospital;
Professor of Surgery in the University of Dublin, &c., &c.

[Read in the Section of Surgery, February 24, 1905.]

I desire to submit to the Surgical Section of the Academy an example of fracture of the tibia only, which is most exceptional in its character.

"It is" (writes Stimson) "quite rare for the tibia alone to be broken when the fracture is by indirect violence, for the force continues to act, if only for a moment, and breaks the weaker fibula all the more easily, and usually at a higher point than the tibia." Every collection of fractures of the leg bones contains examples of fracture of the tibial shaft remote from its articular extremities, in which the tibia alone has been broken by direct injury, and every surgeon has seen examples of these in practice. If the fractures are simple none are more favourable for surgical treatment, for they remain without displacement and unite without deformity. But oblique fractures of the central portion of the shaft, without accompanying fractures of the fibula, are, indeed, rare. The following case came under my care this winter, and I am able to place all the facts before the meeting in these excellent skiagraphs by my colleague, Dr. Watson. I was obliged to get a picture of the skeleton of the entire leg, as someone might say that I had overlooked the possibility of a fracture of the fibula either very high up or very low down. With the picture of all the leg this idea is excluded.
A young girl, fifteen years old or thereabouts, active, and well-grown, was challenging a girl of her own age to jump from a loft window to the ground, a height of about ten to twelve feet. In the play that took place she was shoved and had to jump. She lit on the ground on her feet, and fell backwards sitting. She was able to stand up, but could not put any weight on the injured limb, nor walk a step. A man who lived close by handled the limb, and pronounced that there was no fracture. At the moment no deformity appeared, and no swelling of the limb had time to set in. The girl's mother put her on a car and brought her to hospital which was only a very short distance off. The pain and loss of power indicated the existence of fracture, but no crepitus or abnormal motion in the length of the bone could be obtained. Presently, as doubt existed in the minds of the resident staff, the X-rays were put to work, and disclosed this rare fracture. Knowing my interest in these injuries, the case was very soon placed under my care, and I can present the photographs. The progress was uneventful, and the union was perfect in the ordinary time.

In the photographs of this injury the fracture occupies nearly four inches of the shaft almost at its middle, passing with great obliquity from above and without downwards and inwards. I place here for contrast a recent example of a fracture of the tibia only, a transverse dentate fracture, caused by a direct blow on the bone in the line of fracture, and I show also a good example of the perfect treatment of such an injury possible through the kindly aid of the unbroken fibula. My photograph of the bone, taken about five weeks after the accident, shows that aid of the fibula is just as efficient in the treatment of the oblique fracture as in the case of the transverse.

When an adult leaps for life, and lights on one or both feet the heel bones in one or both feet may break, and, again, the head of the tibia may break after the manner of Heydenreich. Of both these injuries I submit
Fracture of the Tibia.

examples, but of the oblique fracture of the shaft of the tibia from indirect force I possess only these skiagraphs as, with the history I have quoted, the sum of my experience.

Mr. Haughton said that in studying the long bones stereoscopically one found that the oblique direction of the lamellæ was the rule and not the exception, and that when one regarded fractures from the anatomical standpoint it could be seen that their planes ran parallel to the direction of these bone lamellæ.
SOME CASES OF JOINT EXCISION.

By DENIS KENNEDY, F.R.C.S.;
Surgeon to Jervis Street Hospital, Dublin;
Surgeon to the Children's Hospital, Temple Street, Dublin.

[Read in the Section of Surgery, February 24, 1905.]

The havoc that is wrought by tuberculous disease in joints, the number of cases that crop up daily for treatment, and the inefficiency frequently of such treatment is my excuse for bringing briefly under your notice the following cases:—

Case I.—W. G., aged thirty-two, came to me about four years ago with the following history:—About six years previously he sprained his knee breaking a stick across it. Swelling and pain supervening, he consulted a bone doctor, who pretended to reduce a dislocation of the knee-cap. The patient's subsequent history was simply one of tuberculous knee-joint, going from bad to worse. He was constantly under the care of his local doctor, besides being treated in a couple of Dublin hospitals from time to time, the usual routine treatment being apparently followed—counter-irritation, fixation of the joint and opening of abscesses according as they formed. As time went on his condition, both locally and generally, seemed to have got steadily worse. When I saw him he was in a pitiable state. He hobbled about on a pair of crutches, the knee-joint was fixed at right angles, greatly enlarged, and many sinuses were present. He had a bad cough, was very emaciated, suffered from night sweats, and on examination evidence of a large cavity in the right lung was easily obtained. His mental condition was bordering on despair. At his urgent request I decided to undertake an excision of the knee, permission being readily given for amputation if necessary. I carried out the operation in Miss Mason's private hospital in Baggot Street, assisted by Surgeon Tobin and Dr. Dargan, who gave the anaesthetic. The bones were extensively diseased, as well as every part in the neighbourhood
Some Cases of Joint Excision.

of the joint. The great difficulty of the operation was to bring the leg straight even after a section of the hamstring tendons. I used Tobin's calipers and dowel to fix the bones together, and, as a proof of their efficiency, I may mention that just before the patient was brought back to bed he became violent, and actually rolled on to the floor from the operating table; nevertheless, the bones never moved. He made an uninterrupted recovery. In less than six weeks he was up and about, and before three months I showed him at the Academy, then walking without a stick. I found massage of the greatest possible benefit in restoring the wasted muscle of the limb, and consequently enabling the patient to get about much earlier than otherwise. He is now in perfect health, the lung trouble having completely disappeared, and he is able to work all day without the least difficulty on his farm.

The points of interest that appeal to me in this case are: (1) the absolute failure of palliative treatment, though persistently and carefully carried out; (2) his rapid and complete recovery after excision; (3) the fact that in spite of his lung trouble the administration of ether did not apparently do him the least harm; (4) his lung seemed to improve once the source of infection at his knee-joint was removed. The condition of the knee-joint, being riddled with sinuses, and in a state of ankylosis at right angles for a long period, seemed to contraindicate excision, while his general condition seemed a bar to any extensive operation at all. As a matter of fact, he was refused operation in two city hospitals a short time previous to his consulting me. His recovery goes to show that with aseptic surgery results can be obtained which were never dreamt of in pre-aseptic days. I attribute the rapid healing of his wound principally to two causes:—(1) The use of Tobin's clamps in fixing the bones together. (2) Thorough douching of the joint during the operation with an aseptic fluid. In all cases of excision of the knee I consider fixation of the bones by
either clamps such as these, or by wire suture, as an essential detail. I have employed both means, and by both have got satisfaction. For absolute fixation, no suture equals the clamps. By thus fixing the bones, pain after operation is nil, bony union is rapid and complete, and, above all, subsequent flexion of the limb at the site of the operation does not occur. For proof of rapid union after the calipers I need only call your attention to the case of excision of the knee which Mr. Tobin has exhibited to-night. This patient has now been only three weeks operated on; nevertheless, he is up and about without splint or appliance of any kind whatever.

Case II.—Miss A. R., aged fifteen, came to me about three and a half years ago with the following history:—Eighteen months previously, while at a boarding school in the South of Ireland, she wrenched her right shoulder playing at tug-of-war. Some pain and swelling of the joint followed, and for this she was treated by the medical attendant at the school. She improved for a time, but some pain and stiffening seemed to have remained. About four months subsequently, being at home on her holidays, she also consulted a bone-setter, who pretended to reduce a dislocation of the shoulder-joint, using great violence and causing the patient great pain. Improvement followed for a time and she returned to school. Soon, however, her trouble became much worse, and after a short time she had to leave school on account of the pain and stiffening of the joint. When I saw her, her condition was as follows:—She complained of a constant aching pain in the joint, and any attempt at active or passive movement simply produced torture. The muscles round the joint were wasted, the head of the humerus was enlarged and tender. There was no synovial swelling and not a trace of suppuration. Generally she was anaemic and frail-looking, but no organic disease was present. I sent her to a private ward in Temple Street Hospital, and subsequently carried out an excision of the shoulder. The condition I found was very typical of rarefying osteitis. The head of the bone was almost bare of cartilage, and small cavities were present throughout its entire
extent. It was simply honeycombed. There was no pus, nor tuberculous débris, nor effusion into the joint. The glenoid cavity was healthy. Her recovery was protracted, but complete; and I may mention that she is now undergoing a course of technical training in cookery at Kildare Street School, and her teachers have never yet discovered that there is anything wrong with her arm.

Case III.—Miss K., aged twenty-seven, consulted me about eighteen months ago. Her history was as follows:—Seven years previously she noticed a swelling in her left elbow coming on apparently without cause. Some pain was also present on moving the joint, but she continued to perform ordinary household work. After about two years the trouble became worse. She was advised by a surgeon in Chicago, whom she consulted, to return to her native air in the South of Ireland. This she did, but her elbow appeared to get gradually more useless to her. She suffered a good deal of pain, especially on movement, and swelling occasionally appeared. She then placed herself about two and a half years ago under the care of an eminent surgeon in Dublin. He seems to have done everything possible in the way of palliative treatment, including puncture, firing, and fixation of the joint, and subsequently massage, without apparently the least benefit to the patient. After spending about six months in the country, she consulted me, and when I saw her the following condition was present:—Pain nearly always present in the elbow, but greatly increased on the slightest attempt at active or passive movement. The arm was nearly fixed in a straight position, and the muscles were flabby and wasted. There was no swelling about the joint, no trace of sinus or suppuration, but the lower end of the humerus seemed enlarged and tender. The diagnosis was by no means clear, but, fortified by my previous experience of the shoulder-joint, I diagnosed a rarefying osteitis of the humerus, and recommended excision. This I carried out in Miss Mason's Home in Baggot Street, and I found exactly the same condition present that I had found in the shoulder-joint of the other case. The cartilages of articulation had practically disappeared, and all the bones—humerus, ulna and radius—were again in this honeycombed condition. There was not a trace of pus anywhere,
and no synovial swelling. The patient made a rapid and good recovery, and six months ago, when I saw her last, she seemed to be on the high road to having a very useful arm, extensive, active and passive motion being easily carried out.

These two cases are interesting to my mind, first, from the fact that extensive bone disease was present in each case without any swelling of the joint and without any suppuration, although the disease had apparently been present for years. What was the nature of the disease? It was certainly not syphilitic, and I am not at all sure that it was tuberculous. I have begun to think lately that the word tuberculous covers an amount of ignorance. Secondly, the cases were difficult to diagnose. In both cases I was much aided in coming to a decision by a skiagram. I should like to have the opinion of others as to the value of skiagraphy in diagnosing bone disease. Just one word as to the operations. In each case I removed only as little bone as was absolutely necessary. With early passive motion there was no danger of bony ankylosis, and the possibility of a frail limb was averted. The less bone that is removed in excisions of the upper extremity the better will the limb be for the patient.

Case IV.—This case is purely traumatic, and, consequently, different from the others. Molly R., aged five, was admitted to Temple Street Hospital two years ago with the following history:—Sixteen months previously she fell on her shoulder, and from that time she had no movement in her shoulder-joint. When I saw her the joint was completely ankylosed, and attempted movement produced pain. A skiagram showed a fracture of the upper end of the humerus, which I assumed to be epiphyseal separation. I opened her joint and found the following condition:—There was an epiphyseal separation about the line of the surgical neck of the humerus, and, again, the articular head was completely separated from the remainder of the epiphysis, and lying loose in the joint cavity. Some fibrous
bands connected the diaphysis with the lower part of the epiphysis. I removed the articular head, freshened up the opposing surfaces of diaphysis and epiphysis and sutured them with wire. The union was perfect, and the child has an arm difficult to detect that there is anything wrong with it. She is able to perform every movement perfectly.

The subject of tuberculous joints generally is much too great a one for discussion in a short communication such as this. Just one point as to treatment. The difficulty is to know exactly when palliative treatment must give way to radical. There is one conviction, however, that is forced upon me the more I see of tuberculous joints—that is, that a tuberculous joint in a child may be cured by palliative means. In an adult anything short of the excision of the joint is practically useless. Lastly, I must pay my tribute to the utility of massage in restoring a limb to usefulness when the muscles have been wasted from prolonged disease and from disuse.

Mr. Bennett congratulated Mr. Kennedy on the first of his cases—viz., that in which an excision of the knee had been successfully performed in a patient who had symptoms of a phthisical cavity in one lung.

Mr. Tobin had seen some of Mr. Kennedy's cases. The first of these, which had just been recorded, was one which few would have cared to undertake.

Mr. Haughton discussed the diagnosis of early tuberculosis of joints by the aid of X-rays, and gave it as his opinion that a positive diagnosis could be made if the disease had been present for some weeks. Stereoscopy was of great value in these cases. By its aid areas of rarefaction could be discerned, and their true relations to each other and to the joint made out.

In reply, Mr. Kennedy expressed his sense of obligation to Mr. Haughton for his hints as to diagnosis, as cases often cropped up in which it was far from easy to say if bone disease was really present.
PAINLESS HÆMATURIA.

By LEVESON-GOWER GUNN, M.D., F.R.C.S.;
Assistant Surgeon, Adelaide Hospital.

[Read in the Section of Surgery, March 24, 1905.]

The presence of blood in the urine has for a long time been considered as a cardinal symptom of disease of some portion of the genito-urinary system. It is the symptom which, with perhaps the exception of very acute pain, appeals most forcibly to the patient.

Pain, irritation, and frequency of micturition may be, and often are, suffered for months without seeking medical aid, but should the sufferer pass blood in his urine, and recognise it as such, he will, as a rule, lose no time in seeking advice for the relief of his complaint.

To the medical man, to whom such a case comes, hæmaturia as a symptom will arouse both interest and anxiety—anxiety as he knows only too well what severe and even fatal disease may first show itself by this symptom, and interest in so far as he realises that the diagnosis will, as a rule, call for the closest care and observation on his part—the road to correct diagnoses in genito-urinary disease being beset by many pitfalls.

In the majority of cases hæmaturia will be associated with pain, and although in the genito-urinary tract pain is often referred from the actual lesion as from one kidney to the other, or from the kidney to the bladder, or the bladder to the penis, still the presence of pain will, generally speaking, help in making the diagnosis as to the locality that is bleeding.

In cases of painless hæmaturia this difficulty of
diagnosis is greatly increased, while the necessity for an accurate diagnosis is in no way diminished, as the absence of pain does not mean an absence of danger to the patient.

In the first place we must find out whether the blood is coming from the urethra, the bladder, the ureter, the pelvis of the kidney, or the kidney itself, and if from the ureter or kidney, whether from the right or left side.

This problem once solved, then comes the more difficult one of the cause of the haemorrhage.

Bleeding from the urethra can, as a rule, be diagnosed by the fact that the blood may come at times unmixed with the urine; in lesions of the bladder, ureter, or kidney the blood will be intimately mixed with the urine. It is to the differential diagnosis of these cases I wish to call your attention, and to the extent that cystoscopy, kryoscopy, and segregation of the urine will help us in making this diagnosis in the treatment and in the prognosis of such cases.

Five cases of painless haematuria have passed through my hands since last August. All had this common symptom, but the cause of the bleeding differed widely in each case. I have been able to verify the diagnosis absolutely in three, almost certainly in the fourth, while in the fifth I have not yet been able to prove whether the provisional diagnosis is correct or not. This case I now lay before you.

Case I.—In August, 1904, I was asked by Dr. Smyly to see Miss E. R., aged fifty-seven years, a patient in the Adelaide Hospital. Her history is as follows:—She has always been strong herself; her family are all healthy; there is no tubercular disease among any of her relations. Patient works in a factory. In June, 1904, she noticed the urine rather red after a heavy day's work—this happened several times during June and July.
By Mr. Leveson-Gower Gunn.

She consulted a doctor, who told her she was passing blood, and advised her to go up to hospital. She is a thin, spare-looking woman, with good colour in her face, and looks, as she feels, quite healthy. On examining her abdomen both kidneys can be easily felt; both move freely on respiration; the left kidney, which feels rather small, and is quite painless, can be readily held down between the examining hands; no other abnormality. Urine 1024, clear, acid; no albumen, blood, or pus. While she remained quietly in bed the urine remained normal. This she said was always the case, the blood only occurring if she had hard work to do, or much walking or standing. She was sent out for a brisk walk, and that evening the urine contained a little blood, but was clear again next morning. The blood was intimately mixed with the urine; no blood casts could be found.

I decided to examine the interior of the bladder with a new cystoscope. This is Professor Nitze's latest design, which differs in many ways from the older forms of cystoscope, and possesses numerous improvements.

The size is only that of a No. 12 English catheter, and renders the passage of the instrument comparatively painless to the patient and easy to the surgeon.

The telescopic part has been enlarged, and gives a larger and brighter field of vision. It is always in focus, and does not require the constant adjustment of the older instruments.

The lamp is larger and brighter, using a current of 12 volts. It is specially packed round with asbestos, rendering it what is technically called a cold lamp. That is, it will burn in the air for some time without heating, and for a considerable time immersed in water without becoming dangerously hot.

By this lamp one of the greatest drawbacks to cystoscopy has been eliminated.

The speedy way the cable carrying the current can be attached, and as readily removed, greatly facilitates the
ease with which it may be introduced into the bladder, and lastly, the entire surface of the instrument has been kept as smooth as possible, so that it can be efficiently cleaned and sterilised—a point of no small importance.

There are three conditions that must be present before a cystoscopic examination of the bladder can be satisfactorily carried out:

1st. The urethra must allow the passage of a No. 12 catheter. This, I think, is obvious.

2nd. The bladder must be capable of containing at least 4 ozs. of fluid. Less than this amount will not give sufficient room to manipulate the beak of the instrument, and will not allow a proper view of any part of the bladder—the wall of which may easily be injured in the attempt.

3rd. It must be possible to fill the bladder with a clear fluid. To obtain this in most cases is only a matter of patient washing out, but bleeding may be too free or the discharge of pus too continuous ever to allow a proper inspection.

To these I might add, perhaps, that the operator must possess a very thorough knowledge of the normal bladder as seen through the cystoscope—and this differs largely from its appearance in the post-mortem room—or the inspection of the bladder will not give him much information.

Cocain injected first into the urethra, and later on into the bladder, makes the operation almost painless, and renders a general anaesthetic very rarely necessary.

The patient's bladder held 10 ozs. of fluid, and I was able to examine it thoroughly, and satisfy myself that there was no abnormality there that could cause bleeding. The mouths of both ureters were normal, and clear urine issued from each. I examined the bladder again after she had taken some exercise,
and found, as I expected, blood coming from one ureter; the urine on the right side came in a clear eflux, while that from the left ureter was clouded with blood. The appearance of the mouth of the ureter was against the presence of a stone in the ureter or in the pelvis of the kidney, and an X-ray photograph confirmed this, showing no signs of stone. The diagnosis lay between a tubercular deposit in the left kidney, a new growth in the pelvis or in the kidney itself, an angiomatous condition of one of the renal papillæ, or that the bleeding might be due to the mobility of the kidney. The absence of either fever, pus, or tubercle bacilli was against tubercular disease; the small size, smooth surface, and absence of pain was against a new growth in the kidney. I passed a carefully sterilised catheter (by means of this cystoscope) up the ureter into the pelvis of the kidney; this caused no bleeding, as it would almost certainly have done had there been a villous growth in either ureter or pelvis. I believe that in this case the bleeding was due to enlarged blood vessels in the renal papillæ, which probably bleed only when the kidney drops after extra exertion, this misplacement probably causing some engorgement of the vessels. I suggested exploring, and, should there be no sign of a new growth present, fixing the kidney. The patient was unwilling to have any operation done unless I considered it absolutely necessary, and under these circumstances I sent her home with orders to report herself every six months. I saw her again in February, 1905. Her condition is unchanged, and her general health is excellent.

In a case of this kind one must always remember the possibility of malignant disease of the kidney starting in this way, and I have given a guarded prognosis in this case, and will urge that the kidney be at once examined if it shows the least sign of enlarging.

Case II.—Mr. H., aged fifty-one years, a commercial traveller. I was asked to see this patient in September, 1904, by Dr. Bewley. His family history is good; he has had no illness of any importance prior to April, 1904, when his present trouble began. He was staying in London at the time, and was greatly startled one morning to find a quantity of blood passing in his water. He sent at once for a doctor, who kept him in bed for a couple of
Painless Haematuria.

days. The bleeding stopped in 24 hours, and he felt none the worse. At intervals of from two to six weeks these attacks recurred, sometimes preceded for a few hours by a feeling of malaise, but as a rule coming on without any warning. In August the bleeding was very severe, lasting for nine days, and leaving the patient very weak. Another attack occurred in September, when I was asked to see him. Patient is exceedingly pale, and has evidently lost a great deal of blood. He feels weak, but has not lost weight; urine sp. gr. 1012; acid; contains a quantity of blood; no pus; some flat epithelium. He has no pain anywhere, and nothing abnormal can be felt. He says he has been a rather heavy spirit drinker for some years.

I washed out the bladder and examined it with the cystoscope; it appeared quite normal. The mouths of both ureters were normal, and clear urine was coming from the right; from the left ureter clots of blood, moulded to the shape of the ureter, were being forced out with gushes of blood-stained urine. These ureteral blood-casts were lying coiled up in the lowest part of the bladder. X-ray photograph showed, as I expected, no stone. With rest in bed, and large doses of chloride of calcium, the haematuria stopped in a few days. The urine then showed sp. gr. 1012; acid; trace of albumen; a few granular tube-casts.

I believed the trouble in this case was either a new growth in the kidney or one of these rather rare cases of interstitial nephritis which may bleed freely. It was obvious that active measures must be taken to prevent another haemorrhage, as the patient was now so weak that it seemed probable that a further bleeding might prove fatal. As it is a matter of great importance to know the functional soundness of the patient's other kidney, I separated his urine with Professor Luy's segregator, which fits into the floor of the bladder, dividing it into two compartments by this rubber septum, and through these lateral openings collects in these glass tubes the urine as it flows from either ureter.

The urine so obtained from the right kidney I examined by kryoscopy, the principal of which is briefly as follows:

The kidneys, by their osmotic power, separate from the
blood the salts dissolved in the urine. Should this power be impaired this separation will not take place, but as the molecules continue forming in the body and passing into the blood it follows that the total amount in the blood will be increased, and in the urine will be diminished.

The molecular concentration in the blood will be too great, and that in the urine too low.

The greater the molecular concentration in the urine separated by the kidney the lower the temperature required to freeze the fluid will be.

By kryoscopy we can measure the concentration of the blood and of the urine in a rapid and fairly accurate manner, and by comparing the two results gain much information as to the functional activity of the kidney.

The apparatus I use is Bergmann's. It consists of a delicate thermometer graduated to \( \frac{1}{100} \) of a degree Centigrade running from 0 to \(-5\) degrees. This thermometer passes through a rubber cork into about 15 c.c. of the mixture to be frozen. This is surrounded by a freezing mixture of ice and salt.

The fluid is kept well mixed by means of a platinum mixer until it freezes solid right through. The height at which the mercury stands is read off, the fluid is allowed to thaw, and the experiment is repeated again.

Blood normally freezes at \(-0.56^\circ\); an increase of this freezing depression point to \(-0.58^\circ\) or lower shows renal insufficiency.

The depression freezing point of the urine is not nearly so constant as that of the blood, and varies in health from \(-1.2^\circ\) to \(-2.2^\circ\). If it should rise to \(1^\circ\) below zero or higher than this, it shows that both kidneys are diseased. For as long as either kidney has a sufficient amount of healthy parenchyma left to separate these sub-
stances from the blood, there will be no alteration from the normal freezing point of either blood or urine.

It is possible then by this method to estimate the functional capacity of both kidneys, and by the use of the segregator or the ureteral catheter to estimate separately the powers of each. It is claimed that this alteration in the freezing point is present in cases of renal mischief long before any general symptoms of kidney insufficiency occur.

There are two conditions which may cause the urine to freeze at a higher point than normal, but are still compatible with a healthy renal epithelium:

1st. If there has been a very large intake of water.
2nd. If a profound anaemia is present.

There are four conditions also which may cause the blood to freeze at too low a point without kidney insufficiency being present:

1st. Increased abdominal pressure, especially when such an increase is due to the presence of any large abdominal tumour.
2nd. If the blood is not receiving a proper supply of oxygen from the lungs.
3rd. If the patient’s diet does not contain a good supply of carbohydrates.
4th. One-sided renal pain, due to a stone or tumour, may cause a temporary insufficiency in the other kidney.

Some of these conditions can be easily rectified; the intake of water can be regulated for some time before the examination is made. If there is any doubt about the blood receiving enough oxygen from the lungs it is not a difficult matter to allow a little oxygen to pass through the test tube before it is frozen. A liberal carbohydrate diet can be arranged for, and, as a rule, it will be possible
to choose a time during which the patient is free from pain to make the necessary examination.

The two remaining conditions—that of profound anaemia and increased abdominal pressure resulting from a tumour—cannot probably be got rid of, and in these cases abnormal kryoscopic readings must be accepted as of doubtful value.

In no case can a single estimation of the urine alone be taken as of much value, but if done in conjunction with the blood, and especially if abnormal readings are obtained on several occasions, its significance is then of the greatest importance.

In this case, although a severe anaemia was present, the freezing point of the blood and of the urine from the right kidney was normal, showing that there was no insufficiency of this side. In September, 1904, I removed the left kidney with some difficulty, as it had almost no pedicle, Mr. Gordon kindly assisting me. The kidney showed marked interstitial fibrosis, with numerous small haemorrhages scattered through its substance. Sixteen ounces of urine passed the first day, and this amount has steadily increased until now the patient passes daily from forty to fifty ounces of urine; it is free from albumen, and there has been no bleeding since the operation, nor any sign of renal insufficiency.

Case III.—Mrs. M. C., aged forty-seven years, a professional nurse. I was asked by Dr. Purefoy to see this patient in October, 1904. About a month before this date the patient noticed her urine dark in colour on several occasions; always, if from any cause, she had been unable to empty her bladder for some time. She has had some frequency of micturition during the day, and has been obliged to get up and empty her bladder at least once during the night. This has been going on for over a year. Dr. Purefoy, to whom she showed the urine, told her that she was passing blood in it, and sent her on to me. She is a stout, healthy-looking woman; says she has never had any illness, and feels well at the present time. She has no pain; the frequency of micturition does not seem to trouble her,
but she is nervous about the haematuria; urine sp. gr. 1022; alkaline; a few pus cells; some diplococci, and a few short thick bacilli; no blood.

I washed out the bladder, which held only six ounces with comfort, and examined with a cystoscope. The whole surface of the bladder was slightly reddened, vessels were enlarged, and here and there small tags of lymph were hanging from it; it presented the appearance of a chronic cystitis. The mouths of both ureters appeared slightly reddened, but were otherwise normal; clear urine came from each. Scattered over the trigone round the sphincter and the mouth of the left ureter are a number of small raised ulcers, some in process of healing, others breaking down. On injecting a little more fluid into the bladder, and so stretching the floors of these ulcers, several of them began to bleed, and so explained the cause of the haematuria. The ulcers were all superficial, and were, I believe, due to a secondary tubercular infection from one or other kidney. Primary tuberculosis nearly always occurs as a single large ulcer or as scattered submucous patches. Secondary tuberculosis, on the other hand, often occurs as multiple small ulcers, sometimes grouped round the mouth of the ureter, through which the infection has come, the mouth of the ureter itself being often ulcerated. Neither ureter was ulcerated in this case, but from the grouping of the lesions I would suspect the left kidney to be the one at fault. I examined the urine several times, but could not find tubercle bacilli. I have since learned from her doctor that two of her children are tubercular, and that she herself had a smart attack of hemoptysis just three years ago, so I have little doubt but that the diagnosis of secondary tuberculosis is correct. The ulcers have improved considerably under treatment, and there is no sign of fresh infection having taken place.

Case IV.—Mr. M., aged forty-seven years, a retired civil servant. I was asked in November, 1904, to see this gentleman in consultation. He gave the following history:—He has always been rather delicate, and has lived a good deal abroad. In the beginning of 1904 he first passed some blood in his urine; he had some frequency, but no pain. His doctor sounded him twice for stone, but with a negative result. These attacks of haematuria continued up to the time I saw him in November.
They occurred at varying intervals, causing no pain, and giving no clue to the locality that was bleeding. The bleeding often lasts only a few hours, and never for more than a couple of days. The intervals during which the urine has been free from blood have varied from a few days to six or seven weeks.

Patient is a spare man, with a rather sallow complexion, but does not look anaemic. He feels weak, and thinks he has lost some flesh during the past year. On examining the abdomen one could see a marked fulness in the left hypochondriac region, and could feel with ease the edge of the spleen projecting a hand's breadth below the ribs. This enlargement seemed to extend back into the flank, and mask the kidney. We felt sure that the spleen was much enlarged, but were not at all sure of feeling the left kidney at all.

I washed out his bladder and examined it carefully. Unfortunately, about an hour or two before I arrived the bleeding had stopped, and the urine was now quite clear. The bladder walls were quite healthy. A small pedunculated adenoma grew from the back of the middle lobe of the prostate; its surface was unbroken, and it could not be causing the bleeding. The mouths of both ureters appeared normal, and I saw clear urine flow from each.

This examination proved that the hæmorrhage was not vesical. We suspected that the left kidney was at fault—either that there was a growth in the kidney itself or that its circulation was interfered with by the pressure of the enlarged spleen. We decided to do nothing until the next hæmorrhage occurred, when I hoped to be able to ascertain definitely from which side the bleeding was coming. Urine was pale; sp. gr. 1018, acid; contained a trace of albumen; a few white cells, and a good deal of flat epithelium. Three weeks later bleeding recurred. The patient was now complaining of a sense of fulness in his left side and a constant desire to pass water. The swelling in the left side seems to have increased considerably in size. I passed a segregator into the bladder; there was an abundant flow of urine from the right ureter, while from the left only a few drops of blood-stained urine came. It was decided to explore the left kidney at once. Next day I assisted at an operation on his left kidney; we found it enlarged to fully three times its normal size, and obviously the seat of a new growth. The kidney
was removed, and the growth proved to be an adenoma invading the body and pelvis of the kidney. It is interesting to note that the spleen was not appreciably enlarged, and had evidently been pushed forward by the renal growth.

Case V.—Mr. H., aged sixty-four years, a retired Government official. He was sent to me by Dr. Wallace Beatty, in January, 1905, and gave me the following history:—He has always been a strong healthy man. Some two years ago he began to have some frequency and increasing difficulty in passing his water—in fact the usual train of symptoms that follow a senile enlargement of the prostate. Two months before I saw him he had a smart attack of haematuria, lasting for three days; he had no pain, and could assign no cause for its onset. A second rather less severe haemorrhage had just ceased when he consulted me. On rectal examination the prostate could be felt generally enlarged, the right lobe feels very hard; the seminal vesicles were hard and larger than normal. Urine, 1024; acid; trace of albumen; a few white cells, and some cocci; no tubercle bacilli. I examined with a prostatic cystoscope, and found the bladder walls were healthy; the enlarged prostate could be well seen standing up into the bladder. From the back of the middle lobe sprang a red nodular growth, slightly pedunculated, covering the left ureteral orifice, and invading the bladder wall. I believed this growth to be a carcinoma.

Sir Charles Bali saw this case with me, and agreed with this diagnosis, and with his assistance I removed the growth, a portion of the bladder wall, the enlarged prostate, and seminal vesicles through a supra-pubic incision. Microscopic examination of the part removed confirmed the diagnosis. The case has done well, and he is now only troubled with some loss of control if he tries to hold his water for more than two or three hours at a time, and this, I hope, will improve.

This paper has, I fear, already been too long, and I cannot now venture to discuss all the causes of painless haematuria. My object in bringing these cases before your notice at present is to try and demonstrate that these methods of cystoscopy, kryoscopy, and
segregation, although by no means infallible, yet will materially aid in forming a correct diagnosis in this difficult class of urinary cases.

Mr. Edward H. Taylor, in congratulating Mr. Gunn on his communication, said that it confirmed his belief in the great value of cystoscopic examination in urinary cases. In the case recently under his care, Mr. Taylor had derived very valuable information concerning obscure bladder symptoms with Mr. Gunn's assistance. Cystoscopy and ureteral catheterisation appeared to be methods of investigation requiring such a high degree of technical skill as to belong to the specialist rather than to the general surgeon.

Mr. T. E. Gordon agreed with what had been said by Mr. Taylor. He thought that the methods of examination described by Mr. Gunn were essential in a large proportion of cases, and would in future devolve on specialists.

Mr. G. Jameson Johnston congratulated Mr. Gunn on his contribution to the recent advances in renal surgery and the manner in which he had worked out his cases.
ON THE TREATMENT OF PURULENT CAVITIES.

By ROBERT H. WOODS, M.B., F.R.C.S.;
Throat Surgeon to the Richmond Hospital.

[Read in the Section of Surgery, March 24, 1905.]

To judge by recent and current medical literature some confusion of thought exists concerning the principles underlying the treatment of purulent collections. One result of this confusion is that methods of treatment found suitable in particular cases are presumed to be applicable universally. Thus in every text-book I have seen, whether on general or departmental surgery, it is considered that the principle of drainage, so successful in dealing with soft-tissue abscesses, is a prime factor in the cure of chronic purulent collections inside fixed walls.

I shall attempt, among other things, to show that simple drainage is of small value in such cases.

In every soft-tissue abscess the tissues which surround the abscess have been by its growth distorted from their natural position, to which place they always tend to revert. The force of this reversion, or, what is the same thing, the pressure of the pus, is determined as to its degree by the following three factors:—First, the rapidity of the growth of the abscess; secondly, its situation; and, thirdly, its size.

As regards the first, a rapidly-growing collection pushes the tissues more rudely aside, it gives them less time to adapt themselves to its presence; their natural elasticity is therefore less impaired than in a case of a slowly-growing one.
Secondly, the situation in which the abscess grows. If the surrounding tissues are soft and lax the tendency to revert will be feeble and the pus pressure correspondingly lower than when the tissues are denser and tougher. By surrounding tissues is not necessarily meant only those directly bordering on the abscess, but all structures which tend to be distorted by its presence. Fibrous-tissue planes, though remote from the wall, may help it enormously in its reversion.

When we come to consider the influence of the mere size of the abscess on its pressure, we find the question is not quite so simple as it might first appear. It might be thought, since the bigger an abscess grows the more its surroundings are distorted, that the pressure inside would in all cases increase proportionately, but there is an important factor which interferes with this. In cases where we may regard the force of reversion or pus pressure as being due to tension in the sac wall, the pressure varies, according to a well-known physical law, not only as the tension per unit of length, but also inversely as the radius of curvature.

\[ P = \frac{T}{R} \]

Therefore if the sac wall tension remains the same, the larger the sac the lower will be the pressure inside and conversely. Now, in a growing abscess the tissue fibres at the sac wall are always, except at the start, on the point of giving way, and are in other words at their maximum tension. Therefore I think we may say that the tension per unit of length of the sac wall is constant

* For the cases of a cavity having a single curve. If the wall is curved in two directions the formula is \( P = T \left( \frac{1}{R} + \frac{1}{R_1} \right) \) where \( R \) and \( R_1 \) are the radii of curvature.
On the Treatment of Purulent Cavities.

or nearly so for a given abscess, no matter what its size, so long as it is confined to the same tissue.

This law of the pressure varying inversely as the radius only applies to cases where the pressure may be regarded as being due to tension in the sac wall, and where that tension is constant. In the other class—namely, where the pus pressure is due to influences remote from the sac—it is by no means true.

If for simplicity's sake we suppose a small abscess situated in the middle of a much larger and not very resistant structure, which is in turn confined by a tough capsule, a relatively large increase in the abscess will only cause a relatively small stretching of the remote capsule, which will not be distorted beyond its limit of elasticity; its tension will be increased, and the pressure internal to it, and therefore the pus pressure increased. Hence we arrive at the interesting result that increase in the size of an abscess causes a fall of pressure if the resistance to its growth is immediately outside the sac, while if this resistance is some distance from the sac it results in a rise.

This is really only another way of saying that as long as the tissues, whether near or remote, to whose tension the abscess pressure is due are not distorted beyond their limit of elasticity, increase in the size of an abscess is followed by a rise in its pressure, while if that limit be passed it results in a fall.

There is one possible case in which this is untrue, and though it does not occur in the human body it may be mentioned for sake of completeness. The case would arise if the tissues were so elastic that the increase of tension due to stretching was more than counterbalanced by the increase in the radius of curvature.

It need hardly be said that though both these varieties
exist in their pure form, yet most abscesses consist of a combination of both, in which one or the other element predominates.

From the foregoing it only needs a little consideration to show that if the cavity is large enough and the surrounding tissues lax enough, or if the abscess is sufficiently slow in its growth, the tendency after incision to spontaneous evacuation of its contents may be so feeble as to be unable to overcome the effect of gravity if the opening is not near the lowest point; while if the abscess is moderate in size and the surrounding tissues firm the influence of gravity, especially in acute cases, may in turn be quite negligible.

It is in the former class of case that drainage from, or close to, the most dependent point is so important: for it is clear that the abscess will never heal if the head of the pus between the drainage opening and the lowest point of the sac is sufficient either to force the fluid further through the tissues or to prevent the contractility of the tissues from extruding the matter. Examples of this are to be found in every part of the body—abscess beneath the scalp will occur to everyone. A chronic abscess burrowing through the thigh, especially when the wasting of the muscles from inaction has relaxed the deep fascia, is also a case in point; but perhaps the most common region is in and around the abdomen, where the general looseness of the tissues favours its occurrence.

As an example of the latter class—that is, where the influence of gravity may be disregarded—a tonsillar abscess may be cited. The most convenient place for puncture is in the soft palate, and this is rarely the lowest point, yet the healing is never retarded on this account, for the abscess is generally acute, the quantity of pus is
never very great, and the surrounding tissues are never very lax.

If the conditions are favourable even large abscesses may empty themselves in spite of the influence of gravity. I have seen two cases of deep-seated abscess at the root of the neck which from the necessities of the cases had to be opened in front of the trachea low down, at a place which in the upright position must have been quite the highest point, and even in the recumbent position could not have been very far from it. From each of these a quantity of pus escaped on incision, which showed it must have come from deep within the chest, and yet in spite not only of the influence of gravity, but the perhaps more important negative pressure of the thorax, healing took place rapidly. In these cases the abscesses were situated in the connective tissue, and the extrusion of the contents was not so much the result of the contractility of the walls as of the reversion of the deep fascial planes.

This negative pressure of the thorax leads us to consider the part it plays in the empyema of the pleural cavity.

The case of the pleura differs from any we have yet considered in that the outer concave wall is fixed or nearly so. Healing can only take place through obliteration of the cavity by union of the parietal and visceral walls. Now, this could only be accomplished rapidly by expansion of the lung in opposition to the negative pressure, a thing which the lung has no inducement whatever to do since the negative pressure of the thorax is in fact due to the contractility of the lung. So that for obliteration we have to depend on the growth of granulation tissue and the gradual falling in of the chest wall under the influence of the contraction of the fibrous layer deposited on its inner surface.
Here we have the exact converse of the conditions found in abscess beneath the scalp. Here, too, we have a fixed and a movable wall with a semilunar cavity between; but with this important difference, that it is the inner convex wall which is fixed and the outer wall which is movable. Here the contraction of the movable wall tends to obliterate the cavity, while in the case of the pleura it has exactly the opposite effect. This is the sole reason of the difference of difficulty in getting the two cases to heal.

The negative pressure of the thorax is the constant factor in the enlargement of the pleural cavity by contraction of the lung, but in all except recent cases there is another. When the visceral pleura becomes thickened and the organised new tissue tends to contract, its effect is to aid the collapse of the lung, since its concavity is directed towards the root of the lung and away from the pleural cavity. This effect may be very small if the empyema is localised by adhesions, for the contracted surface will be only the chord of a very flat arc, but if the whole cavity be involved the effect will be very great.

From the point of view of its squeezing effect on the lung this new contracting sheet is of importance. In the first place, the thicker it grows and the stronger it gets the harder it will compress the lung; but, in addition to this, its crushing power is increased by the mere fact that the lung gets smaller, because decrease in the radius of curvature of a contracting sheet means increase in the pressure to which the tension in that sheet gives rise. The delicate lung tissue can offer no resistance to the influence of this vicious circle, and we need not doubt that the process of carnification is entirely due to this cause. It is no wonder, then, if such a pleural cavity
On the Treatment of Purulent Cavities.

when opened takes an indefinite time to heal, for the effect of dependent drainage is purely negative.

Up to the present the operation of resection of the ribs as advocated by Estländer is the most important surgical aid to the healing process. It helps the collapse of the rigid outer wall, and so facilitates the obliteration of the cavity.

Let us now consider what would be necessary in order to make such a cavity heal at once. It is clear from the foregoing that if while providing an exit for the pus we could keep the lung continuously distended so that the parietal and visceral layers were in contact, fusion of the granulating surfaces would take place very rapidly, since there would be no cavity to be filled.

Now, there are two ways of keeping the lung distended, and two only. The first is by corking up one bronchus and forcing air into the lung through a perforation in the cork. The other is by applying a suction apparatus to the pleural cavity; and I make this proposal believing it to be quite feasible, for even if some technical difficulties stand in the way it is worth while to try and surmount them when we consider the importance of the end to be attained—namely, aborting the disease, and leaving the patient from the very first with a lung not more impaired than it already was by the disease which occasioned the empyema.

In the first place, the seat of the wound where the drainage tube is inserted should be made airtight. This could be managed by a small pneumatic pad applied round the wound. Through the middle of the pad the drainage tube should pass, the hole in the pad being hermetically sealed to the outer wall of the tube. Suction could be applied by an aspirated reservoir with a manometer tube attached.
In order to fix our ideas, as well as for the purpose of experiment, I have had such an apparatus made for me by Messrs. Smith and Sheppard. It consists of a large bottle, which can be sufficiently exhausted by a small air-pump. It is advisable that the capacity of the bottle should be considerable, so that the minute leakages, such as are inevitable with rubber tubes and joints, as well as the gradual outflow of pus to take the place of air, may have but a small influence on the pressure.

The pressure is gauged by means of a U-shaped mercurial manometer. Into the free end of the manometer a pair of wires, connected with an electric alarm, are lowered to any requisite depth. Should the negative pressure in the reservoir fall below a certain point corresponding with the tips of the wires, the mercury rises, makes contact between the points, and the attending nurse is summoned by the bell.

The air pad, which is fixed to the drainage tube, is
secured to the patient not only by a belt going round the chest, but locally by rubber strapping.

I have had pads made in various shapes and sizes. The best form can only be decided experimentally, but the most promising is a conical one with the drainage tube passing through the axis of the cone. The pad need not necessarily contain air. Water would be less liable to leakage, besides avoiding the necessity of opening the pad to allow for expansion when sterilising by boiling. Great care should be taken to allow no air to enter the pleural cavity either before or during treatment. If it entered it would rise to the highest part of the cavity and prevent the union of the surfaces between which it lay. Furthermore, it would stand no chance of being removed by an apparatus arranged only to abstract the heavy material from the most dependent part. If air gains admission it can be removed by turning the patient on the opposite side and filling the pleural sac with boric lotion, at the same time providing for the escape of air.

It is therefore evident that once the pad has been put in position and the treatment started it ought not to be removed until the case is quite or very nearly cured, for removal of the pad would mean entry of air and ripping up of the adhesions between the granulating surfaces. For the same reason the tube leading from the pad to the bottle should be carefully clamped before removing the bottle for emptying and cleansing, and the bottle again exhausted before undoing the clamp.

Theoretically, the opening in the chest wall should be made at the most dependent point as the patient lies, so that the gravitation of the pus might cause the adhesion of the pleural layers to begin at parts remote from the opening, which should, of course, be the last place to close. But practically, I think, it will be found sufficient
if the opening is some distance away, provided the end of
the empyema tube is at the bottom of the cavity. When
the rest of the pleura has adhered, the track can be made
to close by shortening the tube.

Again, the earlier in the history of such a case the
treatment were tried the better would be the chance of
success, for the more recent the visceral exudation the less
it will be organised and the less will be its tendency to
contract. With regard to the degree of negative pressure
to be applied, it must be at least sufficient to expand the
lung. If the method were applied at the onset of the
disease, the normal negative pressure of the thorax would
be the only resistance to be overcome by the pump. But
since, in many cases, the correct amount will not be pre-
cisely known, a greater negative pressure must, for
certainty sake, be resorted to, and there is no reason, as
far as one can see, against the minimum being consider-
ably exceeded. The determination of the most useful
pressure must, however, be left to experience and experi-
ment.

An approximation to the correct degree may be made
by watching the effect of gradual increase of negative
pressure on the outflow of pus. When it is seen that a
further increase is not responded to by an additional out-
flow, it may be taken as proof that the existing amount
is enough to keep the cavity nearly empty.

In pyopericarditis, in the same way the negative pres-
sure of the thorax keeps the parietal pericardium dis-
tended, and thus retards healing by preventing its con-
tact with the visceral layer.

When we come to rigid cavities, such as the antrum of
Hillmore, where the mucous membrane is degenerated,
the futility of expecting drainage to effect a cure will,
from previous considerations, be apparent. It may be
admitted that in the history of a case of empyema of the antrum from simple infection—that is, where there is no tooth or bone disease, there is a point up to which simple drainage might allow the lining membrane to recover, though I am not at all aware that this has ever been tried. But when once the purulent stage is established it is hard to see how this can ever occur. The truth is that mere drainage, if not actually pernicious in such cases, can be entirely dispensed with. At best it can only get rid of excess of pus; it can never leave the cavity pus-free.

Further, in cases where the membrane at the hiatus semilunaris is degenerated or swollen, no ordinary-sized hole would have the effect of emptying the antrum, for no air could come in from above to take the place of the pus. The rule that lays down that the antra should be tapped at the most dependent point is only an unreasoning extension of what in other circumstances is a highly important practice. The real reason why opening the antrum from below sometimes cures empyema is that it allows thorough flushing and cleansing of the cavity, and by removing the infected matter permits the lining membrane to recover. This end is just as well attained with a solid plug in the opening as with a drainage tube, while the plug has the additional advantage that the pus does not trickle into the mouth and get swallowed by the patient. In cases where degeneration is so well established that regular and frequent flushing does not effect a cure within a reasonable time, continuing the treatment will not bring it about.

Granulation tissue would be no bar to healing if the walls could but collapse and the granulations fuse, but the rigidity of the walls prevent anything of the kind from happening, for after the first few layers granulations
grow so slowly that it would take an enormous time for obliteration to be effected in such a way.

An effort must then be made to curette the diseased tissue and allow the raw surfaces to epithelialise. This can be effected by cutting away the inner wall of the antrum to such an extent as to permit the mucous membrane of the nose to creep in and cover the raw surfaces with a healthy coat. I have lately in three cases supplemented this method by applying large grafts to the inside of the antrum through the opening in the canine fossa a week after the primary operation, and in each case with an excellent result.

Again, let us take the case of the frontal sinus. Here we have the best possible example of the insufficiency of drainage to cure an infected bone cavity. The sinus is provided with an exit at its most dependent point, and if drainage were of any service whatever, one can hardly see how such a cavity could ever become chronically affected. The explanation given by those who attribute its disease to want of proper drainage, is that the concomitant swelling of the mucous membrane of the infundibulum blocks the exit, and so impedes the gravitation of the discharge. So in many cases it undoubtedly does, and in these we have brow ague from pus pressure as a constant symptom; but in many others the infundibulum is large enough to allow perfect drainage, in spite of swelling, and this is borne out by the ease with which large instruments can be introduced into the cavity from the nose, and also by the complete absence of frontal ache, a symptom which could not fail to be present were the outflow in any way obstructed.

That such cases do not tend to spontaneous cure shows that mere drainage is not enough, unless the other neces-
sary condition is present—namely, that one wall of the cavity must be collapsible.

Here, then, we have the key to the correct treatment of such cases. There is no use in trephining the anterior wall and scraping and disinfecting the cavity. The mucous membrane has been destroyed, and when the bone granulates the old condition is re-established as badly as ever. In the same way a diseased mastoid process will never heal from simply letting the pus out by chiselling if its interior is degenerated. Such treatment should be reserved for acute cases, where, on the subsidence of the inflammation, the membrane reverts to its normal condition.

Operations on the mastoid in old-standing cases were heart-breaking to the surgeons until the modern radical procedure was developed. The essence of this beautiful operation consists in throwing the tympanum, external meatus, mastoid cells and attic all into one large accessible cavity over the surface of which epidermis is encouraged to grow by utilising the skin of the external meatus and by transplantation by Thiersch's method from other parts of the body.

So, also, when abscess occurs in bone, the head of the tibia for example, opening and scraping and drainage are of no use, the granulations cannot fuse, for they cannot meet if the cavity is of any but the smallest size. The efforts of the surgeon should be directed towards laying the cavity freely open and planting the raw surface with large grafts.

In the majority of surgical cases pain is a function of tension. Now, tension in cases of infection is determined by two things; the rigidity of the surrounding tissues and the time they get to adapt themselves to their new conditions. There are certain parts of the body noted for
their painfulness when inflamed. These are not necessarily places richly supplied with sensory nerves, but rather parts whose structure is such that it does not readily allow of distortion.

A gouty or rheumatic inflammation of fibrous tissue is always severe, and a patient may suffer a degree of torture from a small furuncle on the fibro-cartilaginous parts of the nose or ear out of all proportion to the size of the affected area. If the affection is virulent the inflammatory products accumulate rapidly, the mechanical disturbance of the tissues is violent, the tension is high, and the pain great. If, on the other hand, the infection is mild the products accumulate slowly, the tissues get time to adapt themselves, the tension is low, and there is but little pain, and if the malady only progresses with sufficient slowness there is no reason why pain should not be entirely absent.

This gives a more reasonable explanation of the fact that tuberculous otitis usually progresses painlessly than the assumption, obviously made to fit the fact, that the tubercle bacillus produces a special anaesthetic toxin which deadens the nerves of the affected area.
ON OPERATION FOR CLOSURE OF CLEFT PALATE IN INFANTS.

By SIR THORNLEY STOKER,

President of the Royal Academy of Medicine in Ireland;
Surgeon to the Richmond Hospital.

[Read in the Section of Surgery, May 12, 1905.]

Operations for the closure of clefts in the hard palate have not undergone any striking developments in late years, except in one particular, and that is the introduction by Mr. Brophy, of Chicago, of a new method of closing the cleft soon after birth by approximation of the superior maxillae and palate bones. The development of the surgical treatment of cleft hard palate divides itself readily into three periods:

1. That commencing with the practice of closing the cleft by operation, introduced by Warren, of Boston, about 1843.

2. That dating from developments of methods during the years immediately preceding 1868.

3. That inaugurated by the operation of Mr. Brophy, described in his paper in The Dental Cosmos of April, 1901.

1. Dr. Warren must be regarded as the pioneer of the operations for cleft hard palate. Up to the time when his paper appeared in the American Journal of Medical Science, in 1843, no method existed of dealing with this deformity, except by the application of obturators, which are never satisfactory, and are only in these days resorted to where surgical measures have failed.

2. The work of Sir Thomas Smith has probably lent itself more to the perfection of operations on the hard palate than that of any English surgeon. His paper, published in the Med. Chir. Trans. for 1868, inaugurated the second period in the
history of uranoplasty. He advocated the operation in patients of tender years, recommended the use of chloroform, and devised his well-known gag to assist the surgeon; and this when Fergusson two years later could only say that he had resorted to the proceeding with partial success, and recommended it for further trial. It is true that in the list of cases published in 1868, Smith in all operated on the soft palate only, and left those in which a cleft hard palate existed to be dealt with later on. This will be admitted by those who have practised Brophy’s operation to be inadmissible to-day, when it will be found better to defer the closure of the cleft in the soft palate until the defect in the hard structures has been corrected.

Since the years preceding 1868, the age selected for closing the hard palate has steadily become earlier. At first it was usually done after puberty. In 1870 we find Mr. Pollock advising that it should be delayed for two or three years after birth, and now it is open to operate by Brophy’s method at any time during the three months after birth.

3. The operation of Mr. Brophy has been done by him at from ten days to three months of age. It constitutes a new departure, not only on account of the early period of life at which it is done, but because it is novel both in principle and method. It consists essentially in drawing the upper maxillae and palate bones of opposite sides together by wire sutures, generally two in number, passed transversely through the alveolar processes above the level of the palate processes of the maxillae and palate bones. There are details of the operation which will be best understood by following Mr. Brophy’s own description. The method cannot be successfully carried out after the child is three months old, as the bones are then too fully ossified to be displaced by any force that could be safely applied. I have had the opportunity of using this treatment in three cases, and may at once say
that I shall use it in future when suitable subjects offer, reserving, however, a few minor points of the procedure about which to offer an independent opinion.

I may epitomize Mr. Brophy’s plea for his method as follows. He advocates its employment during early infancy for the following reasons:—

1. “The surgical shock is less, because the nervous system of a child is not well developed, and it is not, therefore, capable of receiving the same impressions that it would later in life, for young children usually react better. Moreover, all mental apprehension is eliminated, and we know that alarm and dread are among the most powerful factors in producing shock.”

2. “Before the bones are fully calcified they may be bent or moved without fracture. &c.”

3. “If the muscles are very early brought into action they develop instead of atrophy, and hence a good return is secured, with plenty of tissue; whereas if the operation is undertaken later in life, after the parts are shrunken through non-use, they can rarely be made to subserve the same purposes as organs which develop through natural employment, &c.”

4. “When the palatal processes of the maxilae are united, it will be observed that the development of the bones of the alveolar processes of the upper jaw assume a form nearly or quite normal, and when the teeth are erupted they will properly occlude with the lower ones, or nearly so. . . . It was predicted by my surgical friends that the upper jaw would be much narrower than the lower one, and that it must always remain contracted.” Mr. Brophy then proceeds to show that this has not been the case, and that no such deformity is likely to occur beyond what may be corrected by orthodontial methods.

5. “Following early operations there is much less deformity, for all the tissues, bony as well as soft, develop naturally and according to accepted types.”
6. "When the operation is made in early infancy, the parts are sufficiently advanced to give possibility for normal speech when the time comes for learning to articulate. If the operation is not made until faulty habits are acquired it is with difficulty that they can be overcome, even supposing the muscular parts could be made sufficient."

These arguments are too sound to require much comment. With reference to the first I may say I have been struck with the absence of shock or other grave symptoms. In none of the cases I have done has there been any cause for alarm. Even when the anaesthesia was prolonged for nearly three-quarters of an hour, or when a good deal of blood for a tender infant to lose was lost, I have seen far less disturbance than is frequent in shorter operations, with less severe bleeding, at a later period of life. One of the matters, however, requiring attention when operating on very young children is the avoidance of bleeding, and some suggestions later on in alteration of Mr. Brophy’s technique are worthy of attention.

The second observation, with reference to the absence of tendency to fracture, requires a remark. In the last case I operated on the child was six weeks old, and the gap in the hard palate of great width. The sides could not be brought completely together, and in trying to do so I fractured the alveolus of one jaw, but without tearing its soft coverings. This enabled me to get better apposition, and no deformity or ill effect ensued.

The stages of Brophy’s operation, as described by himself, are as follows:—

1. “Thorough paring of the edges of the fissure, including in the incision the edge of the semicartilaginous bone.”

2. “Then raise the cheek, and well back towards the posterior extremity of the hard palate, just back of the maxillary process, and high enough to escape all danger of
not being above the palate bone, insert a large braded silk suture, carrying it through the substance of the bone to the central fissure by means of one of the strong needles, with the opposite needle carrying a corresponding silk suture through the opposite side. We then have two silk suture-loops carried to the centre of the cleft, and passing one through the other enables us to carry the one loop through both of the maxillary bones.

Fig. 1A.—Vertical section of the superior maxillary bones of a child five weeks of age, showing congenital cleft palate. A, A. Silver tension sutures. B, b. Lead-plates. C, c. Germs of the first temporary molar teeth. D. Cleft palate. (From the Dental Cosmos, April, 1901.)

"The silk is more easily introduced by the needle than wire, but a silver wire should always be substituted for it, and drawn through to take its place. The wire should be No. 20, and may be doubled in case the condition of the parts and the tension upon the tissues necessary to approximate them seem to require it. Nearer the front portion of the maxilla insert another wire, carrying it through the

1 The needles he describes are essentially those of Liston.
substance of the bone above the palate plate, and through
the other side in a position corresponding to the place of
entrance. Thus we shall have one wire passing over the
palate in front of the malar process of the bone and another
behind it."

With regard to this stage of the operation I may give
another practical reason for using double wires of moderate
gauge instead of a single thicker one. When much resistance
is experienced it will be found more easy when tightening the
suture to twist two thin wires than one thick one.

3. "The next step is to make lead plates (No. 17, American
gauge) to fit the convexity of the buccal surfaces of the
bones. Have them provided with eye-holes, through which are
passed the protruded ends of the wire upon each side. Twist
these together—that is, the right end of the posterior with
the right end of the anterior wire, and the same on the left
side. These form heavy tension sutures, and the parts, when
once approximated by their use, cannot be separated, as the
sutures do not cut out. If the cleft is a very wide one, and
we are not able to close it by twisting the wires together
on the lead plates, force may be exercised with the thumb
and fingers, or by means of a forceps designed for that purpose.
If by such force the edges of the cleft do not approximate,
there is a further step to be taken which will obviate these
difficulties. After the cheek is well raised, divide the mucous
membrane and the bone, through the malar process, carry
the knife in a horizontal direction, and when well inserted
sweep the handle forward and backward. In this way a
maximum amount of bone and a minimum amount of mucous
membrane will be divided. This done on either side the
bone can readily be moved towards the median line."

1The American gauges referred to in this paper are practically the
same as the Imperial or B.W. gauges, and the lead plates are measured
by a wire gauge and not by a lead-plate gauge.
The next step in the operation is the insertion, should they be found necessary, of some points of interrupted suture in the soft coverings of the hard palate, and, if it be decided to close it at the same time, in the soft palate.

The premaxilla is then dealt with. Its sides are trimmed, a V-shaped piece of bone removed from the vomer so as to allow it to be moved back, and a suture inserted to hold it between the alveolar processes.

At a later date, when the sutures have all been removed, and when no further wide access to the mouth is likely to be required, the hare-lip can be operated on.

In practice I have found certain slight departures from Mr. Brophy's methods advisable. They are as follows:
When performing the operation, paring the edges of the palate should be deferred until the sutures have been inserted and the bones moved towards the middle line. The period for haemorrhage is thus lessened. Also, if it be found that the bones cannot, with reasonable effort, be brought together, it is better not to pare the soft parts at this time. It must be remembered that the sutures act in two ways, at first by drawing the parts together and subsequently by holding them fixed so that they approximate by the process of their growth. In two of the three cases I have operated on I failed to get the maxillary to touch each other, and yet union soon occurred in one, and is being completed in the other and later case. It is therefore better, where full approximation of the maxillae cannot be effected at the time of operation, not to waste tissue by an ineffectual paring, but to wait and do it later on when the bones are touching. Again, the lower edge of the vomer is often very thick and vascular, and in one instance I saw it grow into the cleft and help to close it.

Although he does not explicitly state it, I gather from Mr. Brophy's paper that he advises the soft palate to be operated on at the same time as the hard. In my first case I pared the whole palatal edge, and got union of the soft as well as the hard palate, except the uvula. In the second case the soft palate did not unite, although sutured, and its treatment has been deferred for a year or two. In the third case the gap was so wide that I could not get the sides of the hard palate into apposition. I did not pare or attempt to deal with the soft palate, and propose to operate on it later on.

The proper course seems to be to pass the sutures and move the bones. If they can be got into close apposition to pare the edge of the hard palate and fix the sutures; if they cannot be thoroughly approximated to defer the paring process until the maxillae have grown into apposition, and then do it, if
necessary. If the hard palate is thoroughly closed at the primary operation it would be best to then trim and suture the soft palate, or under less favourable circumstances to defer its closure.

There can be no reasonable objection to the practice of leaving the hare-lip without operation until the hard palate has been closed. Free access to the mouth is thus obtained and the surgeon's work done under the easiest circumstances. Of course, if the soft palate remains deformed, either because the attempt to procure union has failed, or has been deferred, it would be necessary to close the lip as soon as may be convenient after the bony defects have been remedied, and postpone the closure of the soft palate until a later date.

Fig. 2a.—Drawing of a plaster cast of the mouth of a child, showing lead plates and wire sutures in place.

Fig. 2b.—Drawing of a cast of the same mouth one week after the cleft was closed.

(From the Dental Cosmos, April, 1901.)

This shows double the amount of suture described in the text as used in an ordinary case.
I have made extensive use of Mr. Brophy's words, because I am not aware of any previous publication of them in this country, and wish to put his own case. The more so as I only venture to criticise some minor details, and do not take exception to any of his general principles or practice.

The operation requires some neatness, but is by no means a difficult one. In this, as in all procedures of its class, Rose's position should be employed, as it gives both facility and safety. I use no gag, except the jaws are developed enough to permit the use of one such as Lane's. The tongue is best kept out of the way by a depressor, shaped like the curved end of Fergusson's director, which is not bulky, and can be used as a tractor. No special instruments are required, as a couple of Liston's nævus needles of different curves suffice to pass the sutures. The plates and sutures do not seem to cause inconvenience to the child, nor have I seen stenosis of the nasal cavity produced by their use. In the cases I have operated on, the infants have rapidly put up condition. The three cases in which I have done this operation were all of extreme deformity. In each hare-lip, projecting premaxillæ, and complete and wide separation of the hard and soft palates were present.

Case I.—James M'K., aged twelve weeks, was admitted to the Richmond Hospital, October 6th, 1903. He was healthy and well nourished, and suffered from wide cleft of the soft and hard palate, with complete left hare-lip, fissure and separation of premaxilla on the left side. There was much projection of the premaxilla. On October 10th the soft and hard palates were pared, the malar ridges on both sides divided and the clefts closed, the soft one by horse-hair sutures, the hard by two wires and plates. The pre-maxillary bone was then moved back and sutured in its place. The temperature rose to 100° for two days after operation, and then became normal. On November 7th the wires were removed, and on November 11th two points of suture placed
in the soft structures where they were inclined to separate. On December 19th the hare-lip was operated on, and on January 5th the child was taken home. The lip, pre-maxilla, hard palate, and all the soft palate except the uvula being in normal condition. Fig. 3 shows the appearance of this patient at the age of one year and ten months.

Case II.—James M., aged two weeks, was admitted to the Richmond Hospital on March 15th, 1904, but, being in very poor condition, the operation was deferred to May 4th, when at the age of nine weeks he was operated on. The condition of the child was one of much deformity, double hare-lip, projection of pre-maxillary bone, and complete and wide cleft of both hard and soft palates. The steps of the operation were the same as in the first case. There was no subsequent rise of temperature of any moment. The child was taken home five days after operation, and brought back to have the wires removed at the end of four weeks. The lip was then operated on. The result in this case was excellent, the whole of the surfaces uniting thoroughly except the uvula. I cannot show a picture of this child's condition after operation, as it died of bronchitis ten months after its treatment had concluded, a fact only discovered when we sent for it to have a photograph done.

Case III.—Isabella N., aged six weeks, admitted to the Richmond Hospital, February 20th, 1905; suffered from double
hare-lip, pre-maxillary projection, and complete and wide clefts of both hard and soft palates. Figs. 4 and 5. These pictures give a fair average idea of the degree of deformity in all three cases. This child was in unusually good condition, having been fed with human milk, given with a spoon. She was operated on two days after her admission in the same way as the two other cases, except that as the cleft in the hard palate could not be completely closed no attempt was made to deal with the soft palate. The pre-maxilla was moved into line with the alveoli. It is now eleven weeks since the operation; the cleft in the hard palate has become steadily narrower, is quite closed in front behind the pre-maxilla, and will probably soon be closed behind. The hare-lip was operated on on May 10th, and has united favourably. In due course I shall attempt to get union of the soft palate.

The second of the above cases is a test one, as it shows what a good result may be got in even a very feeble and ill-nourished child. It is my usual practice to defer all operations for hare-lip or cleft palate until every effort has been made to improve the condition of the child, but this was a weakly creature in spite of treatment.

Regarding the question as to when the soft palate is to be closed in those cases where it has not united, or in which it has not been thought well to attempt to include it in the primary operation, it is not possible to give a dogmatic opinion. In some instances, no doubt, it may
be secondarily attacked before the lip is closed and while free access is obtainable, while in others a delay of two, three, or four years is unavoidable.

One remark may be made in connection with this operation—namely, not to be discouraged at any unsightliness that may be present at first. It is not always possible to get the case to look pretty at the time of operation, or even always to close the hard palate cleft completely. The parts will gradually fall into line and look nicer, and, as I have before said, the cleft in the hard structures will gradually close more or less completely if it has been securely wired.

Finally, I do not think it is always possible to carry out this operation with academic perfection, but that being said, it is hardly possible to deny that it is of great importance and not to be overlooked.

Mr. T. E. Gordon considered the risks of Brophy's operation somewhat serious, especially in those cases where it was found at first impossible to bring the edges of the palate together, and further steps were necessary.

Mr. Edward Taylor stated that he had operated on a considerable number of cases by the older and better known procedures with various modifications of his own. The all-important thing was to operate at an early age so as to secure closure of the cleft before the child began to speak. The cases he had operated on recently were from twelve to fifteen months old, and in nearly all he had succeeded in obtaining complete closure of the gap and very gratifying results as regards speech. He thought Sir Thornley Stoker's paper would afford a strong inducement to many surgeons to practise Brophy's operation.

Mr. W. S. Haughton said he had performed an operation very similar to Brophy's on a child nine days old. The case was one of emergency, as the child was starving. He had been struck by the ease with which the parts were brought together and with the entire absence of shock.
Mr. Kennedy alluded to the danger attending loss of blood in operations for cleft palate in young children and to the fact that in Brophy’s operation the soft palate remained to be operated on after the closure of the hard palate.

Sir Thornley Stoker, in reply, stated that in his experience there was very much less disturbance and shock following Brophy’s operation than operations performed upon older children. The operation was singularly free from risk. Mr. Kennedy evidently had misunderstood him, as he (Sir Thornley Stoker) considered the risk from loss of blood very serious, and had specially advocated a modification of Brophy’s procedure to obviate it. With the exception of one case he had not deferred operations on the soft palate. In that case he found it impossible to approximate the margins of the two segments of the hard palate.
NOTES ON A CASE OF VOLVULUS OF THE CÆCUM, SECONDARY TO MALIGNANT DISEASE OF THE SIGMOID FLEXURE OF THE COLON.

By JOHN LEO KEEGAN, F.R.C.S.; Surgeon to Jervis Street Hospital.

[Read in the Section of Surgery, May 12, 1905.]

I venture to bring before you the following notes on a case of volvulus of the cæcum, which is the first of its kind that I have met in surgical practice.

J. K., aged fifty, labourer, was admitted to Jervis Street Hospital on December 1, 1904. He had fallen into the hold of a vessel in which he was employed, and had fractured a humerus, both bones of a forearm, and five ribs. He went on well until the morning of December 23, when he complained of griping colicky pains in the lower part of the abdomen and the flanks. The abdomen was much distended, resonant on percussion, and the liver dulness had almost disappeared. His appearance, however, was good; his pulse was strong, and only 72 per minute. At his bedside were two vessels filled with an inky-coloured, sour-smelling fluid, which he had vomited without any effort on his part. His condition suggested some form of intestinal obstruction, and, as I noted that his bowels had not acted for two days, I ordered him castor oil and a turpentine enema, followed by one of soap and water, with the result that the bowels acted, and the pain and swelling rapidly disappeared.

On questioning him he told me that for several years past he had had occasionally similar attacks of pain, which a dose of Epsom salts relieved. He remained well until New Year's Eve, when the symptoms reappeared. I did not see him until the following evening, when I found no vomiting, pain not so intense as before, distention marked, pulse and temperature normal. I treated him as before, but with no result, and then, seeing that the distention was increasing, I advised operation, but to
this his wife would not consent. On the next day his distention became more marked, his temperature was 100° F., pulse 90, and he had slight attacks of vomiting, with distressing hiccough.

On the morning of the third day I asked my colleague, Mr. Kennedy, to see him with me, but, although it was evident that death would soon ensue without operation, it was not until the evening of the third day that consent for this procedure was obtained. That night, being kindly assisted by Mr. Kennedy, I opened the abdomen in the middle line below the umbilicus. On getting through the peritoneum, a quantity of foul gas escaped, there was evidence of peritonitis, and the intestines were deeply congested; but presenting into the wound was an enormously distended portion of bowel, mottled in places by gangrenous patches, which I found to be the caecum. The distention seemed to end just below the hepatic flexure of the colon, and the remaining parts of the colon were apparently normal, as were also the coils of small intestine above. It was clearly a case of volvulus, so I brought the distended bowel as far out of the abdomen as possible, then incised it, and allowed a large quantity of foul gas and faecal fluid to escape. Seeing, however, that the bowel was gangrenous, I resected the entire mass, after clamping the bowel above and below; I was also obliged to remove portion of the mesentery, the vessels of which were thrombosed. I next washed out the abdomen with hot saline solution, and, as the patient's condition was one of extreme gravity, I simply brought out both ends of the bowel, sutured them to the abdominal wall, inserted Paul's tubes, and washed out the intestine. That night he seemed fairly well, the temperature was 99° F., pulse 120; drainage was going on satisfactorily, so he was given a hypodermic of strychnin and morphia, with hot water to sip. He was quiet during the night, and next morning seemed to be holding his own; he was free from pain, and remained so during the day, but in the evening there were some vomiting and hiccough, which continued during the night. On the following day he was weaker, and, lapsing into a semi-comatose state, he died at 7 p.m., having lived for two days after the operation.

The post-mortem examination revealed signs of general peritonitis; the bowel at the site of the wound was healthy, and had drained well, but there was a hard mass just at the brim of the pelvis on the left side, which on removal I found to be cancerous.
A Case of Volvulus of the Cæcum.

This caused an absolute obliteration of the lumen of the bowel, as water would not pass through it at this point.

I received the following report from Professor McWeeney, Bacteriologist to the Hospital:

"Diameter of bowel above stricture, 5½ inches; below, 2 inches. Mucous membrane above stricture is smooth and devoid of rugæ, that below is very rugose.

"Length of stricture, 2 inches: it is so tight that I can hardly push a fine pen-handle through it.

"The greater part of the cancerous tissue is near lower part. The upper part 'tails off' into a sort of collar, which projects into the lumen of the bowel after the manner of a cervix uteri; its microscopic structure is an adeno-carcinoma."

The points of interest in this case are:

1. The fact that the patient showed no signs of having malignant disease until a few days before his death, although he had a cancerous growth which almost occluded the bowel.

2. That the volvulus was limited to the cæcum, and left the remaining portion of the large intestine free—the dilatation mentioned in Professor McWeeney's report being only immediately above the stricture.

3. The enormous size of the cæcum, which seemed to fill the entire abdominal cavity.
ABSTRACTS.

SECTION OF SURGERY.

Friday, November 1, 1904.

The President in the Chair.

Mr. Kennedy exhibited a patient after nephrectomy, and the diseased kidney which had been removed; also cases of excision of the knee and elbow-joints.

Mr. Edward H. Taylor exhibited a man, aged thirty-nine, whose left lower extremity he had amputated through the hip-joint for an osteo-sarcoma of the femur; (2) specimens of rectal cancer removed by the trans-sacral and by the combined sacro-abdominal method; also (3) some unusual forms of urinary (bladder) calculi.

Mr. R. J. Montgomery exhibited a patient showing the result of operation for ectropion of both lower eyelids.

Sir Thomas Myles exhibited (1) some recent cases of gastro-jejunostomy; (2) carinomatous and tuberculons kidneys removed by nephrectomy; and (3) a large fibroma removed from the region of the lesser peritoneal sac.

Mr. W. S. Haughton exhibited (1) case of omentohepatopexy; (2) case of excision of astragalus for dislocation of thirteen months' standing; (3) case showing new method of “screwing” fractured olecranon.

Mr. Maunsell exhibited (1) examples of different forms of goitre; and (2) some pathological vermiform appendices.

Mr. J. B. Story exhibited an improved mask to exclude buccal microbes from operation wounds.

Mr. Seton Pringle exhibited an ano-coccygeal tumour.

Mr. A. B. Mitchell (Belfast) exhibited a specimen showing the condition of parts after gastro-jejunostomy, followed subsequently by jejuno-jejunostomy for the relief of regurgitant vomiting.

Mr. A. B. Mitchell (Belfast) read a paper entitled “Inefficient Operations for Gastric Ulcer” (see page 81, ante).

Mr. W. S. Haughton read a paper entitled “The Healing of Wounds: Aseptic v. Antiseptic Methods” (see page 91, ante).
Friday, December 2.

The President in the Chair.

Mr. R. C. B. Maunsell exhibited a child after operation for spina bifida, also the meningocele which he had removed.

Mr. Kennedy exhibited (a) infant operated on for intussusception; and (b) child treated for hydrocephalus by repeated lumbar puncture.

Mr. Blayney exhibited a Gasserian ganglion removed for epileptiform neuralgia.

Mr. W. Taylor exhibited (a) gall-bladder removed for gangrenous cholecystitis; and (b) hydrencephric kidney.

Mr. Edward H. Taylor read a paper entitled "New Methods of Procedure for the Radical Cure of Inguinal and Femoral Herniae" (see page 111, ante).

Mr. T. E. Gordon read an account of a case of "Acute Intestinal Obstruction from a Gall-stone" (see page 118, ante).

Friday, January 13, 1905.

Mr. G. Jameson Johnston in the Chair.

Mr. Maunsell exhibited a patient upon whom he had operated for acute cholecystitis, and subsequently for gangrenous appendicitis; also a gall-bladder removed for phlegmonous cholecystitis, and another for hydrops.

Mr. C. Arthur Ball exhibited—(a) prostate removed by perineal prostatectomy, (b) prostate and calculi removed by conservative perineal prostatectomy.

Mr. Maunsell read a paper entitled Cholecystotomy for Acute Cholecystitis following Typhoid Fever (see page 125, ante).

Mr. Arthur C. Ball read a paper entitled "Conservative Perineal Prostatectomy" (see page 134, ante).
Abstracts.

Friday, February 24, 1905.

Mr. E. H. Bennett in the Chair.

Mr. L. A. Byrne exhibited patient after operation for epithelioma of the tongue; also the part removed.

Sir Lambert H. Ormsby exhibited patient whose olecranon had been wired after fracture.

Mr. G. Jameson Johnston exhibited patient who had the power of voluntarily producing a partial dislocation of the knee.

Mr. W. I. de C. Wheeler exhibited an extension apparatus for fractured femur.

Mr. Blayney exhibited (a) parts removed in the treatment of acute intussusception, and (b) malignant disease (colloid cancer) of the large intestine.

The Application of Plaster of Paris to Fractured Femur, facilitated by a New Apparatus.

Dr. W. I. de C. Wheeler exhibited the apparatus and read a communication describing it (see page 152, ante).

Mr. Edward H. Bennett read a paper on "Oblique Fracture of the Tibia" (see page 160, ante).

Mr. Kennedy read a paper on "Some Cases of Joint Excision" (see page 163, ante).

Friday, March 24, 1905.

The President in the Chair.

The President exhibited a patient who presented an unusual condition of the hip subsequent to injury.

Mr. Kennedy exhibited—(a) patient operated on for internal strangulation by a band; (b) case after amputation through the shoulder-joint; (c) two cases of Talipes Equino-varus.

Mr. L. G. Gunn read a paper on "Painless Hematuria" (see page 169, ante).

Mr. Robert Woods read a paper on "Purulent Collections" (see page 182, ante).
Section of Surgery.

Friday, May 12, 1905.

THOMAS E. GORDON, F.R.C.S., in the Chair.

Mr. Wheeler exhibited (a) patient upon whom he had performed the Talma-Morrison operation; (b) portion of small intestine removed from a strangulated hernia; (c) transfusion apparatus.

Mr. J. L. Keegan exhibited (a) patient after wiring operation for fractured patella; (b) gangrenous caecum removed for volvulus; (c) malignant stricture of the rectum.

Major Holt, R.A.M.C., exhibited (a) aneurysm of the innominate artery; successful ligature; death from tuberculosis fifteen months later; (b) aneurysm of the innominate artery; unsuccessful distal ligature and wiring by Power and Colt's apparatus; (c) vermiform appendix, proximal end obliterated, distal end perforated; (d) fragments of a broken semilunar fibro-cartilage loose in the knee-joint; (e) kidney with multiple abscesses, and scars after former abscesses, the ureter showing scar of previous ureterotomy for calculus.

Mr. R. A. Stoney exhibited (a) tuberculous caecum, appendix and ileum; (b) goitre; (c) fibro-lipoma of the skin.

Mr. Pasley exhibited an irrigating apparatus.

Sir Thornley Stoker read a paper on "Operation for Closure of the Hard Palate during the three months following Birth, practised by Mr. Brophy, of Chicago" (see page 196, ante).

Mr. J. L. Keegan read notes on "A Case of Volvulus of the Caecum" (see page 210, ante).
SECTION OF OBSTETRICS.

TWO CASES OF CYSTIC ENDOMETRITIS WITH REMARKS ON TREATMENT.

By R. DANCER PUREFOY, M.D., F.R.C.S.; Ex-Master, Rotunda Hospital.

[Read in the Section of Obstetrics, February 3, 1905.]

Endometritis, either alone or as an attendant on, or complication of, some other pathological condition, claims a large share of our attention in nearly every case of uterine disease; and those who have had the widest experience will be the most ready to acknowledge how often our hopes of speedy, or even steady, improvement are disappointed. Whenever we have regard to the complex structure of the endometrium and the periodically recurrent variations in its blood supply, on which its functional activity may be said to depend, we shall feel no surprise in observing how often these variations go wrong in excess or deficiency, and thus induce obstinate structural changes. The precise nature of these changes, the conditions local, constitutional and bactericidal which are potential in causing them, have, as is fitting, engaged the attention of many acute observers with very satisfactory results; but with regard to some forms of endometritis—for instance, that to which I wish to direct your attention in this communication—we must confess their aetiology is still awaiting elucidation.

I propose to bring under your notice two cases illustrating the history and course of what has been termed cystic endometritis.
Case I.—Mrs. F., aged forty-five. In fairly good health at the time of marriage, six years ago; in about seven months afterwards had an early abortion. Some months later, after a long journey, suffered from distressing pruritus vulvae, which, however, yielded to treatment. In October, 1902, after missing one menstruation, distressing dorsal and abdominal pain was felt, attended with slight intermittent bleeding. Later on the bleeding became continuous, sometimes profuse, and so continued till her admission to the Rotunda in January, 1903. At this time her general condition was such as to warrant the use of medicine, at least for a time. in the hope of arresting the bleeding, and accordingly she was given that most useful uterine haemostatic, tincture of Indian hemp, in five minim doses, twice daily, with excellent effect. This was followed by the administration of the muriate tincture of iron, and in about a fortnight the bleeding had quite ceased. Examined under chloroform, the uterus was found in normal position, the cervix healthy, and the appendages likewise. The cavity measured 3½ inches; viscid, glairy discharge poured from it in a large quantity, and the curette brought away in abundance fragments of greatly thickened endometrium. Subsequently, at intervals of three or four days, she was treated on several occasions with iodine and phenol or pyroligneous acid, and in about three weeks from the date of the curettage she was allowed home apparently in a very satisfactory condition. For some months menstruation was normal, but a return of the bleeding rendered a repetition of the treatment necessary, twice in 1903 and once this year (1904), in the month of September. The tissue removed on each occasion, though much lessened in amount, presented the same microscopic characters.

Case II.—K. C., aged twenty-eight, married two years; one early miscarriage. Admitted August 8, 1903, in a very feeble condition, owing to repeated bleedings; indeed her aspect made me apprehend the presence of malignant disease. When some improvement in her general condition had been effected by rest, feeding and tonics, she was examined, and the uterus found to be retroverted and somewhat fixed by numerous adhesions; the appendages were not reached. When dilated and curetted, the amount of pulpy tissue removed was so great that I thought my apprehensions as to the malignant disease were only too well
founded, but examination by my much-lamented friend, the late Dr. Neville, showed only the characteristic appearances of cystic endometritis. This woman was re-admitted a few months later, when it was found necessary to repeat the treatment.

The main points in these two interesting cases may be thus summarised:

1. Both women were married, not advanced in years, and free from other disease.
2. Both had been, at least, once pregnant before the development of this uncommon disease.
3. Bleeding without any considerable pain was the prominent sign in both cases.
4. Recurrence took place at an early date.
5. The microscopic appearances are not like those of malignant disease, especially as regards the epithelium.

It may be urged that more vigorous use of the curette in the first treatment would have prevented recurrence, but, in my opinion, it is better to err in removing too little than to run the risk of rendering the endometrium permanently incapable of fulfilling its highest function by too extensive removal of its glands.

Having regard to the marked tendency to recurrence and the severe bleeding which attend this disease, one feels inclined to describe it as a transitional stage between benign and malignant adenoma.

Though the experience of only two cases is inadequate to warrant positive statements on the matter, I incline to the view that curetting, repeated if necessary, and followed by the application of the caustics commonly in use, will effect a cure.

For the safe and effective use of the latter, I think we are still in want of a safe apparatus. A syringe for intra-uterine injection should be so constructed that the fluid may be measured by drops, and I have not seen one which fulfils this
Two Cases of Cystic Endometritis.

indication. Many years ago Dr. Barnes suggested an apparatus for carrying medicated ointments into the uterine cavity, and recently Dr. Duke has constructed an ingenious appliance by which small quantities of any caustic fluid may be safely diffused in the uterine cavity. For us in Dublin it is, I think, of much interest to recall the fact that a Master of the Rotunda Hospital, Dr. Evory Kennedy, was the first in Europe to devise a syringe for intra-uterine use.

In cystic, as in every other form of endometritis, the first, and perhaps the most important, indication to be fulfilled in treatment is to secure a sufficiently patulous condition of the cervical canal, to admit of free escape of healthy and unhealthy discharges.

As to the choice of caustics, opinions will be found to vary widely. With regard to phenol, it is well to bear in mind the narrowing of the cervical canal, which is very apt to follow its repeated use, unless well diluted. I think glycerine more suitable as a dilutent than spirit or water. In my own practice I have discontinued the application of caustics immediately after curetting, unless in cases of undoubted malignancy.

Dr. Hastings Tweedy said that cystic endometritis was a disease he did not know personally, and of which he could find no account. He objected to fresh nomenclature, and thought they should stick to the term glandular. His objection to the term "cystic" was that it did not lead anyone to a knowledge of what one was dealing with, whilst "glandular" did. The question of treatment was more interesting. A few years ago every case was cured, but of late there had been a reaction against this, and many now preferred to use intra-uterine douches, caustics, &c. He protested against this, and believed that in his study practice the surgeon had little right, and rarely necessity, to pass anything into the uterus. It was impossible to be aseptic in study practice. The vast majority of cases of endometritis,
especially the hypertrophic form, were cured by one cleanly curetting. He left his cases alone after curetting, and did not use medications subsequently. He washed out the debris with salt solution, and did not use antiseptics, which devitalised the tissues, and the less the uterus was irritated with them the better. He could not understand the rationale of taking in a patient every third day to inject iodised phenol after curetting—it seemed to him unnecessary and dangerous. The less often the uterus was entered the better. There was a certain amount of danger in putting anything into it, and if good results were obtained without caustics, why apply them? The term "benign adenoma" is misleading, as it implies growth accompanied by increase in the number of glands, and all such growths of the endometrium will prove in practice to be malignant.

Dr. A. J. Smith said he divided endometritis into glandular and interstitial. There were two kinds of glandular—hyperplastic and hypertrophic. Hyperplastic was adenoma, and in it there was an enormous increase in the number of glands: hypertrophic had a normal number of glands, but greatly increased and distended, and these when occluded formed cysts. The hypertrophic frequently became malignant, therefore these cases had to be seen often, and the uterus removed. The hypertrophic might be merely distended tubes or, if seen late, cysts. The treatment was to mechanically remove the mucous membrane and try to cultivate a healthy mucous membrane. Try to find out the cause of the endometritis. His practice was to curette and wash out once, and wash out again on the third and fourth day to see that nothing was left behind. He used hot sterilised saline solution, and did not employ any intra-uterine medications.

Dr. Purefoy, in replying, said he thought it was of importance for them to study the pathology of these conditions for themselves, and if systematic examination were made in every case good practical results would follow. With regard to the objection to giving the condition a special name, he could not agree with Dr. Tweedy. The condition was one of considerable rarity, and most of the manuals did not allude to it at all. He believed that the condition was not a very advanced stage of glandular endometritis. He did not know what caused it, but thought that if it had been at all common, he would have met with more
Two Cases of Cystic Endometritis.

than two cases of it. The term cystic endometritis was employed in two classes of cases—in gynaecological cases, where the patients were not pregnant, and also as implying a diseased condition of the endometrium occurring in pregnant women. His cases occurred in women apart from pregnancy. The epithelium in the dilated glands was not in the atrophied condition one would expect it would be in from pressure: it was intact. He believed the condition was very rare. With regard to subsequent treatment, he thought it was in the patient's interest to use a mild caustic four or five days after curetting, and its subsequent use was to be determined by the amount of discharge, condition of cervix, character of first menstruation, &c.
OBSERVATIONS ON BOSSI'S DILATOR, WITH NOTES
OF FOUR CASES.

By ALFRED J. SMITH F.R.C.S.;
Pres., Obst. Sect., R.A.M.I.;
Gynaecologist to St. Vincent's Hospital, Dublin.

[Read in the Section of Obstetrics, February 3, 1905.]

The interest which the introduction of Bossi's Mechanical
Dilator has aroused must be my reason for occupying the
time of the Obstetrical Section. As you are aware, Bossi introduced
his original three-pronged dilator in the year 1890. But little interest seems to have been
taken in this question of mechanical dilatation until
Leopold,¹ of Dresden, published his celebrated article in
the Archiv. für Gynäkol., 1902. In this article he claims
for the four-pronged model all the virtues, and he
expresses the wish that it might soon find its way into
the midwifery bag of every medical practitioner; and
he adopts without reservation the five well-known claims
made for the instrument by Bossi, viz.:—(1) It can be
used even when the cervix is closed, and as yet un-
shortened; (2) the operator is sure of obtaining sufficient
dilatation to allow of the delivery of a living child;
(3) this can be secured if necessary in a short time—
15 to 20 minutes; (4) the use of the instrument excites
uterine action even in cases of inertia; (5) dilatation up
to 11 cm. (4 1/3 in.) can be secured without unduly
distending the vagina. The imprimatur of Leopold
stimulated many workers, and soon we find reports of
successful cases appearing in the journals, and hear notes
of alarm being sounded, notably by von Bardeleben and others. Dührssen as the originator of a rival operation—that of incision or vaginal Cesarean section—views with a jealous eye the enthusiasm aroused by the report of these successful cases. He considers the operation by mechanical dilators as a retrograde one, and backs up his opinion by a list of unfortunate occurrences and accidents; and he concludes (1) that the instrument did not always enable the operator to secure enough of dilatation for the delivery of a living child, (2) that the operator was not able to control the process, and (3) that the method was positively dangerous, some of the tears produced being followed by bleeding and some becoming infected.

Such are the views of the rival schools. Enough has been stated to indicate that Bossi's dilator has not met with universal approval or adoption. I consider it to be the duty of each to assist in determining this question, to carefully observe for himself, and not to take for granted the dictum of anyone, however eminent. I had the privilege of showing to this Section some time ago a Bossi's dilator. Since then I have employed it in four cases, which I shall shortly relate:—

Case I.—On April 24, 1904, I saw in consultation with Drs. Cope and Neill, Mrs. ———, 3-para, who was pregnant between 6½ and 7 months. She was seized with convulsions at 4 50 p.m. The first attack lasted about 5 minutes; a second convulsion occurred about 8 30 p.m. I saw the patient at 10 30 p.m. She was then drowsy, and during palpation she was seized by another convulsion. The patient was put under chloroform. On vaginal examination os admitted index finger easily; the cervix was very short and apparently taken up; the head presented. On consultation, I advised dilatation with Bossi's dilator. Accordingly at 11 p.m. I introduced the dilator and arranged to give about two minutes to dilate 1 cm., keeping control by a watch placed in a convenient position. I dilated the os to 8 cm. The forceps
was applied and a child delivered at 12 o'clock midnight. The placenta was expressed. The time from the beginning of the dilatation to the end of the third stage was exactly one hour. I carefully examined for lacerations; none were found. Recovery.

Case II.—On May 7th Dr. Merrin asked me to see with him Mrs. P., in Holles-street Private Hospital. He stated that the patient is a primipara, and is 5½ months pregnant, and that she had two severe convulsions, one at 5 o'clock in the afternoon, another at 9 o'clock, that he administered after the first seizure ½ gr. of morphin hypodermically, and that he followed the recognised routine treatment. On admission she was complaining of pains in the back, and was undoubtedly in labour; the os was the size of a sixpence; cervix taken up. I diluted with Bossi's dilator up to 7 cm. without difficulty. Child delivered by expression. Head too small for forceps. Duration of entire operation—50 minutes. Foetal head damaged by dilator. No lacerations. Good recovery.

Case III.—Mrs. H. was sent up to me by Dr. Hickey, of New Ross. She was married three years and had five full term children. She missed a change on Jan. 14th of last year. About March the 24th a haemorrhage started; it lasted on and off until I saw the patient on June 14th. I kept her under observation in a private hospital. The haemorrhage became heavier, coming in gushes. I asked Dr. Horne to see her with me in consultation. No foetal heart could be heard. It was deemed advisable to empty the uterus. On vaginal examination the cervix was fully an inch in length; the os was so small that the unsheathed dilator was introduced with difficulty, much assistance being obtained by drawing down the cervix by an American forceps and retracting the perineum with a speculum. The dilatation was comparatively easy up to 6 cm., but I experienced much difficulty in dilating up to 8 cm., when the cervix suddenly ruptured. Presentation being transverse, I drew down a leg and delivered. On examination an extensive laceration was found extending up to the right fornix, which bled sharply and required to be stitched. There was a low insertion of the placenta almost marginal. Convalescence normal.
Case IV.—On October 14, 1904, M. H., a primipara at full term, got a convulsion during the first stage of labour. She was quite unconscious on my arrival; her urine was scanty, and loaded with albumen. The child presented in the first position; foetal heart good; os the size of a florin. I introduced a Bossi’s dilator, the patient being under chloroform, and dilated up to 11 cm. in 20 minutes. Forceps applied, and a live child delivered. There was no laceration. Normal convalescence.

Remarks.—It is difficult to dogmatise on such a small number of cases. Nevertheless I consider first impressions are of importance. That we possess in Bossi’s dilator a powerful instrument is admitted on all sides. But is it safe? I am convinced that within certain well-defined limits it has a distinct place in our armamentarium, but these limits must be clearly defined. Take my four cases—three of eclampsia and one of placenta praevia. In all the eclampsia cases labour had started, or at all events the cervix was taken up. Consequently, as I take it, the dilatation was easy—merely a matter of time and patience. On the other hand, take the placenta praevia case, with the cervix an inch in length, and none of it taken up. What was the result? In this particular case I examined continually with my finger up to the time of rupture. I was quite prepared for the event so stretched were the lips of the os. My chief concern was where would the laceration end.

I consider Bossi’s dilator dangerous when the cervix is long and not taken up, especially in the case of placenta praevia. The close proximity of the uterine sinuses adds a new danger, as should the laceration unfortunately extend to and open them up in what a grave position do you place your patient!

Dynamic Action.—Numerous advocates claim dynamic action for this dilator; in other words, that it causes or
sets up uterine action even in cases of inertia. I closely observed for any evidence of this influence, and I am of opinion that the dilator increases the uterine action by stimulating the large cervical when uterine action has already started. It acts much as one's finger does when we massage the region of the internal os to stimulate the uterus to contract in case of post-partum haemorrhage, and I attributed the damage to the fetal head, reported in my second case, to the strong contractions of the uterus pressing the head against the dilator before the sheaths were put on. But does it originate uterine action? I have no evidence in support of this theory.

The Degree of Dilatation.—One great difficulty confronted me in my first three cases. If a full-term head requires the os to be dilated up to 11 cm., how much dilatation will be necessary to deliver the fetal head at the sixth or seventh month? Surely it should not be dilated up to 11 cm.? In none of the monographs at my disposal could I find any light thrown on this question.

In order to formulate a basis for accurate observation I drew up a scale for my own guidance. I took term in the S. O. B. diameter, or 32 cm., which requires for safe delivery a dilatation of 11 cm. I then ascertained by reference to Dr. A. Fancous' thesis that the S. O. B. diameter at the fifth month is 15.9 cm.; at the sixth month 19 cm.; and at the seventh month 20.9 cm. Assuming these figures to be an average, I provisionally proposed to dilate in the fifth month up to 6 cm., in the sixth month to 7 cm., in the seventh month to 8 cm., recognising that during these early months one must consider the cervix as more or less of an elastic tube
which, on the withdrawal of the dilator, has a tendency to contract.


Dr. Kidd said that one disadvantage of Bossi's dilator had been met by changing the number of blades from four to eight, thus having eight points of pressure on the lower zone of the uterus, and less probability of rupture. An advantage of Bossi was that one could watch digitally the amount of tension between the four blades, which was hardly possible when eight blades were introduced. Another objection was that the angle at which the dilator was set to the shaft was not the angle at which dilatation would take place. Seigneux tried to meet this objection in his dilator, but it had its disadvantages also. There was too much resiliency in the lateral blades, and in order to try and gain the advantages of having eight blades one had to change the blades and insert blades with broader flanges, and this was a very troublesome proceeding. He was quite sure that where mechanical dilatation was used for placenta praevia the risks of laceration were greatly increased. He certainly thought that its use in eclampsia would give happy results.

Dr. FitzGibbon said he had used Bossi's dilator in a case in which he had induced labour at seven and a half months. He started by using bougies, but the uterus took no action, and they were introduced again the day following. The membranes had then ruptured, and the following morning he decided to dilate the cervix, into which he could introduce two fingers. He dilated up to 7 cms. He did not intend to complete delivery, and left the patient, having put some gauze in the cervix. Labour set in in three hours, and she was delivered three hours afterwards. He considered that labour had been induced by mechanical dilatation, which started uterine action.

Dr. Hastings Tweedy said he could hardly think that dilators had been responsible for the salvation of even one woman. Medical papers now-a-days teemed with the praises of mechanical
dilatation of the cervix in the treatment of eclampsia. He had treated within the last fifteen months ten cases of puerperal convulsions in the Rotunda Hospital with one death; in this case the diagnosis of eclampsia was doubtful. In a large number of these, it was observed that convulsions became severe after the birth of the child, so that the emptying of the uterus could not be considered a save-all and cure-all. In but one of the successful cases was delivery accelerated even by forceps, in the others labour was allowed to go on naturally, and finish. These results show that recovery will take place even though violent means to extract the foetus are not adopted. As to the dilator’s usefulness in ante-partum hemorrhage, due to placenta praevia, he must dissent from the views of those who recommended its employment; in such cases the cervix often tears without one being conscious of the fact. The treatment could never compare favourably with the plan of bringing down the leg and leaving the case to nature. The cervical epithelium is bruised during the process of forcible dilatation, and such bruising predisposes to sepsis. There was one class of case in which he thought the dilator was sure to be applied—“rigid os;” but its use in such cases was also sure to be repented of. Bossi himself counted the condition as a contrary indication. Seigneux’s instrument appeared to have some slight advantage over the other dilators; but if one desired to open the cervix it could easily be accomplished by any one of the lacerating instruments at present on the market.

Dr. Purefoy said that the range of utility of a dilator was not at all as wide as they had been led to believe. The utmost caution was necessary in its use, and the cervix was often found lacerated without it having been noticed. In cases of eclampsia it might sometimes be of use, but the number of cases was very small. It was useful in cases where labour had started and the pains had come on and afterwards stopped.

Dr. A. J. Smith, in replying, said he had tried the dilator, having heard and read so much about it. He thought that it was in places like Dublin that the principles of new inventions should be tested, as they had to report on them to country practitioners. As to its use in eclampsia, he thought that certain cases would get well no matter what form of treatment was used.
PUERPERAL CONVULSIONS.

By R. J. KINKEAD, M.D. DUBL.;
Professor of Obstetric Medicine, Queen's College, Galway.

[Read in the Section of Obstetrics, February 3, 1905.]

In the communication I have the honour to lay before the Obstetric Section I do not propose to discuss at length the ætiology of eclampsia; the subject is obscure and our knowledge of it defective.

In a very recently published work on obstetrics I find it stated:—

"During gestation the presence of albumen in the urine is due in a great measure to venous congestion of the kidneys, which results from the pressure of the gravid uterus. But, while passive congestion and hydrenephrosis may be thus technically included under the term pregnancy kidney, there is an entirely different affection to which the designation is familiarly restricted, and which most certainly is not due to pressure, nor has it anything in common with ordinary nephritis, whether pre-existent or developed during gestation as a result of exposure or infection."

"For the present, therefore, we may regard pregnancy kidney as due to a toxæmia which is also capable, at times, of causing eclampsia as well."

"Thus we are still in doubt at the present time concerning the essential ætiology of eclampsia. Though all Bouchard's statements may be correct, the true nature of the poison has not been clearly demonstrated."

That we should still be in the dark is not, I think, surprising when we consider the complexity of the
problem and the changes produced by pregnancy and parturition on the functions of the female. If we take account of facts, apart from theories, it seems to me that the wonder is not that eclampsia sometimes occurs, but that so many women escape.

I think it was Robert Barnes who described pregnancy as an experiment performed by nature for our information, which tests every organ of the body and searches out every weak spot. Woman is more emotional than man, her nervous system is more excitable; this normal condition is exaggerated by pregnancy, and she becomes more responsive to stimuli likely to produce reflex movements: digestive disturbances are usual, and thus there is a tendency to a lack of proper balance or equilibrium between the functional activity of parts of the nervous system and to errors of nutrition, whereby the nerve cells may become unstable and prone to undergo discharges of a morbid character.

Not only is there the development and awakening into action of special glands like the breasts, but every gland in the body is forced into greater activity to meet the demands of the uterus and foetus and to contend with the increased metabolism from their growth.

As the chief eliminating glands, vastly increased work is performed by the kidneys. They are, however, handicapped by increased cardiac propulsive power, vascular tension, and more or less anaemia (a condition somewhat similar to what obtains in Bright's disease).

I do not agree with the statement that during gestation the presence of albumen in the urine is due in a great measure to venous congestion of the kidneys resulting from pressure of the gravid uterus, for not only may albumen be found in the urine from vaginal discharge, especially in that passed on getting out of bed, but it is
present and convulsions occur when the uterus is too small to cause pressure. When, however, passive congestion arises from pressure it adds to the embarrassment of the already over-taxd kidneys.

The kidneys appear to be in a state of unstable equilibrium between a physiological and pathological condition: they may remain on the safe side or be forced over into the danger zone, and if they be already damaged by antecedent disease the greater the probability of their giving way.

Then there are apparently contradictory facts—a high degree of albumen in the urine and no convulsions—or convulsions and no albuminuria; in some cases terminating the pregnancy immediately relieves the kidneys and stops the convulsions, in others convulsions commence and albumen appears after the uterus has emptied itself.

"Certain toxic alkaloids, to which Gautier has given the name of leucomains, may be developed during life in the course of certain maladies," and, like ptomaines produced in dead tissue, may be convulsive poisons.

If the dose discharged into the circulation be large enough convulsions shall occur even with physiologically acting kidneys.

Brouardel recounts the case of a druggist:—

"During his wedding night his young wife had an epileptic seizure; the attacks recurred. The husband analysed his wife's urine before, during, and after the attacks. When the attack was coming on the urine always contained an enormous quantity of ptomaines or leucomains." 2

If the toxin be gradually generated no symptoms may be produced if it be eliminated as rapidly as formed, the amount circulating in the blood not being sufficient to cause a nervous explosion; but if the kidneys have
passed into a pathological condition, and their power of excretion is more or less defective, it may be sufficient to eliminate the toxin or it may not; in the latter case the poison accumulates in the circulation, and when an effective dose is attained convulsions supervene.

"A woman who sold poultry in the market, having a stuffed hen turkey that had not been sold, and which she feared would be spoilt, invited her friends and relatives to eat it with her. Twelve persons partook of that repast, and no one noticed any peculiar smell or flavour about the turkey. The woman herself, who had not eaten more than her guests, was taken ill in the night and died. Her guests also became ill, but none of them died.

"We found in the stuffing inside the turkey a product analogous to conicine, and we met with the same product in the viscera of the dead woman. I repeat that this woman had not eaten more of the turkey than the rest, but at the autopsy we discovered that the kidneys were diseased, and unable to eliminate the toxins."

Labour involves powerful and often prolonged muscular exertion, much pain, large expenditure of nerve force, and active and passive congestion of the kidneys. Muscular exertion means combustion of tissues, consequent effete material to be eliminated, and, when prolonged, the production of toxic alkaloids during life as the result of over-work and exhaustion.

Kidneys just able to discharge their functions during pregnancy may be overburdened during labour, or become suddenly blocked after delivery, and this may have happened in the interesting case of post-partum eclampsia for which I am indebted to Dr. Costello of Dunmore.

It appears to me, however, that the supposition of a toxin does not cover the whole ground, and that the
individual factor must be taken into consideration. How far a neurotic temperament or unstable state of equilibrium in the nervous centres tends to the production of convulsions with or without kidney incapacity is an open question. It would be interesting to ascertain the relative frequency of eclampsia in peasant women, town dwellers, and in the educated and wealthier classes; à priori, we would expect environment and habits of life to have considerable influence, and that the cultivation of the emotional and nervous centres at the expense of physical development, tends to produce increased sensibility to pain and a condition of the nerve centres more responsive to stimuli.

So far as I have been able to inquire amongst some of my friends in dispensary practice in rural districts, I find convulsions reported to be very rare among peasant women.

The following cases are interesting as illustrating what I may call the uncertain incidence of eclampsia, the marked effects produced by terminating the pregnancy, and the beneficial effects of chloral and chloroform.

In the first case the result of the death of the fetus seems to indicate that its life is a factor in producing congestion of the kidney.

Case I.—Fourth pregnancy. Ten years had elapsed since birth of her last child, following which she had a desperate attack of septicæmia, with acute nephritis and haematuria; ever since there had been some albumen in the urine, and occasionally casts, but the general health had been fairly good, and there was no evidence of progressing kidney disease. On pregnancy supervening the quantity of albumen increased, and casts became numerous and constant; at four and a half months vision became affected, headache, nausea, oedema and somnolence came on. I brought her to Dublin for consultation, and it was decided to endeavour, by diet and treatment, to tide her over
to the seventh month, when, if labour was induced, there would be a likelihood of the child surviving. In a few days blood appeared in the urine; after a week or ten days of sharp haematuria—about 10 o’clock one night—alarming bleeding from the nose, necessitating plugging, set in, and next morning the child died. Prior to the death of the child the urine looked exactly like blood, and, on cooling, a large coagulum formed; from the death of the child there was a steady diminution of the quantity of blood, so that when labour set in—some 28 hours after—although the urine was red it was clear, and there was no coagulum; within twenty-four hours after the expulsion of the fetus the urine appeared to be free from blood.

We rarely have so decisive a demonstration of the relief to the kidneys which follows the cessation of physiological activity. It cannot be attributed to the nasal haemorrhage, for the urine passed from its cessation until after the death of the fetus next day was as full of blood as before its occurrence.

Case II. was a primipara. As I knew that some fourteen years previously she had had a slight attack of nephritis, subsequent to scarlatina, I examined her urine every fortnight. Prior to pregnancy its sp. gr. was 1020, and it was free from albumen and casts; shortly after, albumen appeared, and although she was at once put on diet and under treatment the albumen increased in quantity; casts were found; later on there was oedema, then headache and defective vision. Labour was induced at the seventh month; the child survived and the mother recovered. In due course she became pregnant again, and although there was a moderate amount of albumen, she went to full time. In her third pregnancy, however, albumen appeared immediately, increased rapidly, and the symptoms became so urgent that I was forced to induce labour in the fourth month. Her fourth pregnancy was normal. On both occasions in which labour was induced the relief to the kidneys was immediate.

In neither of the cases can the albuminuria be attributed to pressure. In both the kidneys had been previously damaged. In the first relief came too late; the
kidsneys went from bad to worse, and the patient died a year afterwards from uræmia and convulsions. In the second, although always delicate, the lady lived several years, and died from pulmonary tuberculosis.

Case III.—Second confinement. As in first pregnancy (she aborted in four and a half months) there had been some albumen in urine and slight oedema. when about a year after she became pregnant for the second time, I examined her urine every fortnight. There was a steady increase of albumen, but it was not excessive until a few hours before attack. At the beginning of the ninth month I was sent for as headache had come on suddenly; it was not very severe, there was no affection of vision, no vomiting or nausea, no increase of oedema; she said she was passing more water than usual. As there was constipation, I ordered her a purgative, and took away a specimen of urine for examination. I was called at 6 a.m. next morning, and on arrival found she had had a convulsion: two more fits occurred before I could get her under chloroform. I then passed a catheter into the uterus, and gave her an enema of mucilage of starch, with 30 grs. of chloral hydrate. Notwithstanding that she was kept under chloroform there were two slight fits in the next hour; she then got a second enema with 30 grs. of chloral, and she got two more in the course of the day, and was kept under the influence of chloroform until the child was born, at 6 p.m. After the second chloral enema there were no further seizures; there were occasional threatenings, but fits were averted by pushing the chloroform. The urine passed in the morning and that drawn off in the course of the day became almost solid on boiling and with nitric acid. After delivery the albumen rapidly disappeared, and the recovery was uneventful. She has since had three children, and although albumen was present it was trifling, and there was no cause of anxiety.

Case IV.—Second confinement, the first at 8 months, reported to have been normal. Convulsions came on suddenly late in the evening without any prodomata, except some headache for two days. Her husband, a medical man, informed me on my arrival, three hours after commencement of attack, that the fits recurred every twenty minutes; he had given a hypoder-
mic of $\frac{1}{2}$ gr. morphin. This was repeated; she was put under chloroform, urine drawn off, and a catheter passed into uterus. During an hour there were three convulsions. I then gave an enema of 30 grs. of chloral hydrate; during the next hour there were warnings of recurrence, which subsided on pushing the chloroform; a second chloral enema was given, and she was kept under chloroform until the birth of the child, at 6 a.m. After the second chloral enema there were no more fits. The urine drawn off became almost solid on heating, marked diminution of albumen followed delivery, and convalescence was uneventful. A couple of years after she again became pregnant; there was no albuminuria, and both pregnancy and delivery were normal.

Case V.—This was also a second pregnancy, the first at full time was normal. Her health in the interval—five years—had been good, as it had also been during her pregnancy (which had arrived at the eighth month) until a couple of days previous to the attack, when, after coming in from boating, she was seized with a sharp pain in the back of her head and neck. The attack was ushered in suddenly by a single convulsion. I saw her an hour after, and she was then conscious and her mind quite clear. While talking to me a second convulsion came on, and the fits recurred rapidly until I got her under the influence of chloroform and chloral. I induced labour, and it was easy to stave off threatening recurrences by pushing the chloroform. After delivery rapid reduction of albumen; there was, however, much suffering for many days from pain in the back of the neck, and along the spine, and convalescence was slow.

Case VI.—Also a second pregnancy, the first going to full time and being perfectly normal. She had a history of chronic dyspepsia from girlhood; diet mostly farinaceous, seldom eating meat; of a nervous temperament, both before and after marriage she suffered from incontinence of urine if going out to walk, or to church, or to dine. During pregnancy her digestive trouble was a little aggravated, but at the fifth month she was feeling well, when suddenly irritability of the bladder set in, necessitating the frequent evacuation of small quantities of urine, especially at night, when she was obliged to get up every half hour. After two nights' loss of rest I was sent for; she expressed
herself as feeling well save for the urinary trouble; though weary from sleeplessness, she had no headache or vomiting, and though the face and hands looked full there was no pitting on pressure. I ordered a purgative and a diuretic, leaving directions for a specimen of urine to be sent to me. Early next morning I was called to her: on arrival I found her dazed and confused, after a couple of sharp convulsions, in which her tongue was rather badly bitten. Very soon there was another fit; I gave her a chloral enema and started chloroform; in half an hour the enema was repeated. I examined a specimen of the urine passed during the night, and one drawn off after the convulsion came on: while the former was loaded with albumen the latter became almost solid. I induced labour, and dilatation of the os uteri proceeded satisfactorily; chloral and chloroform staved off further fits, and the foetus was expelled in about five hours. The urine drawn next morning showed a reduction of albumen to less than a half, and the third day there was only a trace; the irritability of the bladder disappeared at once. On the second day after delivery she described how the attack came on. She had had a poor night from frequent calls to pass water, but, though tired, did not feel unwell: between 8 and 9 a.m. she got up to urinate, when she observed that the edges of the window sashes were zig-zag, then flashes of light shot before her eyes, singing came in her ears, she fell, and remembered no more.

Case VII.—Primipara. Family history good; believes there was slight oedema of legs in later months of pregnancy, but she walked daily, in apparently perfect health, to her school up to the day of her confinement. Labour natural and easy, completed within four hours; placenta expelled in twenty minutes; was perfectly well, laughing and talking. About twenty minutes after the doctor left she got a convulsion; he came back at once and gave her a dose of chloral; in three-quarters of an hour fit recurred, and in it she dislocated her right shoulder. Morphia was given hypodermically, and when she was able to swallow, spts. eth. nit. and jaborandi. Skin dry, catheter passed, no urine in bladder, dry cupped over loins, and nitrite of amyle inhalation, notwithstanding which fits recurred every hour. After two hours skin became moist; catheter passed and pint of urine drawn off, almost solid with albumen; fits less frequent and severe. Skin
got dry again, and temperature ran up to 108°. A cold pack and sponging brought it down to 100°; remained unconscious; died twenty-seven hours after first convulsion; suppression of urine from time catheter was passed.

1 Edgar. Practice of Obstetrics.
2 Brouardel. Death and Sudden Death.
3 Brouardel.
CURATIVE OPERATION FOR PROCIDENTIA UTERI.

By E. HASTINGS TWEEDY, F.R.C.P.I.:
Master of the Rotunda Hospital, Dublin.

[Read in the Section of Obstetrics, March 10, 1905.]

"Every gynaecologist who has much hospital experience must have had cases of severe total prolapse of the uterus and vagina which are intractable to ordinary measures, cases in which no pessary can be retained, and in which the ordinary plastic and suspensory operations fail to give more than temporary relief."

So writes Dr. Christopher Martin in a communication to the British Gynaecological Society, which is fully published in the Society’s Journal of November, 1904. In this paper he describes his very radical operation for the relief of the severest forms of this malady.

In the main it consists of the total removal of the uterus, together with the removal of the mucous membrane of the posterior and anterior vaginal wall down to the urethral orifice. The peritoneum, the stumps of the broad ligaments, and the pelvic fascia are united in separate layers with chromicised catgut, and finally the raw vaginal surfaces are brought together by sutures.

To sum up this operation in Dr. Martin’s own words, “It is a long and difficult operation, and is attended with a good deal of risk to the patients, who are as a rule elderly women and often in feeble health. There is free haemorrhage during the course of the operation. There is considerable risk of wounding the bladder, the uterus, and the rectum. After the operation there is a good deal of shock, and shock in an old feeble woman is a
By Dr. E. Hastings Tweedy.

serious matter. The convalescence is apt to be a tedious one, and in all my cases was complicated with deep-seated suppuration in the wound."

That such an operation should be seriously put forward as a means of relieving a condition not in itself dangerous is sufficiently surprising, but still more remarkable is the apparently favourable manner in which the communication appears to have been received by the members present at the meeting of the British Gynaecological Society.

This Society, of which I have the honour to be a member, exercises no little influence on surgical thought throughout the British Empire, not alone because of the high order of merit of the work submitted to it, but also because of its well-informed and widely circulated Journal. It seems to me the plain duty of those who believe that surgery has at its disposal milder and more efficient methods in dealing with this diseased condition to raise a note of protest against Dr. Martin's operation; as an alternative, I desire to bring to your notice an operation which, on theoretical grounds at all events, I cannot but believe fulfils the requirements of a complete cure.

I do not claim originality for this procedure, but cannot at present recall the source from which I derived my knowledge of it. It probably was in an epitome of current literature in one of our many medical journals; if, therefore, my description differs in important points from its author's, I urge as an excuse that the procedure had to be evolved by me from scanty material.

I have now performed the operation three times, and shall describe the technique as it was carried out on

Mrs. Z., who suffered from a complete procidentia with an ovary prolapsed into the posterior vaginal wall, and extruded
through the vulva. Her health was good, and she had not passed the climacteric. Some days' rest in bed, combined with the usual antiseptic douches, caused a cervical ulcer to rapidly heal.

The first steps of the operation consisted in the removal of a large oval flap of mucous membrane from the anterior vaginal wall, combined with a transverse incision across the cervix and just below the bladder; the latter organ was separated from the cervix, and the abdomen opened between it and the uterus. Access to Douglas's cul de sac was next obtained by a transverse incision made in the usual manner behind the cervix. These transverse incisions were connected at their extremities by lateral incisions, as in the operation for vaginal hysterectomy.

An assistant now pulled the cervix forcibly to the left side, whilst the forefinger passed in front of, and the thumb behind, the broad ligament, were made to firmly grasp the base of this structure.

The mucous membrane of the lateral fornix was now completely detached from the base of the broad ligament by means of a scissors curved on the flat; during this the fingers protected the uterine artery and ureter from injury. A similar procedure was adopted on the corresponding side.

The bases of the broad ligaments, with their ruptured fibrous bands, were now brought into clear view; these robust fibrous bands, known as the ligamenta transversalia coli of Mackenrodt, are composed of dense connective tissue firmly united to the supra-vaginal portion of the cervix, contain unstriped muscular fibres, and constitute by far the most important elements in keeping the uterus from prolapsing.

A curved needle carrying a fine suture (No. 4) was passed through the base of the broad ligament near its pelvic extremity, and the other broad ligament was treated in a similar manner. When these ligatures were brought together and loosely tied in front of the internal os, the cervix was seen to be raised upwards and backwards, slung as it were between these reunited fibres of Mackenrodt's ligaments. The loose knot was now united so as to enable the cervix to be again brought down and permit of its easy amputation. When this was accomplished and all haemorrhage controlled, the cervix was again pushed up, and the threads attached to the broad ligaments at either side of it were
tied and knotted in front of, and on a level with, the internal os. The broad ligaments were still further secured in front of the uterus by uniting them to that structure with several fine silk sutures.

The fundus of the uterus, with its intervening layer of peritoneum, was next secured to the anterior vaginal wall above the urethra, and the further steps of the anterior colporrhaphy completed.

All that now remained was to perform Hegar's colpo-perineorrhaphy and to unite the vaginal skin to that of the cervical mucous membrane.

In my first and last cases I performed this operation without opening into the peritoneal cavity. I do not, however, advise the step, and believe the method above described will yield the easiest and most satisfactory results.

In conclusion, I submit that this operation, when properly performed, will prove in every sense a curative one: it rectifies the abnormal conditions answerable for the prolapse, and I sincerely trust it will obtain a fair trial before surgeons resort to what, in my opinion, is a more serious and more mutilating procedure.

Dr. Glenn said that the original operator passed the stitch through the back of the cervix, and drew it upwards and backwards towards the promontory of the sacrum, the fundus falling forwards. An anterior fixation was also done.

Dr. Jellett said that in many cases of prolapse some form of operation was necessary. He was in favour of vaginal fixation and some form of narrowing operation of the vagina. He had no dread of pregnancy afterwards, never having had any trouble. He had lately opened an abdomen in which anterior fixation had been previously done, and it had had excellent results. He thought that that operation, plus narrowing, was usually quite sufficient, and if not, Dr. Tweedy's operation was an excellent one, as it was on a sound anatomical basis, and got the uterus anteverted, &c., at its normal level.
DR. PUREFOY said that the subject was one of great interest, as some of those cases might be the start of a fatal illness. He had seen one case of peritonitis resulting from such. With regard to the various structures which kept the uterus in position there was some difference of opinion. In Dr. Savage's book there is a series of plates on the subject, and he demonstrates that the structures which chiefly prevent procidentia are the utero-sacral ligaments. The procedure which Dr. Tweedy had shown was in certain cases an excellent one, but was manifestly not suited to all cases of procidentia. Huguiier pointed out that in many cases hypertrophy of the cervix was the cause of procidentia, and this could be dealt with by amputation of the cervix.

DR. A. J. SMITH said that he had found the necessity for some reliable operation for procidentia. He had read the original description of the operation, but had thought it exceedingly theoretical. He had not seen how, in tying the sutures, you avoided compressing the ureters, and he was delighted to hear of Dr. Tweedy's favourable results. He was quite at one with Dr. Jellett with regard to the value of vaginal fixation, plus the ordinary colporrhaphy, in ordinary cases of prolapse. It gave very good results. He had also got good results from abdominal fixation.

DR. TWEEDY, in reply, said he was exceedingly obliged for the favourable way the Section had received the operation. He felt that there were great possibilities in it. Vaginal fixation was not sufficient in the worst forms of prolapse, and he was not at all so convinced as Dr. Jellett that the results of a subsequent pregnancy in all cases would be what one would like. The operation when performed in an ideal manner would give admirable results in pregnancy, but cases were met with in which, before the uterus could be brought forward, one had inflicted such injuries on it that firm adhesions would form. Like Dr. Purefoy, he had formerly attached enormous importance to the sacro-uterine ligaments, but he had now adopted the view that the ligaments he had pointed out were perhaps of more importance in keeping the uterus in proper position, especially in that of elevation. There was frequently a deep laceration of the cervix in bad cases of procidentia, and in such cases Mackenrodt's ligaments were probably torn.
TWO CASES OF WOUNDS OF THE FEMALE GENITALS.

By R. J. KINKEAD, M.D., DUB.;
Professor of Obstetrics, Queen's College, Galway;
Physician and Gynaecologist to the Galway Hospital.
[Read in the Section of Obstetrics, March 10, 1905.]

When a woman falls astride on a hard substance, or receives a kick on the genitals, owing to the small amount of tissue between the surface and the bones, the resulting wound is incised rather than lacerated. The vulva being profusely supplied with blood-vessels, and the veins forming the bulbs of the vagina anastomosing freely with those of the vagina, uterus, and rectum, the bleeding from such wounds is profuse, and if the woman be pregnant is apalling; the nearer to term the larger the vessels, and the greater and more rapid the loss of blood. In all cases dangerous, in advanced pregnancy wounds of the genitals are usually fatal. We not infrequently read in the papers a report that a pregnant woman received a kick in the lower part of her body, was immediately conveyed to hospital, and was either found to be dead on arrival, or died in a few minutes after admission.

The two following cases are interesting, one from a forensic as well as from a surgical point of view; the other from the extensive injuries inflicted:—

About 7 p.m. one evening in the Spring of 1903, I was stopped on my way home and asked to go at once to a woman who had met with an accident, and was "bleeding to death." Her husband, who gave me the message, said he had rushed off at once, it took me two minutes to ride to the house, so I saw her
within ten minutes of her being wounded. She was a married woman, aged thirty-four, the mother of several children, and some months previously had had a bad miscarriage. I found her blanched, pulseless, the heart's sounds almost inaudible, sightless, semiconscious, respiration sighing. There being no visible wound, a trained maternity nurse, who had gone to her assistance, believed the bleeding to be uterine, and had applied wet cloths to the vulva and lower parts of the abdomen; the bleeding had ceased, but the woman was perilously near death. I gave her a hypodermic of ether, had hot bottles applied to feet, limbs and body; some ounces of hot normal saline were thrown up the rectum, and ten ounces transfused under the breasts. As she rallied the bleeding recommenced from a wound in the anterior wall. She was too weak and collapsed for operative interference, so I had to be content with plugging and the application of a "T" bandage.

In the morning, April 24th, there having been no further haemorrhage, she was brought to hospital. I found an apparently incised transverse wound, an inch and a half in length, in the anterior vaginal wall, fully an inch inside orifice, involving the urethra, and which bled freely on removal of plugs. To stop the bleeding, rather than in the hope of obtaining union, I sutured it. Her temperature on admission was 90°, at night 101°. On the 25th there was no bleeding, temperature 100° to 101°, vomiting during night; some oozing on the 26th, which was stopped by adrenalin; wound not looking healthy. Vomiting continued on the 29th; right wrist became painful and puffy; edges of wound sloughing; touched with pure carbolic acid.

She went through a severe attack of septicæmia, in addition to the right wrist the right groin and left shoulder were affected, constant vomiting, profuse sweating. On three occasions the temperature went up to 105.8° and 105.6°, but fell immediately on administration of Warburg's tincture. She was discharged on July 27th. The application of carbolic acid stopped sloughing of wound, which, however, was slow in healing. Although the urethra was cut, and for a considerable time urine escaped through the wound, there is now, after the lapse of over a year, no stricture, nor any difficulty in micturition.

Her history of the occurrence was that she had a dispute with
her husband, that he gave her a push; she had stumbled and fallen over a Windsor chair, the legs of which had been cut down for nursing purposes, and that one of the legs had gone up into her. After her recovery, however, she admitted that the injury was produced by a kick, the story of the overturned chair being invented to shield her husband.

It is here that the medico-legal interest of the case comes in, for the appearance was consistent with the chair story, as there was no injury of the vulva, the only external mark being an ecchymosis, which appeared on the second day, half-way between the right labium majus and the thigh, while, a priori, it was to be expected that the toe of a labourer's boot was bound to have injured the external genitals, and that it could not possibly have inflicted a wound inside the vagina either with or without damage to the vulva. The explanation seems to be that in this case, as in a number of multipara, there was a slight prolapse of the anterior vaginal wall, which caused the lower inch, or inch and half of the anterior column to protrude through the vaginal orifice, when the woman was standing up. A kick, when in the erect position, with the legs separated, if it took effect in the middle line, would drive the protruded portion against the pubic arch, cutting it like a knife. The patient being examined lying down, the wound would be found inside the vagina, and the inference would be that it must have been inflicted by some instrument introduced within that canal.

In the second case the injury was the result of accident.

A servant girl, putting up window curtains, doing what she was warned not to do, stood on the flat top of a step-ladder. She slipped and fell straddlelegs, the corner of the flat top of the ladder evidently struck her inside the right labium majus.
The bleeding was severe, but not excessive. She was brought into town, and I was sent for to see her. A cursory examination showed grave injury, and I had her at once removed to the Galway Hospital. Though she had lost much blood she was not collapsed, and was at once put under ether. The wound and genitals were thoroughly irrigated and cleansed. I found a wound extending from a little below the level of the posterior margin of the vaginal orifice, on the right side of, and within, the vulva, to the level of the pubic arch; its depth at the lower angle was an inch, getting deeper as it passed up, till it attained a depth of quite three inches at its upper angle; nor was this all, for it penetrated under the tissues, and on passing my finger up, I found the pubes laid bare up to the middle margin of the symphysis. The urethra had been torn from its attachments and pushed downwards and over to the left side. The vagina was also displaced and the hymeneal attachment above, to the right and about half way below, torn from the vagina. The hymen was a very perfect one, with a small oval central opening, and could be lifted up, like the lid of the nest of the tarantula spider. I first sutured the deep portion of the laceration under the pubis; I next passed a deep suture along the side of the urethra and out at the upper angle of the wound; another at the anterior margin of the urethra emerging in the vestibule thus bringing it back to, and fixing it in its normal position; and a lateral suture, immediately below the urethra, brought the edges of the wound into apposition to the level of the vaginal orifice. A few fine sutures were then introduced attaching the separated hymeneal margin to its position below the urethra, and at the side; a couple of sutures through the margin of the hymen, along the separated portion of the vagina, under the bottom of the wound, and emerging at the end of the tear, closed the wound, and brought the vagina back to position. The wound was kept constantly irrigated during the stitching, and a catheter was tied in for the first twenty-four hours. Except that the patient was rather collapsed, and vomited a good deal for twenty-four hours, there was no trouble, the wound healed without any suppuration; there was no rise of temperature, the hymen united at the torn margins; and when she left the hospital it needed a close examination to detect any trace of injury.
The restoration of the hymen may possibly be regarded as trivial, but I believe that when dealing with unmarried women we are bound, if possible, to restore the parts to the condition they were in before accident or operation. I was induced to expend some time and care in suturing the hymen to its severed attachments from my experience in another case, with a very well developed hymen, in which, when operating for stenosis of the cervix, I incised the hymen on both sides, making a posterior flap, that, after completing the operation, I replaced, suturing the cut edges. Union was perfect, not even a nick in the circumference of the aperture marking where the hymen had been cut.
NOTES ON A CASE OF LABOUR IN A UNILATERAL SYNOSTOTIC PELVIS.

By HENRY JELLETT, M.D., F.R.C.P.I.;
Gynaecologist and Obstetric Physician to Steevens' Hospital, Dublin.

[Read in the Section of Obstetrics, April 7, 1905.]

Oblique contraction of the pelvis, and particularly that form known as Naegele's pelvis, is a rare condition in this country, and therefore I think that the notes of a case which recently came under my care may be of interest to the Academy. The nature of Naegele's pelvis is shown by the other name that is applied to this form of contraction—namely, the unilateral synostotic pelvis. Its characteristic and distinguishing feature is ankylosis of the sacrum with the ilium on one side, and almost complete atrophy of the lateral mass of the sacrum on the same side. The cause of the ankylosis is usually to be found in a congenital and unilateral failure of the centres of ossification from which the lateral part of the sacrum is normally developed. These changes cause a pronounced unilateral narrowing of the sacrum, and excessive pressure is, in consequence, thrown upon the deformed side. This pressure displaces the sacrum downwards and forwards on that side, and ultimately is responsible for the atrophy of the joint surfaces. In a few cases it is probable that the sacrum in the first instance becomes displaced forwards on one side as a result of injury, and then becomes ankylosed in its new position, thus preventing further lateral development. In a few cases inflammation of the sacro-iliac joint may be the cause of both
displacement and synostosis. The result of these changes
is a marked form of oblique distortion. The sacrum is
depressed and displaced towards the diseased side, towards
which it faces. The symphysis is drawn towards the
healthy side. The inlet assumes the shape of an
obliquely-placed ovoid, the long diameter of which cor-
responds with the oblique diameter drawn from the
diseased joint. The true conjugate may be slightly
increased in length, and the transverse diameters are
shortened, both at the brim and at the outlet. Slight
scoliosis is usually present in the lumbar region.

The patient, L. L., a primipara, aged twenty-four, was admitted
to the maternity ward of Dr. Steevens' Hospital on January 6th
last. On examination her abdomen was found to be unduly
prominent, and the uterus, which was of large size, was so tense
that the foetal parts could not be felt on palpation. As the con-
dition of the abdomen suggested the possibility of the presence
of pelvic contraction I examined her vaginally on the following
morning. At that time she was not in labour, and the os was
closed. A small head was felt presenting at the brim, between
which and the brim there did not appear to be any disproportion.
I measured the oblique conjugate with my fingers, and estimated
the true conjugate to be about or over 10 cms. Unfortunately
I did not palpate the pelvic brim, and consequently I made no
note recording the condition of the transverse diameter. A
diagnosis of twins was made.

The patient came in labour the following day, and the mem-
branes ruptured at 10 30 p.m. Labour pains continued during
the night, and at 7 a.m. the following morning my resident, Mr.
Boyd, telephoned for me to come and see her on account of the
delay in the expulsion of the foetus. I saw the patient at 8 30
a.m. The foetus lay in the second vertex position with the back
behind, the head lay transversely in the pelvic cavity, Naegele's
obliquity was well marked, and the anterior parietal bone pre-
sented. There was a large caput succedaneum. There was some
difficulty in applying the forceps, chiefly owing to a transverse
narrowing of the outlet, but when the blades were applied to the
sides of the head, so that one lay in relation to the symphysis and the other in relation to the sacrum, they slipped on easily, and the head was delivered without difficulty. The child was alive and in good condition. On palpating the uterus a second fœtus could be felt presenting by the head. I then waited for a couple of hours before rupturing the membranes of the second ovum, in order that the patient might obtain some rest, and finally ruptured the membranes at 11 30 a.m. As soon as I did so some meconium came away, and the cord and then a hand prolapsed through the os. I introduced my hand into the uterus with the object of turning and bring down a foot, but on doing so I found that the retraction ring was nearly two inches above the symphysis and the uterine wall very tense, and that, in fact, version was too dangerous a procedure to attempt. As the head was presenting, and as there were no uterine contractions, and had not been any since the birth of the first child, I did nothing further, and the patient was returned to bed. During the foregoing manipulations I recognised the existence of transverse narrowing of the brim. My reason for not delivering the patient immediately was that, in the absence of uterine contractions and in consequence of the partial emptying of the uterus, I did not think delay was likely to prove harmful, while immediate delivery was sure to be followed by uterine atony and possibly by post-partum haemorrhage. At 1 p.m. I saw the patient again, and as her pulse had become more rapid and her general condition was worse I decided to deliver her, although there were still no contractions to speak of. On examination the head presented at the brim, and the hand was prolapsed beside it. I applied forceps, pushed the head slightly upwards, so as to allow the replacement of the arm, and then applied traction. However, although a considerable amount of force was exerted, the head could not be induced even to enter the brim. As the child was dead, I accordingly perforated the head and extracted it. The perineal sutures which I had inserted after the birth of the first child were tied, and the patient returned to bed. At 5 p.m. I was again sent for in consequence of retention of the placenta. Both placenta were adherent, and there were and had been no uterine contractions, but as the patient's temperature had risen to close on 102° F., I thought that it was best not to wait any longer, and so removed the
placentae manually. Very free haemorrhage followed, and the uterus was plugged with iodoform gauze. The perineal sutures, which had been removed to facilitate the placental removal, were re-inserted. The patient's pulse was then 116, and her temperature 102.1°. All the various operative procedures which I had been compelled to adopt were greatly hampered by the narrowing of the pelvic outlet, and consequently took longer than would have been otherwise the case.

The after-history of the case did not become satisfactory until the twentieth day, as, after delivery, the temperature gradually rose until, by the tenth day, it was 103° F. It then fluctuated between 99° F. and 102° F. for ten days more, when it gradually came down to normal. The cause of the temperature was an infection of the vaginal and perineal lacerations, and this infection at no time extended to the uterus.

On February 8th the patient, who was then completely recovered, was anaesthetised, with the object of making a diagnosis of the nature of the pelvic contraction. It was then found that the lumbar spine was deviated to the left side, that the promontory was badly marked and also deviated to the same side, and that there was obvious transverse narrowing and flattening of the brim on the left side. The pelvic measurements which were made then and subsequently are as follow:

**Internal Measurements.**

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Value</th>
<th>Normal Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conjugate of brim</td>
<td>10 cm.</td>
<td>11 cm.</td>
</tr>
<tr>
<td>Transverse of brim</td>
<td>10.5 cm</td>
<td>13 cm.</td>
</tr>
</tbody>
</table>

**External Measurements.**

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Value</th>
<th>Normal Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Greatest distance between ischial tuberosities</td>
<td>8 cm.</td>
<td>11 cm.</td>
</tr>
<tr>
<td>Conjugate of outlet</td>
<td>9 cm.</td>
<td>9.5 cm.</td>
</tr>
<tr>
<td>Between anterior superior spines</td>
<td>22 cm.</td>
<td>26.5 cm.</td>
</tr>
<tr>
<td>Between iliac crests</td>
<td>24 cm.</td>
<td>28.28 cm.</td>
</tr>
<tr>
<td>Between posterior superior spines</td>
<td>5 cm.</td>
<td>9 cm.</td>
</tr>
<tr>
<td>Between trochanters of femora</td>
<td>29 cm.</td>
<td>31 cm.</td>
</tr>
</tbody>
</table>

(The figures in brackets show the normal measurements.)

In addition to these measurements, others were made with the object of determining any disproportion in size between the left
and the right side of the pelvis. These measurements are as follow:—

From left ischial tuberosity to right posterior superior spine, 17 cms. From right ischial tuberosity to left posterior superior spine, 19 cms.

From left anterior superior spine to spine of last lumbar vertebrae, 16 cms. From right anterior superior spine to spine of last lumbar vertebrae, 19 cms.

From left anterior superior spine to right posterior superior spine, 17 cms. From right anterior superior spine to left posterior superior spine, 20.5 cms.

From lower margin of symphysis to right posterior superior spine, 18.5 cms. From lower margin of symphysis to left posterior superior spine, 20 cms.

The last five pairs of measurements are those originally suggested by Naegele for the identification of oblique distortion of the pelvis, and, as will be seen from them, the existence of oblique contraction in this case is very evident.

In order to still more clearly determine the exact nature of the deformity, my colleague, Dr. Haughton, very kindly took for me a skiagram of the case, and this skiagram shows very clearly the condition of the pelvis, the deviation of the spinal column, the flattening of the pelvis on the left side, and the almost complete absence of the left lateral mass of the sacrum. I have submitted the skiagram and the measurements to Dr. Moorhead, who has lately devoted some time to the study of the anatomy of contracted pelves, and to whom I am indebted for the initial description of Naegele's pelvis. He was at first inclined to adopt the opinion that the changes present might be due to scoliosis, and, that being so, that the pelvis should be described as a sciotic pelvis. Against this view, however, must be placed the entire absence of any evidence of rickets to which the sciotic pelvis is practically always due. Subsequently, on comparing the skiagram and measurements with those of other specimens of Naegele's pelvis, he is, I think, satisfied that the case is one of unilateral synostosis.

The cause of the synostosis in this case is not obvious. As I have mentioned, the usually accepted causes are congenital failure of the centres of ossification, displacement of the sacrum to one side, or inflammatory or traumatic change in one sacro-
Skigram of an obliquely contracted pelvis, due to synostosis of the left sacro-iliac joint.

(From a skigram by Dr. W. S. Haughton.)
iliac joint. In the present case the patient states that she had never met with an injury, that she was unaware that she was in any way deformed, and that she never had any severe illness either during her childhood or in adult life. Accordingly, we are thrown for an explanation of the deformity upon either a congenital failure of the ossificatory centres or some unnoticed spinal deviation or injury during childhood. Dr. Moorhead considers that the evidence of the skiagram is against the former explanation since the curvature of the innominate bone is sufficiently well marked to suggest that the synostosis must have taken place subsequent to a period of life during which the curvatures could have been developed by the pull of the sacro-iliac ligaments. In other words, if the synostosis had been congenital the innominate bone on the deformed side would be almost flat from before backwards. We must then, I think, in the absence of further information, attribute the deformity to the second explanation, the absence of the left lateral mass being explained by pressure absorption, and the synostosis being secondary to the displacement and not the cause of it.

Before concluding, I may draw attention to a curious condition of the urine that complicated the puerperium. The urine was examined by Dr. Rowlette, on admission of the patient, and was found to contain a fairly large quantity of albumen, and many granular casts. A fortnight after delivery it was again examined by Dr. Rowlette, and, in addition to the albumen and casts, it was found to contain very large numbers of short motile bacilli resembling the colon bacillus. These were arranged in clumps resembling tube casts in form, so as to suggest that they had been thus moulded in the renal tubules. On further examination, these clumps were found to consist of pure cultures of the colon bacillus. The patient was ordered salol in 10-grain doses every six hours, and subsequently urotropin in 5-grain doses. A week later the urine was again examined, and the bacilli were found still present in large numbers. Shortly after this the patient left the hospital, but continued to take the urotropin, and on examining the urine some three weeks later, all the bacilli had disappeared, but the albumen and tube casts persisted. I regret that the nature of the vaginal infection was not ascertained by bacteriological examination, as it would be of interest to know if it too was due to the colon bacillus.
The year 1903-4 has been in many respects an eventful one to the hospital. We have lost through death two of our esteemed colleagues. Those of us who came in personal touch with Sir Philip Smyly in his capacity of Consulting Surgeon to the Hospital can best appreciate the loss we have sustained by his death.

In the untimely death of Dr. Neville, too, we have been deprived of the services of a most brilliant member of our staff.

These vacancies have been filled by the appointment of Mr. Francis Heuston, F.R.C.S.I., Senior Surgeon to the Adelaide Hospital, and of Dr. Robert Rowlette, Lecturer on Pathology, Queen's College, Galway.

I am pleased to be able to record that our Consulting Staff now includes the names of four distinguished past Masters—Drs. Lombe Atthill, Smyly, Purefoy, and Sir Arthur Macan. All these names are closely associated with the recent progress of our hospital, and to Dr. Smyly in particular we owe the great structural changes which have enabled this institution to maintain its position in the forefront of maternity hospitals.

The new pathological laboratory, generously presented to the hospital by the former Master, Dr. R. D. Purefoy, has proved an incalculable advantage to the institution.
The unsatisfactory condition of our laundry work has ceased since we have entered into our new contract—a change which has, however, involved the hospital in an additional expenditure of £100 per annum.

The deadlock which arose between Irish maternity nurses and the Central Midwives Board in respect of their ability to qualify for the English State examination has been amicably arranged. The subject was first brought prominently to notice by the President of our Obstetrical Section of the Royal Academy of Medicine in Ireland. It was taken up with great vigour by the Governors of the Rotunda Hospital, who, in upholding the interests of their own nurses, were splendidly fighting the cause of all other Irish Maternities.

To Mr. John Gordon, K.C., M.P., is due, in chief part, the satisfactory compromise arrived at; were it not for the personal influence he brought to bear upon some prominent members of the Central Midwives Board it is probable that a less satisfactory termination of the controversy might have resulted. Our thanks are also due to the Editor of the Medical Press and Circular for his able advocacy of our just cause.

A very remarkable increase in the number of patients treated in both the Intern and Extern Maternity occurred throughout the year. In the former department the increase amounted to 193 deliveries, and in the latter to 240. The admissions to the Intern Maternity reached the figure of 2,197. All these occupied beds, and had to be fed and cared for by our nursing staff; 310 were discharged after longer or shorter periods as not being in labour, whilst 1,887 were delivered in the wards of the hospital.

The months of May, July, and August show admissions well above 200 per month, and in the first of these 239 women were admitted and 203 delivered. Such numbers were never contemplated by the organisers of our present hospital system,
Clinical Report of the Rotunda Hospital.

and at times our resources were strained to the utmost. The authorities of the hospital, acting with commendable foresight, have determined on a scheme to enlarge the hospital, so as to still further ensure the safety of our patients and to enhance its usefulness.

The efficiency of a maternity institution is shown more by a low morbidity rate than by an actual mortality. I desire, therefore, at once to direct attention to our results in this respect.

Reckoning our morbidity by the method which has been employed in the hospital for years—viz., the rising of a temperature to or above 100.8 F. throughout the puerperium, we record 159, or a percentage of 8.42 on the total deliveries in the house. These figures compare favourably with the preceding twelve months, in which the morbidity works out at 8.6 per cent. For the sake of comparison with other years we have retained this plan of estimating morbidity, but as a working basis for treatment we have entirely discarded it. It is an arbitrary limit, and differs from the limit of leading Continental Maternities by being two-fifths of a Fahrenheit degree higher, and for it we have substituted the following condition as constituting morbidity—viz., a temperature running above 99° for twenty-four hours (i.e., three morning and evening takings), and accompanied by a pulse rate of over 90 per minute. This, though apparently increasing the morbidity of our hospital, has worked altogether to the advantage of our patients.

Two hundred and two morbid cases have thus been recorded; 91 of these were very slight, and required no active treatment; 26 became normal after one vaginal douching; an additional 25 after a single uterine douching, and 28, being slightly more prolonged, were given two, and sometimes three, uterine douches before the temperature fell to normal. Blood clots, retained lochia or fragments of membrane, accounted for the majority of these temperatures, whilst others arose from
The routine method now in force in dealing with morbid cases consists in a careful examination for the cause as soon as the symptoms present themselves, and if these are acute the thirty-six hour limit is not waited for. If suspicion is directed to the parturient canal a vaginal douche is administered, if not contra-indicated (a culture for microscopical examination having been first taken) a purgative is given, and the head of the bed raised on blocks to promote free drainage; if symptoms persist on the following day the vagina is again douched out, a Fergusson’s speculum inserted, and the cervix wiped dry with sterile wool. Then a sterile glass tube, suitably curved, is passed into the uterus, and its contents are aspirated into the tube by means of an affixed syringe. The tube is next rapidly sealed at either end and sent down to the Pathological Department for bacteriological examination and report of its contents. The uterus is now douched with salt and water, peroxide of hydrogen, or cylinder solutions, according to the predilection of that Assistant Master whose duty it may be to perform the operation.

If symptoms have not abated within twenty-four hours the patient is transferred, with her mattress and all her belongings, to an isolation ward, where she is taken charge of by a special nurse, and the uterus is again douched. If the bacteriological report has been productive of positive results the inside of the uterus is explored by means of a gloved forefinger in order that pieces of retained placenta, membranes, or old blood-clot may be removed if any such should be present.

This treatment differs essentially from the methods employed during the earlier months of my Mastership. Much reliance was placed by my predecessor on the use of the flushing curette, and his views found warm supporters in
the Assistant Masters who were then associated with me. The evidence they were able to adduce in favour of the curette, combined with tight packing of the uterus with iodoform gauze, appeared to be sufficient to permit a continuance of the practice until I could myself personally judge as to its efficiency. My experience of the results obtained by this treatment compelled its abandonment.

The majority of cases no doubt get well under its employment; these are for the most part sapremic, and would yield readily to any form of treatment. When pathogenic organisms are present it rarely, if ever, succeeds in their removal; on the other hand, a grave liability arises that further inoculations and other serious consequences may follow from its employment. Moreover, I have demonstrated by post-mortem examination in two cases that the curette fails at times to fulfil its primary object—namely, the removal of adherent pieces of placenta or even of membrane.

On one occasion I employed the flushing curette in an endeavour to remove a piece of sloughing placenta, the exact locality of which I had ascertained by an examining finger; it caused free haemorrhage, failed even under these circumstances to loosen the mass, and I now look upon its employment in such cases as unjustifiable.

Constitutional treatment was not neglected in any of our serious cases of morbidity; tincture of perchloride of iron in 40 minim doses was administered to all three times a day, and mercurial inunctions, or calomel in small continuous doses by the mouth, were employed in many cases, some of which ended fatally. Stimulants were administered to all, as well as nourishment in large quantities. In one fatal case anti-streptococcic serum was administered immediately on the germ being discovered in the uterus; the results were so discouraging that I feel no inclination to repeat the treatment should occasion arise within the near future.
In one very severe case of pyæmia improvement rapidly followed on the free inunction of Crede’s ointment—collargol, 17 grs. to the oz. of lard.

The treatment of these grave cases, unfortunately, did not prove uniformly successful, for out of our thirty-two serious cases where morbidity was due to septic infection eight died—three of pyæmia and five of acute septïcæmia. One contracted the disease in the month of March, three in April, two in May, one in June, whilst the last case, which occurred in October, was undoubtedly septic on admission to hospital.

A reference to Tables X. A. and I. will show that these months represent our highest percentage of morbidity and the greatest number of admissions.

Our nurses are limited by the accommodation provided for them, having been calculated on a basis of one nurse to three patients at a time when the average number of deliveries did not exceed a hundred per month. It became necessary, therefore, to add to their individual work in consequence of the increased demand on the resources of the institution. Further difficulties arose in the form of an epidemic of influenza, which placed several of our nurses on the sick-list, whilst others developed sore fingers, the result of coming in contact with septic cases.

It is yearly becoming more difficult to empty wards in rotation for the purposes of airing and thorough disinfection—a plan that has worked with such admirable results in the past. Furthermore, our isolation wards are unsuitable and altogether insufficient for the hospital requirements. Persistent and very strenuous efforts were made from the first to deal with this induced unhealthiness of our hospital, and great credit is due to our Lady Superintendent, Miss Ramsden, for the untiring energy she displayed in carrying out new and very irksome alterations in our hospital system.

These changes include the providing of rubber gloves and
finger-stalls for nurses, students, and officers of the house; the substitution of a sand-glass for the rough and ready method before employed for estimating the time spent in washing and disinfecting the hands; the introduction of a large porcelain tank in each labour ward, containing a 1 in 6,000 biniiodide of mercury solution, so that all the trays and other vessels belonging to the labour ward may be continuously left soaking in it when not in use. The complete isolation of each bed, which, in common with all that appertains to it, is now numbered with a number corresponding to one painted on the wall over its head.

The introduction of sterilised Gamgee diapers; the providing of a daily change of towels for drying the buttocks; the introduction of a specially designed bed basin, used exclusively by the patient in the bed to which it belongs; the disinfection by boiling of this basin, together with the face-basin and the chamber, as soon as the patient has been discharged and before the bed is again occupied.

The tow-wipes are no longer disinfected by sublimate solution, but are now boiled daily, and kept steeping in solution of lysol.

To mitigate the liability to sore fingers, nurses wash all morbid cases by the aid of a forceps. These, and many other changes which have now become the routine of our hospital practice, had to be evolved one by one, and needlessly to say did not tend to lessen the work of the nurses.

I feel confident that each of these innovations bore some part in restoring the hospital to its more than normal condition of healthiness, but a noticeable improvement cannot be said to have resulted until the bedchambers and bed-basins were boiled. It seems to me perfectly evident that the process relied on for disinfecting basins by scrubbing with soap and water and rinsing with corrosive antiseptic is altogether inadequate.
MATERNAL MORTALITY.

Thirteen women died in the Maternity Hospital throughout the year. This is an increase of four on the previous year, and even taking into consideration the increased number of women confined in the hospital, the percentage per 1,000 is higher.

In the month of December our first death occurred through accidental haemorrhage, and the case is so remarkable that I shall report it in full:—

K. M., 6-para, aged twenty-nine, admitted December 30th, 1903; normal pulse and temperature, in an early stage of labour, without complication. Two and a half hours after admission sudden and very severe external haemorrhage appeared, the Assistant Master being at the time in the ward. The patient was immediately prepared for examination; the os admitted one finger, and the vertex presented; no placenta to be felt. The vagina was tightly plugged and the binder applied in the usual manner, great collapse supervening before this operation could be completed. The usual well-known methods for combating collapse were resorted to, but despite these the patient's condition became distinctly worse, and her pulse could be felt with difficulty at the wrist an hour and a half later. This seemed clearly to indicate that haemorrhage, though not appearing through the plugs, was nevertheless being poured into the uterus; the plugs were therefore removed, but to our surprise we found that this was not so, the amount of free blood not being greater than could be accounted for by that which was poured out during the process of plugging.

The membranes ruptured whilst the plug was being removed, and it seemed to me advisable, under the circumstances, to perform combined bi-polar version. This was performed with the utmost ease, and occupied but a few moments. Several pints of saline infusion were injected into the cellular tissue; this, combined with stimulants and raising the foot of the couch, caused the patient to rally for a time. During the succeeding four hours no haemorrhage occurred, and she seemed to make satisfactory progress towards recovery; then a sudden change for the worse
took place, and she failed to respond to our utmost efforts at stimulation. Delivery of a dead child was completed whilst the patient was moribund.

No light was thrown on this case by the autopsy. The uterus contained no blood.

Case II. is also of great interest.

A. T., 5-para, aged twenty-eight, admitted January 21st in labour; pulse 108, temperature 99.2°. The head was freely ballotting above the brim, and the history of her last delivery, which occurred in the hospital, was that of a difficult forceps through an insufficiently dilated os.

Three hours after admission the head was still freely ballotting, and a vaginal examination demonstrated the os to be a little more than half dilated; during this examination the membranes ruptured with great force. Some hours later, the pains having decidedly weakened in character, my late Senior Assistant thought it advisable to attempt the application of forceps. Failing to deliver by these means, and having diagnosticated a low insertion of the placenta, he turned and brought down a leg, and retired to bed, believing that the patient would deliver herself.

On the following morning the patient wore an anxious appearance, and labour pains had entirely ceased. I proceeded, therefore, to deliver her—a dead child being born. On examination the vault of the vagina was found to be extensively torn, the tear involving the lower uterine segment in a lateral and transverse direction. I removed the uterus by abdominal hysterectomy an hour and a half later. The patient never properly rallied, and died on the fourth day.

**INDUCTION OF PREMATURE LABOUR.**

This operation was performed in all five times throughout the year.

**Case I.—A. M., 7-para, aged forty-one, was seen in the dispensary six weeks before admission to hospital, and then complained of loss of power in the lower extremities, with pain in the back of three weeks' duration. This loss of power had become absolute on admission. She suffered from incontinence of urine, loss of rectal control, and a bed-sore formation on the buttocks.
The knee-jerk was present in the right, absent in the left, leg; ankle clonus and the plantar reflexes were absent; Babinski's sign was well marked. Tactile sensation was present, but the sense of pain was lost over the lower extremities and over the abdomen. She suffered from cystitis with pus in the urine.

Two bougies were introduced into the uterus three days after admission. On the following morning the cervix was dilated up to seven centimetres by a Bossi's dilator; fresh bougies were then placed in the uterus, accompanied by iodoform gauze plugging of the cervix. On removal of these, in twelve hours, the vagina was tightly plugged with cotton wool. In another twelve hours labour had started, and was completed naturally, a living child being born. It breathed feebly, and died soon afterwards.

The patient regained slight power in her legs on the following day, and movements were fully established by the third day; sensation returned on the seventh day, and rectal and bladder symptoms had disappeared in a fortnight. On the nineteenth day she left hospital completely cured. A diagnosis of acute neuritis was made.

Case II. is remarkable for the complete and prolonged absence of labour pains after rupture of the membranes:—

Case II.—E. H., 3-para, aged thirty-one, was admitted on the 18th of July at full term, with the membranes ruptured. She was kept under observation for five days, and then, as no uterine action had taken place, three bougies were passed into the uterus. These were removed in twenty-four hours; the cervix was then dilated with Bossi's dilator to 5 centimetres and plugged with iodoform gauze. In another twenty-four hours feeble labour set in, the woman being finally delivered by means of forceps of a dead child with a well marked spina bifida.

Case III.—M. M., 7-para, aged thirty-two, was sent up from the country nine days before full term to have labour induced for a slightly flattened pelvis; conjugata vera 9.5 centimetres; all other measurements normal. Premature labour had previously been induced successfully in the Rotunda Hospital on several occasions.

Having regard to her closeness to full term and the comparatively slight degree of contraction, I determined to let her await
normal labour. This had not occurred four weeks after admission, and her great size, together with the accurate dates she was able to furnish, made it highly probable that we had to deal with a case of prolonged gestation.

Similar means to those detailed above were adopted to induce labour, but they failed to excite pains at the end of forty-eight hours. An ounce of sterilised glycerine was then injected into the uterus, and was followed by the rapid onset of labour pains. At the expiration of thirteen hours the os was fully dilated and the head fixed in the brim to an extent that induced my Assistant to believe that delivery with forceps could easily be accomplished. This proved an impossible task, the blades slipping from the head on two different occasions.

* The absence of foetal heart sounds contra-indicated the performance of symphysiotomy or further delay in the hope of obtaining better moulding of the head. Internal podalic version was accordingly performed, and a dead child, weighing 10 lbs., was extracted with difficulty.

* Delivery by craniotomy had been accomplished in her four first children; her fifth child lived for a few moments, and was delivered by forceps; in the sixth and seventh, labour was induced just before full term—living, large, and fully developed infants being born.

Case IV.—M. O., 16-para, aged forty, had labour induced because of extensive and severe kidney trouble of six weeks' duration. Her symptoms included severe öedema, albuminuria followed by hæmaturia. Dr. F. C. Crawley kindly examined her eyes for me, and diagnosed albuminuric retinitis and optic neuritis. All her symptoms having increased in severity during the three days in which she was under observation in the hospital, labour was easily induced by the passage of three bougies into the uterus. Delivery followed in three hours, and she was discharged on the twelfth day from hospital free from öedema and with a normal temperature, but with persistent albuminuria.

Case V.—*See* Hydramnios.

**ACCIDENTAL HæMORRHAGE.**

Amongst the thirteen cases of accidental hæmorrhage one
ended fatally, and is fully recorded under the heading of Mortality.

Nine of these cases required no treatment, as the patients were in good labour and the hæmorrhage not severe enough to warrant interference; in the others labour had not commenced. In these, moist aseptic pellets of cotton wool were inserted into the vagina, and packed tightly round the cervix after a preliminary disinfection. The plugging in each instance was then continued until the vagina became completely filled. A diaper placed between the legs and a tight abdominal binder applied from above downwards completed the operation.

It is unnecessary and mischievous to place a bullet-forceps on the cervix when performing this operation, for the plugs can be readily applied by the aid of the left hand passed into the vagina, with its palmar surface pressed against the posterior wall, so as to make a wide and simple retractor or speculum. During the performance of this the patient is preferably placed on the left side.

I feel more convinced than ever that the circulation in the uterine arteries can be directly impeded by pressure exerted on them by the vaginal tampon. The fact that even in fatal cases the uterus has not been found to contain more blood than can be accounted for by the amount poured out during the application of the plug sufficiently answers the theory that intra-uterine tension due to the out-pouring of blood into the uterine cavity suffices to prevent further flow.

PLACENTA PÆVIA.

There were only four cases of placenta praevia, with one death, due to septic infection. This woman was plugged by a practitioner before being sent into hospital; she fainted several times whilst coming a distance of nine miles in a cab, and reached us in a deplorable condition. Bi-polar podalic
version was easily performed, and she gave birth to a dead child two hours later by natural efforts.

On the fourth day her temperature rose to 101° F. The following day to 102° F.—on this evening cultures taken from the vagina and uterus contained streptococci. She died of acute sepsis on the eleventh day.

Bi-polar version was performed in all other cases, and in one a living child was born sixteen hours after performance of this operation, a proof that the life of the child is not necessarily sacrificed by its prolonged stay in the parturient canal after a foot has been brought down with the membranes ruptured.

**POST-PARTUM HÆMORRHAGE.**

In none of the twenty-nine cases recorded under the above heading was there any effort made to weigh the quantity of blood lost, and so estimate the severity of the disease. A method such as this, even were it practicable, would fail in its object because of the impossibility of ascertaining beforehand the amount of loss that can with safety be sustained in a given case.

Our diagnosis rested on the presence of a continuous flow which natural processes were insufficient to check, and which, if permitted to continue, would inevitably result in the supervention of constitutional disturbance. This seems to me the only practical means at our disposal for determining the condition.

We divide these hæmorrhages into three distinct classes—

1. Traumatic, the result of tearing of the soft parts.
2. Pseudo-atonic, due to inefficient uterine retraction, the result of mechanical obstruction or misplacements.
3. Atonic, a condition of great rarity.

All our cases were successfully treated as follows:—Extern manual massage and ergot in eight cases; in a further three a
hot vaginal douche, with bi-manual massage to the uterus. In eighteen cases the uterus was unable to retract because of mechanical causes; adherent placenta was present in twelve, and blood clots, associated with retained membranes, in six. All save one of these were promptly arrested on the manual clearing out of the uterus, followed by a hot uterine douche; the remaining case necessitated the plugging of the uterus with sterilised gauze bandages three inches wide and six yards long.

There remain to be mentioned two cases of secondary post-partum haemorrhage—one, M. W., occurred on the eighth day in conjunction with septic infection (see Case 9, Mortality Table); the other case had a normal delivery, with a torn perineum, which was united by one suture. Six hours later the patient was discovered to be bleeding, the cause of which was found, on close inspection, to be due to a spouting vessel in the perineum.

MANUAL REMOVAL OF THE PLACENTA.

Manual removal of the placenta was performed thirty-six times. Thirteen of these cases showed a more or less morbid temperature within two days. Three more developed morbidity, but not of a severe kind, after the fifth day. In six post-partum haemorrhage was the indication, while in thirty-one adherent placenta was present, and in five retention was due to spasm of the uterus.

In my opinion this latter complication should not be met with so frequently as we have seen it, nor should it call for forcible removal of the placenta save in the presence of dangerous haemorrhage. Rest, combined with hypodermics of morphine, will in time bring about relaxation of spasm, and so enable the uterus to empty itself by natural efforts.

The employment of gloves, together with the repeated introduction of the gloved hand into the uterus, until every
Clinical Report of the Rotunda Hospital.

particle of adherent tissue has been removed, will, I feel sure, greatly lessen our morbidity in the future.

ECLAMPSIA.

We had no fatality amongst our eight patients who developed eclamptic fits. Five of these were primiparae, two 2-paræ, and one 3-paræ. All were oedematous and suffered from marked albuminuria.

The fewest number of fits was two and the greatest eleven. In two the convulsions were altogether post-partum in type, and in two they were of the mixed variety, occurring both before and after delivery.

Macerated children were born in four of the cases. In one instance forceps was applied, whilst labour was terminated by natural efforts in all the other cases.

In treating this diseased condition the Dublin method was strictly followed. This consists of a rigid adherence to many points of detail. The patient is kept on her side to prevent fluids formed in the mouth from reaching the lungs by inspiration. For the same reason croton oil or other form of medicine or fluid is not permitted to be placed in the mouth of an unconscious patient. I feel convinced that the so-called oedema of the lungs, which is said to close the existence of many eclamptics, results in reality from such mistaken methods of treatment.

Neither a vapour bath, nor pilocarpin, nor other form of diaphoretic is administered, for the advantage derived from each and all seems very problematical, and is certainly more than counterbalanced by other objectionable effects.

We pass a stomach-tube through the mouth in the event of our patient being deeply unconscious, and through the nose if she be in a state of semi-consciousness. In the former condition the soft palate fails to respond to the stimulation of the nose-tube, and the latter is in consequence much more
likely to be passed into the trachea than through the oesophagus. By means of this tube the stomach is thoroughly washed out. Into the stomach is then poured, by means of the tube, a quart of slightly saline hot water, together with castor oil, elaterin, croton oil in four or five drop doses, or some saline purgative, according to the predilection of the operator.

Whilst these preparations are being perfected a hypodermic of morphine in a half-grain dose is injected; this drug is repeated every two hours in quarter grain doses until the fits cease or until a grain has been administered. It is occasionally necessary to continue the administration of the drug, but on no account should a greater dosage than two grains be administered in twenty-four hours.

We attach enormous importance to the thorough and rapid evacuation of the bowels, and the purgatives are stimulated in their action by the administration of copious enemata of soap and water with castor oil. Saline enemata are also given.

In severe cases normal saline, to the amount of two or three pints, is infused once, or oftener, into the cellular tissue beneath the mammary glands. Labour is never induced, nor is its onset to be desired. Should it unfortunately occur its progress is seldom interfered with. In the one case in which we departed from this rule the convulsions seemed to be rather aggravated than diminished by the complete delivery.

PROLAPSE OF THE FUNIS.

This complication arose in seven cases. In only two of these was the cord pulsating when the condition was discovered; both children were born alive—one by forceps and the other by expression on the fundus.

Of the remaining five cases, two occurred with anencephalic monsters, and both were accompanied by ante-partum haemorrhage—one of the accidental and the other of the unavoidable variety. The other cases are of no interest.
HYDRAMNIOS.

Of the seven cases of this condition one occurred in a dropsical woman, with albuminuria and symptoms of threatened eclampsia. An adherent placenta was associated with another. Accidental haemorrhage complicated a third. In a fourth the condition existed in an aggravated form and necessitated the rupture of the membranes at the seventh month of pregnancy. The measurement of the fluid amounted to 22 pints.

Delivery of a dead and hydrocephalic monster was accomplished by podalic version.

HYPEREMESIS GRAVIDARUM.

There was one severe case of this malady; it occurred in a 10-para, aged thirty-seven, sent to us from the country, suffering from complete inability to retain food of any kind in the stomach. She was fed for three days per rectum, and numerous saline enemata were also administered.

Retching and vomiting were entirely relieved by these means, and the patient was given and retained peptonised whey on the fourth day; on the eighth day regular feeding was adopted, with a dietary that included fish. Labour set in four days later in the eighth month of pregnancy. The foetus was macerated. Her convalescence was uninterrupted, and she left the hospital on the eighth day after her confinement.

MANIA.

Mania of a mild type developed in two cases, both primiparae; they required careful watching and isolation. Each left the hospital on the twelfth day completely cured of hallucinations and in sound health.

SYMPHYSIOTOMY.

This operation was necessitated in the case of:—

M.A., a primi para, who was admitted into hospital at the
commencement of labour with the head freely ballotting above the brim. Her pelvic measurements were normal save for that of the internal conjugate, which measured 8 centimetres. The pains were very feeble, but little apprehension was felt that she would not deliver herself naturally. At the end of twenty-four hours pains were still weak, and the head had not fixed in the brim. Morphine was given to promote sleep. At the expiration of twelve hours more the os was fully dilated. Temperature and pulse were normal, and the head still showed no signs of moulding. Walcher’s position was tried, and the membranes having ruptured the patient was left for another six hours, and then forceps were applied in a tentative manner without success, the indication for their use being rapidly recurring pains associated with a quickened pulse. The foetal heart was distinctly audible after efforts at delivery by forceps were discontinued, and nothing remained open to us under the circumstances save symphysiotomy.

This turned out to be a simple operation unaccompanied by haemorrhage or any laceration to the soft parts. The life of the child was not saved. The woman made almost an uninterrupted recovery, marred for a few days by an acute cystitis, the result of the accidental nipping of the bladder between the pubic bone for a few hours during one of the dressings. She was allowed to walk in six and a half weeks, and left the hospital eight weeks after operation.

CæSAREAN SECTION.

M. H., aged twenty-nine, 1-para, sent into hospital because of her small size and abnormal shape before labour had set in. Her pelvic measurements were Crv., 8 cm.; t, 13 cm. Labour set in on the third day after admission. The head showed no sign of fixing twelve hours after commencement of labour, and was found to markedly over-ride the symphysis pubis. It was perfectly apparent that a sufficient amount of moulding could not be brought about by natural efforts to permit the head to pass through the brim, and Cæsarean section was performed. Both child and mother left the hospital in complete health on the fifth week from delivery.

The operation of Cæsarean section must be considered
amongst the easiest and safest of abdominal sections when performed under the favourable condition which obtained in this case.

A very interesting point which was noticed in this, as well as in all my other similar operations, was that the uterine wall thickened rapidly during the process of suturing, and that this thickening occurred quite independently of uterine contractions—a proof that the processes of retraction and contraction are not dependent one upon the other, and that a uterus, though in a relaxed condition, can retract, and so prevent post-partum haemorrhage.

CRANIOTOMY.

Craniotomy was necessitated in the case of a dead child with prolapsed non-pulsating cord; the head was freely ballotting above the brim, though the woman was having strong labour pains for several hours. The extraction was accomplished without difficulty by cranioclav.

ABORTIONS AND IMMATURE DELIVERIES.

Sixty cases are recorded under the above heading. In thirty-one of these the uterus was emptied of its contents by natural methods. In six, bi-manual expression sufficed to remove the ovum, while in twelve, fingers inserted into the uterus caused complete separation of retained contents, and these were subsequently expressed bi-manually or removed by ovum forceps. In eight cases the cervix was too narrow to permit of the entrance of a finger, and a flushing curette was employed successfully to empty the cavity. In one the replacing a retroflexed uterus and the flushing out of its cavity caused the ovum to escape. In the remaining two the cervixes were dilated by Bossi's dilator to extract immature foetus a.
HYDATIDIFORM moles.

Three of these cases occurred during the year, and the first is of particular interest, as the woman subsequently developed chorio-epithelioma:—

M. W. believed herself to be ten weeks pregnant, and had suffered from hæmorrhagic discharge for four weeks before coming under observation; this had developed into severe bleeding two days before admission into hospital, July 10th. A correct diagnosis of her condition was made from the discovery of a cyst in the discharge, and the uterus was immediately emptied of its contents. Her convalescence was normal, and she left the hospital on the eighth day. On October the 18th, she returned complaining of a sanious discharge, which she stated had continued almost since she had left the hospital. On examination the uterus was found to be retroflexed and enlarged with a patulous os. On the following day the uterus was curetted, and a small cystic mass, together with large quantities of old blood-clot, were removed. Rather severe hæmorrhage followed this operation, and necessitated plugging with iodoform gauze. Hæmorrhage then ceased for twelve days, only to again return, and on this occasion another small cyst was removed. The specimen taken from the first curettage was undiagnosable by frozen section, but its more complete preparation demonstrated a marked degree of malignancy. On the 8th November the uterus was extirpated by the vaginal route; its cavity was perfectly healthy save at the fundal extremity, where a small malignant excavation, about \( \frac{3}{8} \) inch wide and a \( \frac{1}{2} \) inch deep, was found. This had almost perforated the uterine wall, its only covering being peritoneum.

Of the other two cases one was delivered naturally, and the other calls for no comment.

HÉMATOMA OF THE VULVA.

Of the three cases in which this complication was met with only one necessitated the opening of the large tumour and the complete turning out of blood clots. The cavity was closed, and hæmorrhagic oozing controlled by obliterating it by cat-gut ligatures in several layers; a certain amount of
suppuration ensued, accompanied by a morbid temperature, which did not at any time exceed 100° F.

PODALIC VERSION.

The ten cases of podalic version met with called for no particular comment.

INFANTILE CONDITIONS.

Amongst the infantile conditions four cases of imperforate anus were met with; in all these the obstruction was relieved by operating through the perineal route. One infant died of secondary haemorrhage, two more sank a few hours after the operation, and one survived, leaving the hospital in perfect health.

In conclusion I desire to convey my sincere thanks to all the officers of the Institution, and particularly to my Assistant Masters, whose loyal support I have at all times been able to count upon.

Table No. I.—Admissions to Maternity Department.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Total deliveries</td>
<td>124</td>
<td>141</td>
<td>148</td>
<td>122</td>
<td>149</td>
<td>149</td>
<td>197</td>
<td>161</td>
<td>176</td>
<td>168</td>
<td>143</td>
<td>150</td>
<td>1,161</td>
</tr>
<tr>
<td>Total abortions</td>
<td>5</td>
<td>3</td>
<td>6</td>
<td>8</td>
<td>3</td>
<td>10</td>
<td>6</td>
<td>4</td>
<td>4</td>
<td>5</td>
<td>4</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Not in labour</td>
<td>21</td>
<td>31</td>
<td>20</td>
<td>28</td>
<td>24</td>
<td>28</td>
<td>36</td>
<td>17</td>
<td>36</td>
<td>20</td>
<td>29</td>
<td>21</td>
<td>19</td>
</tr>
<tr>
<td>Total</td>
<td>150</td>
<td>175</td>
<td>174</td>
<td>158</td>
<td>176</td>
<td>187</td>
<td>239</td>
<td>179</td>
<td>216</td>
<td>202</td>
<td>168</td>
<td>173</td>
<td>2,141</td>
</tr>
</tbody>
</table>

Table No. II.—Dispensary for Out-Patients.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of first attendances</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>5,229</td>
</tr>
<tr>
<td>&quot; repeated</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>6,885</td>
</tr>
<tr>
<td>Special Dispensaries</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1,760</td>
</tr>
<tr>
<td>Total</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>13,814</td>
</tr>
</tbody>
</table>
### Table No. III.—Showing Nature and Number of Cases Treated in the Extern Maternity, 1903-1904.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total deliveries</td>
<td>2,285</td>
</tr>
<tr>
<td>Abortions</td>
<td>268</td>
</tr>
<tr>
<td>Haemorrhage</td>
<td>5</td>
</tr>
<tr>
<td>Presentations—</td>
<td></td>
</tr>
<tr>
<td>Face</td>
<td>4</td>
</tr>
<tr>
<td>Face to Pubes</td>
<td>14</td>
</tr>
<tr>
<td>Brow</td>
<td>1</td>
</tr>
<tr>
<td>Breech and Footling</td>
<td>59</td>
</tr>
<tr>
<td>Transverse and Oblique</td>
<td>9</td>
</tr>
<tr>
<td>Compound</td>
<td>2</td>
</tr>
<tr>
<td>Twins</td>
<td>21</td>
</tr>
<tr>
<td>Prolapse of Funis</td>
<td>6</td>
</tr>
<tr>
<td>Infantile conditions—</td>
<td>1</td>
</tr>
<tr>
<td>Meningocele</td>
<td></td>
</tr>
<tr>
<td>Mortality, maternal</td>
<td>6</td>
</tr>
<tr>
<td>Haemorrhage—</td>
<td></td>
</tr>
<tr>
<td>Accidental</td>
<td>7</td>
</tr>
<tr>
<td>Unavoidable</td>
<td>7</td>
</tr>
<tr>
<td>Post-partum</td>
<td>10</td>
</tr>
<tr>
<td>Operations—</td>
<td></td>
</tr>
<tr>
<td>Version</td>
<td>14</td>
</tr>
<tr>
<td>Forceps</td>
<td>39</td>
</tr>
<tr>
<td>Decapitation</td>
<td>1</td>
</tr>
<tr>
<td>Manual removal of Placentina</td>
<td>24</td>
</tr>
<tr>
<td>Eclampsia</td>
<td>2</td>
</tr>
</tbody>
</table>

### Table No. IV.—Showing Nature and Number of Cases Treated in Intern Maternity.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total admissions</td>
<td>2,197</td>
</tr>
<tr>
<td>Total deliveries</td>
<td>1,887</td>
</tr>
<tr>
<td>Primipare</td>
<td>664</td>
</tr>
<tr>
<td>Abortions</td>
<td>60</td>
</tr>
<tr>
<td>Hydramnios</td>
<td>1</td>
</tr>
<tr>
<td>Hydromnios</td>
<td>7</td>
</tr>
<tr>
<td>Myxoma Chorii</td>
<td>2</td>
</tr>
<tr>
<td>Presentations—</td>
<td></td>
</tr>
<tr>
<td>Face</td>
<td>2</td>
</tr>
<tr>
<td>Face to Pubes</td>
<td>19</td>
</tr>
<tr>
<td>Brow</td>
<td>2</td>
</tr>
<tr>
<td>Breech and Footling</td>
<td>56</td>
</tr>
<tr>
<td>Transverse and Oblique</td>
<td>5</td>
</tr>
<tr>
<td>Compound</td>
<td>2</td>
</tr>
<tr>
<td>Twins</td>
<td>16</td>
</tr>
<tr>
<td>Prolapse of Funis</td>
<td>6</td>
</tr>
<tr>
<td>Eclampsia</td>
<td>8</td>
</tr>
<tr>
<td>Mania</td>
<td>2</td>
</tr>
<tr>
<td>Emphysema (cutaneous)</td>
<td>1</td>
</tr>
<tr>
<td>Puerperal ulcer</td>
<td>7</td>
</tr>
<tr>
<td>Morbidity</td>
<td>292</td>
</tr>
<tr>
<td>Mastitis</td>
<td>3</td>
</tr>
<tr>
<td>Mortality, maternal</td>
<td>13</td>
</tr>
<tr>
<td>Infantile</td>
<td></td>
</tr>
<tr>
<td>Died in Hospital</td>
<td>40</td>
</tr>
<tr>
<td>Premature</td>
<td>20</td>
</tr>
<tr>
<td>Recent</td>
<td>32</td>
</tr>
<tr>
<td>Macerated</td>
<td>48</td>
</tr>
<tr>
<td>Puertril</td>
<td>1</td>
</tr>
<tr>
<td>Infantile conditions—</td>
<td></td>
</tr>
<tr>
<td>Anencephalus</td>
<td>3</td>
</tr>
<tr>
<td>Hydrocephalus</td>
<td>5</td>
</tr>
<tr>
<td>Spina bifida</td>
<td>6</td>
</tr>
<tr>
<td>Meningocele</td>
<td>2</td>
</tr>
<tr>
<td>Cephalhematoma</td>
<td>1</td>
</tr>
<tr>
<td>Cleft palate</td>
<td>2</td>
</tr>
<tr>
<td>Ophthalmia</td>
<td>2</td>
</tr>
<tr>
<td>Talipes</td>
<td>4</td>
</tr>
<tr>
<td>Deformed hands</td>
<td>3</td>
</tr>
<tr>
<td>Hydrocele and hypospadias</td>
<td>1</td>
</tr>
<tr>
<td>Rectropism vesicula</td>
<td>1</td>
</tr>
<tr>
<td>Imperforate anus</td>
<td>4</td>
</tr>
<tr>
<td>Mastitis</td>
<td>1</td>
</tr>
<tr>
<td>Convulsions</td>
<td>2</td>
</tr>
<tr>
<td>Interns</td>
<td>9</td>
</tr>
<tr>
<td>Monsters</td>
<td>1</td>
</tr>
</tbody>
</table>
### Table No. V.—Maternal Mortality.

<table>
<thead>
<tr>
<th>Name</th>
<th>Admitted</th>
<th>Delivered</th>
<th>Died</th>
<th>Cause of death</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>K. M.</td>
<td>Dec. 30</td>
<td>Dec. 30</td>
<td>Dec. 30</td>
<td>Accidental</td>
<td>Plugging and Version</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Haemorrhage</td>
<td>Version, Hysterectomy</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Rupture of the</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Vagina</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Phthisis</td>
<td></td>
</tr>
<tr>
<td>A. I.</td>
<td>Jan. 21</td>
<td>Jan. 22</td>
<td>Jan. 27</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M. E.</td>
<td>Feb. 17</td>
<td>Feb. 18</td>
<td>March 8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>L. M.</td>
<td>March 6</td>
<td>March 7</td>
<td>April 1</td>
<td>Septicæmia</td>
<td></td>
</tr>
<tr>
<td>M. D.</td>
<td>April 13</td>
<td>April 13</td>
<td>&quot;</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Pneumonia</td>
<td></td>
</tr>
<tr>
<td>E. P.</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
<td>Cardiac failure</td>
<td></td>
</tr>
<tr>
<td>E. M'E.</td>
<td>&quot;</td>
<td>&quot;</td>
<td>June 9</td>
<td>Pyæmia</td>
<td></td>
</tr>
<tr>
<td>A. S.</td>
<td>&quot;</td>
<td>&quot;</td>
<td>May 5</td>
<td>Septicæmia</td>
<td></td>
</tr>
<tr>
<td>M. W.</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A. M.</td>
<td>March 16</td>
<td>March 17</td>
<td>&quot;</td>
<td>Pyæmia</td>
<td></td>
</tr>
<tr>
<td>M. M'D.</td>
<td>May 15</td>
<td>May 15</td>
<td>&quot;</td>
<td>Septicæmia</td>
<td></td>
</tr>
<tr>
<td>S. B.</td>
<td>June 23</td>
<td>June 23</td>
<td>July 27</td>
<td>Pyæmia</td>
<td></td>
</tr>
</tbody>
</table>

**Table No. VI.—Application of Forceps.**

<table>
<thead>
<tr>
<th>Indication</th>
<th>No.</th>
<th>Dead Children</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Delay in 2nd stage, with danger to mother or child</td>
<td>50</td>
<td>2</td>
<td>In one case the forceps slipped and had to be abandoned. Delivery completed by version. Child was dead.</td>
</tr>
<tr>
<td>Prolapse of cord</td>
<td>1</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Contracted pelvis</td>
<td>3</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Occipito-posterior</td>
<td>7</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Eclampsia</td>
<td>1</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Cardiac and renal disease</td>
<td>2</td>
<td>1</td>
<td>Applied before the first stage had ended.</td>
</tr>
<tr>
<td>Early rupture of membrane</td>
<td>1</td>
<td>-</td>
<td>Applied on account of child before the first stage was completed.</td>
</tr>
<tr>
<td>Total,</td>
<td>65</td>
<td>5</td>
<td></td>
</tr>
</tbody>
</table>
Sub-table A.—Showing No. of Pregnancy.

| I-para | - | - | 52 |
| II-para | - | - | 2 |
| III-para | - | - | 3 |
| IV-para | - | - | 2 |
| V-para and over | - | - | 6 |
| **Total** | 65 |

Sub-table B.—Ages of Patients.

<table>
<thead>
<tr>
<th>Age Range</th>
<th>-</th>
<th>28</th>
</tr>
</thead>
<tbody>
<tr>
<td>17-23</td>
<td>-</td>
<td>28</td>
</tr>
<tr>
<td>26-30</td>
<td>-</td>
<td>23</td>
</tr>
<tr>
<td>31-35</td>
<td>-</td>
<td>8</td>
</tr>
<tr>
<td>36 and over</td>
<td>-</td>
<td>6</td>
</tr>
</tbody>
</table>

Table No. VII.—Placenta Previa.

<table>
<thead>
<tr>
<th>Name</th>
<th>Age and Para</th>
<th>Variety</th>
<th>Period</th>
<th>Result to Child</th>
<th>Presentation</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. I. B.</td>
<td>32, VII.</td>
<td>Marginal</td>
<td>Term</td>
<td>A.</td>
<td>Breech</td>
<td>Bleeding slightly for three days; foot brought down; delivered in 16 hours.</td>
</tr>
<tr>
<td>2. A. C.</td>
<td>36, V.</td>
<td>&quot;</td>
<td>8 months</td>
<td>D.</td>
<td>Head</td>
<td>Sent in bleeding; cord prolapsed; pulseless; version; delivery in 1 hour.</td>
</tr>
<tr>
<td>3. B. K.</td>
<td>36, VIII.</td>
<td>Central</td>
<td>Term</td>
<td>D.</td>
<td>&quot;</td>
<td>Version; delivery in 6 hours.</td>
</tr>
</tbody>
</table>
### Table No. VIII.—Accidental Haemorrhage.

<table>
<thead>
<tr>
<th>Name</th>
<th>Age and Para</th>
<th>Date</th>
<th>Variety</th>
<th>Treatment</th>
<th>Result to Child</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. A. C.</td>
<td>37, VIII.</td>
<td>Nov. 8 1903</td>
<td>External</td>
<td>None</td>
<td>Alive</td>
<td>In good labour.</td>
</tr>
<tr>
<td>2. M. F.</td>
<td>42, VIII.</td>
<td>21</td>
<td></td>
<td></td>
<td></td>
<td>In labour; membranes had ruptured.</td>
</tr>
<tr>
<td>4. B. G.</td>
<td>35, XIV.</td>
<td>Mar. 22 1904</td>
<td></td>
<td>None</td>
<td></td>
<td>In labour.</td>
</tr>
<tr>
<td>7. M. C.</td>
<td>29, VIII.</td>
<td>5</td>
<td>External</td>
<td></td>
<td></td>
<td>Was in labour.</td>
</tr>
<tr>
<td>10. R. C.</td>
<td>36, VII.</td>
<td>14</td>
<td></td>
<td>Version</td>
<td></td>
<td>Oblique presentation. Delivery in 2 hours.</td>
</tr>
<tr>
<td>11. S. D.</td>
<td>34 XIV.</td>
<td>31</td>
<td></td>
<td>None</td>
<td>Alive</td>
<td>Premature. In labour.</td>
</tr>
<tr>
<td>13. R. L.</td>
<td>27, IX.</td>
<td>3</td>
<td>External</td>
<td>Plugged</td>
<td>Dead</td>
<td>Delivery in 20 hours.</td>
</tr>
</tbody>
</table>
**Table No. IX.—Cases of Contracted Pelvis.**

<table>
<thead>
<tr>
<th>Name</th>
<th>Age</th>
<th>Para</th>
<th>Pelvic Measurements</th>
<th>Mode of Delivery</th>
<th>Result to Child</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>C. V.</td>
<td>Trans</td>
<td>Ext. C.</td>
<td>I-C.</td>
</tr>
<tr>
<td>M. G.</td>
<td>20</td>
<td>I.</td>
<td>8</td>
<td>13</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>M. H.</td>
<td>23</td>
<td>I.</td>
<td>8</td>
<td>13</td>
<td>15</td>
<td>28</td>
</tr>
<tr>
<td>M. M.</td>
<td>32</td>
<td>VII.</td>
<td>9½</td>
<td>13</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>
Clinical Report of the Rotunda Hospital.

Table No. X. A.—Morbidity.

<table>
<thead>
<tr>
<th>Temperature</th>
<th>Nov</th>
<th>Dec</th>
<th>Jan</th>
<th>Feb</th>
<th>March</th>
<th>April</th>
<th>May</th>
<th>June</th>
<th>July</th>
<th>Aug</th>
<th>Sept</th>
<th>Oct</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>100.8° to 101.2°</td>
<td>3</td>
<td>4</td>
<td>3</td>
<td>5</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>37</td>
</tr>
<tr>
<td>101.2° to 102.2°</td>
<td>-</td>
<td>1</td>
<td>4</td>
<td>8</td>
<td>4</td>
<td>4</td>
<td>3</td>
<td>5</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td></td>
<td>38</td>
</tr>
<tr>
<td>102.2° to 104°</td>
<td>1</td>
<td>1</td>
<td>4</td>
<td>2</td>
<td>10</td>
<td>7</td>
<td>6</td>
<td>5</td>
<td>4</td>
<td>5</td>
<td>4</td>
<td></td>
<td>58</td>
</tr>
<tr>
<td>104° and above</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>7</td>
<td>4</td>
<td>3</td>
<td>5</td>
<td>2</td>
<td>4</td>
<td>1</td>
<td>2</td>
<td></td>
<td>26</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>4</td>
<td>6</td>
<td>11</td>
<td>13</td>
<td>26</td>
<td>19</td>
<td>18</td>
<td>19</td>
<td>13</td>
<td>14</td>
<td>8</td>
<td>8</td>
<td>159</td>
</tr>
</tbody>
</table>

Percentage: 8.42%

Table No. X. B.

<table>
<thead>
<tr>
<th>Temperature.</th>
<th>Nov</th>
<th>Dec</th>
<th>Jan</th>
<th>Feb</th>
<th>Mar</th>
<th>Apr</th>
<th>May</th>
<th>June</th>
<th>July</th>
<th>Aug</th>
<th>Sep</th>
<th>Oct</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>90°, P. 90</td>
<td>9</td>
<td>11</td>
<td>13</td>
<td>14</td>
<td>28</td>
<td>19</td>
<td>19</td>
<td>18</td>
<td>15</td>
<td>10</td>
<td>202</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Percentage: 10.70%

Table No. X.—C.

Causes of Morbidity other than Uterine.

<table>
<thead>
<tr>
<th>Condition</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Breast abscesses</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>&quot; inflamed</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>8</td>
</tr>
<tr>
<td>Cardiac disease</td>
<td>...</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Bronchitis</td>
<td>...</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>Constipation</td>
<td>...</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>12</td>
</tr>
<tr>
<td>Eclampsia</td>
<td>...</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5</td>
</tr>
<tr>
<td>Influenza</td>
<td>...</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>6</td>
</tr>
<tr>
<td>Mania</td>
<td>...</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Phthisis</td>
<td>...</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>...</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Pleurisy</td>
<td>...</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Renal disease</td>
<td>...</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>6</td>
</tr>
<tr>
<td>Puerperal ulcer</td>
<td>...</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>7</td>
</tr>
<tr>
<td>Lacerated vagina</td>
<td>...</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>...</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>58</td>
</tr>
</tbody>
</table>
By Dr. E. Hastings Tweedy, Master.

Table No. X.—D.

<table>
<thead>
<tr>
<th>Day of occurrences of Temp.</th>
<th>Day of occurrences of Temp.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st ...</td>
<td>6th ...</td>
</tr>
<tr>
<td>2nd ...</td>
<td>7th ...</td>
</tr>
<tr>
<td>3rd ...</td>
<td>8th ...</td>
</tr>
<tr>
<td>4th ...</td>
<td>9th ...</td>
</tr>
<tr>
<td>5th ...</td>
<td>10th, &amp;c.</td>
</tr>
<tr>
<td>26</td>
<td>18</td>
</tr>
<tr>
<td>36</td>
<td>11</td>
</tr>
<tr>
<td>36</td>
<td>14</td>
</tr>
<tr>
<td>31</td>
<td>5</td>
</tr>
<tr>
<td>26</td>
<td>2</td>
</tr>
</tbody>
</table>

Dr. Jellett said he had to congratulate Dr. Tweedy on his first report. He saw that a certain proportion of deaths had occurred in excess of what would probably occur in future years, but most of them were due to causes which were outside the control of the Master. He would specially like to congratulate him on the steps which he had taken to prevent the recurrence of sepsis, as he had introduced many important reforms, such as rubber gloves, finger stalls, separate utensils for each patient, &c. Going on to criticise the report seriatim, he referred to the fatal case of accidental haemorrhage, and asked Dr. Tweedy how it was that because the membranes ruptured while the plug was being removed he did bipolar version? Had the adoption of this treatment any relation to the theory of compression of the uterine artery which Dr. Tweedy brought forward to explain the action of the vaginal tampon in accidental haemorrhage? Personally he did not think that the circulation in the uterine artery could be impeded by a vaginal tampon, nor did he think the application of a bullet forceps and pulling down the cervix could exert any pressure on the uterine artery. He considered that Dr. Tweedy’s theories that one could bring down the angle of the uterine artery within reach of a plug was fantastic, since when a vaginal tampon was put in, and a binder applied outside, the uterus was compressed between the two, and did not change its position. Did Dr. Tweedy still hold to his theory? He criticised the arrangement of the Rotunda Reports, saying that he had had occasion to read up many of them to compile statistics, &c., and found it very annoying when there was always a different arrangement or no arrangement at all. It was very difficult to collect information from a whole series of them, as the subjects did not follow one another
in a systematic order, and he thought the Reports would gain enormously in value if Dr. Tweedy would classify his subjects in a systematic order and record them in the same way through his whole Mastership.

Sir A. V. Macan congratulated Dr. Tweedy on his report. He noted the enormous increase there was in the number of cases, and this was very striking. An interesting point also was the efforts made to combat sepsis. Personally he would think the important thing would be first to make a thorough investigation as to where the fault lay. He agreed with Dr. Jellett as to the value of the improvements. He did not consider rubber gloves of such great importance, and could not regard the stoppage of sepsis as being due to those measures. Rubber gloves were the fashion, but he himself did not consider them at all essential. As to the use of antiseptic dressings, he did not think their outward application to the vulva could be very important in parturition; if it was, the matter had been very insufficiently provided for by nature. He thought there should be free drainage for the lochia. As to the maternity mortality, he still regarded the first case as having died from accidental haemorrhage, and if plugging took so long to apply that a woman could collapse we should at once go back to Caesarean section. It failed in its object if it could not be applied quickly, and you could leave it, and say it had stopped the haemorrhage. He quite agreed with Dr. Tweedy about the abandonment of curettage, and thought it was quite time to give it up. He, like Dr. Tweedy, had been disappointed in the antistreptococcic serum, and to be of any use it would have to be a mixed one. Crédé's ointment might turn out to be of use. With regard to the treatment of eclampsia, the simpler the treatment was the better, and if we agreed with regard to the injection of large doses of morphine, why wash out the stomach also? He said he had seen a case in which hebotomy was performed, and it seemed to him immeasurably superior to symphysiotomy. The only objection seemed to be that a haematoma might form.

Dr. R. D. Purefoy said he would like to congratulate Dr. Tweedy on his clearness in enunciating his opinions and his courage in defending them. With regard to some of the new measures, he might say that during his Mastership separate
utensils were in use, and sterilised dressings for the vulva had been prepared and were partly in use. He was still a believer in the use of the flushing curette in suitable cases. He said he was at a loss to know what was the contra-indication to which Dr. Tweedie referred. The subsequent treatment of these cases was douching the uterus and constitutional treatment. Dr. Tweedie stated, page 260, that "the majority of cases, no doubt, get well . . . yield readily to any form of treatment." He thought this a very sweeping statement, and was not prepared to admit that most of the cases were merely sapraemic. During his Master-ship one of his assistants, Dr. Lloyd, prepared a series of cases of rise of temperature in which bacteriological and microscopic examinations were made of the contents of the uterus, taken with every necessary precaution, and in most of the cases the examination was made by Dr. Earl. Out of 130 cases 73 had streptococci. He believed that the curette, when used in time, removed most of the organisms, and the patient was then able to resist the small dose which reached her circulation; therefore he was not prepared to give up the flushing curette. He would also like to know what were the dangers which Dr. Tweedie believed followed from its use. The report also said:—"If the bacteriological report has been productive of positive results, the inside of the uterus is explored by means of a gloved fore-finger in order that pieces of retained placenta, membranes, or old blood clot may be removed if any such should be present." That was a very mild proceeding judging by the description of it. With the flushing curette the amount of pain was very slight and shock to the patient absolutely absent. In the proceeding advocated the patient was anaesthetised, the hand was passed into the vagina, there was the shock to the patient, the opening up of the innumerable vents after parturition, the vagina was distended and air freely admitted, a number of avenues for infection made or opened up, and then the finger was passed into the uterus. Besides, he did not believe that the finger would remove the debris in the same short space of time as the curette. The Master urged against the curette that in the two cases in which it was used it failed. That was possible; it sometimes might fail, but the risk in skilled hands was infinitesimal. He thought that the risks of the curette were
ininitely magnified by the new method. He was glad to find the Master had not lost faith in constitutional treatment; but why did he recommend mercurial inunction? As to the use of serum, he also had come to the conclusion that it was perfectly useless. He thought the case of accidental haemorrhage was very interesting. It demonstrated the use of the plug and binder, but also the utter unsuitability of version in these cases. He was also glad to see that the Master had continued the use of Bossi’s dilator in helping delivery and in inducing labour. As to the use of the bullet forceps in plugging the vagina, he had never heard of its being done, and he would not do it. In his time, after manual removal of the placenta, there was seldom a rise of temperature. As to rubber gloves, he did not use them himself. He differed strongly from Dr. Tweedy’s plan of a “repeated introduction of the gloved hand into the uterus.” Surely one ought to do it with as few introductions as possible and without removing the hand if possible. With regard to the treatment of eclampsia, he thought the practice of passing fluid into the stomach a very valuable one, but he considered the statement—p. 270—“Neither a vapour bath . . . objectionable effects” was a very sweeping one. What were the objectionable effects of a vapour bath? He knew of no drawbacks to it. He had always condemned the use of pilocarpin. Again, Dr. Tweedy differed from most authorities as to the wisdom or otherwise of hastening labour in these cases. He himself did not think that labour should be induced if the convulsions had come on, but otherwise he thought that labour should be assisted. He noticed in the Report that practically there was very little information about the extern maternity.

Dr. Smyly congratulated Dr. Tweedy on his most interesting and excellent report. He had made many changes, and he thought they were all improvements. Rubber gloves were of great use, and especially in handling anything that was septic. The using of sponge holders by the nurses when washing the vulva was also of importance. The statement that there were only three cases of mastitis out of over 1,800 deliveries was different from his own experience in his own private practice, and he thought that it was caused in nearly all cases by the nurses, who frequently contaminated their hands when washing the
patient, and then infected the nipple, and mastitis was brought about.

Dr. Horne, referring to the question of mastitis, said that these cases occurred usually in the second or third week, and after the patient had left hospital, so that probably many returned to the extern department. He congratulated Dr. Tweedy on his report. It was interesting to see that notwithstanding all the advances that had been made a certain proportion of cases did suffer from various forms of puerperal trouble.

The President was glad to see that rubber gloves were used, and thought them a great step in advance. On the question of reckoning morbidity, he thought there should be a uniform standard on the Continent and here, and he considered the German method a good one, which should be adopted. He congratulated Dr. Tweedy on his very excellent report.

Dr. E. H. Tweedy, in replying, said he first had to thank the members for the way in which they had received his report. Replying to Dr. Jellett, he said the question of the arrangement of the report did not occur to him, and he would be very glad to follow out any suggestions that Dr. Jellett had to offer as to the future arrangement. Dr. Jellett differed from him on the question of plugging the uterus for accidental haemorrhage, and, as to the first case, Sir Arthur Macan had objected to his having taken out the plug. This treatment was comparatively new, and he lacked the courage of his convictions. He was now more firmly convinced than ever as to its efficacy, and believed that the plug stops the haemorrhage immediately. He thought the case exceptional, and had taken out the plug to see if bleeding still continued, and whilst doing so the membranes ruptured. It was then perfectly easy to turn, and he did it instinctively. He still did not know of what the patient had died. Sir Arthur Macan had said that there was no necessity for completely isolating the patients, but he must remember that the beds, mattresses, &c., could not be rendered surgically clean. In a series of four cases of infection which had occurred it was traced to the pan used for catching the antiseptic lotion with which the patients were washed, and it was the only utensil in common use among them. The fact remained that the moment they began to boil the bed chambers, &c., and isolate the patients, the septic condition
ceased. The aseptic dressings to the vulva might not be necessary, but were certainly expedient. In dealing with morbidity, he said that no morbidity was ever based on a first twenty-four hours' rise of temperature, and nothing else. A temperature of 100.4° was the basis of the morbidity standard of many of the Continental clinics, but with us 100.8° had marked the limit until lately. A serious condition of septic disease could not exist with a pulse of 90 or below it. He would certainly like to see a common standard adopted. He attached importance to the temperature and pulse taken in conjunction, as the one was a check on the accuracy of the other. He considered that in giving morphine in eclampsia, 2 grains in the 24 hours was plenty. He agreed with Dr. Purefoy and Dr. Jellett that all the so-called improvements were in process of introduction when he went into the Rotunda—it was simply a matter of evolution—but he took exception to the statement that separate utensils were in use, as they were changed from one bed to another and from one patient to another. As to sepsis, Dr. Lloyd had counted 130 cases of poisoning of the uterus, with 73 cases of streptococcus poisoning. He thought that that was very high. Dr. Williams, of America, once held that streptococci were very common in the vagina, but he had since said he had pushed the germs from the vulva, and this was what probably happened to Dr. Lloyd, and he did not think that anyone would admit the accuracy of Dr. Lloyd's 130 cases. As to the description that Dr. Purefoy had given of his efforts to remove a piece of placenta in the case alluded to, the uterus had been previously curetted by an assistant-master trained in curettng by Dr. Purefoy. Dr. Lloyd's statistics and Dr. Purefoy's belief in the curette were at variance with the very latest statistics. The serious dangers from curettng were thrombosis and rupture of the uterus. One might curette away the softened muscular tissue as he had seen done. The case, also, that Dr. Purefoy referred to was not an ordinary case—she was in a very bad condition, and had been previously curetted. As to passing the hand frequently into the uterus, he did not believe that anyone could say that the uterus was empty so long as there was anything in his hand. Sensation was abolished if he had anything in his fingers, and it was infinitely more important to take away everything than to exclude air
germs. He had never seen bad results from putting the hand in frequently. As to the use of vapour baths, everyone knew they were most depressing, and eclamptic women died from heart failure. Dr. Smyly had fallen into an error in stating that only one case of mastitis was recorded in this report. He again thanked the members very much for their reception of his report.
ABSTRACTS.

SECTION OF OBSTETRICS.

Friday, November 18, 1904.

The President in the Chair.

Dr. A. J. Horne showed a case of elephantiasis of leg and vulva.

Dr. A. J. Horne opened a discussion on

*The Influence of Fibro-myomata on Pregnancy and Parturition.*

Dr. W. J. Smyly said that the general impression was that women with fibroids were less likely to conceive than others, but these women were generally sterile long before. The cause of sterility appeared to be the condition of the mucous membrane. Another point was that these women went on bearing children to a later period in life than others, and it was attributed to ovulation and menstruation going on to a later period. His own experience did not either support or contradict these opinions. He thought that these tumours did not often cause obstruction during labour, even when growing low down in the pelvis, as they were usually drawn up out of the way. He related a case in which there was a subperitoneal myoma pushing the peritoneum up and the vaginal mucous membrane down. He had to do Caesarean section and remove the uterus and tumour. In another case there was an interstitial myoma in the lower uterine segment. The patient went into labour for a few hours, and then labour pains apparently ceased; the pains came on again in a few hours, and the membranes ruptured. She was in labour on and off for a week. The os then allowed two fingers in, and a foot was pulled down, and after a long time the fetus came away. He thought the chief danger of myomata was during the puerperium. In one case a hospital patient died from post-partum haemorrhage, and, post mortem, a submucous myoma was found the size of an orange. He had also found that portions of the membranes were retained sometimes in these cases, which decomposed and caused sepsis. Retained placenta was also common. He did not agree with Bland Sutton that all myomata should be removed, though the risk of operation was not great.
Dr. Purefoy thought the influence of fibroids in causing sterility was over-rated. An interesting aspect of the question was the greatly increased difficulty in diagnosticating pregnancy in the first three months. If a myoma invaded the cervical region it was most difficult, as he thought the cervix did not undergo the characteristic amount of softening of pregnancy. He related a case in which a fibroid occupied the pelvis, and he did Cæsarean section. A fibroid in the uterine wall also enfeebled the uterine contractions, and they often caused marked interference with the course of labour. He related another case of a primipara, aged thirty, with a fibroid in the lower uterine segment. She went five weeks beyond full term. The presenting part could not be reached, and there was a complete absence of labour. The uterus was removed along with the child, and the patient made a good recovery. Displacement should be done in these cases if possible, and he thought myomectomy was advisable only in the early months of pregnancy. He tabulated these cases in the Rotunda, and notified an absence of post-partum haemorrhage. Most of the patients were women aged between thirty and forty years. He suggested that the occurrence of pregnancy rather hastened the development and increased the size of the tumours.

Dr. E. H. Tweedy related a case in which he had removed the uterus at the fifth month, as the patient could not have gone on to full term. As to fibroids causing sterility, he said it was not the fibroids, but the endometritis, that caused it in a certain number of cases. If there was a subperitoneal myoma it would not cause endometritis, and there would be no sterility. Another point was that they might directly cause twi\textipa{stting}, or pressing, or stretch-\textipa{ing} of the tubes, which might cause sterility.

Dr. Kidd agreed as to the difficulty of diagnosticating pregnancy in the early months, when associated with myomata. As to sterility, he thought the question was one of comparative and not absolute sterility. He related a case of a primipara, aged forty-four, with two large tumours at the fundus. They caused transverse presentation; version was done. The placenta had to be removed manually. The patient made a good recovery. He had examined the patient since, and found that the tumours had entirely disappeared. When pregnancy was complicated by cancer, the cancer usually
increased rapidly, owing to the hyperæmia. He thought the same increase usually occurred in the case of other tumours under similar circumstances. He related another case in which the tumour sprang from the posterior wall of the supravaginal portion of the cervix. Panhysterectomy was performed, and the patient made a good recovery. In another case in which a tumour was present which could not be lifted out of the pelvis Cæsarean section was performed. Two years later the patient again came into hospital, after she had been in labour for 73 hours. On this occasion it was possible to deliver her with forceps. In another case labour was induced, but soon ceased. A tumour the size of a hen’s egg was then enucleated from the cervix; after ten days labour came on again, and the patient delivered herself.

Dr. Jellett said that another aspect of the question was the effect of myomata on the life of the ovum during the last months of pregnancy. Quite recently he had had a case in which death of the foetus in utero apparently resulted from a myoma of the fundus. He saw a patient in February last who was expecting to be delivered about May 1st. She went all through May, and then a myoma the size of a cocoa-nut was found on the anterior wall of the fundus. A dead and macerated child was born on June 3rd, and the placenta was little more than half the normal size. He was interested to know if such a case would be attributed to the myoma, or whether it was a mere accident of pregnancy. If the former, there were two views of the case. First, was the patient’s history correct, and should she have been delivered on May 1st, and did the myoma affect the innervation of the uterus and prevent labour coming on at the proper time? Secondly, did the myoma interfere with the development of the placenta? This was probably the correct view. Four months after the labour he found another myoma on the posterior aspect of the fundus, so that there was very little healthy area at the fundus for the placenta. It then became a question of how close the myomata lay to the mucous membrane, as if they lay very close there might not be sufficient room for the uterine sinuses to develop, and hence an insufficient circulation. After a certain period, then, the foetus would die from interference with the placenta. One cause of sterility in cases of uterine myoma was probably to be found in cystic degeneration of the ovaries.

Dr. A. J. Smith related two cases. The first had a large fibroid
tumour, the size of an eight months' pregnant uterus. There were no urgent symptoms, but there was no room for a pregnant uterus along with the tumour. He removed the tumour, and the following year the patient was delivered of a full term child, and had had four children since. The second case was one of six months' pregnancy, complicated by a large fibroid tumour. There was great distress and dyspnoea, and the tumour was removed along with the pregnant uterus. This was another example of the danger of a large fibroid tumour in pregnancy.

Dr. A. J. Horne, in replying, said he never knew of a primipara aged more than forty-seven. He said there were two questions he had avoided in his paper: first, the diagnosis of fibroids with pregnancy, and, second, their after-effects on the puerperal state. In the case he had with Mr. McArdle, the patient was six months pregnant. There was a tumour in the right hypochondrium, which overshadowed the uterus, but there was a distinct line of demarcation. It gave rise to only slight pressure symptoms, and the pregnancy went on to full time. There was a breech presentation. As to post-partum haemorrhage, he had always referred to the danger of it in these cases, but had seen many without it. He did not see how sterility was caused by pressure on the oviducts, as in these cases there was excessive menstruation.

**Friday, February 3, 1905.**

**The President in the Chair.**

**Specimens.**

Dr. Alfred J. Smith showed a large physometric uterus.

Dr. E. H. Tweedy showed an ovarian cyst.

Dr. Purefoy read a paper on "Two Cases of Cystic Endometritis" (see page 217, ante).

Dr. Alfred J. Smith read a paper on "Observations on Bossi's Dilator" (see page 223, ante).

Professor R. J. Kinkead read a paper on "Puerperal Convulsions" (see page 230, ante).
Friday, March 10, 1905.

THE PRESIDENT in the Chair.

Specimens.

Dr. E. Hastings Tweedy—Tubo-ovarian abscess; Myoma of broad ligament removed by myomectomy; Maldevelopment of large intestine—enterostomy; Ovarian cystoma.

Dr. Henry Wilson—Fibro-myomatous tumour excised from labium majus; uterus removed by panhysterectomy for adenoma malignum.

Dr. Jellett showed an infant on whom cleidotomy had been performed in consequence of impaction of the shoulders at the pelvic brim. Traction failed to deliver, and so cleidotomy was performed. Traction still failed, and accordingly the posterior muscular attachments of the scapula were divided. This enabled the whole scapula and shoulders to move forward on to the chest, and delivery was then easily accomplished. The foetus measured 45 cms. round the shoulder girdle with the shoulders in their normal position, and 34 cms. when compressed after the division of the bones and soft parts.

Dr. Hastings Tweedy read a paper on "A Curative Operation for Procidentia Uteri" (see page 240, ante).

Professor R. J. Kinkead read a paper on two cases of "Wounds of the Female Genitals" (see page 245, ante).

Friday, April 7, 1905.

THE PRESIDENT in the Chair.

Specimens.

Dr. Jellett showed a Fallopian tube with calcified nodules filling its lumen, the remains of tuberculous infection.

Professor E. J. McWeeney showed a specimen of Ruptured Tubal Pregnancy, with Demonstration of Chorionic Villi.

Dr. Jellett read notes on a case of "Labour in a Unilateral Synostotic (Nægele's) Pelvis" (see page 250, ante).

Dr. E. Hastings Tweedy read the Report of the Rotunda Hospital for the year 1903-4 (see page 256, ante).
Abstracts.

Friday, May 26, 1905.

The President in the Chair.

Specimens.

The President (Dr. A. J. Smith)—An Ovarian Cyst.

Dr. E. Hastings Tweedy.—Ruptured Uterus.
I have been fortunate in observing during the last couple of years a series of cases representing most of the recognised morbid conditions of the blood. These cases occurred for the most part in the wards of the Mater Misericordiae Hospital, to which I am pathologist.

In view of the great interest attaching to them, and of the rapid development of our knowledge as to their mutual relations, classification, and pathogenesis which we owe to the methods associated with the name of Ehrlich, I have thought it well to lay my observations before the Royal Academy of Medicine in Ireland. When giving notice of this paper I intended to include all the cases in the one comparative study, but on looking over the mass of notes and specimens which I have accumulated I saw that I could not adequately deal with them in the time at my disposal, and I have accordingly decided to confine myself on this occasion to the description of two cases of lymphatic leukæmia, one of chronic and one of acute character.

J. N., aged fifty-five, farmer, was admitted Sept. 2nd, 1904, to Mr. Blayney’s Ward, complaining of abdominal tumour. Patient
was a tall, spare man, with pallid face and stooping shoulders. He had no serious illness previous to the present attack. In January last he fell from a hay-rick, when his left side struck the handle of a hay-fork, since which time he felt uncomfortable in that region. About Easter he began to notice his neck swollen, and first detected a distinct tumour in the left side of the abdomen on May 20th. He complained of dragging sensations, dyspnoea on exertion, loss of appetite and general weakness.

On examination the left side of the abdomen was found occupied by a large solid tumour, evidently the spleen, extending down two inches below the umbilicus. The liver was also distinctly enlarged. On both sides of the neck was a chain of enlarged lymphatics extending from the suboccipital region to the clavicle. The glands were harder and smaller on the right side. Both axillæ also contained glands, which were small and superficial on the right side, whilst on the left they formed a mass as big as a hen’s egg, more deeply situated. In the right groin was a mass of glands, each of which was nearly as big as a walnut. Those in the left groin were quite small and shot-like. He did not complain of hæmorrhage from the mucous membranes, but there was a spot of suffusion under one of the conjunctivæ.

The blood-count, which I made on the day after admission, gave reds, 3,908,000; whites, 163,200; whereupon the case was diagnosed as leukaemia, and removed to a medical ward under the care of Dr. Martin Dempsey. The blood was noticed to be pale, watery, and coagulated slowly. The differential leucocyte count made two days later yielded no less than 96 per cent. of mononuclears, of which 81 per cent. were lymphocytes and 15 per cent. large mononuclears, whilst only 4 per cent. were polymorphs, the relation of mono- to poly-nuclears being as 24 to 1. The following table gives the details of the several blood-counts that were made. In the first differential count, which was made on a Jenner-stained preparation, it seemed possible to differentiate between lymphocytes and large mononuclears; the former were smaller, averaging about 8 μ., but reaching in some cases 10.8 μ., with deeply-staining nucleus and scanty hyaline protoplasm; the latter were larger, 11 to 15 μ., with larger, less deeply-staining nucleus, which was often coarsely lobulated. Their protoplasm was more abundant, and in triacid and Jenner preparations it often presented a minute basophilic
granulation. In haematoxylin-eosin preparations the distinction between large and small lymphocytes could not, however, be maintained. Intermediate forms occurred in every field, and I was obliged to abandon the attempt to discriminate between these two varieties of leucocytes. Granule cells were extremely few; at no time did the finely granular polymorphs ever reach 10 per cent. of the total leucocytes. Myelocytes were extremely scarce, and such as were found seemed very fragile. I missed them altogether from the preparations made towards the end of the case, though they occurred in the lung-blood taken post mortem. Eosinophiles were even scarcer. Out of several hundred leucocytes examined on a haematoxylin-eosin preparation only one was seen, and it was remarkable in several respects. It was 13.2 μ in diameter, with a small deeply-staining nucleus, 6.2 μ in diameter, nearly central in position. The aspect of the cell was totally unlike that of any eosinophile I can remember to have seen. The nucleus exactly resembled that of the neighbouring lymphocytes. No undoubted mast-cell was seen. The red corpuscles showed but little alteration. On the one occasion (September 7th) when the haemoglobin was tested with Sahli's modification of Gower's instrument, it worked out at about 65 per cent., with a red count of somewhere about 3,000,000. The specific gravity (Roy-Hammerschlag) on that occasion was 1.047. Nucleated forms were, considering the anaemia, surprisingly scarce. Megaloblasts were absent altogether, and normoblasts were very uncommon. A few, with much fragmented nucleus, were, however, detected. Granular degeneration and polychromatophilia were not observed. Glycogen was not present to a very marked extent at any time. It was seen only in the polynuclears, and never in the lymphocytes. The urine showed no marked abnormality save the presence of amorphous urates in large amount. Crystals of uric acid were not seen. There was no albumen. The total nitrogen, estimated on one occasion by my pupil, Dr. Farnan (Student in Pathology of the Royal University), was found to be 1.428 per cent. by Kjeldahl.

Progress of the Case.—On September 25th the patient, who had been allowed to walk in the garden, developed lobar pneumonia of markedly asthenic type, his temperature rose to 103ø. On the 27th the respirations were gasping and hurried, pulse 128, with a tendency to intermit, face and lips pale. The breathing was
much embarrassed by the abundant muco-purulent exudate, which he was unable to expectorate. He died on the following day.

His blood was examined about 36 hours ante mortem, when, as will be seen from the table, there was not only no diminution of the total leucocytes, such as usually occurs when pneumonia or other infective process supervenes on leukemia, but they were actually increased in number. Of the polynuclear leucocytosis so characteristic of pneumonia, there was no trace in the peripheral blood. This, of course, means absence of reaction against the infective agency, and accounts for the rapid collapse of the patient, who may be said to have sunk without a struggle, being deprived of his normal defensive mechanism—the polynuclear leucocytes. The vast majority of the few he did possess were found at the autopsy, as might be expected, in the blood and exudate of the lung. Another point noted in the differential blood-count made on that day was the obviously degenerate condition of the nucleus of many of the lymphocytes and large mononuclears. It was often incurved, reniform or bilobed, or else it presented a highly stained or unstained belt dividing it in two, as though it had just undergone direct division. It was often vacuolated, and a darkly stained granule, resembling a nucleolus, sometimes appeared in the centre of a large vacuole. The nodal points of the nuclear reticulum seemed also unduly prominent. The protoplasm of many of the smaller lymphocytes was reduced to a hardly visible ring. Many of the polynuclears were unusually large, with completely fragmented nucleus and protoplasmic granulations tending to be basophilic in their colour reaction to the Jenner stain.

**AUTOPSY.**

This was done within 12 hours after death, and was complete, with the exception of the nervous system. Only the points of importance are here given. All the lymphatic gland groups were enlarged: those in the neck, axillae and groins moderately so (pea to cherry size); those in the mediastina, retro-peritoneal tissue, portal and coeliac regions were larger (up to a hen's egg). The bronchial glands were three times as big as normal, slate-grey in the middle, pinkish and pulpy towards the periphery of the cut surface; they were highly vascular. The capsule of all
**Table of Blood-counts in a Case of Chronic Lymphæmia.**

<table>
<thead>
<tr>
<th>Date</th>
<th>Dilution used in making count</th>
<th>Reds, in thousands</th>
<th>Whites, in thousands</th>
<th>Relation of whites to reds</th>
<th>No. of Leucocytes examined in making differential count</th>
<th>Polymorphs, per cent.</th>
<th>Large mono-nuclears, per cent.</th>
<th>Lymphocytes, per cent.</th>
<th>Total mono-nuclears, per cent.</th>
<th>Relation of mono-nuclears to polymorphs</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sept., 1901</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>1:100</td>
<td>3,908</td>
<td>163</td>
<td>1:24</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Blood, pale, watery—coagulated slowly.</td>
</tr>
<tr>
<td>5</td>
<td>1:10</td>
<td>—</td>
<td>154</td>
<td>—</td>
<td>200</td>
<td>4</td>
<td>15</td>
<td>81</td>
<td>96</td>
<td>24:1</td>
<td>—</td>
</tr>
<tr>
<td>10</td>
<td>1:100</td>
<td>2,848</td>
<td>206</td>
<td>1:14</td>
<td>307</td>
<td>7.8</td>
<td>2.3</td>
<td>89.5</td>
<td>91.8</td>
<td>124:1</td>
<td>Differential count made on a Jenner prep.</td>
</tr>
<tr>
<td>15</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>94.7</td>
<td>18:1</td>
<td>Toluidene-blue prep. Mononuclears not sub-divided.</td>
</tr>
<tr>
<td>20</td>
<td>1:10</td>
<td>—</td>
<td>167.2</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>92.4</td>
<td>15:1</td>
<td>—</td>
</tr>
<tr>
<td>27</td>
<td>1:100</td>
<td>3,048</td>
<td>332</td>
<td>1:9</td>
<td>477</td>
<td>6.2</td>
<td>—</td>
<td>—</td>
<td>92.4</td>
<td>15:1</td>
<td>Hämatox-cosin prep.—a few (1.6 per cent.) very large mononuclears seen; but, generally speaking, it was impossible to distinguish large from small mononuclears.</td>
</tr>
<tr>
<td>29</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>175</td>
<td>20.5</td>
<td>—</td>
<td>—</td>
<td>75.5</td>
<td>3.7:1</td>
<td>Film of lungblood taken post mortem and stained with Jenner. It contained 0.5 per cent. of eosinophils and 3.5 per cent. of myeloocytes.</td>
</tr>
</tbody>
</table>
the glands seemed intact, and the glands, though swollen and soft, could not be said to be confluent; their intense vascularity was a marked feature. The largest masses were as usual found along the course of the aorta in the posterior mediastinum, and from the coeliac axis to the bifurcation.

The *lungs* showed red hepatisation of the whole left lower lobe, and of the lower fourth of the right upper, and upper two-thirds of the right middle lobe. They were elsewhere oedematous, and presented obsolete tubercle in both apices. The left pleura was nearly full of fluid; none in the right.

*Heart.*—Beyond some irregular raised white plaques on the surface of the right auricle, this showed no abnormality. There was slight mottling of the myocardium, but no pronounced condition of fatty degeneration.

*Spleen.*—This weighed 51 ozs. It was adherent to the diaphragm and omentum. Without and within it presented an appearance that might perhaps be most fittingly described as "marbled"—studded over with small irregular grey and red areas, with here and there a larger bright-yellow cheesy area of necrosis, surrounded by a vivid red zone of hæmorrhage. Its consistency was decidedly firmer than normal. Neither follicles nor trabeculae were visible on the cut surface. In the hilum were several enlarged glands as big as a horse-bean.

The *liver* weighed no less than 83 ozs. It was soft and pale, with slight "nutmeg" discoloration. Though extensively altered, as will be seen lower down, it presented no marked change to the naked eye.

The *kidneys* were about normal in size, and studded over with small yellowish-white patches, which proved to be leukæmic infiltrations. These were mostly superficial, not penetrating more than about 2 mm. beneath the capsule.

The *mesenteric glands* were all moderately enlarged. The lymphoid structures in the small and large intestine were enlarged, and projected about 1 mm. above the surface of the mucous membrane. They were greyish-pink in hue, and not ulcerated.

*Marrow.*—That of the left femur and left humerus was examined, 6-inch lengths of each bone being removed. It was found to be yellow, fatty, non-vascular, and quite normal, both macro- and micro-scopically.

The *manubrium sterni* was bisected, squeezed in a vice, and
A Case of Chronic Lymphæmia.

films made from the pinkish marrow-like fluid that exuded. They will be described lower down.

HISTOLOGICAL EXAMINATION.

This was done by means of films and sections. The former were all air-dried and fixed in alcohol and ether, equal parts. The tissues were fixed in triplicate:—(a) In 10 per cent. formalin; (b) in saturated sublimate; (c) in Flemming’s solution.

1. Bone-marrow.—(a) Of the Long Bones.—After removal of the fat by immersion in alcohol and ether only a few red discs and lymphoid cells were left. Neither eosinophiles, nor myelocytes, nor nucleated red cells were discoverable. The normal fatty character of the marrow had evidently persisted. (b) Of the Sternum.—This was seen to be of lymphoid character, most of the cells being small, non-granular, mono-nuclear elements, with a narrow rim of protoplasm, mostly more basophilic than the nucleus. There were also much larger cells with a single faintly stained nucleus which often nearly filled the cell. Their protoplasm was either (1) hyaline or (2) studded with extremely fine basophilic or neutrophilic granules (myelocytes). A few eosinophiles, both poly- and mono-nuclear, were also seen. Polynuclear neutrophiles of the ordinary kind were almost absent. There were many nucleated red cells, all of normoblastic character, and possessing either a single or a fragmented nucleus.

2. Pleural Exudate.—By means of the centrifuge an abundant deposit of leucocytes was readily obtained. These were mostly lymphocytes, the polymorphs being relatively few and degenerate, their granules being scanty and often basophilic in affinity. There were also many large mononuclear cells with non-granular protoplasm so basophilic as to be hardly distinguishable from the nucleus. These cells were often much vacuolated. They were probably desquamated endothelium. Pneumococci were present in small number outside of the cells.

3. Lymph-glands.—The films showed only lymphoid cells of moderate size, with a large nucleus and a narrow fringe of basophilic protoplasm. Mitoses were not seen, nor was there any difference between films taken from different glands. In material fixed whilst still warm with Flemming, paraffined, and cut in ribbons, not exceeding 5 mikra in thickness, mitoses were readily found, especially in safranin preparations. In some sections they were
By Dr. E. J. McWeeney.

quite numerous, several in each high-power field. The lymph-channels were filled with structureless coagula, containing many lymphoid cells, some of which lay near the outer wall, and seemed as though wandering into or out of the sinus. The distinction between endothelioid germ-centres and ordinary small-cell lymphoid tissue was obliterated. The lymphoid tissue in some of the glands was divided up into follicles by trabecule coming in from the capsule, and many of these follicles contained large thin-walled vessels. Giant and eosinophile cells were not seen in the glands.

4. Spleen.—Films made at the autopsy presented mainly two sorts of cells—viz., ordinary red corpuscles and lymphocytes—mostly small, many being reduced to a nucleus with a barely discernible ring of protoplasm. Occasionally there occurred much larger, more faintly-staining elements, with a large faint nucleus, nearly filling the cell; the protoplasm was charged with exceedingly fine acidophil granules. These cells would therefore appear to be of myelocytic nature. Their significance in the spleen is doubtful in a case of lymphatic leukaemia. They were often vacuolated. A single large mononuclear with eosinophil granules was also found. The presence of these two last-mentioned varieties of cell suggests the question: Could there have been a myeloid transformation going on in the spleen? One would hardly expect it to occur in lymphatic leukaemia unless, indeed, the bone-marrow had undergone such an amount of lymphoid transformation as to react by an overgrowth of its granule-cell-forming tissue, and that this had then become generalised. None of the sections made from portions (four) cut from various parts of the spleen showed any myeloid cells. In this connection it is also to be noted that there were extremely few polynuclears in the spleen. Sections showed (a) reduction of the trabecular system, the cords being few and small. (b) Reduction of the follicles, very few being present, and these of small size. The general appearance of the sections was one of nearly equal distribution of the lymphoid cells, between which a fine reticulum of nucleated fibres, often reinforced by unstriped muscle, could readily be made out. Long lines and columns of large mononucleated cells, perhaps identical with Mall's lymph-cords, could be seen all over the sections, but a lobular division, as described by that writer, could certainly not be detected. (c) Presence of patches of
necrosis corresponding to the opaque patches seen on the whole specimen. The centre of these stained a diffuse violet with hematoxylin, and showed complete karyorrhexis. The periphery showed evident signs of re-organisation in the shape of many layers of fibroblasts and new thin-walled vessels filled with lymphocytes. Here and there was an extra-vascular polynuclear leucocyte, though the majority, even in these re-organising parts, were mononuclear. Outside of the fibroblastic area were effused red corpuscles, which became more and more numerous and distinct as one passes outwards, until finally they formed a bright purple-red ring, visible to the unassisted eye, round the necrosed patch. (d) Presence of brown amorphous pigment in granules, lumps and scales, especially at the periphery of necrotic parts and near the hæmorrhages. This pigment gave a typical iron-reaction with ammonium sulphide, and a less distinct one with ferrocyanide of potash and HCl. (e) Amongst the red corpuscles, so numerous in the spleen-sections, nucleated forms were not seen. (f) On the reticulum a large few mononucleated endothelial plates could be made out, but no giant cells. The general impression left by a study of the spleen sections was that of an enormous and uniform increase of lymphocytes, forcing apart the trabeculae, obliterating the follicles, and producing a uniform microscopic picture, varied only by patches of hæmorrhage, necrosis, pigmentation, and fibroid transformation. Giant-cell formation, so often encountered in cases of Hodgkin's disease, was not found. In concluding this description of the spleen I wish to lay special stress on the rarity of mitotic figures, even in thin paraffin sections of Flemming-fixed material stained with safranin, whereas they were readily demonstrated in sections of the lymphatics, similarly treated. Apart from the hæmorrhages there was very little necrosis.

5. Liver.—Despite the macroscopically unaltered appearance of this organ, the sections showed the most exquisite lymphocytic infiltration of the portal canals. The walls of the veins, and occasionally of the arteries, were infiltrated, and, indeed, replaced by the small-cell growth, in which the bile-ducts were immersed; the cells composing it were typical lymphocytes, with scanty protoplasm and darkly-stained nucleus, and they lay upon a distinct reticulum. They did not penetrate between the liver cells, which, save for the presence of golden-brown pigment,
seemed quite normal. The pigment was amorphous, and did not give the iron reaction. It lay chiefly near the centre of the lobules. In many of the liver cells there were also isolated granules of iron-containing pigment. Amongst the lymphoid cells, which so abundantly infiltrated the liver, it was very difficult to find any in a state of mitotic activity, yet we must suppose them to be actively proliferating.

6. Kidneys.—These were thickly studded with small patches of lymphoid infiltration, which for some reason were strictly confined to the sub-capsular region of the cortex, into which they penetrated in a wedge-like manner, like minute infarctions, for the distance of a millimetre or two. The lymphoid cells seemed to be insinuating themselves between the tubules and forcing them apart. Degenerating tubules and glomeruli, quite immersed in lymphoid cells, could be seen here and there. Typical mitoses were again very difficult, almost impossible, to find amongst these closely-crowded lymphoid cells, even on Flemming preparations stained with safranin and iron-hæmatoxylin.

7. SuprarenaIs.—These were normal, save for the presence of lymphoid infiltrations amongst the large cells of the medulla.

8. Pancreas.—A minute patch of lymphoid cells was found amongst the acini, and doubtless others would have been seen if sought for. The islands of Langerhans seemed unduly prominent and large.

9. Lungs.—In view of the rarity of polymorphs in the general circulation, the histological examination of the pneumonic lungs promised to be of great interest. The alveoli were found distended with coagulated fibrin, in which were entangled many red corpuscles and great numbers of polymorphs, apparently nearly all the patient had to dispose of. Many of them seemed degenerate, with completely fragmented nuclei. In every field there were small numbers of pneumococci. They were mostly extra-cellular. The contrast between the snake-like vessels crowded with lymphocytes and the alveoli packed with polynuclears, between which they coursed, was very curious.

This case must be looked upon as a typical one of chronic lymphæmia. In the course of events the only departure from normal was the absence of any polynuclear leucocytosis or diminution of the lymphocytosis
A Case of Chronic Lymphæmia.

as the result of pneumococcal infection. From the anatomical standpoint the chief departure from the conditions usually found in such cases was the state of the marrow of the long bones, which, instead of undergoing a lymphoid transformation, retained its normal fatty character. Possibly the lesion was unevenly distributed focal, and might have been detected had a more complete examination been made. Another point of interest is that whereas fresh blood-preparations always contained leucocytes crammed with large refractive granules, which might readily have been taken for eosinophiles, stained films showed practically no trace of this variety of cell.

I have to express my thanks to Dr. Martin Dempsey for permission to bring this case forward.
That acute yellow atrophy of the liver is a comparatively rare disease in this country is sufficiently demonstrated by the fact that no case has been reported at any of the Sections of the Royal Academy of Medicine in Ireland since its inception twenty-one years ago. Murchinson in his writings about this disease stated that during a period of nine years, and out of a total number of 27,000 patients admitted into the London hospitals, he had only seen one case, and Dr. Wickham Legg could only trace one case in the records of St. Bartholomew's Hospital for a similar period.

It is an interesting as well as an historical fact, according to Dr. William Hunter of Charing Cross Hospital, that it was Dublin physicians who in the beginning of the last century first in this country made observations about the disease—Cheyne and O'Brien in the year 1818, and Marsh four years later; Morgani had previous to this described cases presenting features of the disease. The first accurate clinical account recorded was that by Bright in the year 1836. He described the changes which took place in the liver as a "diffuse inflammation affecting more the glandular portion than the connective tissue, leading to great diminution in the size of the organ, and accompanied by intense jaundice, severe
nervous symptoms, and often a hæmorrhagic tendency."

Two English observers—Busk in the year 1845, and Handfield Jones two years later—accurately described the characteristic microscopic appearance of the degenerated liver cells, and about the same time Continental investigators confirmed these observations and added more to what had already been made known.

The patient, E. B., was admitted into Jervis Street Hospital on the 24th August last. She was a well-nourished woman of medium height and build, with dark hair and complexion, aged twenty-seven years. She was born in Dublin, and had a good family and personal history. She had been married five years, had three children, the youngest of whom was four weeks old; the other two, aged respectively four and two years, were strong and healthy. She had not had any miscarriage, nor was there a history of a rash or sore throat, so that syphilis as a cause must be excluded. Her husband, a labourer by occupation, was not always steady or at regular work, which necessitated her doing washing, and in this way she contributed to the support of her family. She had not been in the habit of taking either porter or any other stimulant. She resided in a tenement house situated in one of the most congested parts of the city, where the surroundings could not be anything other than detrimental to a robust state of health. While carrying her last baby she enjoyed average health—as she did on the two former pregnancies. Parturition seemed to have been normal, and she was able to be up and about at the end of the week, and, as with the other two children, she nursed this child. During the third week after her confinement she felt sickness of stomach, loss of appetite, languor, and the jaundice first appeared at the end of three weeks after childbirth. A week later she first came under my care. At that time there was nothing in her condition to suspect anything other than an ordinary case of catarrhal jaundice. The liver dulness was not affected; no hardness or resistance of the organ could be detected, and the gall bladder was not distended; there was no pain on pressure. The tongue was thickly coated, enlarged, flabby and indented with teeth marks. The teeth were exceptionally good; the tonsils were normal, and no enlarged glands could be discovered. The
abdomen was soft and flaccid; no rigidity at or about the liver region; all the abdominal organs seemed normal, as well as the heart and lungs. There was marked constipation, and rather more sickness of stomach perhaps than one usually gets in a simple case. The skin and mucous membrane presented all the appearance of a well-marked case of jaundice, as also did the urine. The quantity of urine passed per day was below the average, 35 ounces. Urates were abundant, and there was a slight trace of albumen; no sugar, sp. gr. 1022, and the quantity of urea was less than normal. At this time leucin and tryosin were not looked for. There was one other symptom which the patient complained of on admission—viz., a shooting, acute, spasmodic pain, lasting only a short time, apparently in the liver. She stated that the pain was deep-seated, and scarcely ever occurred in the exact place a second time, and her description of the pain was as it were the stab of a sharp instrument. External pressure had no effect in either relieving or increasing it, nor did the deep breadth affect it. All through her illness this symptom was prominent, and towards the end became sometimes so severe that she had to cry out. Her temperature was normal, and pulse 62 per minute. For the first week in hospital this patient seemed to improve; took light nourishment well; the tongue became cleaner, and the jaundice was apparently not so intense. The bowels however required strong purgatives. There was no trouble with the breasts, as the milk ceased coming at once, although she had been suckling her child until her admission. During her second week in hospital the patient did not feel so well, the nausea and epigastric discomfort was very marked, the bowels became more stubborn, and the jaundice became more intense. From this until her death the patient got gradually worse, vomiting became almost incessant, the liver dulness diminished in area, and it was now found the amount of urea was diminished, and about half what it was on admission and leucin and tryosin were present. The temperature now became irregular, varying from 96.5° to 102°, and the pulse became intermittent and fluctuated between 80° and 120°. There was no direct relationship between the temperature and pulse; when the temperature was high the pulse might be low, &c. At the end of the third week in hospital cerebral symptoms developed.

She gradually became drowsy and semi-conscious, was rest-
A Case of Acute Yellow Atrophy of the Liver.

less and had slight delirium; the jaundice became very intense, and appeared as a greenish hue; the tongue became dry and brown, and the amount of urine passed diminished to 13 ounces per day. The liver dulness practically disappeared in front and only posteriorly could it be slightly detected. She remained in this condition for three days, but gradually becoming more unconscious and weaker, and died about four and a half weeks after the jaundice first appeared. The vomiting was always gastric. At no time had she any hæmatemesis or any other hemorrhagic manifestations. In the post-mortem room the body was not much wasted, considering the severity of her illness and the small quantity of nourishment she was able to make use of. On opening the abdomen there was found a small quantity of greenish-coloured fluid. The liver instead of presenting as usual was small, baggy-like and collapsed, and could only be seen by its being kept in position by the suspensory ligament. When removed it felt soft and spongy-like, with a wrinkled capsule, and weighed twenty-nine ounces. The stomach was small, and on opening it the mucous membrane was found to be pale and apparently healthy. The other abdominal organs seemed to the naked eye to be in a healthy condition. The spleen, the pancreas, and the kidneys seemed normal in size and on section. The uterus was normal, and there was a small ovarian cyst about the size of a marble. None of the abdominal glands were enlarged. The heart and lungs were healthy, and there was not any fluid in the pleural cavity or pericardium. I did not get examining the brain.

The clinical points of interest about this case are:

I. The rather protracted course—about 4½ weeks—although Hunter in his 29 collected cases gives 9, or 31 per cent., as running a course of from 3 to 8 weeks.

II. The acute, short, spasmodic liver pain which was persistent throughout.

III. The total absence of any hæmorrhagic symptoms.

Professor E. J. McWeeney then described the character of the specimen as follows: Liver weighed 29 ozs., small, very soft,
and pulpy; capsule wrinkled, pale red with yellowish mottlings. On section, mostly reddish with bright yellow patches, lobular marking obliterated. Dimensions of right lobe, 6 in. from side to side, 5½ in. from before backwards, 1½ in. thick. The left lobe was at most only half an inch thick. Gall-bladder normal. The microscopic examination was made by means of teased preparations, frozen and paraffin sections. It showed complete necrosis of the liver cells; patches of round-cell infiltration, which still preserved their nuclear staining; patchy pigmentation of the liver cells, with yellow granular matter which did not give an iron reaction (bile); moderate fatty degeneration; and the presence of minute, yellowish-brown globular concretions in groups and rows. These were very abundant in the tissue, and varied from 12μ to 30μ in diameter. Some of them seemed radially cleft. others were coated with a prickly layer of minute needle-shaped crystals, whilst others presented obscure concentric striation. There were also minute colourless acicular crystals lying singly and in sheaves. These objects were doubtless crystals of leucin and tyrosin. The globules were soluble in alkalies and weak H₂SO₄; insoluble in ether, alcohol, and chloroform; partly soluble in water. A few bacilli were demonstrable on the sections, and of the numerous culture-tubes inoculated a few showed colonies. Study of the organisms, however, convinced him that they were merely ordinary putrefactive germs.

Professor O'Sullivan said he remembered one case of the kind, which belonged to Dr. Wallace Beatty. He had some sections of it which considerably resembled those shown, but his were more advanced. He was not sure that he would have taken Professor McWeeney's view of the condition of the liver cells, as he did not think the process was very far advanced. There was certainly not so much disintegration shown in the sections as in those of the case in his possession. The process seemed to be starting from the heptic veins, the portal areas being less affected.

Dr. H. C. Earl had seen portions of a liver from an undoubted case of this disease. A large portion of the right lobe was involved, and on section of this, more than half the affected portion was a brilliant red colour, and the rest an equally brilliant yellow. In the red area the liver cells had disappeared, and in the portal canals there was small round-cell infiltration. In the yellow areas were cells which looked absolutely normal. He was surprised at
the small amount of fat he found in the cells. He noticed in some parts of the yellow areas, where they bordered on the red, that the centres of the lobules were certainly more affected than the margins. The process seemed to be progressing from the centre of the lobule to the periphery.

Professor McWeeney, in replying, said he considered that the cells were completely necrotic, though their outlines were preserved. They did not give the ordinary nuclear stain. The protoplasm had stained diffusely with haematoxylin stain. The preparations showed no well-preserved liver cells. The yellow pigmentation was well marked in one of the sections. The bacteriology of these cases was very interesting, because it was very important to eliminate the theory of living bacteria. The generally accepted theory was that the disease was due to toxins. He had set going a number of aerobic and anaerobic cultures, but the material was not perfectly fresh. The anaerobic tubes remained sterile, and the aerobic showed some small white colonies which he considered unimportant. He cut some sections in paraffin, and found occasional bacilli, which he thought were ordinary putrefactive organisms. He thought the toxæmia theory still held the field.
A CASE OF GLANDERS.

By EDWARD F. STEPHENSON, F.R.C.S., D.P.H.;
Dunmore East, County Waterford.

[Read by permission in the Section of Pathology, November 25, 1905.]

P. M., a farmer's son, aged twenty-one years, of powerful make and robust constitution, sickened on October 1st, 1904, with general malaise and pains, but no rigors. His mouth became sore, and hard swellings formed on the gums and roof. A few days later a large tumour appeared in the right submaxillary region, and smaller tumours beneath the left jaw. He continued to work on the farm till October 15th, when I was called to see him. I found the gums and hard palate covered with nodules; the teeth were loose, and fetid pus welled up between them and the gums; the breath was fetid; a hard, nodular mass filled the right submaxilla; it was fixed to the lower jaw, and was painless on pressure; a dusky red phlegmonous patch partly covered the chin. The patient was unable to breathe through the left nostril, from which there was an offensive discharge; the chest, abdomen and back were covered with subcutaneous nodules, varying in size from a pin's head to that of a bean; the skin over these was red, except in a few instances; subcutaneous nodules were also present on the forehead, over both eyes, and on the left temple. The temperature was normal; pulse, 100; respirations, 24. He complained of noise and deafness in the left ear, and of having sweated freely on a few occasions. He had suffered from neuralgia for some days before the onset of his illness; and he was accustomed to pick his teeth with his finger nail. Glanders was diagnosticated, and as the relatives would not permit his removal to the fever hospital the patient was promptly isolated in his own house. The strictest inquiry failed to elicit information of the illness of horses, donkeys or mules on the farm, nor did his movements for some days prior to the onset throw light on the origin of the disease. The farmyard showed an entire absence of sanitation. Some days later, after very minute inquiry, I learned that a horse had been sick in 1902 in a small adjoining farmyard
belonging to his father, but quite distinct from the farm dwellings. This horse had been sick for three or four months; he had suffered from a sore throat and nose, and the breathing had been considerably embarrassed; abscesses had formed on the head and body, and were, from time to time, lanced by members of the family, "the matter shooting across the yard." The horse recovered, and was sold. The stable was twice limewashed, and in due course the manure removed, with the exception of about two feet, which remained in the yard. The stable was soon occupied by another horse, and the remains of the old heap became quickly covered with fresh manure. Four days before P. M. became ill he was engaged in removing this heap, and on this occasion none was left behind. He was suffering from neuralgia, and picked his teeth with his finger nail. I visited the patient frequently. He became progressively worse. Nodules appeared on the soft palate, fauces, and in the second nostril; dyspnœa became extreme. The nodules on hard palate softened, and were lanced, discharging pus and blood; those on the soft palate and fauces and the "farcy buds" on body showed no sign of suppurating. Antiseptic gargles and mouth washes were freely used, a plentiful supply of nourishment and stimulants allowed, nux vomica, quinine and strophanthus administered. On October 24th the pulse quickened (140) and grew feeble, the temperature rose to 101° F., and delirium supervened. Dr. Jackman, of Waterford, saw the case with me on the 28th. He was then sinking. No improvement occurred, and at 2 a.m. the morning following he succumbed to exhaustion.

The points of interest in this case are:—(1) The source of infection, and (2) the seat of the primary inoculation.

A CASE OF ACUTE LYMPHÆMIA.

By E. J. McWEENEY, M.A., M.D., R.U.I., F.R.C.P.I.;
Professor of Pathology, C. U. Medical School, Dublin;
Pathologist, Mater Misericordiae Hospital.

[Read in the Section of Pathology, November 25, 1904.]

A boy, B. F., aged nine years, was admitted to the Mater Hospital on 1st October, 1904, under the care of Dr. John Murphy. He had swellings on both sides of the neck, breathlessness, and bleeding from the nose.

Family History.—His father, a tailor, hurt his leg when four years old, and subsequently developed hip disease, for which amputation through the hip joint had to be performed. The mother, who was described as having been always very pale, died in February, 1903, about a fortnight after the birth of a child, from what cause I was unable to ascertain. Patient’s elder sister, aged ten years, was ill with pneumonia when patient was admitted to hospital, and her life was despaired of, but she recovered. His younger sister, aged five years, was described as suffering from swellings in the neck. I had her brought to the hospital and found her to be a ruddy, healthy child, with perfectly normal blood. In the upper cervical and suboccipital regions there were a few enlarged glands, which were obviously associated with irritation of the scalp, due to pediculi.

Personal History.—Six weeks before admission lumps were first noticed in the neck, armpits, and groins. For a week prior to admission he had been suffering from epistaxis and bleeding from the gums. The swellings under the left arm were painful.

Condition on Admission.—A good-looking, well-nourished, fair-haired boy, the lower part of whose face presented a peculiar square outline owing to the large glandular swellings beneath the jaw on each side. The face and mucous membranes were very pale. The tonsils were enlarged, and the post-nasal adenoids evidently present and large. Tongue dry, and coated at the sides; lips cracked on the inside. The skin of the abdomen and legs is mottled with dozens of small petechiae, which are said to have
A Case of Acute Lymphæmia.

appeared before the glands began to swell. Masses of enlarged glands are seen and felt at the sides and back of the neck, in the submaxillary region, in both axillae and groins. The glands are moderately hard, discrete, and freely movable. One gland in the left axilla is both painful and tender, and the glands in the right are somewhat tender. Pressure over the other glands causes no pain; no glands palpable in the antecubital or popliteal fossa. The spleen is decidedly enlarged, its edge being felt about midway between the lower border of the ribs and the umbilicus. Temperature on admission, 99° F. (100° F. the same evening); pulse, 120; respirations, 26. The patient was very dull and apathetic. The urine deposited an abundant sediment of uric acid crystals, and contained a distinct trace of albumen.

BLOOD EXAMINATION.

At the first glance the case might have been taken for Hodgkin's disease, but the fresh blood preparation at once settled the diagnosis. On the day after admission the blood count was R. 1,898,000, W. 212,000, the proportion being as 8.9 to 1. The stained preparation showed hardly any other form of leucocyte save lymphocytes—large, faintly staining, and evidently degenerating forms being very numerous. The diagnosis was therefore, lymphatic leukaemia, and in view of the rapid development of the symptoms, temperature, haemorrhages, and general condition, the case had to be classed as an acute form of the disease. The haemoglobin was about 40 per cent., specific gravity 1.0435 (Roy-Hammerschlag). The fresh blood showed much fine molecular matter floating in the plasma, and displaying remarkably active motility, probably Brownian.

The first differential leucocyte count was made on a triacid preparation three days after admission, and showed that no less than 98 per cent. of all leucocytes were mononuclear. Polymorphs were only 0.5, transitionals 1.1 per cent., whilst out of several hundred cells examined only a couple of doubtful myelocytes were detected.

Characters of the Mononuclears.—Somewhat less than half of them (42 per cent.) were of medium size, about as big as a red corpuscle or a shade larger. Their nucleus was strongly basophilic, often delicately incurved or reniform, and sometimes double, with the apposed sides of the nuclei flattened as
though direct division had recently occurred. The protoplasm of these lymphocytes was scanty and hyaline. It was often more basophile than the nucleus, and in Jenner and triacid preparations there was little distinction. A certain number (about 0.6 per cent.) had the same sort of nucleus, but a much larger amount of protoplasm. These might be described as large mononuclears. The other kind of lymphocyte was present in larger numbers (56 per cent.). It was larger, more faintly stained, and so easily broken down that most of them looked like mere irregular bluish expansions, about twice as big as a red disc, and scarcely showing any difference between nucleus and protoplasm. Their margins were often fringed with irregular protuberances. Subsequent observation showed that there was no fundamental difference between these two classes of mononuclear leucocyte, their relative proportions seeming to vary with the amount of pressure used in making the film. The large faint variety I consider to consist of the more labile, degenerating, or necrotic individuals.

A very few large mononuclears appeared to contain very fine granulations of acidophile character—possibly myelocytes. In others there were basophilic granules of various sizes. Polymorphs were remarkably few and hard to find, whilst eosinophiles and typical mast-cells were absent altogether. The red corpuscles were also labile, readily deformed, apt to run together in clumps, and did not stain well, being evidently poor in haemoglobin. With few exceptions, they did not display any basophilic tendency. Nucleated forms were, considering the profound anaemia, surprisingly uncommon, and all that I came across were of normoblastic character.

Progress of the Case.—On October 6th, a bluish patch of ecchymosis, 2¾ in. by 3½ in. in diameter, appeared in front of the left elbow round the vein from which blood had been taken for bacteriological examination. Another large ecchymosis appeared spontaneously over Poupart's ligament, on the same side. The temperature fluctuated between 99° and 101°, the pulse was between 130 and 140, respirations about 24. The sensorium was generally dull, with occasional revivals of interest in his surroundings—on one or two days he even played with his toys. Towards the middle of the month he became steadily weaker, and moaned continually with pain, which he referred to the ears,
A Case of Acute Lymphamia.

teeth, neck, and right arm and hand. The glands increased in size, and there was almost continual weeping of blood in small quantity from the nose and mouth. On the 18th he vomited twice. On the 19th I examined his blood for the last time. He was then quite conscious, but the respirations were hurried and gasping. Death took place shortly afterwards.

Development of the Blood Condition.—On October 7th, the reds were 1,648,000, and the whites 310,000, the relation being 5.3:1. On October 11th the count was—reds, 1,444,000; whites, 245,000, or nearly 6 to 1. The last count was done on the 19th, when the little patient was moribund. It yielded the remarkable figures—reds, 820,000; whites, 783,000, or nearly 1:1. The blood looked decidedly milky, and in the fresh preparation most of the field was occupied by the leucocytes, patches of pale, degenerate-looking, coherent, red discs being visible here and there. Stained preparations showed practically nothing but large lymphocytes of the two varieties described above. Polymorphs were very few—perhaps 1 for every 200 or 300 lymphocytes. An occasional myelocyte was seen, the fine acidophile granules being few and faint. Both Jenner and triacid preparations showed a few small cells with haemoglobin, containing protoplasm of basophilic tendency, a nucleus of normoblastic type and coarse basophilic granules. Normoblasts were fairly abundant, but megaloblasts were absent. Despite the enormous numbers of lymphocytes which must have been the products of active proliferation, I could find very few mitotic figures, and these were not well preserved. The nuclei rather gave the idea of multiplying by direct division. The films were dry ones, however, fixed by heat or alcohol and ether, not with Flemming's solution.

Bacteriological Examination of the Blood.—On October 6th rather more than 5 c.c.m. of blood was obtained aseptically from a vein in front of the elbow, and at once introduced into 500 c.c.m. of sterile bouillon which was then placed in the incubator at 37°C. No growth whatever occurred, and at the end of a few days the corpuscles, both red and white, appeared quite unaltered in the sediment. Stained films of this showed nothing resembling protozoa.

Effect of Stains on the Fresh Blood—On October 6th the patient's finger was punctured through a drop of Unna's poly-
chrome methylene blue, and the mixture of blood and staining fluid examined at once. The nuclei of the lymphocytes were then seen to have become deep violet, whilst those of the polymorphs remained colourless. After the lapse of a few minutes, the protoplasm of the polymorphs began to present a fine violet granulation—evidently due to staining of the neutrophile granules by the red constituent of the polychrome blue. Control preparations with healthy blood showed no staining of any leucocyte nuclei, though a similar tinging of the neutrophilic granules appeared after a time. The rapid penetration of the stain into the lymphocyte nuclei of this patient I am inclined to ascribe to their being in a necrotic, devitalised condition.

**Necropsy.**

This was done a few hours after death. The body was still warm, and the blood only partially coagulated. Its volume seemed much increased, and that contained in the heart looked exactly like anchovy sauce. On standing in a jar it deposited a layer of red corpuscles about an inch thick, whilst the supernatant fluid was cream coloured. The coagula in the heart resembled lumps of fat. Charcot-Leyden crystals never formed.

**Heart.—** Much distended, with discoloured blood, its surface thickly sprinkled with petechiae. Endocardium normal. No valvular lesion. Muscle seemed unaltered. The pericardium contained about 2 ozs. of turbid serum. On its apex was an enormously enlarged thymus, consisting of two well-defined triangular lobes and a connecting central part. Its colour was pale pink without, white within; its consistency quite soft, almost brain-like. Behind it a mass of enlarged glands adhered to the innominate veins, and seemed almost to penetrate them.

**Lungs.—** These showed no sign of tubercle. They were sprinkled over with large subpleural petechiae, and contained many small hæmorrhagic nodules like small infarctions. Bronchial glands enlarged to the size of good-sized beans, pinkish-grey on section. Glands in the posterior mediastinum moderately enlarged. Tonsils much enlarged.

The Peritoneum was studded over with petechiae. It contained no fluid. The great omentum contained many glands as large as peas, and bright red in hue.
Case of Acute Lymphœmia.

Spleen.—Moderately enlarged (12 oz.), pale-red without and within, consistency normal. The trabeculae and follicles were quite visible on the cut surface, which did not differ markedly from the normal. One small white infarct 6 by 2 mm. was seen.

Liver.—Enlarged and hyperæmic, colour pale, with yellow dots, as though fatty. Lobular marking obliterated. Gall bladder small, contained dark-green bile. A chain of enlarged lymphatics occupied the portal fissure and accompanied the vessels to the duodenum.

Suprarenals.—Small, pale, flat.

Kidneys.—Rather large; capsule stripped easily, leaving smooth pale surface; cortex rather wide, pinkish, with large irregular, ill-defined pale patches; boundary layer distinct, bright red, a millimetre wide. Pyramids pink with white streaks. Weight of both organs, 10 ozs.

Stomach.—Small, very pale externally; lesser curvature and posterior surface thickly studded with enlarged lymphatic glands. Mucous membrane cream-colour, mottled with petechiae.

Intestines.—Peyer's patches and solitary glands raised about a millimetre above the surface, swollen, grey, and pulpy; no ulcers.

Mesenteric Glands.—As big as hazel nuts, very red. The largest (size of a pigeon's egg) were at the ileo-cæcal valve.

Cæliac and Lumbar Glands.—Moderately enlarged, soft, bright red on section, hyperæmic.

Marrow of the long bones was dark-red in hue, and resembled spleen pulp.

C. N. S. and external lymph glands, in deference to request of the friends, were not examined.

Minute Anatomy.

The tissues selected at the autopsy (which was done whilst the body was still warm) were fixed, some in Flemming's strong mixture, others in saturated sublimate, and the remainder in 10 per cent. formalin. Smear preparations of the organs were also made.

Marrow (of Femur).—(a) Smears—These showed practically only one sort of leucocyte—namely, large mononuclear cells, which at first sight appeared to consist entirely of a large, faintly-stained nucleus. On closer examination, a zone of proto-
plasm could be made out, of approximately the same affinity for basic dyes as the nucleus, and therefore, inconspicuous. It was non-granular. The margins of these cells (or nuclei) were often peculiarly fringed or crenate. They took the stain in a patchy manner, and altogether gave one the idea of being labile, degenerate structures. Here and there one of their nuclei, more darkly-stained than its neighbours, looked twisted or contorted as though undergoing mitotic change. But in none of the films (alcohol and ether fixation, staining with Ehrlich's haematoxylin) did I observe a genuine mitosis. On the other hand, many of these cells rather gave one the idea of multiplying by fission or budding, and this impression was still further increased by the frequent occurrence of masses of these nuclei lying close-packed together in an ill-defined protoplasmic mass, the whole structure closely resembling an immature giant-cell. The films also showed many red corpuscles, a large proportion being nucleated, and of normoblastic type. Their nuclei were, as usual, opaque black with haematoxylin, and many were rosette-shape or otherwise fragmented, but no genuine mitoses were found on the films. Megaloblasts were not seen. Here and there was a very large cell (macrophage) with single or lobed, vesicular, faintly-staining nucleus, and an abundance of strongly acidophile protoplasm, in which were embedded irregular masses of deeply-stained chromatin, evidently nuclear débris undergoing intracellular digestion.

(b) Sections of sublimate-fixed marrow presented an aspect which at first sight differed widely from that of the films. This was because the cells looked so much smaller in sections than in the spread films. Closer examination, however, showed that the prevailing type of cell was the same—large lymphocytes with round or lobulated nuclei and scanty protoplasm. The fringed and crenate appearance of the cell borders met with in the films was not seen on the sections. The cells were supported on a very delicate stroma, through which coursed many wide vessels, the walls of which were composed of a single layer of endothelial plates. The cells within these vessels were the same as those without—namely, mononuclear leucocytes, similar to those in the peripheral blood. Some of the larger vessels in the marrow were literally packed with them. Scattered through the mass of lymphocytes of which the marrow was
composed were very large masses of protoplasm, with single or lobulated vesicular nucleus and acidophile protoplasm (megacaryocytes).

The most remarkable feature about the marrow was that it contained no granule cells of any sort. The absence of neutrophile myelocytes was proved by the treatment, not only with Ehrlich's triacid, but also with Dominici's eosin-orange toluidin blue. It also contained very little fat.

With regard to evidences of mitotic division, the only preparations that yielded satisfactory results were those stained with Heidenhain's iron hematoxylin, after Flemming or sublimate fixation. In these plenty of mitoses were seen both within and without the vessels. It must, however, be remarked that the chromatin loops were mostly more or less "lumped," and that good diasters seemed unusually hard to find as compared with, for example, a rapidly-growing epithelioma.

*Lymph Glands.*—These were packed with large mononuclear cells with vesicular nucleus, containing several nucleoli. The protoplasm was often reduced to a narrow rim. These cells were evidently the same large lymphocytes which swarmed in the blood and marrow to the almost complete exclusion of almost every other sort of cell. Not only was the capsule of the lymph glands infiltrated with these cells, but also the fat cells which occurred in the vicinity. The glands also contained large endothelial plates belonging to the reticulum, and large mononuclear elements with acidophile protoplasm often containing deeply-stained fragments of chromatin or vacuoles (macrophages). No distinction between germ centres and ordinary lymphoid tissue could be made out, nor could the lymph sinuses be distinguished. The only channels seen were large or small thin-walled blood vessels crammed with red discs and lymphocytes. A remarkable character of these blood vessels was that they contained many macrophages.

*Mitoses* were very numerous in preparations stained with iron hematoxylin, as many as 20 being seen in a single oil-immersion field, whilst as many as 17 were counted in the cross section of a blood vessel 0.06 mm. in diameter. In many cases the daughter nuclei seemed to be again dividing without going through a resting stage. The fat outside the capsule of some of the lymphatic glands was also infiltrated with proliferating lymphocytes.
By Dr. E. J. McWeeney.

323

The Thymus presented, so far as examined, appearances identical with the lymph glands. Some concentrically whorled structures (Hassall’s corpuscles) could be made out among the swarming lymphocytes. Mitotic figures were readily demonstrable.

The Spleen presented the same minute structure as the lymph glands plus patches of extravasated red discs. Mitoses were, however, not so readily found in it. The distinction between pulp and follicles had disappeared.

Heart.—There were large perivascular accumulations of lymphocytes, which seemed to be forcing their way in between the muscle fibres, and to be compressing or eroding them. Similar accumulations were seen beneath the epicardium and pericardium. Many of these extravascular lymphocytes were dividing.

The Nodules in the Lungs proved to be due to the occupation of superficially-placed groups of air cells by (1) oedema; (2) haemorrhage; and (3) lymphocytic accumulations. The vessels in the vicinity were stuffed with lymphocytes, which were dividing both within and without the vessels.

The Liver sections presented an appearance all the more remarkable because the naked-eye characters were not strikingly altered. There was most extensive infiltration of the lobules with large lymphocytes, which distended the capillaries, and in places so compressed the liver cells as to cause them to atrophy and melt away. Whole groups of lobules were thus converted into lymphoid tissue, through which coursed strands of atrophic hepatic cells forming a scarcely visible network. The infiltration was not confined to any one region; both the centre and the periphery of the lobules, as well as the portal canals, seeming to suffer equally from the lymphoid intrusion. In places the capillaries were plugged in a very remarkable manner with large ovoid, oblong, or linear nuclei, hyperchromatic and degenerate, and seemingly devoid of protoplasm. These peculiar nuclei were often twisted together like cords, and seemed as though dividing by transverse direct division. Mitotic figures could not be seen in them even in iron haematoxylin preparations. There were transitions between these and accumulations of ordinary round lymphocytes more closely packed than usual, and of course, between these and less closely-packed individuals.
The capsule was intensely infiltrated, and cord-like masses of lymphocytes penetrated from it into the body of the organ. Mitoses were readily discoverable in all these accumulations by appropriate staining.

In the less deeply infiltrated parts the liver cells seemed healthy, and in iron preparations showed the intercellular and intracellular bile capillaries with great distinctness. Giant cells were not seen in the liver, nor did the capillary endothelium seem to share in the proliferative process.

The Kidneys showed, mutatis mutandis, precisely the same minute appearances as the liver. Proliferating lymphocytes seem to have actually found their way, here and there, into the tubules. Mitoses were readily found.

Sections of the swollen intestinal lymphoid apparatus showed appearances similar to those already described in the lymphatic glands.

**SUMMARY.**

This case was typical of the acute form of the disease in the following characters:—

1. The youth of the patient (9 years).
2. The duration (9 weeks).
3. The moderate enlargement of spleen and glands.
4. The clinical course, characterised by fever, haemorrhages, and increased uric acid excretion.
5. The predominance in the blood of the large mononuclear cell with hyaline basophile protoplasm. At no time were these less than 95 per cent. of all leucocytes.
6. The complete destruction or suppression of the myeloid element in the marrow, and the consequent total absence of granule-cells from the blood.

The following characters seem to me, from a study of such already recorded cases as are accessible to me, exceptional:—

1. The severe and rapidly progressive anaemia, the reds falling from 2,000,000 to 820,000 within three weeks. The total absence of megaloblasts is highly interesting,
as showing that the production of these elements is no part of the normal regenerative process, but takes place under the influence of some special stimulus which was absent in the case. (There were plenty of normoblasts in the marrow.)

2. The large proportion of leucocytes to erythrocytes. Osler states that it is never as great in the lymphatic as in the myelogenous form of leukaemia, and regards 1 white to 10 reds as quite excessive. In this case the first count yielded 1 white to 8.9 reds, and the ratio at the end was practically 1 to 1.

3. The absolute number of lymphocytes—over 700,000 at the end—was also higher than is usually met with in acute cases. Dominici says in his article in the second volume of the new edition (1902) of Cornil and Ranvier's *Pathological Histology*, p. 674, "le nombre des mononucléaires mis en circulation, ne dépasse guère quarante ou cinquante mille." It is only fair, however, to add that higher numbers than those mentioned by Dominici are given in some recent observations of the acute form of the disease—one by Januskiewicz with 165,000 whites, of which 87 per cent. were large mononuclear, and the others by Kelly with 119,000, 567,000, and 378,000 leucocytes respectively, nearly all being lymphocytes.

4. The great enlargement of the thymus.

*Concluding Remarks.*—1. The essential feature of the morbid state in this case is the abnormal activity of the lymphoid tissues throughout the body and the flooding of the blood and tissues with their products, the lymphocytes.

2. This activity in the recognised seats of lymphoid tissue is accompanied by a rejuvenescence of that tissue in places where only traces of it persist during adult
A Case of Acute Lymphæmia.

life—for example, the marrow of the long bones, the liver and kidneys. In the bone marrow, this revival takes place with such revolutionary energy as to invade and completely suppress the other variety of hæmatopoietic tissue—the myelogenous; hence the profound oligocythaemia, and absence of granule-cells from the blood.

3. The behaviour of the lymphoid tissue in this case strongly resembles that of malignant new-growths, and the resemblance is still further accentuated by the fact that the stimulus, whatever it may be, does not appear to be of bacterial nature (Cf. the negative result of the culture experiment). Like the cells of a malignant tumour, the proliferating lymphoid cells show a tendency to spread in all directions round their focus of origin, whether that focus be a lymphatic gland or a blood-vessel (Cf. the appearances described in the heart).

4. The proliferating cells in this acute case are of distinctly embryonic type, resembling the mother-cells from which both varieties of blood-forming tissue spring, rather than differentiated lymphocytes. This feature still further accentuates the resemblance to malignant tumours in which the malignancy is the more pronounced—the more anaplastic (to use Hansemann's expression)—or undifferentiated are its cellular constituents. On these lines one might compare a case of chronic lymphæmia with typical medium-sized or small lymphocytes to a benign or histoid tumour—the lymphocytes being fully developed—whilst the present acute case would resemble a tumour process of sarcomatous nature affecting the lymphoid tissue throughout the body.

5. As regards the seat of origin of the disease: looking at the remarkable condition of the marrow alone one might be inclined, with Benda, to regard the large
lymphocytic cells, of which it was almost exclusively composed, as primitive marrow cells, his myelogonia, the "indifferent lymphoid cells" of A. Wolff, and to regard the disease as one primarily affecting the bone marrow. Morphology alone can hardly help us here, for it is not possible to distinguish undifferentiated embryonic cells of one kind of tissue (myeloid) from those of another kind (lymphoid). The objection to Benda's view is that it leaves the intensely active participation of the lymph glands unexplained. It seems more natural to regard acute leukemia as a primary disease of lymphoid tissue wherever situated—a disease with distinct affinities to the malignant tumours.

REFERENCES.


Professor White said that during the past year he had seen a case of the so-called spleno-medullary leukemia in which there was apparently a direct connection between an injury and the disease. The patient fell down the hold of a ship, and hurt himself about the abdomen, but was able to work on for a fortnight, after which he went into hospital, and died in a few weeks of leukemia. Another point in Professor McWeeney's case was the fact that the patient died so quickly of pneumonia. In one of the cases of leukemia (common variety) he had seen, the patient, a woman, left hospital to go home, but missed her train and had to come back; she almost immediately developed a sharp attack of erysipelas, and died in twenty or thirty hours, showing the little resistance these patients have against infectious diseases. With regard to Hodgkin's disease, he did not think it was typical to find giant cells, and believed that when giant cells were found we were probably dealing with some chronic infection, possibly
tuberculous, instead of Hodgkin's disease. With regard to the acute case the point which interested him most was the section which showed what was probably mitosis. He was convinced of the fact that mitoses did occur in the circulating blood under certain conditions.

Prof. O'Sullivan suggested the possibility that the giant-cells in the liver capillaries might have been marrow cells carried there.

Dr. Craig said that Professor McWeeney's cases rather upset the ordinary clinical teaching; for instance, he had been accustomed to teach that the increase of leucocytes was not so great in lymphatic as in spleno-medullary leukæmia, but Professor McWeeney had said that in the acute case, at least, the red and white corpuscles were almost equal in number. Another point was that myelocytes were never present in lymphatic leukæmia. With regard to the case of acute leukemia, he said it was of special interest, because occasionally this disease occurs among children, and when the enlarged glands appear they are supposed to have acute general tuberculosis.

Dr. Earl agreed with Dr. McWeeney as to the presence of giant cells in Hodgkin's disease, and he was certain that in the two or three cases of real Hodgkin's disease which he had examined he had found glands which were not those of tuberculosis.

Dr. McWeeney, in replying, said that Professor White's observation as to the traumatic origin of a case of the spleno-medullary variety was very interesting, especially the short duration. The case would appear to have been acute myelæmia, which is very rare, whereas lymphæmia is sometimes acute; but he was not certain that any acute spleno-medullary cases had been recorded. With regard to his statement about the presence of giant cells in Hodgkin's disease, he based it on a report of the pathologist of the Ayer Pathological Laboratory, who had examined fifteen cases of the disease, and had met giant cells which it was easily seen were not tuberculous. They arose from some irritation of the large endothelial plates in lymphatic glands. If the same cause produced leukæmia which produced Hodgkin's disease giant cells might be expected to occur in the glands, but they do not. The explanation sometimes given is that in Hodgkin's disease, owing to some peculiarity of the capsule of the gland, the greatly increased lymphoid cells in it and in the
sinus are so pressed on that they cannot get out into the blood; whereas if the same stimulus attacks a gland with a soft capsule the new lymphocytes do get out, causing leukæmia. In reply to Dr. Craig, he had striven to deal with the pathological and clinical aspects. There could be no doubt that there are many cases of lymphatic leukæmia which give very large counts.
NOTE ON A LARGE FIBROMYOMA FROM THE RECTUM.

By H. C. EARL, M.D., F.R.C.P.I.;
Pathologist to the Richmond, Whitworth, and Hardwicke Hospitals.

[Read in the Section of Pathology, January 6, 1905.]

This tumour was removed from the rectum of a woman during labour by Dr. Golding, of Headford, Co. Galway, who has kindly supplied me with notes of the case, of which I give the main facts:—

The patient was aged about forty years, and had previously had two children. In her first labour version had to be performed after failure of the forceps, and in the second delivery was by forceps. When Dr. Golding was called to her in August last, on vaginal examination he felt what he believed to be a mass of feces in the rectum, and gave an enema. As labour was not advanced he left the patient, and returned about five hours afterwards. He then found the os dilated, the head at the brim of pelvis, and labour severe. The mass he had felt in the rectum was still there, and appeared to prevent the head advancing. He gave chloroform, and examined the rectum, and found the mass was a tumour held in position by a membranous attachment a good finger length from the anus. The forceps was applied, and as the head neared the perineum the tumour was delivered per rectum, and the membranous attachment clamped, ligatured, and divided. The patient had never noticed anything unusual about herself. She has recovered perfectly, and has no rectal or uterine trouble.

The tumour is somewhat kidney-shaped, and its surface is irregular. There are some points of calcification close to the surface. There is a membrane attached to one surface by a long attachment. Microscopic examination showed this
membrane to be composed of loose fibrous tissue, with a good deal of adipose tissue. The tumour itself is composed of smooth muscular fibres, with a varying amount of fibrous tissue in the different parts examined.

The tumour measures nearly three and a half inches in its longest diameter, its shortest diameters being two and a quarter inches and two inches respectively.
NOTE ON A CASE OF CIRRHOSIS OF LIVER.

By GEORGE PEACOCKE, M.D., F.R.C.P.I.,
Assistant Physician, Adelaide Hospital.

[Read in the Section of Pathology, February 17, 1905.]

Mrs. C., aged thirty, was admitted to the Adelaide Hospital on the 24th August, 1904, under my care. Sixteen months previously she had been confined, and for some time before the birth of her baby her legs and hands were swollen. These swellings quite cleared up after her confinement, and she remained in good health up to June of last year, when she noticed she was getting thin and was very easily tired. No further symptoms developed until early in August, when she observed her abdomen was becoming larger. She consulted Dr. Brew, of Enniskerry, who, recognising that ascites was the cause of her abdominal enlargement, advised her to come into hospital. I may say briefly that beyond the discomfort caused by the ascites she had no symptoms. She felt well; her appetite was good and bowels regular. She had no vomiting and no history of any hæmatemesis. Her urine was sp. gr. 1030, and contained no albumen, sugar or bile. It was fairly high-coloured. Her heart was healthy. There were some distended venules on the cheek, but nothing else to give any clue as to the cause of the ascites. On account of the ascites it was impossible to make any detailed examination of the abdomen, and so I asked Mr. Gordon to remove the fluid by a laparotomy. No ovarian disease or tubercular peritonitis was discovered, but the liver was found to be cirrhotic. The removal of the fluid caused a considerable change in her symptoms. She became rather seriously ill, lost all appetite, her pulse was exceedingly small and compressible; she became jaundiced, and bile appeared in the urine. After remaining in this condition for a few days she gradually recovered. The same symptoms, only rather more intense, developed after her abdomen was a second time tapped, but this time only with a trocar and canula, and though I thought on one day she was not going to survive, she again
rallied. On the fourth occasion that the fluid was removed Mr. Gordon again performed a laparotomy, and grafted the omentum on the peritoneum, but she never really rallied from the operation, and died on the 25th November. A limited post-mortem was made and the liver removed. It is, as you can see, a typical example of atrophic cirrhosis.

The great interest in this case is the presence of a marked multilobular cirrhosis of the liver in a young woman, who was a life-long teetotaller. No history of syphilis was obtained, nor do I think there was the slightest reason to suspect any such taint.

On looking up the article on cirrhosis of the liver in Clifford Allbutt’s System of Medicine, I find it stated “that the excessive use of alcohol is by far the most common cause of cirrhosis of the liver in all countries; by its side all the other causes together are insignificant”; and again, “in conclusion, it may be safely stated that in nearly all the patients who present definite signs of cirrhosis the disease has arisen as a consequence of alcoholic excess, syphilis, or chronic malarial infection.”

Dr. H. C. Earl said that the connection between alcoholic excess and cirrhosis of the liver had been assumed, and partly proved, but he thought that too much importance was placed on the alcoholic excess. In quite half the cases he had seen there was certainly none. It was hard to say whether there was syphilis or not, but at any rate it was impossible to get a history of it. He had seen cases of typical cirrhosis in very young people—for example, in a child of seven and in a child of eleven. It was quite obvious that there was something else besides alcoholic excess, because one found patients in hospital who drank all they could get, and had no cirrhosis, and it was by no means a common affection among them.

Dr. Matson asked if there was any tubercular history, or whether any tubercular foci existed in any part of the body? Also, was there
any enlargement of the spleen, and how soon after the draining did the patient die? In view of acute yellow atrophy could the pregnancy throw any light on the causation? As regards alcohol producing cirrhosis, he wondered if the kind of alcohol made any difference, as in England it was usually whisky or brandy that was drunk, and in Ireland stout.

Professor O'Sullivan said that his own experience coincided with that of the President. One made post-mortem examinations on numbers of drunkards in this city, and fatty infiltration of the liver was usually found, while cirrhosis of the liver was the exception; and he was inclined to agree with Dr. Matson that it might be due to the kind of spirit consumed. At the same time in experimental cirrhosis the disposition of the animal appeared to exercise a certain amount of influence on the result—that is, some animals of the same species would get cirrhosis and others would not, as if there was a tendency in some individuals to the formation of increased connective tissue. One ought not to forget the interesting work that was being done in Montreal by Professor Adami on the effect of organisms of low virulence acting for a long time on the organs. He remembered that there was an announcement made that cirrhosis of the liver was due to an extremely minute coccus, but this was subsequently corrected, as it was found that it was a form taken by the colon bacillus. Adami and his pupils believed that many of the chronic interstitial changes in the body are due to micro-organisms of low virulence, no matter what the specific nature is.

Colonel McNeece said that during the last 15 years he had made many post-mortem examinations, but had never seen a case of cirrhosis of the liver. The cases which did occur at Netley were in men who had served 15 years in India. Formerly each man was served out with a dram of rum in the morning (and many men besides bought more drink), but that had been done away with for the last 12 years or so, and now they saw nothing of the hob-nailed liver in the service, so that he thought it was due to a sort of crude alcohol manufactured in India from beet-root. He had read the accounts of the investigations in Canada, and it might have something to do with the case before them—of cirrhosis occurring in a strictly temperate person.

Dr. Kirkpatrick asked if Dr. Peacocke could give them any explanation of the occurrence of ascites in these cases of cirrhosis.
of the liver? The teaching they had had was that the fibrous tissue pressed on the portal veins and caused a very chronic form of ascites. Now, it seemed that many cases ran almost their whole course without any ascites appearing; also that in those cases where it did appear it was a terminal symptom. A few years ago some cases were collected together to show the results of Talma's operation, and these seemed to show that it gave bad results in cases of cirrhosis of the liver, but in cases due to perihepatitis it had given brilliant results, so that he thought the old explanation—that the ascites was due to mechanical obstruction—could no longer be supported, and he believed that it was a terminal symptom. Whether the cirrhosis started primarily as a result of change in the liver cells or as a result of an irritant reaching the connective tissue in the portal systems through the blood-vessels, it did not seem to him that that could afford a full explanation of the symptoms. A patient would go on for a considerable time without any symptoms, then, more or less suddenly, he began to lose flesh and present symptoms of loss of vitality; ascites supervened, and death resulted in a short time. If the symptoms were due to a crowding out of the liver cells by connective tissue, one would expect the process to be more gradual; and if this crowding out of the liver cells was to be a full explanation, then surely the ascites ought to be an early symptom and last for a long time; but in many cases we found that it occurred as a terminal symptom. Could Dr. Peacocke give any explanation of the mode of onset?

Dr. Peacocke, replying, said that anyone who had experience of post-mortem examinations knew that cirrhosis of the liver occurred in cases without any alcoholic history, and vice versa. In this case he had got no tubercular history. The post-mortem was limited, but they saw no tubercular foci. The spleen was, perhaps, slightly enlarged. The ascites was first noticed early in August. The patient came into hospital on the 24th of August, and died on the 25th of November, and it was a day or two after the 4th tapping that she died.
A CASE OF INFECTIVE ENDOCARDITIS.

By GEORGE PEACOCKE, M.D., F.R.C.P.I.;
Assistant Physician, Adelaide Hospital.

[Read in the Section of Pathology, February 17, 1905.]

On the 30th July, 1904, a man was admitted to the Adelaide Hospital under my care. He was forty-eight years of age, had been a soldier, and served in India, and was now employed at some work in connection with one of the military departments. He appears to have been a healthy man, except that he had suffered considerably from malaria whilst abroad, and had had some attacks since he returned home about fifteen years ago. His bowels were always rather constipated, requiring medicine before they acted. Some few weeks before admission he appears to have first shown any signs of ill-health. He suffered from neuralgia in various parts of the body, and an attack of severe pain in the stomach on the 25th July induced him to take a large dose of castor oil. Diarrhoea resulted from this, and as it continued he came to hospital.

On examination I made out the following points:—There was a very faint mitral systolic murmur, but the heart was not enlarged. The liver could be felt below the costal arch on the right side. The urine contained a trace of albumen, and during the week he remained in hospital there was slight irregular pyrexia, the temperature on one occasion reaching 99.8°. The diarrhoea ceased the day after his admission, and he left hospital on the 5th August. After returning home he was able to resume his work for a week, but then a return of neuralgic pains in his head, chest, but especially in his legs, compelled him to give up. I again admitted him on the 30th August, as he was evidently suffering acute pain, especially in the calves of both legs. His condition was otherwise unaltered from the time I had last seen him. He remained in hospital for three weeks, and during this time he had again irregular fever; on one occasion the temperature rose to 103.8°, but fell to normal the following morning. He
had no fever during the third week of his stay in hospital on this occasion. I thought that possibly the pains and fever might be accounted for by an attack of neuritis.

After leaving hospital he remained at home for a month, and then went back to his work. He did not, however, seem to be in good health, was very cranky and short-tempered, complained of the pains in his head and chest, but his appetite continued as good as usual. On the 26th November he returned home about 12 o'clock in the day, and his wife noticed he seemed queer; he was unsteady on his feet, and she could hardly make out what he was saying. He spoke very little, and kept muttering—"I'm done for." He remained in this condition, so I admitted him to hospital for the third time on the 28th Nov. His condition was as follows:—He was very thin and sallow-looking, and had evidently lost considerable flesh during the past two months. He complained of pains in his head and chest. He had the same type of irregular pyrexia. The mitral systolic murmur was louder than when I last examined him; there was no other cardiac murmur. His pulse was about 100, and the arterial walls were slightly thickened. The liver was slightly enlarged, the bowels rather constipated, and the urine pale in colour, and contained a trace of albumen. He had some cough, and a few rhonchi could be heard through his chest. There was no oedema. He was aphasic—the form of aphasia being word deafness. He could speak; sometimes what he said was fairly intelligible, but as a rule had no reference to any question he was asked. At other times he muttered an unintelligible jargon. He was quite unable to write correctly from dictation, but could read fairly well. He would put out his tongue when asked to do so, and apparently understood some things that were said to him. He never spoke unless spoken to, and remained all day in a semi-stupid state. He slept well, and took his food up to a few days before death. Mr. Swanzy kindly examined his eyes, and found the fundus normal.

A blood examination showed haemoglobin, 65 per cent.; red blood corpuscles, 3,150,000; white blood corpuscles, 9,200. Professor Scott kindly examined his blood for micro-organisms, but got no growth.

He gradually became more and more stupid, passed under him, refused food, and died on the 8th January.
A Case of Infective Endocarditis.

Post-mortem Examination.

Heart.—Numerous vegetations on tricuspid, mitral and aortic valves—the vegetations being not confined to the valves, but also found in left auricle and ventricle. There were some small superficial ulcers on the endocardium.

Lungs.—A few haemorrhagic infarcts.

Spleen.—A fairly large infarction.

Kidneys.—A few small infarctions.

Brain.—Several infarcts in brain on right side; in frontal, temporo-sphenoidal and occipital lobes.
ON A CASE OF PRIMARY ACTINOMYCOSIS OF THE RIGHT KIDNEY WITH EMBOLICAL CEREBRAL ABSCESS; WITH A SHORT ACCOUNT OF SOME OTHER CASES OF THE DISEASE BACTERIOLOGICALLY DIAGNOSED.

By H. C, EARL, M.D., Univ. Dub., F.R.C.P.I.; Pathologist to the Richmond, Whitworth and Hardwicke Hospitals; Pathologist to the Royal Victoria Eye and Ear Hospital.

[Read in the Section of Pathology, March 17, 1905.]

Actinomycosis in the human being has been a well-recognised condition for 25 years, but until 1900 no case had been communicated to this Academy, and, so far as I know, no case had been clinically or bacteriologically diagnosed in Dublin. In 1900 Dr. Littledale communicated to this Section a case which had been under the care of Dr. Bennett, who had made the clinical diagnosis (the affection in this case was in the neck and lungs), but the bacteriological diagnosis had not been established during life. Since Dr. Littledale made this communication I have made the bacteriological diagnosis of several cases of this disease, and have thought it worth while to lay before the Academy a short account of these.

In February, 1901, I received a specimen of pus from Dr. Orr, of Omagh, who gave the following history of the case from which it came:—

About the previous September a tumour began to grow in the anterior abdominal wall above the umbilicus. It continued to grow, and was then removed, or partially removed, and sent to a histologist, who pronounced it a fibro-fatty tumour. It subsequently softened and dis-
Primary Actinomycosis of the Right Kidney.

charged, and some of the discharge was sent to me. The discharge was sero-purulent in character, slightly blood-stained, and contained minute yellow grains which were readily recognised in fresh and stained preparation to consist of the actinomycetes fungus. The clubs in this case were very small, and were only to be found in a few of the grains. About a year afterwards I learned from a relative of the patient that he had developed actinomyces of his lungs, of which he subsequently died.

In May, 1901, a man, aged about thirty, was admitted into the Richmond Hospital under Sir Thomas Myles. He had a brawny purplish swelling of his neck, with some sinuses discharging. Sir Thomas made the diagnosis of actinomycosis, and bacteriological examination confirmed this. Clubs were present in some of the grains, but were small. Sometimes the discharge contained no trace of the organism, and sometimes no yellow grains were seen, but the branching threads were present in films from the discharge. With the small amount of material that could be obtained, tubes of glycerine agar were inoculated, but remained sterile. The patient was rapidly cured.

In June, 1901, Dr. Fannin brought me material from a patient of his, of which he has kindly supplied me with notes. The following are the main features of the case:—

The patient was a lady, aged forty-five years; had always lived in Dublin. A swelling, beginning like a boil, occurred in the anterior abdominal wall. It was seen by Dr. Fannin one month after the patient first noticed it, and was then a hard, flattened tumour of roughly circular shape, and about five inches in diameter, situated to the left of, and below, the umbilicus. The swelling contained pus, and a large quantity of greenish, yellow pus, with a fetid smell, was evacuated. Healing of the cavity was slow, and even when completed, a fibrous nodule, the size of a small walnut, remained, in which occasionally small sinuses opened. Subsequently the patient suffered from pains and oedema of the right leg, and
later on an abscess pointed on the right side of the abdomen, above the crest of the ilium. It was opened, and a large quantity of pus evacuated. The abscess showed no tendency to heal, and was scraped. Later on the patient suffered from severe diarrhoea, and the motions contained pus and yellow grains. She shortly afterwards died.

The yellow grains in this case were generally very large—much larger than in any other case I have had material from. Clubs were not always present, and were only occasionally fairly distinct. Dr. Fannin kindly brought me a good supply of material, and I inoculated numerous tubes of different culture media, and kept tubes both under aerobic and anaerobic conditions, but I did not obtain a single culture, though only a very few tubes showed a growth of accidental impurities.

Dr. Fannin also brought me a small portion of affected skin, of which I show a section. It will be seen that the liquefaction of tissue is in it very considerable, and the clubs are not to be made out in the colonies it contains. It forms a marked contrast in these respects to the sections of renal actinomycosis, to which I shall presently allude.

In October, 1901, I received a piece of cotton wool which was saturated with the discharge of a sinus in the neck. It was sent to me from the County Clare, and I was informed that the sinus had apparently healed several times, but always opened up again.

On moistening the wool the yellow grains were readily seen, and the diagnosis of actinomycosis established.

Since October, 1901, I have made the bacteriological diagnosis of four other cases of the disease, in all of which the neck only was affected. One of these was in the Drumcondra Hospital last year, and Dr. Fannin, who made the diagnosis, brought me some material in which the actinomycosis was readily found. I have also seen
two cases of affection of the neck which were probably actinomycosis, but it was not possible when I saw them to obtain any material for bacteriological examination. Besides these cases that I have mentioned, I have heard of, I think, three other cases, one of which was under the care of Dr. Wallace Beatty, and it appears to me very probable that the disease is, though of course not very common, certainly more common in this country than has been generally thought to be the case.

The case which I shall now describe is the first case of actinomycosis which I have met with post mortem. In it the diagnosis was not made, nor, as the description of the case will show, could it have been made, during life.

J. G., a porter aged forty-nine years, was admitted into the Whitworth Hospital on December 28th, 1904, under the care of Dr. Coleman. He complained of pain in his left side, and in his precardial region, and of breathlessness on slight exertion. His family history was good, and his previous history. He stated that for a month before his admission he had been troubled with general weakness of his limbs and body. He had also had a cough, with a small amount of expectoration. He had lost weight considerably during this month. He had no night sweats. On admission he was very feeble and much emaciated. The heart sounds were very weak. There was flattening and diminished movement over the left apex, and slight dulness over the left lower lobe behind and over the left apex, and crepitation was heard over both these regions. Expiration was prolonged. The sputum contained tubercle bacilli. The urine contained a trace of albumen, and showed a slight deposit of mucus on standing, but the albumen was not found a week after his admission. On the 26th January, 1905, he had a series of epileptiform fits (about 35 in all) which came on in rapid succession. Each fit lasted 60 to 80 seconds, and intervals of 20 to 30 seconds intervened between the fits, with the exception of the last two, the intervals in these being 8½ and 10 minutes. These fits generally involved the left side of the body only, but
the last two involved both sides. The pupils were dilated, and the patient was quite unconscious. He recovered consciousness about three hours afterwards, and was able to answer questions. His left conjunctival reflex was then found to be absent, and his left arm and leg were completely paralysed. The paralysed limbs subsequently recovered to a considerable extent, but some paresis remained up to February the 5th, when patient died. From the physical signs and the presence of tubercle bacilli in his sputum, the diagnosis of phthisis was made, and the convulsions and subsequent paralysis were thought to be due probably to tubercular disease in his brain, or meninges.

The following are the notes taken at the post-mortem examination:

The body is much emaciated. The pericardium contains about 6 ounces of clear fluid. The heart is small, the sub-pericardial fat is gelatinous, and the muscular tissue pale. The valves are normal. There is well-marked atheroma, with some calcification of the coronary arteries and their branches. There is a chronic partial heart aneurysm in the left ventricle on its posterior surface, and just below the coronary sinus. The aorta is dilated, and shows a few small patches of atheroma. The right lung is adherent by firm adhesions to the chest wall and diaphragm. The lungs are emphysematous and oedematous. The larger bronchi contain a thin muco-pus; there is some hypostatic congestion at the base of left lung. In the upper lobe of each lung there are several separate masses of caseating broncho-pneumonia, each mass being about the size of a walnut. These masses are situated near the apices of the lungs, and in the left apex one had formed a small cavity. There were a few grey tubercles in the lung tissue around and between the broncho-pneumonic patches. The bronchial glands are enlarged and caseous. The spleen is adherent to the diaphragm, but otherwise is normal. The liver, stomach, intestines, suprarenals, and left kidney are healthy. The bladder contains a small quantity of urine, which is quite clear. The lower third of the right kidney is occupied by a greyish, firm mass, in which are seen a number of small cavities, generally lying in groups, and containing a thick yellow pus. The kidney is slightly
adherent to the quadratus lumborum muscle, due to the extension of the new growth into this muscle. The arteries at the base of the brain are very atheromatous. The lateral ventricles are dilated and filled with a turbid fluid. The third and fourth ventricles, and the iter, are also dilated. The fourth ventricle contains a fibrinous mass. There is a small abscess, about \( \frac{3}{4} \) inch in diameter, below and to the outer side of the posterior horn of the right lateral ventricle. It has opened into the ventricle by a wide opening; a thick, creamy pus, almost colourless, adhered to the abscess cavity, and filled the descending and posterior horns of the ventricle. The tissue surrounding the descending cornu is infiltrated with pus, and there is a small patch of white softening near the tip of the temperosphenoidal lobe.

In the pus from the ventricle no yellow grains and no clubs were found, but threads with true branching, as well as bacillary and coccal forms, were readily found when films were stained by Gram's method. In the pus from the kidney the yellow grains were seen, but they were few and small. A few cultures were attempted and failed, as the material was not pure.

The cavities seen in the kidney are filled with pus cells, red corpuscles, and fibrinous shreds, and contain actinomycoses colonies. The clubs are, in many of the colonies, very large. The cavities are lined with a granulation tissue which is partly necrotic, and outside this is renal substance greatly altered by the growth of a rather dense fibrous connective tissue containing masses of small round cells. The renal tubes that remained contained shed epithelial cells and leucocytes, but the fungus was not found in any of them, nor was it found in a small vein containing pus, which was seen in sections.

Sections of several portions of the wall of the brain abscess were made, but no colonies of the fungus were found embedded in the tissues, though in the pus adher-
By Dr. H. C. Earl.

ing to the wall branching threads were easily found, and in one case showed a somewhat radial arrangement.

Except in the brain and kidney no actinomycotic condition was found in the body, and though a careful examination was made of the body no point of entrance of the fungus was found. So the case must be considered as cryptogenic infection, which occurs, according to Schlegel, in 7 per cent. of the cases of actinomycosis. Of the two actinomycotic foci found, that in the kidney, on account of its much larger size and its great formation of fibrous tissue, must be considered the primary, and that in the brain the secondary condition. The amount of fibrous tissue formed in the kidney was much greater than it generally is in human actinomycosis, and the condition resembled very much the actinomycotic tumours found in cattle. The clubs were much larger than any I had previously seen in any of the cases I have alluded to, and their large size is no doubt due to the unusual formation of fibrous tissue. Embolic abscess in the brain appears to be fairly common in actinomycosis. It occurred in three out of fifteen cases recorded by Godlee. Primary actinomycosis of the kidney appears to be very uncommon. I have only been able to find one record of a case, that by J. Israel.

Sir Thornley Stoker inquired whether the rarity of cultures was due to the want of a suitable medium.

Dr. Craig was forced, from the number of cases seen by Dr. Earl, to assume that cases of human actinomycosis were commoner than he hitherto imagined.

Mr. Gunn related particulars of cases which he had seen, and also mentioned a case where, owing to the discovery of mycelial threads in the discharge, a mistaken diagnosis of actinomycosis of the rectum was given. Later on it proved to be a carcinoma.
Dr. Kirkpatrick mentioned a somewhat similar case occurring in the lip, where a diagnosis of actinomycosis was at first made, but subsequently this also was shown to be cancer.

Dr. Earl, in reply, stated that many of the peculiar yellow grains were made up of dead organisms, and so cultivations were not easy to obtain. He also considered the increase in the number of cases reported was due to better observation.
MELANO-SARCOMA OF THE CONJUNCTIVA.

By ARTHUR H. BENSON, M.B. Univ. Dub., F.R.C.S.,
Ophthalmic Surgeon, Royal City of Dublin Hospital,
Surgeon, Royal Victoria Eye and Ear Hospital;

AND

HERBERT C. MOONEY, M.B. R.U.I., F.R.C.S.,
Ophthalmic Surgeon to the Children's Hospital, Temple Street,
Assistant Surgeon and Curator, Royal Victoria Eye and Ear Hospital.

[Read in the Section of Pathology, March 17, 1905.]

A. G., a married woman, of slight build, aged forty, was admitted to the Royal Victoria Eye and Ear Hospital, Dublin, on 4th February, 1905. Protruding between the eyelids of the left eye was a jet black tumour, about the size of a large split pea, partially flattened and lobulated, and adherent to the globe at the corneo-scleral junction at the temporal side. The attachment to the globe was rather small, and only by the edge of the tumour. The tumour could be freely moved about, and the conjunctiva with it, showing that it had no deep attachment to the sclerotic. There were a few large dark vessels on the conjunctiva, running to and from the tumour, otherwise the conjunctiva was normal in appearance. When she shut the eye in the ordinary way, the growth protruded out through the lids, but by a slight manoeuvre she could, with her finger, slip the upper eyelid over the tumour, and this she did when she wanted to sleep. There was no soreness about the eye, only a small central corneal ulcer, produced, no doubt, by the inability to close the eye properly in winking, &c., through the day. The tumour was abscised. A Grady's forceps was closed under it, and it was so raised off the sclerotic that with a scissors it was cleanly separated from the sclerotic. The surface where it lay on the sclerotic was cauterised as a precaution, and the conjunctiva sutured over the place. There was no intra-ocular complication, nor was the iris involved in any way.
"Sections of the tumour showed it to be composed of two parts. The larger was typical small spindle-celled sarcoma tissue, with abundant pigmentation, which was most marked near the periphery, and again round the smaller portion or excentrically placed non-pigmented area. This non-pigmented area, or core, which was placed towards the base of the tumour, was made up of large swollen-looking epithelioid cells, loosely arranged with plentiful intercellular substance. Many of these large cells had one or two vacuoles. In none of the sections was this area in touch with the surface of the tumour, and its limits were sharply defined. In bleached sections the structure of the pigmented portion was more easily determined. Mixed with the tumour cells were large epithelioid cells, some having vacuoles, and others deeply staining granules. These large cells were much more numerous where the pigment was most plentiful. Many small empty dilated spaces were scattered throughout the growth, which had very few blood vessels. The conjunctiva covering the tumour was, for the most part, thickened, and at places cornified.

"H. C. Mooney."
This year is a very proper time to take note of our knowledge of fractures of the costal cartilages, for this is the centenary of these injuries. In 1805 Lobstein published the first exact observation of them at Strasbourg. I have a special right to refer to the details of one of these injuries—fracture of the first costal cartilage—for I find that I am credited by a writer in Guy's Hospital Reports with being the first to describe the injury. He says: "I can only find one instance of fracture of the first costal cartilage published, and, curiously enough, I think that there is no doubt that it was not an example of fracture of this cartilage, but an instance of dislocation of the joint which I have shown is developed in the ossified first costal cartilage, and which was not recognised at that time. The case I refer to is one published by Dr. Bennett in the Dublin Quarterly Review, March, 1876." Apart from the inaccuracy of the reference, for as we all know there is no such Review, I feel that my claim for priority in this matter is rudely shaken in the very first detail, for the next sentence assumes that I could not recognise a recent fracture of the cartilage of the ribs on the table of the post-mortem room.

The author of this criticism quotes some one to this effect: "Fracture of the first rib does not occur because it is sheltered by the clavicle." And sets up the opposite: "Curiously enough, the reverse of the teaching of the time is true, for the
very reason given—namely, 'the first rib is frequently broken because it is sheltered by the clavicle.'" These passages evidently contemplate the fracture of the first rib, or of its cartilage, by direct injury—or the more ordinary forms of indirect injury. I would present to the section a few very brief notes on the subject of fractures of the first costal cartilage as the accidents which have come under my observation during a very few months illustrate them. I will, if anyone likes, accept the view that a false joint, or rather a new joint, may be formed in the cartilage of the first rib and present difficulties of diagnosis when met on the dead-house table or in the formal progress of dissection. This concession may be made to the author I have quoted in regard to the class of the laborious hodman and the like. Of the life histories of these examples our knowledge is very uncertain. The facts of the cases I would now record admit of no doubt, even though they have been observed by so incompetent an authority as myself; and, in passing, I would direct attention to the fact that more of my subjects were females than males—not dock labourers or hodmen, or the like. I have in hands the pathological details of over five and twenty actual specimens of fractures of the costal cartilages in general, including the first, many of which I have published from time to time in the old Pathological Transactions or in the Records of this Academy. To-day I bring before the Section six specimens of fractures of the first cartilage, and I would record an example observed in the living which presents special interest from the mode of its cause.

To take the last first.

I have already recorded the observation of the rare occurrence of fracture of the costal cartilage, from muscular action (Dublin Journal of Medical Science, Vol. LXI., page 207, 1876).
This winter (14 January) I had the good fortune to meet with a perfect example of this rare accident, confined to the clavicle and first rib on the left side:

M. B., aged fifty-seven years, a carter, was loading wheat from his cart into some canal craft. In doing this he had to place on the scales each sack with its mouth open full of the regular twenty stone of grain which goes to make a sack. In delivering the sack after it passed the scales, M. B. caught the corners of the open mouth of the sack and lifted it on to his right shoulder. On doing this he lost his balance and fell backwards with the sack to the stage on which he should step down—some three feet. In the effort he felt his collar bone and rib yield without his striking on his chest. He had a fracture of the clavicle between the conoid and trapezoid ligaments, and of the cartilage of the first rib on left side. He left hospital on 7th February fairly recovered. In giving the account of his accident he was very clear and precise, being certain that no blow struck his collar bone or chest wall on the left, and that he did not strike these parts in his fall.

My next case is as follows:

On 30th January last at 2 p.m. a labourer, aged fifty years, was admitted to Sir Patrick Dun's Hospital. He had been crushed by a piece of timber weighing over a ton. He and others were engaged in shifting this when it slipped and crushed his chest. He lived until 12 30 a.m. in the night, and died with failure of breathing and rapidly developing surgical emphysema. Four or five ribs on each side were broken, and the pleurae and lungs were much torn; the liver and spleen were also lacerated.

I now place before the meeting the recent fractures of the first costal cartilages taken from his body, which could not by the merest novice in pathology be mistaken for new or false joints.

Next, I present the sternum, and portions of the ribs attached, which were removed from the body of an aged woman who was killed on, or very close to, Baggot Street.
Bridge by being run over by a motor car one day this winter. Here are the recent fractures of the two first cartilages, and no doubt can be entertained of their characters, nor any suggestion that the specimens are examples of new or false joints produced by a specially laboriously spent life. The patient was an old and feeble woman, and was killed on the spot where she fell.

Her first costal cartilages have been broken after the constant pattern of such lesions, and bear all the traces seen in such recent injuries. With them were many other rib fractures and a fracture of the sternum; but at this time we are not concerned with these. I want only to establish before the Academy the verity of my specimens and of my records of them.

3449 (1892). Front of the thorax of an old woman who was killed last week at the gas works. "On the left side the cartilage of the first rib is broken transversely about its middle; on right side the ribs are broken from 1–10."—Verdict, not guilty—Chief Baron, &c.

129. Sternum and ribs; fractures of first ribs at their necks, and of the cartilages. Sternum separated at the joint of the manubrium and gladiolus.

1639. Dislocation of clavicle backwards, with fracture of the first costal cartilage on the left side.—Trans. Path., 1881.

2544. Ununited fracture of the first costal cartilage at the junction of bone and cartilage; the convex imbedded end of the cartilage alone remains attached to the bone. (See Klopsch. Dissertatio inauguralis.) This specimen was given to me by Mr. ——, who found it in the dissection of the neck of a female subject who had fractures of the lower ribs, also the right side. No life history.
Man killed loading timber on North Wall. Recent fracture of both first ribs at cartilages.
Old woman killed at Baggot Street Bridge. Both first cartilages fractured.
ABSTRACTS.

SECTION OF PATHOLOGY.

Friday, October 28, 1904.

The President in the Chair.

Dr. McWeeney read a paper on "Hæmatological Observations on Lymphatic Leukæmia" (see page 296, ante).

Aortic Aneurysm Perforating the Oesophagus.

Dr. Matson showed a specimen of the above taken from a woman, aged fifty. History of alcoholism, but none of syphilis. Patient had been subject to attacks of rheumatic fever. Two years ago she had an attack of bronchitis, in which she expectorated a good deal of blood.

Dr. Travers Smith asked did the patient vomit blood?

Dr. Matson said, in reply, his patient vomited a considerable amount of blood, and there was a good deal of blood in her stomach.

Aortic Aneurysm Rupturing into Pleura.

Dr. Gillman Moorhead exhibited a case of aneurysm of the aortic arch, which had ruptured into the pleura. The specimen was obtained from a man, aged thirty-five, by occupation a sailor, and who had contracted syphilis ten years previously. The only symptom complained of was that of cough. Rupture occurred suddenly during sleep after the patient had been under treatment for four days. Almost the entire wall of the sac was necrotic, brittle and very thin, and only a small amount of soft clot was present. One hundred ounces of blood were found in the left pleural cavity. The curious point about the case was that rupture had not occurred while the patient was at work, as he had worked to within a week of his death.

Friday, November 25, 1904.

The President in the Chair.

Dr. W. J. Thompson read a paper on "A Case of Acute Yellow Atrophy of the Liver" (see page 307, ante).
Abstracts.

Glanders.

The Secretary (for Dr. E. F. Stephenson) read notes of a fatal case of "Glanders in a Man" (see page 313, ante).

Mr. Story and the President exhibited a glioma of the retina with microscopic sections.

Glioma of the Retina.

Dr. Mooney exhibited a specimen, which was a right eye removed from a female child, aged three, for glioma retina. Four months before admission to the Children's Hospital, Temple-street, Dublin, the eye squinted, and for four weeks the pupil had been white and the eye blind. The eye showed no irritation, nor was there pain, although the tension equalled + 2. No proptosis. Microscopically, the tumour showed the usual appearances, much of it being necrotic and fluid, with spots of hyaline degeneration. The optic nerve was wholly invaded by gliomatous cells up to the point of section, only the fibrous trabeculae remaining.

Professor McWeeney said he never saw a prettier example of infiltration of the optic nerve. The resemblance between the cells of the tumour and the nuclear cells of the retina was very striking. He agreed as to the rarity of finding tubes lined with epithelium. These tumours, when not necrotic, were formed of small, round nuclei, broken up here and there by patches of necrosis. In Dr. Mooney's case patches of hyaline degeneration could be seen. It was not so well marked in Dr. Earl's case.

Professor O'Sullivan thought the resemblance to angiosarcoma was due to the fact that the cells in the neighbourhood of the vessels were well nourished, while those away from them were not, and therefore became necrotic. This should be borne in mind in examining all cases of angio-sarcoma. The cells thus seemed to exist as tubes round the vessels.

Dr. Mooney, in replying, said he had omitted to mention that there were some calcareous spots in his specimen.

The President, in replying, said he agreed with Professor O'Sullivan as to necrosis occurring in those cells which were far away from the vessels.

Dr. McWeeney communicated further Haematological Observations on Lymphatic Leukæmia (see page 315, ante).
Section of Pathology.

Friday, January 6, 1905.

The President in the Chair.

Anaemic Infarction of Liver.

Professor O'Sullivan, for Dr. Roy Dobbin, showed specimens from a case of extensive anaemic infarction of the liver. The case was one of puerperal eclampsia, with jaundice. A large gall-stone was impacted in the upper end of the gall-bladder. The smaller branches of the hepatic artery showed an extensive degeneration of the walls, commencing in the muscle cells of the middle coat, and accompanied by a similar change in the walls of groups of capillaries in the neighbourhood of the branches of the artery. The degenerated material took on Weigert's fibrin stain deeply, and gave none of the amyloid reactions. Hyaline thrombi were present in some of the arteries. The walls of the hepatic and portal veins were healthy. Some of the portal veins inside the infected areas were thrombosed.

Endothelioma of Uterus.

The President and Mr. Maunsell showed an endothelioma of the uterus.

Professor O'Sullivan thought that in the present state of knowledge in regard to endotheliomata, unless one could establish the transition from the ordinary endothelium of the lymph space to the tumour endothelium, the diagnosis was not justified.

The President agreed with Professor O'Sullivan's remarks, and admitted that there were many parts of the tumour from which nobody could possibly establish a diagnosis.

Myoma of Rectum.

The President made a communication on the above (see page 330, ante).

Dr. Travers Smith asked was there any possibility of it being a uterine tumour which had found its way out per rectum?

The President said he did not think it was a uterine myoma, because he believed that if a patient had had a myoma like that eating into her bowel she would have complained about it. Dr. Golding had also given a distinct history of its having a membranous attachment, which would not have been present if the tumour had eaten its way into the rectum.
Abstracts.

Pott's Caries.

Mr. Gunn exhibited a case of Pott's caries.

Gastric Ulcer.

Mr. Gunn showed a gastric ulcer which he thought was of a tuberculous nature.

Professor O'Sullivan said he would like to ask the reason for supposing the ulcer to be tuberculous. The rarity of tuberculous conditions in the stomach made it worth while to examine it carefully if it was supposed to be tuberculous.

Mr. Gunn said the reason he thought it so was that there was old tuberculous disease of the abdomen, and active tuberculosis of the lungs, the patient probably swallowing a lot of the sputum. The ulcer was also tuberculous-looking, having thin, undermined edges.

Friday, February 17, 1905.

The President in the Chair.

Mr. E. H. Taylor exhibited specimens removed by operations of (1) a benign tumour of the mammary gland of unusual development; (2) malignant growth in caecum; and (3) a malignant stricture of large intestine (Pelvic Colon).

Dr. Peacocke read papers on (a) cirrhosis of liver; (b) infective endocarditis (see page 332, ante).

Dr. R. Travers Smith exhibited a specimen showing perforation and aneurysm of an aortic valve.

Friday, March 17, 1905.

The President in the Chair.

Carcinomata.

Sir Thornley Stoker, President of the Academy, exhibited specimens removed by operation of:—A columnar cell carcinoma of the uterus in a very early stage; and a columnar cell carcinoma of rectum and sigmoid flexure and involving the left ovary.

Actinomycosis.

Dr. Earl made a communication on cases of human actinomycosis (see page 339, ante).
Mr. Arthur Benson and Dr. Moohey made a communication on sarcoma of the conjunctiva (see page 347, ante).

Gangrenous Intussusception.

Mr. L. G. Gunn showed a beautiful specimen of the above, removed from a child.

Cancer of the Prostate.

Mr. L. G. Gunn showed sections, together with a model of a cancer of the prostate implicating the wall of the bladder.

Friday, May 5, 1905.

Professor Bennett in the Chair.

Professor Bennett presented a note on fracture of the cartilage of the first rib (see page 349, ante).

Dr. T. G. Moorhead asked how frequently Prof. Bennett had found a false joint, and also whether he had ever found a synovial cavity.

Prof. Bennett replied by showing a specimen which illustrated an attempt at the formation of a false joint.

Anomaly of Cardiac Valve.

Dr. Moorhead exhibited a case of anomaly of the mitral valve. On the ventricular aspect of the large anterior cusp a small foramen with smooth edges was present which led into a sac about 3rd inch in diameter. The site and appearance suggested a valvular aneurysm, but the presence of muscular tissue in the wall, the absence of fibrin and the smooth endothelial lining, precluded that view.

Tuberculous Disease of Cæcum, Appendix and Ileum.

Mr. R. Atkinson Stoney showed a specimen of above removed by operation from a female of 23 years of age. A tumour was found in the right iliac fossa, freely movable towards the middle line, to a less extent in an outward direction, but not either upwards or downwards. Patient complained of chronic constipation and occasional colicky pains. There was marked hyperæsthesia of the 11th dorsal area of the right side. At operation a thickened band was found round the ileum, one and a half inches from the cæcum. The cæcum itself was large, nodular, and thickened. The diseased parts were
removed, and a lateral anastomosis was performed. On opening the specimen a stricture of half an inch in diameter was found in the ileum, the cavity of the cæcum was almost obliterated, and a second stricture was found in the commencement of the colon, with a diameter of a third of an inch. No ulceration of mucous membrane was found macroscopically, and the specimen was thought to be malignant. Some glands were found enlarged behind the cæcum. When examined microscopically both the tumour and the glands were found to be infected with typical tubercle.

Dr. W. J. Thompson said the specimen was unusual and interesting, and congratulated Mr. Stoney on the successful result of the operation.

Dr. Kirkpatrick said that during the past winter session he had seen two cases of tubercular disease of the cæcum, both successfully treated by operation. In one case, after removal, the specimen was considered to be malignant, and it was only after very careful microscopic examination that the diagnosis was finally established. This bore out what Mr. Stoney had said. He thought the conditions could not be so rare as Mr. Stoney would lead them to believe, as this was the third specimen he himself had seen during one winter session.

Mr. Stoney, in replying, said that he was sorry he had given the impression that tubercular disease of the cæcum was a rare condition. Indeed two London surgeons had lately published the results of eighty-four cases.

**Recrudescent Ulceration of Stomach.**

Dr. W. J. Thompson stated that the specimen exhibited was taken, post-mortem, from a patient, aged 29 years, clerk by occupation, who was admitted to Jervis Street Hospital on the 21st of February last. He had contracted syphilis five years ago, and for two or three years previously had been drinking heavily. While under specific treatment for about one year he ceased taking alcohol. Since then he drank freely. During the past six months he lost flesh rapidly. Physical examination showed the heart to be enlarged and organically diseased; also enlarged liver, dilated stomach, peripheral neuritis and albuminuria.

At the post-mortem examination the stomach was found enlarged, with a well-marked contraction near the pylorus. On opening it there were seven ulcers (two as large as a sixpence)
in the region of the pylorus. These ulcers were on the cicatrices of two very large ulcers.

Prior to death patient showed practically no sign of gastric ulceration.

Congenital Sarcoma of the Eyelid.

Mr. Arthur Benson and Dr. H. C. Mooney showed a tumour removed from the upper eyelid of an infant 17 days old. It was then the size of a small walnut. There was no family history of any form of malignant growth in either parent. At birth it was the size of a split pea, situated in the centre of the left upper eyelid. It was darkish in colour, and somewhat resembled a hematoma. It changed little for a week, and then grew rapidly. It was lobulated, very elastic in feel, and had entirely lost its dark look, and more resembled a cyst in feel and appearance. Three weeks afterwards recurrence took place in the neighbourhood of the wound; also over the malar bone and in the orbit. A second operation failed to eradicate it, and a sub-maxillary gland enlarged, also one in the axilla. The orbital tumour was, after three months, the size of a small potato, and similar in character to the original growth removed.

Sarcomata of the eyelid, they said, were rare, and congenital unpigmented carcinomata of the eyelid had not before come within their knowledge.

The tumour, irregularly oval, measured 25mm. × 20mm. × 15mm. On section it was creamy white, with small signs of vascularity. The skin removed with the tumour was non-adherent, and under the microscope showed no signs of infiltration. Sections of the tumour showed nothing unusual—it was a mixed-celled, non-pigmented sarcoma with few blood vessels, but with a fair number of large dilated empty spaces, the walls of which seemed to be composed of the compressed surrounding tumour cells. Primary sarcoma of the lid is rare, about fifty cases having been reported, many of which were pigmented. As far as can be ascertained this case is the only one of congenital sarcoma of the lid recorded.

Dr. W. J. Thompson said he had seen the case shortly after birth. The tumour looked then like a small hematoma. The skin over it was freely movable, and its base seemed firmly attached to the eyelid. For a week afterwards there was no advance in size, and then it began to grow rapidly.
Black = Mean Temperature of each week.  Red = Number of Diarrhoeal Deaths registered each week.

* These lines indicate the beginning and end of the critical Temperature 56° at 4 feet.
AUGUST, 1904.

DIAGRAM I.

SEPTEMBER, 1904.

Air

Temp.

68°F

1 foot Therm.

40 deaths 4 feet Therm.

30 deaths

20 deaths

10 deaths

0 deaths

Black = Air Temperature (Fahrenheit). Green = Earth Temperature 1 foot below the surface. Blue = Earth Temperature 4 feet below the surface. Red = Number of Diarrheal Deaths.
SECTION OF STATE MEDICINE.

EARTH TEMPERATURE AND DIARRHOEAL DISEASES IN DUBLIN DURING 1904.

By SIR JOHN W. MOORE, M.D. DUBL.; F.R.C.P.I.;
D.Sc. Oxon. (Honoris Causa);
Physician to the Meath Hospital and County Dublin Infirmary.

[Read in the Section of State Medicine, February 10, 1905.]

In an elaborate report to the Local Government Board for England upon the causation of the annual mortality from "diarrhoea," which is observed principally in the summer season of the year, the late Dr. Edward Ballard in 1887 advanced the proposition that the temperature of the soil is a far more effective element in raising the death-rate from diarrhoeal diseases than any other meteorological factor. He constructed for London and many other towns in the kingdom a large number of charts showing week by week for many years the earth-temperature at a depth of 1 foot from the surface and at a depth of 4 feet also, each chart showing in addition the diarrhoeal mortality of the corresponding weeks. The general result shown by these charts is as follows:

(a.) The summer rise of diarrhoeal mortality does not commence until the mean temperature recorded by the 4-foot earth thermometer has attained somewhere about 56° F., no matter what may have been the temperature previously attained by the atmosphere or recorded by the 1-foot earth thermometer.

(b.) The maximal diarrhoeal mortality of the year is usually observed in the week in which the temperature
recorded by the 4-foot earth thermometer attains its mean weekly maximum.

(c.) The decline of the diarrhoeal mortality coincides with the decline of the temperature recorded by the 4-foot earth thermometer, which temperature declines very much more slowly than the atmospheric temperature, or than that recorded by the 1-foot earth thermometer. The epidemic mortality may in consequence continue (although declining) long after the last-mentioned temperatures have fallen greatly, and may extend some way into the fourth quarter of the year.

(d.) The atmospheric temperature and that of the more superficial layers of the soil exert little, if any, influence on the prevalence of diarrhoea until the temperature recorded by the 4-foot earth thermometer has risen to 50° F. Then their influence is apparent, but it is a subsidiary one, notwithstanding the statement made by Dr. August Hirsch that the summer diarrhoea of children makes its appearance as an epidemic only in those districts whose average temperature for the day in the warm season is rather more than 15° C. (59° F.).

Dr. Ballard believed that a working hypothesis, or provisional explanation, that would best accord with the whole evidence then in his possession bearing on the production of epidemic diarrhoea, may be stated as follows:—

1. The essential cause of diarrhoea resides ordinarily in the superficial layers of the earth, where it is intimately associated with the life processes of some micro-organism not yet detected, captured, or isolated.

2. The vital manifestations of such organism are dependent, among other things, perhaps principally, upon conditions of season and on the presence of dead organic matter which is its pabulum.
3. On occasion, such micro-organism is capable of getting abroad from its primary habitat, the earth, and having become air-borne obtains opportunity for fastening on non-living organic material, and of using such organic material both as nidus and as pabulum in undergoing various phases of its life-history.

4. In food, inside of as well as outside of the human body, such micro-organism finds, especially at certain seasons, nidus and pabulum convenient for its development, multiplication, or evolution.

5. From food, as also from the contained organic matter of particular soils, such micro-organism can manufacture, by the chemical changes wrought therein through certain of its life processes, a substance which is a virulent chemical poison.

6. This chemical substance is, in the human body, the material cause of epidemic diarrhoea.

To the foregoing we have only to add Dr. Meinert's words: "The poison, or a combination of poisons, appears to work upon the medulla oblongata, for there lies the centre for intestinal secretion, vomitings, convulsions, respiratory and vaso-motor phenomena." This is a quotation from an article on "Cholera Infantum" by Dr. E. Meinert, of Dresden, which appeared in the Medical Annual for 1893. In that article Meinert fully accepts Ballard's views, especially his statement that density of buildings, whether dwellinghouses or otherwise, upon area—quite apart from density of population upon area—promotes diarrheal mortality to a remarkable degree, particularly because crowding together of buildings of whatever sort restricts and offers an impediment to the free circulation of air.

On January 1, 1904, through the liberality of the Provost and Senior Fellows of Trinity College, a Normal
Climatological Station was established within the precincts of the University of Dublin. The station, which is under the supervision of Professor W. E. Thrift, M.A., F.T.C.D., occupies an open space in the Fellows' Garden, Trinity College, and is fully equipped. At the suggestion of Dr. William Napier Shaw, F.R.S., Secretary of the Meteorological Office, London, the equipment included two earth thermometers. One of these has its bulb at a depth of 12 inches (1 ft. earth thermometer) below the surface of the ground. The bulb of the other is sunk in a metal tube to a depth of 4 feet.

So far, of course, the observations for only one year—1904—are available for discussion. Nevertheless, I thought that the State Medicine Section of the Royal Academy of Medicine in Ireland would be interested in the attempt to test the accuracy of Ballard's theory by a comparison of the death-curve of diarrheal diseases in the Dublin Registration Area during the summer and autumn of 1904 with the earth temperature records in Trinity College—the site of the Observatory being in the very centre of the Metropolitan District.

The observer, my son, Arthur Robert Moore, B.A., has thrown the figures into two diagrams. The first of these (Diagram I.) gives three temperature curves for each day of the three months, July to September, 1904. The continuous black line is that of the air temperature at 4 feet above the ground recorded in a Stevenson's screen at 9 a.m. daily. The upper green line shows the earth-temperature at 9 a.m. each day at a depth of one foot below the surface of the ground; the lower blue line similarly gives the earth-temperature four feet below the surface. At the base of the diagram will be seen in red the number of deaths from "diarrheal diseases" registered week by week in the Dublin Registration District.
Diagram II. contains two weekly curves for the whole year 1904. The upper of these represents the weekly march of underground temperature at a depth of four feet. The lower red curve gives the number of deaths from diarrhoeal diseases registered in each week of 1904 in the Dublin Registration Area.

In both diagrams especially heavy rainfalls have been entered, and in the epidemic season weekly measurements of an inch or upwards on two occasions seem to have been closely followed by a decline in the number of deaths from diarrhoeal diseases. Thus in the week ended September 3, the rainfall amounted to one inch (0.999 inch); a fortnight later the weekly diarrhoeal deaths fell from 26 to 14. Again, in the week ended September 17, the rainfall exceeded an inch and a quarter (1.285 inches); a fortnight later the weekly diarrhoeal deaths fell from 28 to 16.

Reference to the curves in Diagram II. shows that diarrhoeal mortality in the Dublin District in 1904 was trifling till the week ended August 6—that is, the third week after the subsoil temperature at 4 feet had passed above 56° F. The mortality rapidly increased till the week ended August 27, in which 35 deaths from diarrhoeal diseases were registered, or about 10 per cent. of all the deaths from those diseases in the whole year 1904. This maximum of mortality followed the maximum of warmth of the soil at 4 feet (58.5°) by an interval of just a fortnight. Such a coincidence is remarkable. Diarrhoea kills very young children quickly—usually within a week. Then, allowing a few days for delay in registration, we come to the close of the second week.

Diagram II. also shows that the 4-foot thermometer stood at 56° or upwards from the 10th of July to the 24th of September—a period of eleven weeks. Starting a
similar period of eleven weeks a fortnight later (to allow time for the malady to attack and kill and for registration of the resulting deaths), we find that in the eleven weeks beginning July 24 and ending October 8, the diarrhœal deaths were 249, or 71.7 per cent. of the total deaths from diarrhœal diseases registered in 1904, 339 in number. Of these, only 18 were registered in the first quarter of the year, only 10 in the second, 243 in the third, and 68 in the fourth quarter. In his Quarterly Summary of the weekly returns of births and deaths in the Dublin Registration Area, under date October 1, 1904, the Registrar General observes:—"The deaths from diarrhœal diseases form a feature of the mortality statistics, this being a usual coincidence at the period of the year included in this report (July 3 to October 1, 1904, inclusive). The total number of deaths assigned to above causes was 243—namely, 104 deaths from epidemic diarrhœa, epidemic enteritis, and 139 deaths from 'diarrhœa dysentery.' The total equals a rate of 2.6 per 1,000 of the population (estimated) of the Dublin Registration Area, and exceeds the average for the corresponding quarter of the past 10 years by 33 deaths. Of the total deaths, 204, equal to an annual rate of 2.8 per 1,000, appertained to City Districts, and 38, equal to an annual rate of 1.8 per 1,000, appertained to the Urban Districts which, with the City, constitute the Dublin Registration Area." The mean of the readings of the 4-foot earth thermometer in these thirteen weeks ending October 1, 1904, was 57.0°—a remarkable coincidence, to say the least, this figure being one degree above the "critical" temperature at 56° F. In my opinion, "Diarrhœal Diseases" should be "notifiable" from July 1 to September 30 in each year, so as to give even more timely warning of an epidemic tendency.
Dr. Edward W. Hope, Medical Officer of Health for Liverpool, some years ago investigated the influence of the mode of feeding of young infants on the prevalence and fatality of diarrhoea, and came to the conclusion that infants fed solely from the breast are notably exempt from fatal diarrhoea, whereas infants fed with artificial food to the exclusion of breast milk suffer most severely from his dread disease. In relation to this Dr. Ballard’s observations go to show that the direct or indirect exposure of food to telluric emanations tends to render it liable to produce diarrhoea, particularly when the storing place is dark and ill-ventilated.

According to Flexner the bacillus of dysentery can be isolated from the intestinal discharges and the intestinal mucosa of a large percentage of children suffering from the diarrhoeal diseases prevalent along the Atlantic seaboard of the United States of America in summer. The type of bacillus most commonly found is that known as the Flexner-Harris. The Shiga type is exceptionally met with, and both types may occur in association. There is some reason to believe that the dysentery bacillus may be found among the saprophytic intestinal bacteria. So long ago as 1890, Weichselbaum (Wien. klin. Wochenschrift, 1890, Vol. III., page 187) isolated the Diplococcus pneumoniae in a case of membranous enteritis. It will be well in future for this organism to be sought for in the stools of “summer diarrhoea.”

Dr. Kirkpatrick said that the chief interest and value of the observations was to give an indication of the onset of these attacks of summer diarrhoea in children to the authorities, who could then take precautionary measures, and give timely warning of its approach. This was possible, owing to the interval which elapsed between the rise of the earth-temperature to the critical line of 56° and the incidence of the diarrhoea. He did not think there was sufficient evidence to show that the rise of the temperature to the critical line and the summer diarrhoea were connected casually. It was more probable that the earth-temperature reached a certain height only during conditions when epidemic diarrhoea was liable to arise also, and there was probably an organism which could develop when the earth-temperature had been a sufficient height for a certain length of time. This was borne out by the fact that the mortality from summer diarrhoea was much less amongst breast-fed than bottle-fed children, and everyone knew the difficulty of giving bottle-fed children even moderately sterile food. There could be no doubt that this epidemic diarrhoea was started by the introduction of poisonous food into children, and it was probably caused by organisms capable of producing decomposition in food stuffs, and not by any specific bacillus. He hoped that active measures would be taken next year to prevent the onset of summer diarrhoea and warning given in time to take due precautions.

Dr. Craig was sure that Sir John Moore did not mean to convey that the earth-temperature alone caused this epidemic diarrhoea. It was well recognised that this very fatal affection was due to micro-organisms. He thought that the heightened temperature tended to increase putrefaction, and that flies had a great deal to do with carrying the infection. People who were careful with regard to their food were rarely affected with summer diarrhoea. He thought that children used often to be affected through the state of the bottles themselves.

Dr. M'Vittie said he would like to have an explanation of the statement that diarrhoeal diseases were more markedly on the increase in those districts where the houses were closely built than where there was an excess of population. This would indicate that the earth-temperature could not be the main factor, because a large portion of earth covered by closely-built houses could not rise to the same temperature as places where there
was a lot of open space between the houses. Closely-built houses maintained a more equable earth-temperature in summer and winter. Therefore there must be a number of other factors in the causation of these diseases, such as increase of fruit supply, &c. Another factor was that young people in cities became exhausted at that time of year, and their capacities for mastering those diseases were lowered. Small children played much in the sun, got into heats, and then got chilled.

Dr. Langford Symes thought that those who suggested that there was a multiple series of factors producing those diseases held the correct view. These diseases seemed to destroy more children than adults, and, perhaps, a milk diet accounted for that. We seemed to be more exempt from them in Ireland than in other places, such as New York, where the infant death-rate was much higher. The climate here seemed to minimise them, there being no great difference between the summer and winter temperatures in Dublin. The earth-temperature certainly seemed to have a great deal to do with the development of the organisms which caused the diseases and predicts their onset. As to food, it was found by Holt that this factor greatly influenced their onset amongst infants. The ground had recently been gone over again in America by bacteriologists, and they had come to the conclusion that not only the earth-temperature, but also food, had a good deal to say to their causation. Again, Holt had noticed in his series seven cases of breast-fed infants who had also got water from the tap without boiling, and the seven had died; therefore the diseases might be water borne. Another factor was sanitation, and indeed these diseases used to be taken as a sanitary index. With regard to infection, light had been recently thrown on this question by Shiga in Japan, who had found a bacillus which gave the Widal and Pfeiffer reactions with blood serum, and which he considered to be the specific bacillus of the diseases. It was called the *Bacillus dysenteriae* of Shiga. Besides this bacillus there was also the Flexner-Harris type, which was found principally in the mucous portions of the stools, and in scrapings from the mucosa after death. An acid medium was necessary for their growth, and we know that many children's foods developed acidity, which might account for their ready growth. A serum had been made from horses, and injected in these cases, but it
had not proved to have much value. He thought that Dr. Craig had struck a good note in referring to flies, insects, &c., as a means of conveying serious infection.

Dr. Matson asked Sir John Moore if he thought that the nature of the ground had anything to do with those diseases. He himself, whilst in a London hospital, had seen a severe epidemic, and had gone from that to Hampshire, where a similar epidemic occurred. The soils in the two places were very different. The following year, whilst in Las Palmas, where the temperature was 80° to 85°, he asked a doctor there what his experience of those diseases was. He told him that he had never seen them in children nursed by their mothers, whilst there was a considerable amount of fatal diarrhoea amongst older children who were liable to infection from fruit, garbage, &c.

Dr. Winter said that Sir John spoke of a heavy rainfall seeming to have been followed by a decline in the death-rate. While in England, twelve years ago, he himself had had occasion to examine the reports on the Tees Valley epidemic of enteric fever, and a heavy rainfall there was followed by a great increase in the number of cases. If these two facts were correct, he would say that enteric was water borne, and these diarrhoeal diseases were not water borne, but were carried through the air, contaminating the food.

Dr. Ninian Falkiner said he would like to call attention to the nomenclature of these so-called diarrhoeal diseases. Some years ago statisticians found a number of cases returned as gastro-enteritis, enteritis, and muco-enteritis, and it soon became evident that these terms were loosely used. A special committee was appointed, the net result of whose labours was that these three divisions were all placed amongst the local diseases of the digestive system.

Sir John Moore, in replying to Dr. Kirkpatrick, said that last year the observations had actually been used to give warning of the onset of diarrhoeal diseases. He had been careful in his paper not to commit himself to the view that the earth-temperature was the absolute cause of these diseases, but he said there was a remarkable coincidence. He agreed that various organisms might cause the diarrhoea. A very interesting point was that the symptoms were referable to poisoning of the centres in the medulla oblongata. He also agreed with Dr. Craig as to the mischief
caused by flies, and thought that this was very far-reaching. He believed that small-pox infection had often been carried by them. He could not quite agree with Dr. M'Vittie that the mere placing of houses over the soil would very materially interfere with the rise of the subsoil temperature. With regard to Dr. Symes' observations, he said that everyone agreed that food was of extreme importance. As to the nature of the soil it was certain that clayey soil always warmed up more slowly than gravel or sand, so that diarrhoeal diseases might be expected earlier in the latter case. With regard to what Dr. Winter had said, he thought that the mortality had been checked last year by heavy rains, this fact telling in favour of contamination by dust or flies.
SICILY AS A WINTER HEALTH RESORT.

By E. PARLATO, M.D., Ph.D., Palermo.

[Read in the Section of State Medicine, February 10, 1905.]

A winter resort which in the last few years has been attracting a growing number of visitors to its shores is Sicily. There is no doubt that the many natural beauties of the island, the historical interest attaching to the monuments it contains, together with the mildness of its climate, are factors which will make it in the near future a still more popular point for a prolonged winter stay.

Thanks to their insular position, the people have retained many of the characteristics of the different races which have succeeded each other on the island, so that there is no lack of local colour.

The object of these lines is not to repeat what quite a number of books recently published on Sicily have said, but to lay before the profession some details drawn from the writer's personal experience and from the most recent official reports.

The climate of Palermo, Messina, Catania, and Taormina belongs to the warm sea-coast class: moderate degree of humidity, absence of fogs, small diurnal variations of temperature. Within the last five years the thermometer has, at Palermo, never actually reached the freezing-point—the lowest recorded temperature within this time having been 0.4° Centigrade = 32.7° F.

The great charm of the climate is certainly its mildness, which allows one to sit in a room by the open window on a great number of days throughout the winter. The following figures are taken from the report
of the Royal Meteorological Observatory of Valverde, Palermo:—

**Averages of Max. and Min. Temperature.**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1900</td>
<td>16.5</td>
<td>7.1</td>
<td>18.4</td>
<td>7.8</td>
<td>16.9</td>
</tr>
<tr>
<td>1901</td>
<td>14.6</td>
<td>4.4</td>
<td>15.6</td>
<td>5.2</td>
<td>19.9</td>
</tr>
<tr>
<td>1902</td>
<td>15.9</td>
<td>5.6</td>
<td>17.9</td>
<td>7.9</td>
<td>18.1</td>
</tr>
<tr>
<td>1903</td>
<td>16.6</td>
<td>5.8</td>
<td>16.2</td>
<td>5.1</td>
<td>18.2</td>
</tr>
<tr>
<td>1904</td>
<td>14.8</td>
<td>6.1</td>
<td>17.1</td>
<td>8.0</td>
<td>17.9</td>
</tr>
</tbody>
</table>

Average of 5 years: 60.2 42.6 62.7 44.3 64.8 44.9 68.7 49.8 63.5 46.3

The following table gives the relative moisture of the winter months at Palermo; Saturation = 100:—

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%</td>
<td>%</td>
<td>%</td>
<td>%</td>
<td>%</td>
</tr>
<tr>
<td>1900</td>
<td>71.7</td>
<td>68.8</td>
<td>69.3</td>
<td>74.8</td>
<td>78.7</td>
</tr>
<tr>
<td>1901</td>
<td>78.1</td>
<td>73.0</td>
<td>65.5</td>
<td>74.4</td>
<td>72.7</td>
</tr>
<tr>
<td>1902</td>
<td>78.7</td>
<td>73.2</td>
<td>72.3</td>
<td>78.5</td>
<td>77.3</td>
</tr>
<tr>
<td>1903</td>
<td>75.9</td>
<td>75.9</td>
<td>74.3</td>
<td>70.4</td>
<td>71.9</td>
</tr>
<tr>
<td>1904</td>
<td>79.4</td>
<td>63.3</td>
<td>67.4</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

Average of 5 years: 76.7 70.8 69.7 74.5 75.1

*Rain* is more prevalent during the winter months—the number of rainy days being more numerous in November
and December. The average number of rainy days is 116 for Palermo and 86 for Catania.

The relative humidity at Catania and Taormina is slightly lower than at Palermo. This last has the advantage of a greater uniformity of temperature.

Winds.—The great superiority of Sicily over the Riviera in this respect is the absence of any cold winds, such as the Mistral or Bise. The north-east wind, the so-called "Greco," is very rare at Palermo, and lasts but a very short time. The much-dreaded Sirocco is rarely observed during the winter months, but is more prevalent in September and in April. It is really much less frequent than is generally supposed, and is more talked about than experienced. It lasts one, two, or, very rarely, three days, during which the temperature rises to a point which is disagreeably felt owing to the suddenness of the change. The writer's personal experience is that the Sirocco is most trying to people of full habit and plethoric constitution, whereas nervous, delicate individuals feel quite comfortable.

Talking of the winds, attention must be called to the dust of some of the streets of Palermo, which, during a high wind, becomes inconvenient and irritating. The municipal authorities have of late years been taking the dust problem in hand, and by paving and by watering of the streets have done much towards allaying this nuisance.

From the foregoing remarks the reader will be able to draw some conclusion as to the cases which will derive some benefit from a stay in the "Pearl of the Mediterranean."

Indications.—Convalescents from infectious diseases will find that the balmy and sunny atmosphere of Sicily renders possible and agreeable an outdoor life during the
winter season, with all the advantages it brings to the general health.

Although for chest complaints (tuberculosis, pneumonia, and pleurisy residues, &c.) the writer is a partisan of the Alpine health resorts (Davos, Arosa, Leysin, &c.) the preference should be given to Palermo in those cases of tuberculous or other affections of the throat and chest, which are combined with a nervous temperament. The climatic influence will have a sedative action also in cases of bronchial asthma and irritability of the throat, although in cases accompanied by a copious catarrhal discharge (bronchorrhoea) a prolonged stay at Palermo is not to be recommended.

One class of invalids who will find Palermo very suitable as a winter resort is composed of the numerous sufferers from insomnia, neurasthenia, &c., caused by excessive irritability of the nervous system. Patients coming from the Riviera soon notice the change and the soothing effect of the Palermo climate on their sleep.

For cases of chlorosis and anaemia, combined with reduced vitality, a stay at Palermo or Taormina will be found very beneficial.

Sicily has an ideal climate for old people during the winter months, when in colder latitudes they would be obliged to remain indoors. Patients with compensated valvular trouble will find a winter spent in Sicily, free from the cold and fogs of the Northern climate, very pleasant and of benefit to the general health. The writer would also recommend a stay at Palermo or Taormina to the many Anglo-Indians who, for the first winter after leaving India, would find the climate of England too cold. Chronic rheumatic affections form a contraindication to a stay in Palermo. In these cases Taormina would be more suitable.
A CASE OF CARBON MONOXIDE POISONING.

BY W. J. THOMPSON, M.D. Univ. Dubl., F.R.C.P.I.;
Physician to Jervis Street Hospital.

[Read in the Section of State Medicine, April 14, 1905.]

During the past 4 or 5 years quite a number of cases of carbon monoxide poisoning have occurred in Dublin owing to the mixture of water gas with the coal gas as supplied by our Gas Company, resulting, unfortunately, in the loss of life. Last Session this Section of the Royal Academy of Medicine had the privilege of hearing a most valuable and scientific paper from Professor McWeeney on this subject, in which he minutely describes the changes he had observed in the blood, as well as other changes in different organs as found by him at post-mortem examinations.

I have had under my care in Jervis Street Hospital three cases within the past few years, and as even the larger text-books are somewhat indefinite in the description of symptoms and treatment, I thought, perhaps, the description of a case admitted under my care last year might be of some interest to the members of this Section. The case, I may say, is almost unique in how the gas poisoning was contracted, for in looking up the literature on the subject I can find no similar case recorded.

At 7 30 a.m. on the 12th November, 1904, F. H., a strong, healthy, able-bodied, seafaring man, aged thirty-seven years, was admitted to hospital, and I saw him shortly afterwards. The following is the history of his case we got from his companions:—He was by occupation a cook in one of the dredgers
used at the mouth of the Liffey, and belonging to the Port and Docks Board. On the previous evening, as was his usual custom, he retired to the skipper's room about 9 o'clock, and not making his appearance at 6 o'clock next morning one of the crew knocked at the door, and getting no answer the door was burst open. The patient was found on the floor, lying on his felt side, apparently lifeless. That night the dredger was anchored some distance down the river, and although all possible haste was made it was 1½ hours afterwards before he was brought to the hospital in the city ambulance. It seems that on these dredgers there are usually eleven of a crew—the skipper, cook, and nine others—and at night only two of the crew, who act as watchmen, remain on board, as well as the cook. It was always the custom for the cook to retire to this room for the night. The room is described as being about 17 feet long by 8 feet wide, by 7 feet high (cubical contents 952 feet), and when the door is closed there is no ventilation. At one end of the room there is a small stove in which, during the winter months, there is always a fire. We found out from the patient afterwards that when retiring that night he closed and bolted the door, and as the fire was low he put on one large block of Scotch steam coal. He remembered nothing more until he found himself in hospital.

When I saw the patient he was in a state of deep coma; the colour, not only of his face, but also of the whole body, was dusky, dark and greyish; he had well-marked short, shallow, and sterterous breathing (24 per minute); there were slight tremors of all the muscles of the body, specially of the masseters, and this tremor was rhythmical with an interval of about two minutes. The pupils were irregular, that of the right eye being slightly dilated, and that of the left eye being slightly contracted; both were insensible to very strong light. The abdomen was retracted. The surface of the body was icy cold, although the temperature taken in the rectum was 99.6°. The mouth was rigidly closed; the mucous membrane of the nose, as well as the inside of the ear, was in a state of anaesthesia; besides, when a catheter was passed the patient apparently did not feel it. The whole surface of the body was in the same state of anaesthesia. There was also analgesia and the sensibility to heat and cold was lost. The pulse was thready, small, easily compressible, and so quick that it was impossible to count it. The heart sounds were al
Case of Carbon Monoxide Poisoning.

inaudible; that of the first sound was practically lost. The superficial reflexes, including the conjunctival, were lost, and there was well-marked rigidity of all the muscles of the body—more particularly those of the arm. There was no urine in the bladder, although it was at least twelve hours since he had micturated. The blood when drawn off had the bright red cherry colour characteristic of carbon monoxide poisoning, and Professor McWeeney concurs in this view.

The other two cases which came under my care were affected from inhaling the ordinary gas, as supplied to the Dublin consumers, which, as was pointed out by Professor McWeeney, is charged with carburetted water gas, sometimes to the extent of 16 per cent., whereas in ordinary coal gas (as used formerly to be supplied in Dublin) the percentage is rarely more than 6 per cent. This case illustrates how carbon monoxide poisoning may accidentally take place. Dr. Glaister, Professor of Forensic Medicine and Public Health in the University of Glasgow, in his well-known and popular text-book, describes the celebrated Snaefel case of CO poisoning as occurring from burning timber, in which combustion was not active because of imperfect supply of air. The probable explanation of my case which I venture to give is that there was slow combustion of the large lump of coal owing to the deficient supply of air due to the smallness of the room and the total want of ventilation. As a matter of fact, there was still a smouldering fire in the stove when the door was burst open.

It is scarcely necessary to mention that carbon monoxide poisoning is a typical example of a cumulative poison. The powerful toxic action of carbon monoxide is due to its affinity for haemoglobin. Some authorities state that this affinity is from 150 to 200 times greater than the affinity of oxygen for haemoglobin. When CO is
inhaled it gradually displaces the O of the hæmoglobin, and forms carboxyhaemoglobin—a much more stable compound than oxyhaemoglobin. In the living body carboxyhaemoglobin neither takes up nor gives off O, and is, therefore, incapable of acting as an O carrier to the tissues. According to Haldane it is necessary in human beings that the blood be one-third saturated before the characteristic symptoms become urgent, and this explains the delay in the appearance of any serious symptoms. The combination is sufficiently intimate to resist the action of reducing agents, but it gradually yields to the action of O. This fact is most important, as it gives us a forcible hint in treatment, for the administration of O is of paramount importance. To be of any marked service it must be used continually for a long time. It is proved experimentally that if air or O is passed for a long time through a solution of carboxyhaemoglobin the CO is slowly and gradually separated from the hæmoglobin, and O takes its place. This point was well illustrated in the first case that came under my care in hospital, and which Professor McWeeney mentions in his paper last year. The patient was three days in hospital before death, and during that time had inhaled large quantities of O. At the post-mortem it was found that there was no carboxyhaemoglobin, showing that the use of O inhaled had doubtless affected the complete removal of the CO.

Dr. Nixon Mann, Professor of Forensic Medicine and Toxicology in Owens' College, Manchester, in his valuable and instructive text-book says that it has been stated that CO possess an intrinsic toxic action in addition to its power of depriving the tissues of O. Linossier, of Lyons, from experiments, concludes that CO does produce such an action; while, on the other hand, such a dis-
tiguisished authority as Haldane states that CO acts solely by combining with hæmoglobin, and in no other way.

The treatment adapted for this patient was the almost continuous use of O, the prolonged carrying on of artificial respiration, hypodermic injections of digitaline, strychnine, and æther. Nutrient enemas with rectal injection of stimulants. In addition, heat was applied all round the body, and, what I consider most useful, the application of electricity along the course of the phrenic nerves and the spine. I have used saline transfusion, but, in my opinion, without any beneficial result. Copious bleeding is also important and useful.

As I have stated before, the patient's bladder was empty on admission. During his first day in hospital he passed 9 oz., on the second day 15 oz., on the third day 23 oz. The quantity passed on the fourth day was normal. No trace of sugar or albumen could be detected, although some authorities hold that sugar is present. The amount of urea was about normal per ounce. This is a contrast to my first patient, who, during the three days before death, secreted no urine, and at the post-mortem Dr. McWeeney found extreme congestion of the interlobular vessels of the kidneys.

The patient did not regain consciousness until late in the afternoon of the day of admission, and from that time he made a rapid recovery. He left hospital on the 27th November, having been a patient for fifteen days, apparently nothing the worse of his illness. In many of the recorded cases convalescence has been very slow, and frequently some organic disease resulted.

I cannot conclude these short notes without paying a well-deserved tribute to our senior house surgeon, Dr. Loughnan, and my resident pupil, Mr. Stuart, both of
whom so assiduously and perseveringly carried out the treatment.

Professor McWeeney stated that he had made an examination of the blood from Dr. Thompson's patient. The first specimen he examined was too small in amount for detailed examination, but presented all the appearances of the presence of CO gas combined with the haemoglobin. At a subsequent examination no trace of CO gas was found, and the leucocyte count was also normal. Dr. McWeeney thought that Dr. Thompson deserved the greatest credit for his active and successful treatment of the case.
SECTION OF ANATOMY AND PHYSIOLOGY.

THE ANATOMY OF A SIRENOMELIAN MONSTER.

By T. GILLMAN MOORHEAD, M.D., DUBL., M.R.C.P.I.;
Physician to the Royal City of Dublin Hospital.

[Read in the Section of Anatomy and Physiology, April 28, 1905.]

While the occurrence of double monsters and of monsters with multiple arms and legs is comparatively common, those forms, in which fusion, more or less complete, of the limbs of a single foetus constitutes the anomaly, are undoubtedly rare. Up to the period at which the work of Geoffrey St. Hilaire on Monstrosities made its appearance in 1836 no adequate attempt had been made at a classification of monsters in general, and, in particular, those monsters in which the two lower limbs are united to form a single terminal and median appendage were only casually and generally referred to under such terms as monopodia and sympodia. To St. Hilaire belongs the credit—as has been recognised by all succeeding writers—of distinguishing the sub-varieties of this class and of applying the name Symeles to the group. Both this name and also those which he applied to the sub-varieties have been universally adopted.¹

Early references to the sympodia are not at all common, and almost all those which are found scattered in the literature before 1800 deal more with mythical and legendary reports than with actual verified occurrences.

¹Since writing the above I find that Ballantyne in the second volume of his Antenatal Pathology, just published, prefers the term sympodia.
The fact, however, that even legends exist points to the occasional appearance of nereid-like human foetuses, and the structure of such is certainly sufficiently striking to afford a more than usually solid basis for much folk-lore.

Perhaps the earliest systematic attempt to explain the origin of human and other monstrosities is that of Aristotle in Chapter III. of the fourth book of his work "On the Generation of Animals." In it he discusses, in a truly scientific manner, the various theories then current, and, after advancing one of his own, enumerates the various forms of monstrosities with which he was acquainted. In his fairly extensive list no reference to the symelian type is to be found, and since Aristotle was the exponent of all scientific knowledge at that time we must conclude that no authentic case was then known. Pliny, on the other hand, in addition to his numerous references to the true sea nereids and tritons of the ancients, states in Chapter II. of the seventh book of his Natural History, on the authority of Ctesias, that there existed near the country of the Trogogclytes a race of men called Monocoli, who, although possessed of only one median lower limb, were able to walk and jump with agility. These Monocoli were also, he states, called Sciapodae, because they were accustomed to recline on their backs during the heat of the sun and to protect themselves from its rays by their overspreading foot. This legend, I think, may fairly be regarded as being inspired by the occasional occurrence of symelian foetuses, especially in view of the tendency of writers in these days to look upon monstrosities as the chance occurrence, among men, of beings belonging to another race or type. As such it may be looked upon as the earliest reference to sympodia, and it appears to have impressed subsequent writers, for it is referred to, and even figured
in the work of Lycosthenes in 1557 (Prodigii Obstentorum Chroncon) under the heading "Sciopodes and Monomeri," and also in that of Aldrovandus, which appeared in 1642. Before either of these last-mentioned dates, however, a short but unequivocal account of a typical sirenomelian foetus appeared in the work entitled "Julii Obsequentis Prodigiorum Liber." In this book the specimen described is figured, and resembles in all respects the foetus that I am about to describe, with the exception of the fact that, in addition to the lower limb deformity, it was devoid of arms. In 1836, as already stated, St. Hilaire adopted a definite name and classification, and, furthermore, collected most of the previous references in the literature with the exception of those I have recorded above. The last important English paper upon the subject was that by Manners Smith in the Journal of Anatomy for 1896. It consists of a description of two symelian monsters, and contains an almost complete bibliography. Since that date only very few have been described, and after a fairly complete search through the literature I have only been able to discover one or two additional papers, the most important of which consisted of an accurate description of a formalined sirenomelian monster, and appeared in Bd. 171 of Virchow's Archiv," under the title "Ein Fall von Sirenbildung," by Abramav and Rjisanow. For a full bibliography Ballantyne's work on Antenatal Pathology may be consulted.

The foetus now described belongs to the sirenomeles, or third variety of St. Hilaire, in which the lower limbs are united in a single median extremity which tapers below to a point or rounded stump without a distinct foot. This variety is more common than either of the other two forms (symeles and uromeles), in which less perfect union of the lower extremities is present. The
present specimen was born at full term in the Rotunda Hospital, Dublin, and had the appearance of being dead for some days. For permission to dissect and describe it I am indebted to the late Dr. W. Neville, pathologist to the hospital.

Superficial Appearance.—In general appearance the foetus was normal in every respect above the level of the umbilicus. Below that level it gradually tapered to a rather sharp point, which presented somewhat the appearance of a toe, and which was marked at its extremity by a dorsal depression resembling the bed of a nail. This median limb projected forwards, making an angle of about 150° with the trunk, and presented on each side, in the neighbourhood of the knee-joint, a rounded swelling. On its anterior aspect there was situated in the middle line, just below the symphysis pubis, a puckered scar, which, as the sole representative of the external genitals, may be called the genital scar. Posteriorly no anus was present, but high up over the back of the coccyx there was placed an anal scar similar to that described in front.

Thoracic and Abdominal Viscera.—The thoracic viscera were normal in every respect, except that the thymus gland was of unusually large size. In microscopical structure this gland was, however, quite normal.

In the abdomen the stomach and small intestines were quite normal, and had normal peritoneal connections. The appendix was also normal. The caecum lay above the right iliac fossa, and was distended with meconium, as was also the transverse colon. The descending colon was enormously distended, and terminated below in a pointed extremity which lay in the left iliac fossa. Almost the whole of the descending colon was free, being only attached by a loose mesenteric fold to the peri-
The Anatomy of a Sirenomelian Monster.

toneum over the hypogastric artery. The liver, gall-bladder, pancreas and spleen were normal. No kidneys, ureters, or bladder were present. The suprarenal capsules were very large, and lay in the normal position on the posterior abdominal wall. In microscopical structure they were normal.

The aorta entered the abdomen in the normal manner, and, immediately after it emerged from under cover of the pancreas, gave off a large single branch, which passed almost directly forwards towards the umbilicus. This branch was much larger than the continuation of the aorta within the abdomen. Shortly after its origin it divided into two branches, which ran side by side for a short distance and again united into a single trunk before reaching the umbilicus, so that only a single artery entered the umbilical cord. The hypogastric artery in its intra-abdominal course was covered by a peritoneal fold which practically divided the lower part of the abdomen into two symmetrical lateral halves. In the right half were contained the small intestines, while in the left half lay the distended sigmoid flexure, attached by its mesentery to the fold of peritoneum over the artery. The aortic trunk below the origin of the hypogastric artery continued downwards on the posterior abdominal wall and divided on the fourth lumbar vertebra into the two common iliac vessels. These last-named, after giving off small internal iliac branches to the pelvis, were continued as the external iliac vessels to become continuous with the femorals under Poupart's ligament.

The cavity of the pelvis was only represented by a slight depression in the peritoneum on each side of the median peritoneal fold. In this depression there lay on each side the testes, which were normally developed, and
which presented on their posterior aspect well-formed epididymes. From the epididymis the vas deferens passed upwards on each side in the peritoneal fold over the hypogastric artery, and, gradually narrowing, finally disappeared from view close to the neighbourhood of the pancreas. Microscopically the vas was traced a little further up as a small duct lined with columnar cells. The microscopical appearance of the testis and epididymis was normal.

Among the special points of interest in connection with the viscerae the following may be noted:

1. No trace of kidneys or bladder could be met with, although searched for most carefully, and that in a foetus preserved in formalin. This is in accord with what has generally been found in this class. Manners Smith, however, described the presence of cystic kidneys and of a small bladder in one of his cases, and in Abramov and Rjisanow’s case cystic kidneys were also found, but neither bladder nor ureters. It is noticeable that in neither of these two cases were suprarenal capsules present. In the present case kidneys were absent, and the suprarenal capsules were of unusually large size.

2. The condition of the lower end of the intestinal canal in sirenomelians varies considerably. In the majority of cases no anus or rectum is present, and the sigmoid flexure terminates in the manner described. In one case, however, a narrow canal, closed both above and below, was present as the representative of the rectum, and in another the sigmoid flexure terminated in a narrow canal which opened at the surface just underneath a tail-like process.

3. The presence of well-formed testes within the pelvis is rare. In most of the male cases described these organs have been found within the upper part of an
imperfect inguinal canal. In the present case no trace of an inguinal canal could be detected.

Muscular System.—The muscles of the upper portion of the body, including the latissimus dorsi and the abdominal muscles, were normal, except that there was no inguinal canal present among the last-named.

Muscles of the Inferior Extremity.—In describing these muscles considerable difficulty is experienced in determining the names to apply to them. On the whole, I think it would be better to avoid doing more than indicating the group to which any given muscle is supposed to belong, but, in accordance with other writers, I have placed the supposed name of each muscle in brackets after a description of its origin and insertion. These found on the anterior aspect of the lower limb are as follows:

1. A muscle which arose from the anterior superior iliac spine and the portion of the ilium below it, and which passed downwards and inwards to be inserted into the tibiae below the knee-joint (Sartorius). This muscle was supplied by the anterior crural nerve.

2. A muscle which arose from the front and side of the body and horizontal ramus of the os pubis, and running down parallel to the first-named muscle, was inserted into the front of the tibiae below the knee-joint (M. Gracilis). This muscle was also supplied by the anterior crural nerve.

3. A muscle which lay on a deeper plane than the two preceding, and which, arising from the front of the body and descending ramus of the os pubis, passed outwards to be inserted by a wide insertion along the outer border of the fused femora. This muscle probably represented fused adductors as it was supplied by the obturator nerve.

4. A muscle which arose from the front of the lower-
two-thirds of the fused femora and from a strong fibrous cord which intervened between it and its fellow on the opposite side. The fibres of this muscle passed downwards and outwards, and ended in a strong tendon, which was inserted into the outer aspect of the fused tibiae just below the knee-joint. The muscle was supplied by the obturator nerve, and must, therefore, be regarded as belonging to the adductor group.

5. The psoas muscle arose normally within the abdomen on each side, and was inserted into the posterior aspect of the horizontal ramus of the os pubis.

6. The iliacus muscle arose normally within the pelvis, and, after passing under Poupart's ligament, was inserted into the side of the fused femora just below the head of the bone and deep to the superior adductor group.

7. A small muscle, supplied by a twig of the anterior crural nerve, lay on each side on the front of the fused tibiae. The pair blended below into a single tendon which was inserted into the terminal cartilaginous nodules of the lower limb.

On the posterior aspect the following muscles were found:—

1. A muscle, corresponding to the gluteus maximus, arose from the lower part of the dorsum ilii on each side. The muscles of each side blended with one another below the coccyx and were inserted into the deep fascia of the back of the thigh. Above the origin of these muscles a large mass of fat covered over the dorsum ilii.

2. Deep to the gluteus maximus a muscle (gluteus medius and minimus) arose from the dorsum ilii, and was inserted into the summit of the great trochanteric region of the femora. This muscle was fused with its fellow in the middle line. It had no intrapelvic origin.

3. The whole of the back of the femora was covered
by a large muscular mass (extensor group), which arose from the back and sides of the fused bones, and was inserted below into two separate patellæ, which covered over the back of the joint. Into this mass there passed backwards on each side a muscle which arose from the ilium immediately behind the origin of the so-called Sartorius, and which probably corresponded to the rectus femoris muscle. It is not represented in the drawing, but lay parallel and behind the upper portion of the Sartorius before blending below with the extensor group. The two extensor groups were united in the middle line by a strong fibrous septum, but separated somewhat as they approached the patellæ. Each patella was united by a distinct ligamentum patellæ to the tibiae. This entire group of muscles was supplied by the anterior crural nerve.

4. A small slender muscle arose from the back of the tibia on each side. The two united into a single tendon, which was prolonged along the posterior aspect of the end of the limb. No nerve was traced to this muscle.

Other muscles:—

1. The quadratus lumborum muscle was present and normal.
2. Covering over the region of the thyroid foramina within the pelvis was an irregular muscular mass, which passed directly backwards and was inserted into the fascia of the pelvis. No other intra-pelvic muscles were present.

Nervous System.—The brain was so soft when the fetus was obtained that no systematic examination of it could be carried out. A superficial examination of the cortex, however, revealed nothing abnormal.

Spinal Cord.—This terminated at the lower end of the fourth lumbar vertebra. The lumbar swelling appeared
unusually big; otherwise the cord was quite normal. Sections revealed nothing abnormal either in the nerve cells or tracts of nerve fibres. So far as I am aware, only one other observation has been made on the spinal cord in sirenomelians, and that by Odisio (Stud. Anat. ed. Istol. Sopra un Sirenomeli, Torino). In his case the cord presented in the lumbar region a longitudinal group of fibres lying between the central canal and the anterior fissure. This was carefully looked for in my case, but was not present. The large size of the lumbar swelling corresponds with the large size of the cervical enlargement found by Theunier in a case of amelia (Bull. Soc. Anat. de Paris).

Peripheral Nerves.—The cutaneous nerves of the abdominal wall and thorax were normal. The external cutaneous nerves arose normally on each side from the lumbar plexus, and were distributed on the outer side of the lower limb as far as the knee. No genito-crural nerve was present. The anterior crural nerve was very large; it arose normally on each side from the second, third, and fourth lumbar nerves, and left the pelvis on the surface of the iliacus muscle. The main trunk, after giving off twigs to supply the sartorius, gracilis, and rectus femoris muscles, and also a branch which accompanied the femoral artery and supplied the muscle on the front of the tibiae and the skin over the upper region of the front of the leg, turned directly backwards and ended in the mass of extensor muscles at the back. The obturator nerve arose from the third and fourth lumbar nerves on each side. It supplied the upper and lower adductor group of muscles on the front of the thigh, after its emergence from the pelvis in the usual manner. The lumbo-sacral cord was normal: it turned downwards into the pelvis and joined with the remaining sacral nerves
to form a single nerve band which left the pelvis through a foramen immediately in front of the sacrum. This trunk terminated in the gluteal muscles at the back.

Vascular System.—The general direction of the common and external iliac vessels has already been described. A
small internal iliac vessel was present on each side, and emerged from the pelvis along with the great sciatic nerve to end in the gluteal group of muscles. The femoral artery immediately below Poupart's ligament gave off a rather large muscular twig which accompanied the anterior crural nerve into the extensor group of muscles. The main trunk then continued down along the front of the thigh between the sartorius muscle and the adductor groups of muscle, and, reaching the front of the tibia below the knee, was continued down as a very slender trunk on each side towards the extremity of the limb. A superficial saphenous vein was present on each side, and joined the main femoral vein below Poupart's ligament.

Osseous System.—The bones of the head and arms were normal. There were thirteen ribs on each side, thirteen dorsal vertebrae, and five lumbar vertebrae. This abnormality in number of ribs and vertebrae is in accordance with what is usually found in these monsters, and is of importance, I think, in showing that the anomaly is more than a local one. The body of the last lumbar vertebrae articulated on each side with the ala of the ilium. The sacrum and coccyx were represented by four incomplete bony vertebrae, and by four cartilaginous nodules, the whole structure being directed almost straight backwards between the iliac bones.

The appearance of the pelvis is well seen in the accompanying figure. The iliac fossae were expanded and directed more forwards than inwards, and the iliac crests were very slightly curved. The bodies of the pubic bones were directed forwards in the shape of a beak projecting over the upper part of the femora. The true pelvis was very shallow, and had an almost complete osseous floor owing to the union in the middle line of the
tubera ischii and of the ischial spines. The union took place in such a way as to leave three outlets from before backwards. The first constituted the subpubic angle, between the tubera ischii and the ischial spines, and the

A. Pelvis and skeleton of the fused lower limbs as seen from the front.
B. Pelvis and skeleton of the fused lower limbs as seen from behind.

and was filled in by fibrous tissue; the second intervened third between the ischial spines and the front of the sacrum. Through this last-named the gluteal nerves escaped from the pelvis.
By Dr. T. Gillman Moorhead.

The femora were completely fused, the only evidence of their bilateral origin being a slight groove along the middle line, both in front and behind, and the presence of a small foramen in the lower third of the shaft. The cartilaginous heads projected outwards on each side from the upper end of the bone to articulate with the acetabula, and posterior to them an extensive shelf of bone was present immediately below the pelvis, representing the fused great trochanters. Each hip-joint was surrounded by a distinct capsule, and both the acetabular surfaces of the innominate bones and the heads of the femora were much flattened. The lower ends of the femora presented three distinct articular surfaces, separated from one another by septa of fibrous tissue which passed down to be attached to the intervals between corresponding articular surfaces upon the tibiae. The two lateral articular surfaces were of the normal shape of the internal condylar surface of the femora, while the middle surface was triangular in shape, and presented in the centre a slight ridge as indication of its bilateral character. As already stated, two separate patellae were present on the back of the joint.

Both tibiae were completely fused to form a single bone, large and expanded at its upper end, and gradually tapering below. No trace of the fibulae was present. The lower end of the limb was composed of five small cartilaginous nodules united to one another and to the lower end of the tibiae by fibrous tissue. In the upper two of these nodules a bony centre was present.

It is not my intention to discuss here the various theories that have been advanced to account for the occurrence of sirenomelian monsters, but before concluding I would wish to express the opinion that no mechanical compression theory can account for all the abnormalities
found in this type, and that, therefore, it is necessary to refer the cause back to some defect of the early ovum. How compression by the amnion, or a fusion of lower limbs after original separate development, can account for the irregularities in number of the vertebrae and ribs is, I think, impossible to conceive. The irregularity of the bones of the trunk is indeed, I think, the strongest evidence that exists in favour of primary as against secondary malformation, inasmuch as all the other peculiarities present can to some extent be explained by a theory of compression.

REFERENCES.


Steinhaus, F. Virchow’s Archivs., 143. P. 227.


Lyceosthenes. Prodigiî Obstantorum Chronicon. 1557.

Julii Obsequentis Prodigiorum Liber. 1552.

Aldrovandus. Historia Monstrorum. 1642.


HISTOGENESIS OF THE GREY MATTER OF THE CEREBELLUM.

By D. J. COFFEY, M.B. R.U.I.;
Professor of Physiology, Catholic University Medical School.

[Read in the Section of Anatomy and Physiology, April 28, 1905.]

The present paper is a preliminary communication on the histogenesis of the grey matter of the cerebellum, touching incidentally on the relations of the structural connections of the functions carried out by the cerebellum as a central mechanism of co-ordination. In the mammal at birth the general anatomical formation of the hemispheres and their laminae is well established. The neurone structure is, however, in a most rudimentary condition. The grey matter of the cortex consists of close-packed layers of cells, which, as yet, have only imperfectly asserted their neurone nature. The chromate of silver method reveals nothing of the dendritic growth so characteristic a few weeks afterwards. The layer of Obersteiner, the superficial germinal layer, as described by Herrick and others, consists of small cells in several rows, underneath which a layer of large cells is identified consisting of elements of Pürkinje, but devoid of branching processes, and of others of epithelial or ependyma nature. The latter shows a profusion of vertical, outwardly directed, processes which reach to the surface, and form the first clearly recognisable scaffolding along which the development of the cells produced in the superficial layer is determined. It results from it that the small neurones produced in the superficial layer acquire a vertical orientation, and, becoming bipolar, are gradu-
ally made to descend into the cerebellar substance until the cell body lies below the large Pürkinje elements. Synchronously with this determination of direction to the cells, which are now the granule cells, there is an increasing ramification of the fibres of the white matter lying underneath. This white matter consists mostly of afferent fibres from the inferior and middle peduncles. These afferent fibres were exhibited in the sections as the only neurone elements stainable in the mammal at birth whose trend could be positively asserted. Many of them acquire their ultimate character as the "moss fibres" when they meet with the developing granule cell. Other cells derived from the superficial germinal layer, somewhat larger, acquire the neurone differentiation a little later, synchronising with the development of the outspreading dendrites of the Pürkinje's elements. It would seem that this association in growth determines a horizontal orientation of those latter superficial cells, and that such is probable is borne out by their ultimate relation as "basket cells" to Pürkinje's elements. The next neurone, the Golgi cell, does not apparently come from the superficial germinal layer, but lies from the first near the Pürkinje element, and its development is related to the later stages of the granule cell development. Its rôle of "accumulator cell" for groups of granule cells suggested by Cajal seems most appropriate. The second class of afferent fibres, the tendril fibres, as demonstrated by Cajal, are developed in the closest relation to the growth of the Pürkinje cells. If, as described by Cajal, they bring cerebral stimuli to the cerebellar cortex, is it possible that, bringing impulses to the protoplasmic substance of the Pürkinje cells, rather than to the terminations of the dendrites, they are nutritional, that is influencing irritability of the protoplasmic substance,
rather than bringing impulses which pass further to peripheral organs? This view would accord well with the physiological relation of cerebrum to cerebellum. As the unfolding of this cell structure goes on during the few weeks following birth in the domestic mammals, the completion of the connections coincides with the development of co-ordinating power.
IRREGULAR FORM AND POSITION OF THE COLON.

By ALEC FRASER, F.R.C.S.,
Professor of Anatomy, R.C.S.

[Read in the Section of Anatomy and Physiology, April 28, 1905.]

In the specimen exhibited (a male past the meridian of life) the position of the large bowel is normal from the cæcum to the splenic flexure, at which point the irregular course begins. The bowel from this flexure passes straight towards the terminal duodenum, to the lower margin of which it is attached, then over the structures in front of the vertebral column, along the attachment of the mesentery to which it is adherent, until it almost reaches the ilio-colic junction. At this point it forms a long loop, the upper end of which reaches up as far as the right vault of the diaphragm, passing in front of the transverse colon, and lying between the muscle and the liver. The terminal end of the loop passes over the pelvic brim on the right side, then forms the pelvic colon, and reaches the middle line at the beginning of the rectum. In this case then the left half of the posterior abdominal wall is entirely clear of the colon, and any one expecting to reach the bowel through the posterior abdominal wall would have been disappointed. Almost the whole length of the kidney in front and the psoas muscle is covered by what is called parietal peritoneum. During the last fifteen years every abdominal cavity (nearly 1,500 in number) passing through the School of the Royal College of Surgeons has been examined and noted by me, and I have found frequent irregularities of
the ascending, the transverse, and the descending division of the large bowel both in form and position. The most numerous as regards form are due to the formation of the U-shaped loops which may be present in one of the divisions in one subject, in two of the divisions in a second, and in all three divisions in a third. As regards position, the transverse colon may reach as high as the left vault of the diaphragm, generally in young powerful males with the stomach empty, or as low down as the pubes, or even the pelvic cavity, in old females. I once found the cæcum implanted in the left iliac fossa, and this was no developmental irregularity. In all these cases this is the first time I have found the left lateral half of the abdominal wall clear of the colon behind.
INDEX.

[The names of Contributors are printed in Small Capitals.]

Actinomycosis of the right kidney, H. C. Earl, 339.
Acute intestinal obstruction by a gall stone, T. E. Gordon, 118.
Anæmic infarction of liver, A. C. O'Sullivan, 356.
Anatomy of sirenomelian monster, T. G. Moorhead, 382.
Aneurysm, aortic, perforating the oesophagus, J. A. Matson, 354.
Aneurysm, aortic, rupturing into pleura, T. G. Moorhead, 354.
Aneurysm, innominate, sequel to, James Craig, 31.
Anomaly of cardiac valve, T. G. Moorhead, 358.
Aseptic v. antiseptic methods in healing of wounds, W. S. Haughton, 91.

Ball, C. A., conservative perineal prostatectomy, 134.
Bennett, E. H., fractures of first costal cartilage, 349.
Bennett, E. H., oblique fracture of tibia, 160.
Benson, A. H., and H. C. Mooney, congenital sarcoma of the eyelid, 360.
Benson, A. H., and H. C. Mooney, melano-sarcoma of the conjunctiva, 347.
Bewley, H. T., syringomyelia, 74.
Bossi's dilator, A. J. Smith, 223.

Cecum, tuberculous disease of, R. A. Stone, 358.
Caisson disease, J. B. Coleman, 71.
Carbon monoxide poisoning, W. J. Thompson, 376.
Carcinomata, Sir Thornley Stoker, 357.
Cholecystotomy for acute cholecystitis, R. C. B. Maunsell, 125.
Chorea, case of hysterical, James Craig, 73.
Cirrhosis of liver, George Peacocke, 332.
Clinical experiences, R. Travers Smith, 42.
Closure of cleft palate in infants, Sir Thornley Stoker, 196.
Coffey, D. J., histogenesis of the grey matter of the cerebellum, 397.
Coleman, J. B., caisson disease, 71.
Colon, irregular form and position of the, Alec Fraser, 400.
Index.

Colour tests for the chief nitrogenous constituents of urine, Walter G. Smith, 50.

Congenital sarcoma of the eyelid, A. H. Benson and H. C. Mooney, 360.

Conjunctiva, melanoma-sarcoma of the, A. H. Benson and H. C. Mooney, 347.

Costal cartilage, fractures of first, E. H. Bennett, 349.

Craig, James, case of hysterical chorea, 73.

Craig, James, sequel to a case of innominate aneurysm reported in 1893, 31

Cystic endometritis, R. D. Purefoy, 217.

Diarrhoeal diseases in Dublin, 1904, Sir J. W. Moore, 361.

Drury, H. C., pneumonia in pregnancy, 35.

Dunne, F. J., tuberculosis of lungs and knee treated by Deny's tuberculin, 76.

Earl, H. C., large fibromyoma from the rectum, 330.

Earl, H. C., primary actinomycosis of the right kidney with embolical cerebral abscess, 339.


Earl, H. C., and J. B. Story, glioma of the retina, 355.

Earth temperature and diarrhoeal diseases in Dublin, 1904, Sir J. W. Moore, 361.

Endocarditis, infective, George Peacocke, 336.

Endometritis, cystic, R. D. Purefoy, 217.


Fibromyoma from the rectum, large, H. C. Earl, 330.

Fibromyomata, influence of, on pregnancy and parturition, 290.

Fraser, Alec. irregular form and position of the colon, 400.

Gall stone, intestinal obstruction by a, T. E. Gordon, 118.

Gastvic ulcer, L. G. Gunn, 357.

Gastric ulcer, inefficient operations for, A. B. Mitchell, 81.

Genitals, wounds of female, R. J. Kinkead, 245.

Glanders, a case of, E. F. Stephenson, 313.

Glioma of the retina, J. B. Story and H. C. Earl, 355.

Glioma of the retina, H. C. Mooney, 355.

Gordon, T. E., intestinal obstruction by a gall stone, 118.

Grey matter of the cerebellum, D. J. Coffey, 397.

Gunn, L. G., gastric ulcer, 257.

Gunn, L. G., painless haematuria, 169.

Gunn, L. G., Potts' caries, 357.
Index.

Hæmatological observations, E. J. McWeeney, 297.

Haematuria, painless, L. G. Gunn, 169.

Haughton, W. S., healing of wounds; aseptic v. antiseptic methods, 91.

Herniotomy, E. H. Taylor, 111.

Histogenesis of the grey matter of the cerebellum, D. J. Coffey, 397.

Hodgkin's disease occurring in twins, George Peacocke, 1.

Hysterical chorea, a case of, James Craig, 73.

Infected endocarditis, George Peacocke, 336.

Innominate aneurysm, James Craig, 31.

Intestinal obstruction by a gall stone, T. E. Gordon, 118.

Irregular form and position of the colon, Alec Fraser, 400.

Jellett, H., case of labour in a unilateral synostotic pelvis, 250.

Joint excision, D. Kennedy, 163.


Kennedy, D., joint excision, 163.

Kidney, large white, George Peacocke, 25.

Kindead, R. J., puerperal convulsions, 230.

Kindead, R. J., wounds of female genitals, 245.

Kirkpatrick, T. P. C., sporatic cretinism, 70.


Liver, cirrhosis of, George Peacocke, 332.

Lymphæmia, acute, E. J. McWeeney, 315.

Lymphæmia, chronic, E. J. McWeeney, 297.

McWeeney, E. J., case of acute lymphæmia, 315.

McWeeney, E. J., case of chronic lymphæmia, 297.


Matson, J. A., aortic aneurysm perforating the oesophagus, 354.

Maunsell, R. C. B., cholecystotomy for acute cholecystitis during convalescence from enteric fever, 125.

Maunsell, R. C. B., and H. C. Earl, endothelioma of uterus, 356.

Mitchell, A. B., inefficient operations for gastric ulcer, 81.

Mooney, H. C., and A. H. Benson, congenital sarcoma of the eyelid, 360.

Mooney, H. C., melan-sarcoma of the conjunctiva, 347.

Mooney, H. C., and H. C. Earl, glioma of the retina, 355.
MOORE, SIR J. W., earth temperature and diarrheal diseases, 361.
MOORHEAD, T. G., the anatomy of a sirenomelian monster, 382.
MOORHEAD, T. G., anomaly of cardiac valve, 358.
MOORHEAD, T. G., aortic aneurysm rupturing into pleura, 354.
MOORHEAD, T. G., Bacillus coli communis as a cause of septicemia, 54.

Nitrogenous constituents of urine, some colour tests for the chief, WALTER G. SMITH, 50.

Observations on Bossi’s dilator, A. J. SMITH, 223.
O’CARROLL, JOSEPH, syringomyelia, an uncommon case of, 6.
Operation for procidentia uteri, E. H. TWEEDY, 240.
O’SULLIVAN, A. C., anaemic infarction of liver, 356.

Palate, closure of cleft, SIR T. STOKER, 196.
Paratyphoid fever, A. R. PARSONS, 16.
PARLATO, E., Sicily as a winter health resort, 372.
PARSONS, A. R., typhoid and paratyphoid fever, 16.
PEACOCKE, GEORGE, cirrhosis of liver, 332.
PEACOCKE, GEORGE, Hodgkin’s disease occurring in twins, 1.
PEACOCKE, GEORGE, infective endocarditis, 336
PEACOCKE, GEORGE, large white kidney, 25.
Perineal prostatectomy, C. A. BALL, 134.
Pneumonia in pregnancy, H. C. DRY, 35.
Pott’s caries, L. G. GUNN, 357.
Pregnancy, pneumonia in, H. C. DRY, 35.
Procidentia uteri, operation for, E. H. TWEEDY, 240.
Puerperal convulsions, R. J. KINHEAD, 230.
PUREFOY, R. D., cystic endometritis, 217.
Purulent cavities, treatment of, R. H. WOODS, 182.

Recrudescence ulceration of stomach, W. J. THOMPSON, 359.
Rotunda Hospital Report, 1903-4, 256.

Septicaemia, Bacillus coli communis as a cause of, T. G. MOORHEAD, 54.
Sicily as a winter health resort, E. PARLATO, 372.
SMITH, A. J., observations on Bossi’s dilator, 223.
SMITH, R. TRAVERS, some miscellaneous clinical experiences, 42.
SMITH, WALTER G., on some colour tests for the chief nitrogenous constituents of urine, 50.
Index.

Sporadic cretinism, T. P. C. Kirkpatrick, 70.
Stephenson, E. F., a case of glanders, 313.
Stoker, Sir Thornley, carcinomata, 357.
Stoker, Sir Thornley, closure of cleft palate in infants, 196.
Story, J. B., and H. C. Earl, glioma of the retina, 355.
Synostotic pelvis, case of labour in a unilateral, H. Jellett, 250.
Syringomyelia, Joseph O’Carroll, 6.
Syringomyelia, H. T. Bewley, 74.

Taylor, E. H., new methods for the performance of herniotomy, 111.
Thompson, W. J., case of carbon monoxide poisoning, 376.
Thomson, W. J., recrudescent ulceration of stomach, 359.
Tibia, oblique fracture of, E. H. Bennett, 160.
Tuberculosis of lungs and knee treated by Deny’s tuberculin, F. J. Dunne, 76.

Tweed, E. H., operation for procidentia uteri, 240.
Tweed, E. H., Rotunda Hospital Report, 256.
Typhoid fever, A. R. Parsons, 16.

Unilateral synostotic pelvis, case of labour in, H. Jellett, 250.
Urine, some colour tests for the chief nitrogenerous constituents of, Walter G. Smith, 50.


Woods, R. H., treatment of purulent cavities, 182.
Wounds, healing of, W. S. Haughton, 91.
Wounds of the female genitals, R. J. Kinkead, 245.

Printed by John Falconer, 53 Upper Sackville-street, Dublin.