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NEW YORK

ACADEMY OF MEDICINE,

INSTITUTED 1847.

SECOND SERIES. VOLUME THREE.

PRINTED FOR THE ACADEMY.

1883.

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1858....*JOHN P. BATCHELDER, M.D.
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1867....ALFRED C. POST, M.D., LL.D.
1869....*HENRY D. BULKLEY, M.D.
1871....*EDMUND R. PEASLEE, M.D., LL.D.
1873....AUSTIN FLINT, M.D., LL.D.
1875....SAMUEL S. PURPLE, M.D.
1879....FORDYCE BARKER, M.D., LL.D.

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* Deceased.

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1847.	JOHN B. BECK, M.D.
1847.	JOHN W. FRANCIS, M.D., LL.D.
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1850.	JAMES C. BLISS, M.D.
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1856.	JOHN WATSON, M.D.
1856.	JACKSON BOLTON, M.D.
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1857.	FORDYCE BARKER, M.D., LL.D.
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1877 ARNOLD, JOHN W. S., M.D.
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1877 ASSENHEIMER, AUGUSTUS, M.D.
1875 AYERS, SAMUEL, M.D.

1882 BACON, GORHAM, M.D.
1878 BADGER, WILLIAM, M.D., Flushing, N. Y.
1865 BAHAN, THOMAS S., M.D.
1867 BALL, A. BRAYTON, M.D.
1880 BANGS, L. BOLTON, M.D.
1862 BANKS, JAMES L., M.D., Trust. 1869–'79.
1854 BARKER, FORDYCE, M.D., LL.D., V. P. 1857, 1876–'79. P. 1879, now in office.
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1854 *BARRY, ROBERT A., M.D., *Obiit* 7th January, 1882, *æt.* 57.
1862 BARSTOW, JOSIAH W., M.D., Flushing, L. I.
Original. *BATCHELDER, JOHN P., M.D., V. P. 1851–'52; P. 1858, *Obiit* 8th April, 1868, *æt.* 83.
1868 BAYLES, GEORGE, M.D.
Original. *BEADLE, EDWARD L., M.D., C. S. 1850–'52; Trust. 1850; V. P. 1853 to 1858, Poughkeepsie, N. Y., *Obiit* 5th April, 1882, *æt.* 74.
Original. *BEALES, JOHN C., M.D., *Obiit* 25th July, 1878, *æt.* 74.
Original. *BEALS, GORHAM, M.D., *Obiit* 9th January, 1848, *æt.* 29.
1870 *BEARD, GEORGE M., M.D., *Obiit* 23d January, 1883, *æt.* 43.
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1880 BECKWITH, FRANK E., M.D., New Haven, Conn.
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1871 BELL, CHRISTOPHER M., M.D.
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1877 BERMINGHAM, E. J., M.D.
1864 *BIBBINS, WILLIAM B., M.D., Trust. 1867–'71, *Obiit* 16th January, 1871, *æt.* 46.
1882 BILLINGS, JOHN H , M.D.
1871 BILLINGTON, CORNELIUS E., M.D.
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Original. *BLOIS, SAMUEL, M.D., *Obiit* 19th October, 1873.

1857 BLUMENTHAL, MARK, M.D.

1873 *BOGERT, CORNELIUS R., M.D., *Obiit* 10th November, 1877, *æt.* 77.

Original. *BOLTON, JACKSON, M.D., R. S. 1852; V. P. 1856, *Obiit* 16th February, 1866, *æt.* 51.

1848 *BOORAEM, AUGUSTUS C., M.D., *Obiit* 16th December, 1871, *æt.* 46.

1880 BOSWORTH, FRANK H., M.D.

Original. *BOYD, THOMAS, M.D., *Obiit* 18th March, 1856, *æt.* 83.

1869 BOZEMAN, NATHAN, M.D.

1876 BRADLEY, EDWARD, M.D.

1847 *BRADY, PATRICK J., M.D., *Obiit* 23d October, 1856, *æt.* 42.

1882 BRANDEIS, RICHARD C., M.D.

1878 BRANDIS, ADOLPH C., M.D.

1880 BREAKELL, JAMES A., M.D.

1880 BRONSON, EDWARD B., M.D.

1863 BROWN, D. TILDEN, M.D.

1867 *BROWN, JAMES L., M.D., *Obiit* 4th February, 1873, *æt.* 41.

1848 *BROWN, WILLIAM K., M.D., Brooklyn, L. I., *Obiit* 4th July, 1879, *æt.* 72.

1848 *BRUENINGHAUSEN, CHARLES, M.D., *Obiit* 20th August, 1876, *æt.* 67.

1880 BRUSH, EDWARD F., M.D.

1875 BRYANT, JOSEPH D., M.D.

Original. *BUCK, GURDON, M.D., C. S. 1848; V. P. 1852, 1855, 1856, 1859, 1860, *Obiit* 6th March, 1877, *æt.* 69.

1855 *BUDD, CHARLES A., M.D., *Obiit* 17th May, 1877, *æt.* 43.

Original. *BULKLEY, HENRY D., M.D., V. P. 1862–'68; P. 1870–'71; Trust. 1871, *Obiit* 4th January, 1872, *æt.* 67.

1874 BULKLEY, LUCIUS D., M.D.

1880 BULL, CHARLES S., M.D.

1880 BULL, WILLIAM T., M.D.

1879 BULLARD, WILLIAM E., M.D.

1848 *BULLUS, EDWARD, M.D., *Obiit* 25th September, 1854, *æt.* 50.

1879 *BUMSTEAD, FREEMAN J., M.D., LL.D., *Obiit* 28th November, 1879, *æt.* 53.

1879 BURCHARD, THOMAS H., M.D.

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1861 BURKE, JOHN, M.D.
1865 BURRALL, FREDERICK A., M.D.
1877 BUTLER, GEORGE H., M.D.
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Original. *CAMERON, JAMES, M.D., *Obiit* 12th December, 1851, *æt.* 66.
1880 CAMERON, EDWARD M., M.D.
1862 *CAMMAN, GEORGE P., M.D., *Obiit* 14th February, 1863, *æt.* 58.
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Original. *CAMPBELL, JAMES, M.D., *Obiit* 12th March, 1853, *æt.* 59.
1869 *CARO, SALVATORE, M.D., *Obiit* 1879.
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1858 *CARRINGTON, WILLIAM A., M.D., *Obiit* 17th July, 1866.
Original. *CARTER, GALEN, M.D., V. P. 1849–'52; Trust. 1851, *Obiit* 2d April, 1870, *æt.* 74.
1879 CASTLE, FREDERICK A., M.D.
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1869 CHADSEY, ALONZO J., M.D.
1847 CHALMERS, THOMAS C., M.D.
1879 CHAMBERLAIN, GEORGE W., M.D.
1862 CHAMBERLAIN, WILLIAM M., M.D., R. S. 1865–'68.
1879 CHAMBERS, PORTER F., M.D.
Original. *CHAPIN, JOHN R., M.D., *Obiit* 23d June, 1852, *æt.* 41.
1872 CHAUVEAU, JEAN F., M.D.
1847 *CHEESMAN, JOHN C., M.D., *Obiit* 11th October, 1862, *æt.* 75.
1871 CHEESMAN, TIMOTHY MATLACK, M.D.
1882 CHEESMAN, TIMOTHY M., Jr., M.D.
1865 *CHILDS, TIMOTHY, M.D., *Obiit* 3d September, 1865, *æt.* 42.
1856 CHURCH, ALLEN S., M.D.
1859 *CHURCHILL, CHARLES W., M.D., *Obiit* 27th October, 1859.
1878 CISNÉROS, JUAN, M.D.
1856 CLARK, ALONZO, M.D., LL.D.
1870 CLARK, JAMES G., M.D., W. New Brighton, S. I., N. Y.
Original. *CLARKSON, CORNELIUS V., M.D., *Obiit* 1st June, 1877, *æt.* 63.
Original. *CLEMENTS, JAMES W. G., M.D., *Obiit* 1882.
1879 CLEVELAND, CLEMENT, M.D.
1859 *COCHRAN, GEORGE, M.D., Brooklyn, *Obiit* 19th November, 1872, *æt.* 40.
1883 COLLINS, STACY B., M.D.
1854 *CONANT, DAVID S., M.D., *Obiit* 8th October, 1865, *æt.* 40.
1864 *CONNOLLY, JAMES J., M.D., *Obiit* 18th June, 1871, *æt.* 36.

ELECTED.

Original. *COOPER, JAMES S., M.D., *Obiit* 11th April, 1867, *æt.* 49.
Original. *COVEL, JOHN C., M.D., *Obiit* 4th November, 1860, *æt.* 64.
1876 COWLES, EDWARD O., M.D.
1862 *COX, HENRY G., M.D., *Obiit* 29th May, 1866, *æt.* 47.
1870 CRAMPTON, HENRY E., M.D.
1849 CRANE, JAMES, M.D., Brooklyn, N. Y.
1847 CRANE, JOHN J., M.D.
1866 CRANE, JOSEPH S., M.D.
1851 *CREVELING, ABRAHAM, M.D., *Obiit* 28th April, 1853, *æt.* 39.
1880 CUSHMAN, WILLIAM F., M.D.
1880 CUTTER, GEORGE R., M.D.
1877 CYPERT, JOHN R., M.D.

1868 *DALTON, EDWARD B., M.D., *Obiit* 13th May, 1872, *æt.* 37.
1856 DALTON, JOHN C., M.D., Orator 1873; V. P. 1874 to 1877.
1867 DANA, SAMUEL W., M.D.
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1879 DAWSON, BENJAMIN F., M.D.
1879 DEGARMO, WILLIAM B., M.D.
Original. *DELAFIELD, EDWARD, M.D, *Obiit* 13th February, 1875, *æt.* 80.
1880 DELAFIELD, FRANCIS, M.D.
1880 DELAVAN, D. BRYSON, M.D.
1876 *DELUNA, ABELARDO B., M.D., *Obiit* March, 1883.
1880 DENSLOW, LEGRAND N., M.D.
1879 DENNIS, FREDERICK S., M.D.
1859 DERBY, EDWARD W., M.D.
1877 DERBY, RICHARD H., M.D.
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1879 DESSAU, S. HENRY, M.D.
1880 DEXTER, B. F., M.D.
1880 DIEFFENBACH, R. G. L., M.D.
1860 *DONAGHE, WILLIAM R., M.D., *Obiit* 18th July, 1866, *æt.* 36.
1856 DOUGLAS, JOHN H., M.D.
1848 *DOUGLAS, ROBERT, M.D., *Obiit* 25th July, 1861, *æt.* 47.
Original. *DOWNS, HENRY S., M.D., *Obiit* 2d May, 1879, *æt.* 67.
Original. *DRAKE, BENJAMIN, M.D., C. S. 1847, *Obiit* 11th January, 1871, *æt.* 65.
1858 DRAPER, WILLIAM H., M.D.
1847 DUDLEY, WILLIAM H., M.D., Brooklyn, N. Y.
1875 DU BOIS, M. B., M.D.
1880 DUNCAN, WILLIAM F., M.D.

ELECTED.

1877 DURANT, GHISLANI, M.D.
1847 *DWIGHT, WILLIAM W., M.D., *Obiit* 11th July, 1861, *æt.* 54.

1847 *EARLE, EDWARD, M.D., *Obiit* 21st August, 1849, *æt.* 37.
1878 EDEN, JOHN H., M.D., Fordham, N. Y.
1851 *ELDER, ALEXANDER, M.D., *Obiit* 3d February, 1875, *æt.* 70.
1858 ELIOT, ELLSWORTH, M.D.
1858 *ELLIOT, GEORGE T., M.D., *Obiit* 28th January, 1871, *æt.* 43.
1870 *ELLIS, HENRY A., M.D., *Obiit* 25th January, 1876, *æt.* 41.
Original. *ELLIS, SAMUEL C., M.D., *Obiit* 20th July, 1874, *æt.* 71.
1862 ELSBERG, LOUIS, M.D.
1856 EMMET, THOMAS ADDIS, M.D.
1880 EMERSON, J. H., M.D.
Original. *ENOS, DEWITT C., M.D., Brooklyn, N. Y., *Obiit* 14th December, 1868, *æt.* 48.

1864 FARNHAM, HORACE P., M.D., T. 1878; V. P. 1883, now in office.
1878 FARRINGTON, JOSEPH O., M.D.
Original. *FERGUSON, JOHN T., M.D., *Obiit* 11th October, 1859, *æt.* 55.
1847 *FERRIS, FLOYD T., M.D., *Obiit* 6th November, 1855, *æt.* 62.
1855 FINNELL, THOMAS C., M.D.
1847 *FISK, LYMAN, M.D., *Obiit* 1st August, 1859, *æt.* 36.
1847 *FITCH, JAMES D., M.D., *Obiit*, 1881.
1879 FLEMING, MARTIN J., M.D.
1862 FLINT, AUSTIN, M.D., Orator 1868; V. P. 1871 and 1872; P. 1873 and 1874; Trust. 1875 to 1880.
1862 FLINT, AUSTIN, Jr., M.D.
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1870 FOSTER, FRANK P., M.D.
Original. FOSTER, JOEL, M.D., V. P. 1859 to 1862; Trust. 1862 to 1866.
Original. *FOSTER, SAMUEL CONANT, M.D., R. S. 1855 and 1856; V. P. 1858 and 1859; Orator 1861 and 1862, *Obiit* 18th April, 1873, *æt.* 56.
1874 FOWLER, GEORGE B., M.D.
1880 FOX, GEORGE H., M.D.
1856 *FOY, MICHAEL E., M.D., *Obiit* 9th June, 1861, *æt.* 37.
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1880 Frazer, John Gordon, M.D.
1870 Frothingham, William, M.D.
1879 Fruitnight, J. Henry, M.D.
1871 Fuller, Robert M., M.D.
1865 Furman, Guido, M.D.

Original. Garrish, John P., M.D.
1879 Garrigues, Henry J., M.D.
1870 Gay, Harvey S., M.D.
1848 *Gescheidt, Anthony. M.D., *Obiit* 20th August, 1868, *æt.* 68.
1877 Gibney, Virgil P., M.D.
1863 Gilfillan, William, M D., Brooklyn, N. Y.
1847 *Gilford, Jacob T., M.D., *Obiit* 11th June, 1869, *æt.* 63.
1877 Gillette, Walter R., M.D.
1879 *Glazier, W. C. W., M.D.
1882 Goldsmith, W. B., M.D.
1880 Goldthwaite, H., M.D.
1856 Gomez, Horatio, M.D.
1882 Goodwillie, D. H., M.D.
1881 Gorton, O. A., M.D.
1856 Gouley, John Wm. S., M.D.
1847 *Graham, John, M.D., *Obiit* 20th May, 1847, *æt.* 46.
1848 *Green, David, M.D., *Obiit* 18th October, 1856, *æt.* 60.
Original. *Green, Horace, M.D., LL.D., *Obiit* 29th November, 1866, *æt.* 63.
Original. *Greene, Isaac, M.D., *Obiit* 2d July, 1854, *æt.* 40.
Original. *Griscom, John H., M.D., Orator 1854; V. P. 1854; Trust. 1851, and 1856 to 1859, *Obiit* 28th April, 1874, *æt.* 64.
1872 Griswold, Henry, M.D.
1883 Griswold, Gaspar, M.D.
1847 *Guernsey, Peter B., M.D., *Obiit* 26th November, 1873, *æt.* 69.
1847 *Gunn, Alexander N., M.D., *Obiit* 21st December, 1871, *æt.* 60.

1880 Habirshaw, John, M.D.
1875 Hadden, Alexander, M.D.
Original. Hall, Edwards, M.D.
1874 Hall, William H., M.D.
Original. *Halsted, Jonathan, M.D., *Obiit* 10th April, 1856, *æt.* 46.
1873 Hamilton, Allen McL., M.D., Statistical Secretary 1874 to 1879.
1864 Hamilton, Frank H., M.D., LL.D., V. P. 1880.

ELECTED.

1874 HANKS, HORACE T., M.D., A. S. 1874–'77; R. S. 1877 to 1879; V. P. 1882.
1857 HARRIS, ELISHA, M.D.
1881 HARDY, W. L., M.D.
1879 HARRISON, GEORGE T., M.D.
1859 *HARSEN, JACOB, M.D., Trust. 1862 to 1866, *Obiit* 31st December, 1862, *æt.* 54.
1879 *HART, H. LE BARON, M.D., *Obiit* 21st February, 1880, *æt.* 38.
Original. *HART, JOHN, M.D., *Obiit* 9th August, 1867, *æt.* 57.
1877 HARWOOD, EDWARD C., M.D.
1865 *HAZLETT, JOHN, M.D., *Obiit* 4th March, 1870, *æt.* 53.
1865 HEDGES, DAVID A., M.D.
1882 HEALY, T. F., M.D.
1880 HEINEMAN, HENRY N., M.D.
1883 HEITZMAN, CHARLES, M.D.
1881 HENNA, J. J., M.D.
1871 HENRY, MORRIS H., M.D.
1847 *HENSCHEL, CHARLES, M.D., *Obiit* 18th September, 1872, *æt.* 63.
1867 HERRICK, EVERETT, M.D., T. 1882.
1857 *HERZOG, MAX, M.D.
1863 *HEWIT, HENRY S., M.D., *Obiit* 19th August, 1873, *æt.* 47.
1856 HEYWOOD, CHARLES F., M.D., R. S. 1857 and 1858.
1879 HICKOK, GEORGE B., M.D.
1879 HILLIS, T. J., M.D.
1856 †HINTON, JOHN H., M.D., R. S. 1861 to 1865.
1854 *HIRSCH, SIMON, M.D., *Obiit* 23d April, 1878, *æt.* 61.
1880 HITCHCOCK, URBAN G., M.D.
Original. *HOBART, WILLIAM H., M.D., *Obiit* 21st January, 1857, *æt.* 52.
1877 HODGMAN, ABBOTT, M.D.
1847 *HOGAN, DANIEL M., M.D., *Obiit* 1849.
1871 HOGAN, EDWARD J., M.D.
1867 *HOGAN, MICHAEL, M.D., *Obiit* 1883.
1854 HOLCOMBE, WILLIAM F., M.D., A. S. 1856.
Original. *HORSFIELD, THOMAS W., M.D., *Obiit* 19th February, 1868, *æt.* 64.
1872 HOWE, JOSEPH W., M.D.
Original. HUBBARD, SAMUEL T., M.D., C. S. 1853 to 1858; V. P. 1873 to 1876; Trust. 1862 to 1873, 1876, now in office.
1874 HUDSON, E. DARWIN, Jr., M.D., Orator 1875, L. 1883.
1866 HULL, JOSEPH J., M.D.
1867 HUMPHREYS, GEORGE H., M.D.
1878 HUNTER, ALEXANDER S., M.D.

ELECTED.

1849 *HUNTER, ABRAHAM T., M.D., *Obiit* 1st August, 1849, *æt.* 52.
1879 HUNTER, JAMES B., M.D.
1879 HUSTACE, FRANCIS, M.D.
1856 HUSTED, NATHANIEL C., M.D.
Original. *HUTCHINSON, EUGENE F., M.D., *Obiit* 2d March, 1848, *æt.* 24.
1857 HUTCHISON, JOSEPH C., M.D., Brooklyn, N. Y., V. P. 1869 to 1873.
1880 HUTTON, ALLAN C., M.D.
1882 HYDE, F. E., M.D.
1848 *HYSLOP, JAMES, M.D., *Obiit* 17th May, 1870, *æt.* 53.

1850 *ISAACS, CHARLES E., M.D., Brooklyn, N. Y., V. P. 1858, *Obiit* 16th June, 1860, *æt.* 48.
1875 IVES, FRANK L., M.D.
Original. *IVES, GEORGE W., M.D., *Obiit* 6th December, 1874, *æt.* 55.

1880 JACKSON, GEORGE F., M.D.
Original. JACKSON, WILLIAM H., M.D.
1857 JACOBI, ABRAHAM, M.D.
1880 JACOBI, MARY P., M.D.
1879 JACOBUS, A. M., M.D., A. S. 1883.
1853 JANES, EDWARD H., M.D., A. S. 1865–'68; R. S. 1868 to 1871.
1879 JANEWAY, EDWARD G., M.D.
1867 JANVRIN, JOSEPH E., M.D.
1882 JEWETT, CHARLES, M.D.
1852 *JENKINS, J. FOSTER, M.D., Yonkers, N. Y., *Obiit* 1882.
1875 JOHNSON, LAURENCE, M.D., L. 1877, now in office.
1855 *JOHNSON, WILLIAM J., M.D., *Obiit* 22d September, 1860, *æt.* 55.
1882 JOHNSON, I. G., M.D.
Original. *JOHNSTON, FRANCIS U., M.D., V. P. 1847, *Obiit* 7th January, 1858, *æt.* 61.
1852 *JONES, ALANSON, S., M.D.
1856 *JONES, E. LEE, M.D., *Obiit* 30th January, 1876, *æt.* 46.
1847 JONES, WILLIAM W., M.D.
1872 JUDSON, ADONIRAM B., M.D.
1882 JUDSON, E. A., M.D.

1882 KALISH, R., M.D.
1857 *KAMMERER, JOSEPH, M.D., *Obiit* 10th June, 1875, *æt.* 53.
1876 KATZENBACH, WILLIAM H., M.D., R. S., now in office.
Original. *KEARNY, RAVAUD, M.D., *Obiit* 21st March, 1849, *æt.* 26.

ELECTED.

1879 KELLOGG, THEODORE H., M.D.
1880 KEMP, WILLIAM M., M.D.
1847 KENNEDY, JAMES, M.D.
1863 *KERRIGAN, JOSEPH A , M.D., *Obiit* 17th January, 1879, *æt.* 46.
1878 KERSHNER, EDWARD, M.D., U. S. Navy.
1880 KETCH, SAMUEL, M.D.
1882 KEYES, E. L., M.D.
Original. *KILBOURNE, J. SAGE, M.D., *Obiit* 12th June, 1877, *æt.* 71.
1854 *KIMBARK, EVERETT H., M.D., *Obiit* 29th August, 1872, *æt.* 53.
1851 *KINGSBURY, GEORGE H., M.D., *Obiit* 4th May, 1852, *æt.* 31.
1882 KINNICUTT, F. P., M.D.
1880 KINNICUTT, FRANK P., M.D.
Original. *KISSAM, RICHARD S., M.D., Trust. 1853 to 1860, *Obiit* 28th November, 1861, *æt.* 53.
1874 KNAPP, HERMAN, M.D.
1876 KNIGHT, JAMES, M.D.
1880 KNIGHT, C. H., M.D.
1854 *KRACKOWIZER, ERNST, M.D., *Obiit* 23d September, 1875, *æt.* 53.

1880 LANGMAN, GUSTAVE, M.D.
1865 LAWRENCE, JONATHAN S., M.D.
1869 LEALE, CHARLES A., M.D.
1854 LEAMING, JAMES R., M.D., V. P. 1879.
1869 LEE, CHARLES C., M.D.
1874 LEFFERTS, GEORGE M., M.D.
1872 LEO, SIMEON N., M.D.
1850 *LEO WOLF, GEORGE, M.D., *Obiit* 14th March, 1855, *æt.* 40.
Original. LEO WOLF, MORRIS, M.D.
1851 *LEVERIDGE, BENJAMIN C., M.D., *Obiit* 16th April, 1862, *æt.* 63.
1880 LEWIS, DANIEL, M.D.
1866 LIDDELL, JOHN A., M.D.
1879 LINCOLN, RUFUS P., M.D.
Original. LINSLY, JARED, M.D., Trust. 1865 to 1870.
1864 LITTLE, JAMES L., M.D.
1882 LIVINGSTON, B., M.D.
1855 LIVINGSTON, WATTS C., M.D.
1876 LOCKROW, ARTHUR V. B., M.D.
1861 *LOINES, JONAS P., M.D., *Obiit* 15th December, 1873, *æt.* 52.
1863 LOOMIS, ALFRED L., M.D.
1883 LORING, P. G., M.D.
1876 LUDLUM, WILLIAM S., M.D.
1871 LUSK, WILLIAM T., M.D.

ELECTED.

1874 LYNCH, PATRICK J., M.D.
1847 *LYON, JAMES L., M D., *Obiit* 24th December, 1858, *æt.* 50.
1881 LYONS, F. A., M.D.

1864 MACGREGOR, JAMES R., M.D.
1857 *MCALLISTER, GEORGE, M.D., *Obiit* 29th July, 1864, *æt.* 37.
1880 MCBRIDE, THOMAS A., M.D.
1866 MCCLELLAN, CHRISTOPHER R., M.D., Brooklyn, N. Y.
Original. *MCCLELLAND, JOHN, M.D., *Obiit* 20th February, 1875, *æt.* 69.
1882 MCDONALD, A. E., M.D.
1847 *MCDONALD, JAMES, M.D., *Obiit* 5th May, 1849, *æt.* 45.
1880 MCLAURY, WILLIAM M., M.D.
1882 MCLEAN, MALCOLM, M.D.
1857 MCLEOD, S. B. WYLIE, M.D.
1865 MCMILLAN, CHARLES, M.D., Rome, Italy.
1847 *MACNEVEN, WILLIAM H., M.D., *Obiit* 12th May, 1854, *æt.* 38.
1848 *MCNULTY, JOHN, M.D.
Original. *MANLEY, JAMES R., M.D., Orator 1848; V. P. 1849, *Obiit* 21st November, 1851, *æt.* 69.
1876 MANN, MATHEW D., M.D., Hartford, Conn.
Original. MARKOE, THOMAS M., M.D., L. 1847.
1847 *MARTIN, JOSEPH, M.D., *Obiit* 26th April, 1864, *æt.* 67.
1872 MARTIN, T. DWIGHT, M.D.
Original. *MARVIN, DAVID D., M.D., *Obiit* 21st October, 1852, *æt.* 40.
1877 *MASON, THEODORE L., M.D., Brooklyn, N. Y.
1882 MASON, L. D., M.D.
1847 MAXWELL, WILLIAM H., M.D.
Original. *MEIKLEHAM, DAVID S., M.D., *Obiit* 20th November, 1849, *æt.* 45.
1878 MERRILL, JOHN N., M.D., Skowhegan, Me.
Original. METCALFE, JOHN T., M.D.
1879 MILHAU, JOHN J., M.D.
1848 *MILLER, JOHN, M.D., *Obiit* 13th January, 1863, *æt.* 56.
1848 *MILLER, WILLIAM ELLISON, M.D., *Obiit* 16th January, 1852, *æt.* 52.
1879 MILNE, CHARLES, M.D.
1848 *MINER, WILLIAM, M.D., *Obiit* 16th November, 1859, *æt.* 45.
Original. *MINER, WILLIAM W., M.D., V. P. 1848, *Obiit* 20th March 1863, *æt.* 83.
1880 MIRANDA, RAMON L., M.D.
1847 MITCHELL, CHAUNCEY L., M.D., Brooklyn, N. Y.
1852 *MONELL, JOSEPH A., M.D., *Obiit* 1882.

ELECTED.

1848 *MOORE, SAMUEL W., M.D., *Obiit* 26th August, 1854, *æt.* 67.
1849 *MORAN, THOMAS, M.D., *Obiit* 1853.
1870 MORRIS, MOREAU, M.D., Rowaton, Conn.
1870 MORRIS, STUYVESANT F., M.D.
1879 MORTON, WILLIAM J., M.D.
1880 MORROW, P. ALBERT, M.D.
1874 MOSHER, JACOB S., M.D., Albany, N. Y.
Original. *MOTT, VALENTINE, M.D., LL.D., P. 1849 and 1857, *Obiit* 26th April, 1865, *æt.* 79.
1882 MOURRAILLE, G., M.D.
1875 MUNDÉ, PAUL F., M.D.
1882 MUNN, J. P., M.D.

1871 NEFTEL, WILLIAM B., M.D.
1848 *NEILSON, JOHN, M.D., *Obiit* 19th June, 1857, *æt.* 82.
1852 *NELSON, JAMES B., M.D., *Obiit* 28th September, 1874, *æt.* 61.
1874 *NESMITH, ROBERT D., M.D.
1879 NEWCOMB, GILBERT L., M.D.
1847 *NICHOLS, ELIAS S., M.D.
1880 NICHOLS, CHARLES H., M.D.
1859 NICHOLS, TRUMAN, M.D.
1873 NICOLL, HENRY D., M.D.
1861 NOEGGERATH, EMIL, M.D.
1871 *NOTT, JOSIAH CLARK, M.D., *Obiit* 31st March, 1873, *æt.* 69.
1862 NOYES, HENRY D., M.D.

Original. *OGDEN, BENJAMIN, M.D., Trust. 1853 to 1859, 1861, *Obiit* 18th June, 1867, *æt.* 69.
1873 ORTON, SAMUEL H., M.D.
1861 OTIS, FESSENDEN N., M.D.
1857 *O'REILLY, JOHN, M.D., *Obiit* 6th December, 1868, *æt.* 55.
1861 O'SULLIVAN, RICHARD J., M.D.

1871 PACKARD, CHARLES W., M.D.
1880 PAGE, R. C. M., M.D.
1864 *PAINE, MARTYN, M.D., LL.D., *Obiit* 10th November, 1877, *æt.* 83.
1869 PARDEE, CHARLES I., M.D.
1873 *PARIGOT, JULIUS, M.D., *Obiit* 30th September, 1877.
Original. PARKER, WILLARD, M.D., LL.D., Trust. 1851; V. P. 1853; P. 1856.

ELECTED.

1874 PARKER, WILLARD, Jr., M.D.
1847 *PARKINSON, WILLIAM B., M.D., *Obiit* 11th May, 1856, *æt.* 45.
1878 PARTRIDGE, EDWARD L., M.D.
1847 *PAUL, JAMES C., M.D., *Obiit* 5th May, 1859.
1858 *PEASLEE, EDMUND R., M.D., LL.D., Orator 1858; V. P. 1868 to 1871; P. 1871 and 1872; Trust. 1873, *Obiit* 22d January, 1878, *æt.* 63.
1878 PEASLEE, EDWARD H., M.D.
Original. *PENNELL, RICHARD, M.D., *Obiit* 11th April, 1861, *æt.* 62.
1880 PERRY, I. G., M.D.
1879 PERRY, JAMES L., M.D.
1870 PETERS, JOHN C., M.D.
1879 PETERS, GEORGE A., M.D.
1874 *PEUGNET, EUGENE, M.D., *Obiit* 10th October, 1879, *æt.* 42.
Original. *PHILLIPS, SAMUEL B., M.D., *Obiit* 3d March, 1857, *æt.* 54.
Original. *PIATT, WILLIAM F., M.D., *Obiit* 6th May, 1848, *æt.* 59.
1875 PIFFARD, HENRY G., A.M., M.D.
1867 PINKNEY, HOWARD, M.D., London.
1873 POLK, WILLIAM M., M.D.
1865 POMEROY, OREN D., M.D.
Original. *POND, JAMES O., M.D., T. 1848 to 1877, *Obiit* 1881, *æt.* 90.
1869 POOLEY, THOMAS R., M.D.
1880 POORE, CHARLES T., M.D.
1856 *PORTER, MORTIMER G., M.D., *Obiit* 24th November, 1863, *æt.* 37.
1880 PORTER, P. B., M.D.
1847 POST, ALFRED C., M.D., LL.D., Orator 1849; V. P. 1861 to 1866; P. 1867 and 1868.
1870 POST, WILLIAM H. B., M.D., A. S. 1871 to 1874.
1881 POWELL, S. D., M.D.
Original. *POWER, WILLIAM, M.D., *Obiit* 14th September, 1858, *æt.* 60.
Original. *PRATT, PETER, M.D., *Obiit* 1860, *æt.* 52.
1882 PRATT, H. R., M.D.
1871 PURDY, ALFRED E. M., M.D.
Original. PURDY, ALFRED S., M.D.
Original. PURDY, SAMUEL A., M.D., R. S. 1853 and 1854.
Original. †PURPLE, SAMUEL S., M.D., V. P. 1872 to 1875; P. 1875 to 1879; Trust. 1879, now in office.
1847 PUTNAM, FREDERICK A., M.D.
1879 PUTZEL, LEOPOLD, M.D.

1876 QUACKENBOS, HENRY F., M.D.

ELECTED.

1876 RABORG, SAMUEL A., M.D.
1875 RAMSDELL, EDWIN D., M.D.
1851 *RANDOLPH, ISRAEL, M.D., *Obiit* 2d June, 1877, *æt.* 76.
1882 RANNEY, A. L., M.D.
1851 *RANNEY, MOSES H., M.D., *Obiit* 7th December, 1864, *æt.* 50.
1856 *RAPHAEL, BENJAMIN I., M.D., *Obiit* 1881.
1859 *RAY, ROBERT, Jr., M.D., *Obiit* 3d July, 1860, *æt.* 27.
1879 READ, IRA B., M.D.
Original. *REESE, DAVID MEREDITH, M.D., LL.D., *Obiit* 13th May, 1861, *æt.* 60.
1880 REID, J. J., M.D.
1879 *REID, KENNETH, M.D.
1872 REINFELDER, MAX J., M.D., Yonkers, N. Y.
1866 *REYNOLDS, JAMES B., M.D., *Obiit* 1882.
1855 RICHARDS, JOSEPH W., M.D.
1874 ROBERTS, NATHAN S., M.D.
1878 ROBERTS, STEPHEN M., M.D.
Original. *ROBERTS, WILLIAM C., M.D., Orator 1859; V. P. 1870 to 1873, *Obiit* 9th December, 1873, *æt.* 63.
Original. *ROBESON, ABEL B., M.D., *Obiit* 22d March, 1853, *æt.* 36.
1880 ROBINSON, A. R., M.D.
1876 ROBINSON, BEVERLY, M.D.
1880 ROBINSON, G. W., M.D.
1869 ROCKWELL, ALPHONSO D., M.D.
Original. *ROCKWELL, WILLIAM, M.D., Trust. 1854, 1855, *Obiit* 30th December, 1867, *æt.* 67.
1873 *RODENSTEIN, CHARLES F., M.D., *Obiit* 18th March, 1876, *æt.* 49.
1862 RODENSTEIN, LOUIS A., M.D.
Original. *RODGERS, JOHN KEARNY, M.D., V. P. 1848 to 1851; Trust. 1851, *Obiit* 9th November, 1851, *æt.* 58.
1847 *ROGERS, J. SMYTH, M.D., *Obiit* 29th March, 1851, *æt.* 58.
1872 ROOF, STEPHEN W., M.D.
1880 ROOSA, D. B. ST. JOHN, M.D.
1858 ROSS, JAMES, M.D.
1880 ROWE, ALVAH, M.D.

Original. SABINE, GUSTAVUS A., M.D.
1874 SABINE, THOMAS T., M.D.
1879 SANDERS, EDWARD, M.D.
1873 SATTERLEE, F. LE ROY, M.D.
1864 *SATTERLEE, RICHARD S., M.D., U. S. A.
1882 SATTERTHWAITE, T. E., M.D.

ELECTED.

Original. SAYRE, LEWIS A., M.D.
1882 SAYRE, L. H.
1852 *SCHILLING, ERNEST, M.D., *Obiit* 25th April, 1872, *æt.* 62.
Original. *SCHMIDT, JOHN W., Jr., M.D., *Obiit* 1857, *æt.* 50.
1847 *SCHIRMER, WILLIAM, M.D., *Obiit* 8th July, 1878, *æt.* 70.
1873 SCHULTZE, LOUIS F., M.D.
1882 SCHOENEMANN, C. P. R., M.D.
1873 SEGUIN, EDWARD C., M.D.
1882 SCHUYLER, W. D., M.D.
1870 SELL, E. H. M., M.D.
1856 *SEWALL, JOHN G., M.D., *Obiit* 18th January, 1874, *æt.* 51.
1879 SEXTON, SAMUEL, M.D.
Original. *SHANKS, JOHN, M.D., *Obiit* 10th August, 1870, *æt.* 69.
1862 *SHEPPARD, JOHN W., M.D., *Obiit* 5th October, 1868, *æt.* 46.
Original. *SHERWOOD, BURRITT, M.D., *Obiit* 10th August, 1854, *æt.* 53.
1879 SHRADY, JOHN, M.D.
1856 SIMS, J. MARION, M.D., Orator 1857.
1880 SILVER, HENRY M., M.D.
1879 SKENE, ALEXANDER J. C., M.D., Brooklyn, N. Y.
1876 SMITH, ABRAM A., M.D.
1877 SMITH, ANDREW H., M.D.
Original. *SMITH, DAVID, M.D., *Obiit* 16th January, 1867, *æt.* 57.
1870 *SMITH, DAVID A., M.D., *Obiit* 9th April, 1872, *æt.* 28.
Original. *SMITH, GILBERT, M.D., *Obiit* 16th July, 1851, *æt.* 80.
1858 SMITH, GOUVERNEUR M., M.D., L. 1861, 1862; Orator 1869; V. P. 1875 to 1878; Trust. 1878, now in office.
1882 SMITH, G. DE F., M.D.
1864 SMITH, HANBURY, M.D.
1853 SMITH, JAMES O., M.D.
Original. *SMITH, JOSEPH MATHER, M.D., Orator 1850; V. P. 1850 to 1852; P. 1854; Trust. 1859, *Obiit* 22d April, 1866, *æt.* 77.
1856 SMITH, J. LEWIS, M.D.
1866 SMITH, OSCAR G., M.D.
1855 SMITH, STEPHEN, M.D., Orator 1867.
1870 *SNELLING, FREDERICK G., M.D., *Obiit* 26th November, 1878, *æt.* 47.
1847 *SNOWDEN, JOHN, M.D., *Obiit* 22d January, 1848, *æt.* 33.
1864 SPIER, S. FLEET, M.D., Brooklyn, N. Y.
Original. *SPRING, EDWARD, M.D., *Obiit* 13th February, 1850, *æt.* 42.
1876 *STANLEY, C. GRAHAM, M.D., *Obiit* 24th May, 1877, *æt.* 30.
Original. *STEARNS, JOHN, M.D., P. 1847, *Obiit* 17th March, 1848, *æt.* 78.
1880 STEDMAN, T. L., M.D.

ELECTED.

1880 STEIN, ALEXANDER W., M.D.

Original. *STEPHENSON, MARK, M.D., *Obiit* 28th August, 1865, *æt.* 62.

Original. *STEVENS, ALEXANDER H., M.D., LL.D., P. 1851, *Obiit* 30th March, 1869, *æt.* 79.

1847 *STEWART, JAMES, M.D., *Obiit* 12th September, 1864, *æt.* 65.

1847 *STICKNEY, JOSIAH DWIGHT, M.D., *Obiit* 30th September, 1849, *æt.* 34.

1865 *STILES, R. CRESSON, M.D., *Obiit* 17th April, 1873, *æt.* 42.

1875 STIMSON, DANIEL M., M.D.

Original. *STIMPSON, EDWIN B., M.D., *Obiit* 15th May, 1858, *æt.* 36.

1865 *STIRLING, THOMAS B., M.D., *Obiit* 10th June, 1877, *æt.* 47.

Original. *STONE, JOHN O., M.D., *Obiit* 7th June, 1876, *æt.* 63.

Original. *STORER, EBENEZER, M.D., *Obiit* 20th May, 1879, *æt.* 77.

1874 STRACHAN, A. RUSSELL, M.D.

1880 STURGIS, F. R., M.D.

1878 SUSSDORFF, G. E., M.D.

1879 SWASEY, JOHN H., M.D.

Original. *SWEENY, HUGH, M.D., *Obiit* 15th September, 1857, *æt.* 52.

1851 *SWEENEY, JAMES, M.D., *Obiit* 1872.

1847 *SWETT, JOHN A., M.D., Orator 1853, *Obiit* 18th September, 1854, *æt.* 45.

1866 *SWIFT, FOSTER, M.D., *Obiit* 10th May, 1875, *æt.* 41.

1876 SWIFT, SAMUEL, M.D., Yonkers, N. Y.

Original. *TAFT, MARCUS L., M.D., A. S. 1848, *Obiit* 8th February, 1850, *æt.* 29.

1872 TAUSZKY, RUDOLPH, M.D.

1867 TAYLOR, CHARLES F., M.D.

Original. TAYLOR, ISAAC E., M.D., V. P. 1857 and 1858; Trust. 1864, 1872 to 1877, now in office.

1877 TAYLOR, JAMES R., M.D.

1862 *TEATS, SYLVESTER, M.D.

1865 TELLER, SELIGMANN, M.D.

1847 *THAYER, HENRY W., M.D., *Obiit* 21st May, 1857, *æt.* 50.

1859 *THEBAUD, JULIUS S., M.D., *Obiit* 20th October, 1876, *æt.* 48.

1857 THOMAS, T. GAILLARD, M.D., R. S. 1858 to 1861; Orator 1877; V. P. 1879.

1869 *THOMPSON, GEORGE, M.D., *Obiit* 13th January, 1877, *æt.* 37.

1864 THOMS, WILLIAM F., M.D., Statistical Secretary 1868 to 1873.

1864 THOMSON, WILLIAM H., M.D., Orator 1878.

1880 THURMAN, WILLIAM, M.D.

1867 *TOWNSEND, JOHN F., M.D., *Obiit* 8th January, 1874, *æt.* 64.

ELECTRD.

Original. *TOWNSEND, PETER S., M.D., *Obiit* 26th March, 1849, *æt.* 54.
1857 TUCKER, CARLOS P., M.D.
1863 *TUCKER, GEORGE H., M.D., *Obiit* 25th January, 1863, *æt.* 34.
1854 *TUTTLE, JOHN T., M.D., *Obiit* 27th January, 1870, *æt.* 68.

1854 *UHL, DAVID, M.D., *Obiit* 17th September, 1858, *æt.* 36.
Original. *UNDERHILL, ALFRED, M.D., V. P. 1863 to 1866; Trust. 1866 to 1873, *Obiit* 7th December, 1873, *æt.* 64.

1847 *VACHÉ, ALEXANDER F., M.D., *Obiit* 9th June, 1857, *æt.* 57.
Original. *VAN ARSDALE, HENRY, M.D., Morristown, N. J., *Obiit* 25th January, 1864.
Original. VAN ARSDALE, HENRY, M.D.
1847 *VAN ARSDALE, PETER, M.D., *Obiit* 1858, *æt.* 77.
Original. *VAN BEUREN, THOMAS, M.D., *Obiit* 1848.
1856 *VAN BUREN, PETER, M.D., *Obiit* 5th December, 1873, *æt.* 71.
Original. *VAN BUREN, WILLIAM H., M.D., LL.D., V. P. 1858, *Obiit* 1883, *æt.* 64.
1859 *VAN DOREN, MATTHEW D., M.D., *Obiit* 1882.
1879 *VAN HOUTEN, JACOB A., M.D.
Original. *VAN KLEEK, JOHN R., M.D., Trust. 1861 to 1866, *Obiit* 2d January, 1876, *æt.* 66.
1847 VAN PELT, MOSES D., M.D., V. P. 1860 to 1863; Trust. 1864 to 1869.
1879 VAN SANTVOORD, RICHARD, M.D.
1847 VAN WINKLE, EDWARD H., M.D.
1847 VANDERPOEL, EDWARD, M.D.
1882 VANDERPOEL, S. O., M.D.
1859 *VANDERVEER, JACOB H., M.D., *Obiit* 20th August, 1873, *æt.* 55.
Original. VANDERVOORT, JOHN L., M.D., R. S. 1849.
1847 VARICK, THEODORE R., M.D., Jersey City, N. J.
1880 VARLEY, C. D., M.D.
1862 *VEDDER, JOSEPH H., M.D., *Obiit* 18th July, 1864, *æt.* 33.
1854 *VON ROTH, WOLDEMAR, M.D., *Obiit* 1857.

1876 WAGNER, CLINTON, M.D.
1873 WARD, EDWIN F., M.D., A. S. 1877 to 1879; R. S. 1880.
1853 WARNER, EVERARDUS B., M.D.
1879 WARNER, JOHN W., M.D.
1880 WARREN, J. S., M.D.
Original. *WASHINGTON, JAMES A., M.D., *Obiit* 30th August, 1847, *æt.* 45.

ELECTED.

1853 *WATSON, JOHN, M.D., Orator 1855 and 1860; V. P. 1856; P. 1859 and 1860, *Obiit* 3d June, 1863, *æt.* 56.

Original. *WATTS, ROBERT, JR, M.D., T. 1847, *Obiit* 8th September, 1867, *æt.* 55.

1876 WEBB, Z. SWIFT, M.D.

1867 WEBER, LEONARD, M.D.

1879 WEBSTER, DAVID, M.D.

Original. *WEEKS, CYRUS, M.D., *Obiit* 20th September, 1875, *æt.* 68.

1877 WEEKS, GRENVILLE M., M D.

1880 WEIR, ROBERT F., M.D., V. P., now in office.

1870 WEISSE, FANEUIL D., M.D.

1879 WELCH, WILLIAM H., M.D.

1880 WELD, FRANCIS M., M.D.

Original. WELDS, OVID P., M.D.

Original. *WHITE, AMBROSE L., M.D., *Obiit* 2d June, 1865, *æt.* 61.

1858 WHITE, FRANCIS V., M.D., Statistical Secretary 1879, now in office.

1877 *WHITE, JOHN P. P., M.D.

1882 WHITE, J. B., M.D.

1876 WHITE, OCTAVIUS A., M.D.

Original. *WHITE, OLIVER, M.D., V. P. 1866 to 1870; Trust. 1871 to 1876, *Obiit* 7th November, 1879, *æt.* 69.

Original. *WHITE, SAMUEL P., M.D., Trust. 1853 to 1858, *Obiit* 6th June, 1867, *æt.* 65.

1867 WHITE, WILLIAM T., M.D., A. S. 1868 to 1871; R. S. 1871 to 1877; Orator 1876; V. P. 1877 to 1880; Trust. 1880, now in office.

1875 WIENER, JOSEPH, M.D.

1883 WIENER, R. G., M.D.

1880 WILDER, RUFUS L., M.D.

1847 *WILKES, GEORGE, M.D., *Obiit* 30th November, 1876, *æt.* 75.

Original. *WILLIAMS, MERRILL W., M.D., *Obiit* 3d December, 1873, *æt.* 72.

1860 *WINCHELL, MARTIN E., M.D., *Obiit* 1st May, 1864, *æt.* 34.

1869 WINSTON, GUSTAVUS S., M.D.

1880 WITTHAUS, R. A., M.D.

1878 WOOD, CHARLES S., M.D.

1882 WINTERS, J. E., M.D.

Original. *WOOD, ISAAC, M.D., V. P. 1849; P. 1850 and 1853; Trust. 1851 to 1852, and 1859 to 1863, *Obiit* 25th March, 1868, *æt.* 74.

Original. *WOOD, JAMES R., M.D., LL.D., V. P. 1857, *Obiit* 1882.

ELECTED.

1880 WOODBURY, EDWARD C., M.D.

1852 *WOODWARD, GEORGE F., M.D., *Obiit* 1857.

Original. *WORSTER, JOSEPH, M.D., *Obiit* 7th August, 1877, *æt.* 73.

1869 WRIGHT, CHARLES, M.D., Treasurer of Trustees, 1879, now in office.

1875 WYLIE, W. GILL, M.D.

1873 YALE, LE ROY M., M.D., Orator 1879.

Original. †YOUNG, WILLIAM, M.D.

☞ In order to secure accuracy in the list of Fellows of the Academy, it is requested that any change of title or residence be reported to the Secretary without delay.

NON-RESIDENT FELLOWS.

BARSTOW, J. W., M.D., Flushing, L. I.

*BERGER, FRANCIS E., M.D., Paris, France, *Obiit* 1st February, 1866, *æt.* 77.

CLARK, J. G., M.D., New Brighton, S. I.

*CORSON, JOHN W., M.D., Orange, N. J.

*DRAPER, JOHN W., M.D., LL.D., Orator 1863, Hastings-on-Hudson, N. Y., *Obiit* 1882.

DUNSTER, EDWARD S., M.D., Orator 1872, Ann Arbor, Mich.

FIELD, HENRY M., M.D., Newton, Mass.

FRANCIS, S. W., M.D., Newport, R. I.

HEPBURN, JAMES C., M.D., Japan.

HART, G., Saybrook, Conn.

JOHNSTON, FRANK U., Jr., M.D., N. Y.

*LEE, CHARLES ALFRED, M.D., Peekskill, N. Y., *Obiit* 14th February, 1872, *æt.* 71.

*LEWIS, WILLIAM B., M.D., *Obiit* 16th June, 1874, *æt.* 32.

MARTIN, T. D., Morrisania, N. Y.

MASON, JOHN J., Newport, R. I.

MANN, M. D., M.D., Hartford, Conn.

*MERRITT, J. KING, M.D., Flushing, N. Y., *Obiit* 1882.

MERRIL, J. W., Skowhegan, Me.

Metcalfe, F. J., M.D., Florence, Italy.
North, Nelson J., M.D., Brooklyn, N. Y.
Ordronaux, John, M.D., Roslyn, N. Y.
Reinfelder, M. J., M.D., Yonkers, N. Y.
*Sands, Austin L., M.D., Newport, R. I., *Obiit* 20th December, 1877, *æt.* 5[2].
Shrady, George F., M.D., N. Y.
Sloan, William J., M.D., U. S. A.
Smith, Jerome C., M.D., Philadelphia, Pa.
Stewart, F. Campbell, M.D., Florence, Italy.
St. John, Samuel B., M.D., Hartford, Conn.
Swift, S., M.D., Yonkers, N. Y.
Thompson, Bradford S., M.D., Salisbury, Conn.
*Van Arsdale, Henry M., M.D., Morristown, N. J., *Obiit* 25th January, 1864.
Varick, T. R., Jersey City, N. J.
Vermilye, William E., M.D., Pittsfield, Mass.

BENEFACTORS.

1878 *Purple, Edwin R., *Obiit* 20th January, 1879, *æt.* 47.
1879 Dubois, Abram, M.D.

HONORARY FELLOWS.

Elected.[1]

1878 *Barnes, Joseph K., M.D., Surgeon-General U. S. A., Washington, D. C., *Obiit* 5th April, 1883.
1871 Bowditch, Henry I., M.D., Professor of Clinical Medicine in Harvard University, Boston, Mass.
1881 Cuyler, John M., M.D., Surgeon U. S. A., N. Y.
1879 Dubois, Abram, M.D., New York City.
1859 *Gibson, William, M.D., Professor of the Principles and Practice of Surgery in the University of Pennsylvania, Philadelphia, Pa., *Obiit* 2d March, 1868, *æt.* 80.
1876 Gross, Samuel D., M.D., LL.D., D.C.L. Oxon., Professor of Surgery in Jefferson Medical College, Philadelphia, Pa.

ELECTED.

1857 *IVES, ELI, M.D., Professor of Materia Medica and Botany in Yale College, New Haven, Conn., *Obiit* 8th October, 1861, *æt.* 82.

1860 *JACKSON, JAMES, M.D., Professor Emeritus of the Practice of Physic in Harvard University, Cambridge, Mass., *Obiit* 27th August, 1867, *æt.* 90.

1859 *LA ROCHE, RENE, M.D., Member of the American Philosophical Society, Philadelphia, Pa., *Obiit* 9th December, 1872, *æt.* 77.

1859 *MUSSEY, REUBEN D., M.D., Professor of Surgery in the Medical College of Ohio, Cincinnati, O., *Obiit* 21st June, 1866, *æt.* 86.

1857 *SPAULDING, MATHIAS, M.D., of Amherst, Mass., *Obiit* 22d May, 1865, *æt.* 95.

1871 STILLÉ, ALFRED, M.D., Professor of Clinical Medicine in the University of Pennsylvania, Philadelphia, Pa.

1874 VANDERPOEL, S. OAKLEY, M.D., Professor of Theory and Practice of Clinical Medicine in Albany Medical College, Albany, N. Y.; Health Officer of the Port of New York.

1878 *WHITE, JAMES PLATT, M.D., Professor of Obstetrics and Diseases of Women and Children in University of Buffalo, N. Y., *Obiit* 1882.

1871 *WOOD, GEORGE B., M.D., LL.D., Professor Emeritus of the Theory and Practice of Medicine in the University of Pennsylvania, Philadelphia, Pa., *Obiit* 30th March, 1879, *æt.* 81.

CORRESPONDING FELLOWS.

1873 ACLAND, HENRY W., M.D., F.R.S., Regius Professor of Medicine in the University of Oxford, England.

1856 *ADAMS, FRANCIS, M.D., LL.D., Surgeon, Banchory, Scotland, *Obiit* 26th February, 1861, *æt.* 66.

1847 *AGASSIZ, LOUIS JOHN R., M.D., F.R.S., Professor of Natural History in Harvard University, Cambridge, Mass., *Obiit* 14th December, 1873, *æt.* 66.

1880 ALDEN, C. H., M.D., U. S. A.

1882 ALTHAUS, JULIUS, M R.C.P., London.

1850 *AMUSSAT, JEAN ZUILME, M.D., Member of the Royal Academy of Medicine, Paris, France, *Obiit* 12th May, 1856, *æt.* 58.

ELECTED.

1854 *ANDRAL, GABRIEL, M.D., Professor of Pathology in the Faculty of Medicine, Paris, France, *Obiit* 13th February, 1876, *æt.* 78.

1854 *BARTHEZ, ERNST, M.D., Paris, France.

1847 *BECK, THEODRICK ROMEYN, M.D., LL.D., Professor of Medical Jurisprudence in Albany Medical College, *Obiit* 19th November, 1855, *æt.* 64.

1857 *BENNETT, JOHN HUGHES, M.D., Professor of Medicine in the University of Edinburgh, Scotland, *Obiit* 25th September, 1875, *æt.* 63.

1854 *BIGELOW, JACOB, M.D., LL.D., Professor of Materia Medica in Harvard University, Cambridge, Mass., *Obiit* 10th January, 1879, *æt.* 91.

1847 *BLATCHFORD, THOMAS W., M.D., Troy, N. Y., *Obiit* 7th January, 1866, *æt.* 71.

1867 BROWN-SÉQUARD, C. E., M.D., Paris, France.

1882 BROWNE, J. CRICHTON, M.D., London.

1849 *BUREAUD-RIOFREY, A.M., M.D., Paris, France.

1871 CHAMBERS, THOMAS K., M.D., Physician to and Lecturer on Medicine at St. Mary's Hospital, London.

1880 CHARCOT, JEAN M., M.D., Faculty of Medicine, Paris.

1847 *CIVIALE, JEAN, M.D., Honorary Member of the Academy of Medicine, Paris, France, *Obiit* 13th June, 1867, *æt.* 75.

1866 CUNHA, JOSÉ DE, M.D., Rio Janeiro, Brazil.

1868 DAVIS, NATHAN S., M.D., Professor of the Principles and Practice of Medicine in Chicago Medical College.

1872 DICHIARA, FRANCESCO, M.D., Palermo, Italy.

1850 *DICKSON, SAMUEL H., M.D., Professor of the Institutes of Medicine in South Carolina Medical College, *Obiit* 31st March, 1872, *æt.* 74.

1871 DICKINSON, WILLIAM H., M.D., Physician to and Lecturer on Pathology at St. George's Hospital, London, England.

1854 *DUBOIS, Baron PAUL, Dean and Professor of Clinical Midwifery in the Faculty of Medicine of Paris, France, *Obiit* 29th November, 1871, *æt.* 76.

1867 DUMONT, HENRI, M.D., Havana, Cuba.

1848 DUPIERRIS, MARTIAL, M.D., Paris, France.

1876 ERICHSEN, JOHN ERIC, F.R.S., late Professor of Surgery in University College, London, England.

1850 *FENNER, ERASMUS D., M.D., Professor of the Theory and Practice of Medicine in New Orleans School of Medicine, *Obiit* 4th May, 1866, *æt.* 59.

ELECTED.

1849 *FERGUSON, Sir WILLIAM, F.R.S., Surgeon to King's College Hospital, London, England, *Obiit* 10th February, 1877, *æt.* 69.

1877 GALLARD, T., M.D., Paris, France.

1882 GARROD, ALFRED BARON, M.D., F.R.C.P., F.R.S., London.

1879 GOODELL, WILLIAM, M.D., Professor of Clinical Gynæcology in the University of Pennsylvania.

1851 GROSS, SAMUEL D., M.D., late Professor of Surgery in the Medical Department of Louisville University, Louisville, Ky.

1855 *GUGGENBUHL, JEAN L., M.D., Abendberg, Switzerland, *Obiit* February, 1863.

1847 *HARRIS, THOMAS, M.D., Surgeon-General U. S. N., Washington, D. C., *Obiit* 4th March, 1862, *æt.* 72.

1848 *HOLLAND, Sir HENRY, Bart., M.D., D.C.L., LL.D., F.R.S., Physician to H. M. the Queen, London, England, *Obiit* 29th October, 1873, *æt.* 85.

1850 *HOOKER, WORTHINGTON, M.D., Professor of the Theory and Practice of Medicine in Yale College, New Haven, Conn., *Obiit* 6th November, 1867, *æt.* 61.

1880 HUNT, EZRA M., M.D., New Jersey.

1881 HOWARD, B., M.D., London.

1874 JENNER, Sir WILLIAM, Bart., M.D., D.C.L., F.R.S., Professor of Clinical Medicine in University College, London, England.

1880 LA LOGGIA, GAETANO, M.D., Palermo, Italy.

1880 LENTE, F. D., M.D , Florida.

1852 *LEROY (D'ETIOLLES), JEAN J. J., M.D., Paris, France, *Obiit* July, 1860, *æt.* 62.

1879 LITTLE, WILLIAM J., M.R.C.P., M.R.C.S., London, England.

1880 MARSH, ELIAS J., M.D., Paterson, N. J.

1877 MERCIER, L. AUGUSTE, M.D., Paris, France.

1874 MITCHELL, S. WEIR, M.D., Philadelphia, Pa.

1871 *NÉLATON, AUGUSTE, Professor of Clinical Surgery in the University of Paris, France, *Obiit* 20th September, 1873, *æt.* 65.

1874 OWEN, RICHARD, M.D., Hunterian Professor in the Royal College of Surgeons, London, England.

1874 PAGET, Sir JAMES, Bart., D.C.L., LL.D., F.R.S., Consulting Surgeon to St. Bartholomew's Hospital, London, England.

1857 *PEASLEE, EDMUND R., M.D., Professor of Surgery in Dartmouth Medical College, Hanover, N. H., *Obiit* 22d January, 1878, *æt.* 63.

1853 PRINCE VIROMMA LUANG SI TIRAT SANIK, Siam.

ELECTED.

1868 POST, GEORGE E., M.D., Beirut, Syria.

1874 REGO, JOSÉ PEREIRA, FILHO, M.D., Rio Janeiro, Brazil.

1850 *REYNOLDS, EDWARD, M.D., Boston, Mass., *Obiit* 1881.

1852 RICORD, PHILIPPE, M.D., Member of the Royal Academy of Medicine, Paris, France.

1854 *RILLIET, FRANÇOIS, M.D., Paris, France.

1878 RIZZOLI, FRANCISCO, M.D., Bologna, Italy.

1860 ROESSER, D. P., M.D., Athens, Greece.

1849 *ROGET, PETER MARK, M.D., F.R.S., Professor of Physiology in the Royal Institution of Great Britain, London, *Obiit* 12th September, 1869, *æt.* 90.

1871 *ROKITANSKY, CARL, M.D., Professor of Pathology in the University of Vienna, *Obiit* 23d July, 1878, *æt.* 74.

1856 *SIMPSON, JAMES Y., M.D., Professor of Midwifery in the University of Edinburgh, Scotland, *Obiit* 8th May, 1870, *æt.* 59.

1848 SMITH, ASHBEL, M.D., Texas.

1874 STEWART, F. CAMPBELL, M.D., Florence, Italy.

1878 THOMPSON, Sir HENRY, F.R.C.S., Emeritus Professor of Clinical Surgery in University College, London, England.

1872 TILT, EDWARD J., M.D., Consulting Physician to the Farringdon General Dispensary, London, England.

1852 *TOWNSEND, JAMES C., M.D., Glen Cove, N. Y., *Obiit* 1882, *æt.* 86.

1852 *VELPEAU, ALFRED A. L. M., Professor of Clinical Surgery in the Faculty of Medicine of Paris, Surgeon to the Hôpital de la Charité, Paris, *Obiit* 23d August, 1867, *æt.* 72.

1871 VIRCHOW, RUDOLPH, M.D., Professor of Pathological Anatomy in the University of Berlin.

1869 VOSS, LOTHAR H., M.D., Berleberg, Prussia.

1880 WALES, P. S., M.D., Surgeon-General U. S. N., Washington, D. C.

1882 WELLS, T. SPENCER, M.D., London.

1874 *WHITE, JAMES P., M.D., Professor of Obstetrics and the Diseases of Women and Children in the Medical Department of the University of Buffalo, N. Y., *Obiit* 1882.

1857 *WING, JOEL A., M.D., late President New York State Medical Society, Albany, N. Y., *Obiit* 6th September, 1852, *æt.* 64.

1867 WORTABET, JOHN, M.D., Beirut, Syria.

LIST OF CONTRIBUTORS

TO THE

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WITH AMOUNTS PAID TO JANUARY, 1883.

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Laurence Johnson, M.D.	15 00
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CONTENTS.

TRANSACTIONS

OF THE

NEW YORK ACADEMY OF MEDICINE.

THE GALVANIC ACCUMULATOR FOR STORING DYNAMICAL ELECTRICITY FOR CAUTERY AND ILLUMINATING PURPOSES.

By LOUIS ELSBERG, A.M., M.D.,

Fellow of the New York Academy of Medicine, Member of the American Academy of Medicine, President of the New York Laryngological Society, etc., etc.

(Read April 21, 1881.)

Gentlemen: Of all the instruments I became acquainted with during my stay in Europe last summer, perhaps the most useful, certainly the most remarkable, is the apparatus I have the honor to exhibit to you this evening. The idea of storing up galvanism in a box, of having an apparatus—as it were, a Leyden jar—full of dynamical electricity, that can be taken out in graduated doses for effective cautery or illumination, must strike as wonderful all who are familiar with the ordinary employment of the "galvanic current."

What is electricity? Perhaps this question would be answered differently by different persons; what I think it is, I have published more than ten years ago, viz.: a mode of motion of particles or molecules of matter—a to-and-fro or vibratory motion, the vibrations being more frequent than forty thousand, and less frequent than sixty-five trillions in a second. It is well known that vibrations less rapid than at the rate of forty thousand in a second, provided not less than sixteen full vibrations occur

per second, affect our auditory organ as sound ; while vibrations more frequent than sixty-five trillions in a second impress our consciousness as heat up to four hundred trillions, and as light between four hundred and eight hundred trillions in a second. It is also well known of some of these vibrations, that on being propagated through certain media, their rate can be altered ; thus, some vibrations beyond the visible extreme violet color of the spectrum, which are more frequent than eight hundred trillions in a second, can be slackened in their speed, and transformed into vibrations which can affect our eye, by being passed through a solution of sulphate of quinine. If this were the proper place to enter into details on these points, I might cite many instances of the convertibility of vibrations ; but my object is attained, if, by making reference to them, I facilitate understanding of the suggestion, as to how electricity is correlated to heat and light.

It has been ascertained that the velocity of electricity is much less than that of heat and light. I have no doubt that electrical vibrations are propagated by progressive wave-motion, the propagation requiring a substance the particles of which take up and transmit the vibratory movement. Substances differ very much as to their facility of propagating or conducting electricity ; some offer greater resistance than others to the wave-propagation, or, as it is called, to the passage of the electric current. Those that offer most resistance, and are, therefore, the worst conductors, are called non-conductors, or insulators, for they are used as supports or surroundings for electrified bodies, when the propagation is to be prevented. Such non-conductors are gutta-percha, caoutchouc, shellac, etc. ; of course, no substance insulates perfectly, and the electricity of every electrified body is dissipated more or less rapidly. Glass is a very bad conductor, unless moisture condenses upon it, and, in practice, the insulating power of glass is improved by coating it with shellac varnish. Dry air is a good insulator, but as the atmosphere usually contains moisture, it conducts electricity ; and this is the principal manner in which electricity is lost. Among conductors, charcoal offers more resistance

than the metals; of these, the following order shows relatively lessening conductivity, or, what is equivalent, increasing resistance, viz.: silver, copper, gold, zinc, platinum, iron, lead, German-silver. (In parenthesis, I may here remark that for conduction in electrical apparatus, copper-wire and cords containing copper-threads are used almost without exception, instead of the more expensive silver, copper conducting nearly as well as silver, and far better than any other metal; while for very long wire, as for telegraphing, iron—the specific resistance of which is five or six times as much as copper, and which, therefore, requires the wire to be five or six times as thick to conduct as well—is preferable on account of its cheapness. On the other hand, for interposing artificial resistances in electrical instruments, which, as I shall explain further on, is often useful, thin wire of German-silver is preferable.)

It is well known, that if to a metal ball increasing temperature is imparted, the vibrations constituting heat become, under certain circumstances, so much accelerated that they impress our consciousness as light; similarly, the vibrations of electricity, propagated under certain circumstances through metallic wires, become so much accelerated, that they impress our consciousness as heat. When, to each of the plates of a galvanic battery, a good conductor, as a copper-wire, is attached, on bringing the ends of the two wires together, they become heated and emit a spark. If a comparatively bad conductor, such as a platinum-wire, is connected to the ends of the copper-wires, the rate of the electrical vibrations is so much increased, that the platinum becomes very hot; the heat increasing under favorable conditions so much as to cause incandescence, and even fusion. With a sufficiently powerful galvanic battery, all metals, without exception, can be melted.

For medical purposes of cautery and illumination, platinum is especially adapted, because it is not easily fused, is not liable to rust, and is not brittle; because a thin wire is comparatively strong, and because it has a low specific heat, on account of which it is raised to a higher temperature than a wire of greater specific heat by the same amount of heat.

Galvanic cauterization seems first to have been performed in the year 1830, by Pravas, of Alfort, and Fabré Palaprat, of Paris. Since then, others have occasionally used and recommended it, particularly Heidler, of Vienna, in 1845; Crusell, of Cronstadt, and afterward of St. Petersburg, in 1848; and John Marshall, in 1850; but the merit of really introducing it into surgery belongs entirely to Middeldorpf, of Breslau, in 1854.

Nobody seems to have thought of making use of the light of incandescent platinum for illuminating the cavities of the body previous to 1867. In that year, the Breslau dentist, Dr. Bruck, constructed an apparatus which he called "stomatoscope," for the examination of the oral cavity by means of this light; and Millot, of Paris, published his successful experiments on cats and dogs. In 1868, Lazarevitch, of Charkow, applied the electric light, and in the same year I made use of it, in connection with Dr. Charles A. Budd, of this city, in laryngological and gynecological examinations. But, although these, and a number of other attempts were made, with more or less success, it was not until the ingenious electrical-instrument maker of Paris, M. Gustave Trouvé, in the year 1870, took up the problem, that it was satisfactorily solved.

It has been ascertained that, in accordance with Ohm's law,[1] the maximum effect of a galvanic battery is obtained when the total internal resistance (the intra-elemental resistance, *i.e.*, the resistance offered to the conduction of electricity by the contents of the element, viz.: the liquid between the two plates and the plates acted on) is equal to the total external resistance (the interpolar, *i.e.*, the resistance offered to the conduction by all the substances included in the circuit connecting the two plates). Now, although the platinum wire used in galvano-cautery and galvano-illumination offers, compared with silver or copper, a good deal of resistance to electrical waves, or currents, as they are commonly called, it offers only very little resistance as com-

[1] $E = \frac{nP}{nR + R'}$ in which formula E represents effectiveness; n, number of elements; P, production of electricity; R, internal resistance; and R', external resistance.

pared with water, the epidermis, or the human body generally; hence, when the object to be accomplished is to heat the platinum, we must, in order to obtain the best effect from our battery, diminish the internal resistance more than is required for any other purpose in electro-therapeutics. This diminution of intra-elemental resistance is accomplished by arranging the elements at our disposal so as to have a comparatively small number with comparatively extensive surfaces of action. A battery thus arranged is called a battery for quantity, while one arranged in a series of single cells is called a battery for intensity or tension; but the terms quantity and intensity, or tension, as applied to electricity, have arisen from the erroneous conception of the nature of electricity—namely, that it is a peculiar fluid matter; *the difference in the effect of batteries thus differently arranged is due entirely to the difference of intra-elemental or interpolar resistance.*

The main reason why galvano-cautery is not more frequently practised by physicians, lies unquestionably in the inconveniences, difficulties, and disappointments attending on the employment of all the galvanic batteries hitherto in use for this purpose. As the intra-elemental resistance requires to be small, we have had to use either an *inconstant* battery, *i.e.*, one with only one liquid interposed between the two plates, which, in spite of all sorts of devices,[1] is objectionable on account of its polarization and inconstancy, or else a constant battery requiring strong acid, in which the chemical action is very great, etc. In January, 1874, I said, in the *American Journal of Syphilography and Dermatology*, p. 15: "The desideratum of a galvanic battery for cautery purposes, entirely satisfactory, is still unsupplied. By far the best that I am acquainted with, for our purposes, is Voltolini's (Middeldorpf's) carbon-zinc battery of two elements. I have had experience with a large number of others, and if it were not for its requiring two acids, viz.:

[1] I have invented such a galvano-cautery battery, in which a rocking movement of the plates is kept up by an extra electro-magnetic device; and I must not fail to recommend Dr. Cutter's large portable battery, which I have found the most constant of all the inconstant batteries I have ever tried.

nitric and dilute sulphuric, and a good deal of care to keep it in order, I would not desire a more convenient or efficient apparatus. Dr. Sass, of this city, in whose ability and ingenuity I have great confidence, is experimenting with a view of perfecting a new galvano-caustic battery, which is to obviate all objections." The hope then expressed as to Dr. Sass has not, so far as I am aware, been realized; but at that very time already, Trouvé had begun to supply the desideratum in a very ingenious way.

It had long been known that the chief cause of enfeeblement and final cessation of action in inconstant batteries is the so-called *polarization* of the inactive plate, *i.e.*, the deposition on its surface of hydrogen from the decomposition of water of the liquid in which the plates are immersed; the experiments of Ritter, Faraday, Poggendorf, and others, had shown that by taking pairs of pieces of metal of the same kind, each pair being separated by a piece of moistened cloth, and each end of the system connected with the poles of a battery, a so-called "secondary battery" can be constructed; the constituents of the water with which the cloth is moistened, oxygen and hydrogen, accumulate on opposite plates of the circuit, and, after some time, even if separated from the battery itself, the apparatus can be made to exhibit a so-called "local," "secondary," or "polarized current." Gaston Planté had already made use of such an arrangement for producing effects of a galvanic battery. Now it occurred to Trouvé, that such a secondary battery might be used for the purpose of galvano-cautery and galvano-illumination. He constructed, after the manner of the secondary battery of Planté, an apparatus of appropriate dimensions, and found that by connecting it for a sufficient length of time with even a very feeble battery, it became capable of producing, for a short time, powerful effects of heating platinum.

By this device has been utilized for a purpose for which it was heretofore inapplicable, a constant battery with great intra-elemental resistance, in which but little chemical action goes on, and which, therefore, remains without material alteration for a very considerable period of time. Those only who

have experienced the annoyances hitherto inseparable from galvano-cautery batteries, can appreciate the innovation. To use the language of older electrology, we may say that a large quantity of current electricity accumulates and is confined in the secondary apparatus, and that "a battery for intensity" can be used, with this go-between and time, to accomplish work of "a battery of quantity." A battery which gives no trouble whatever, and needs hardly any attention for six months or a year, does work which hitherto required a battery that involved constant attention, care, and trouble.

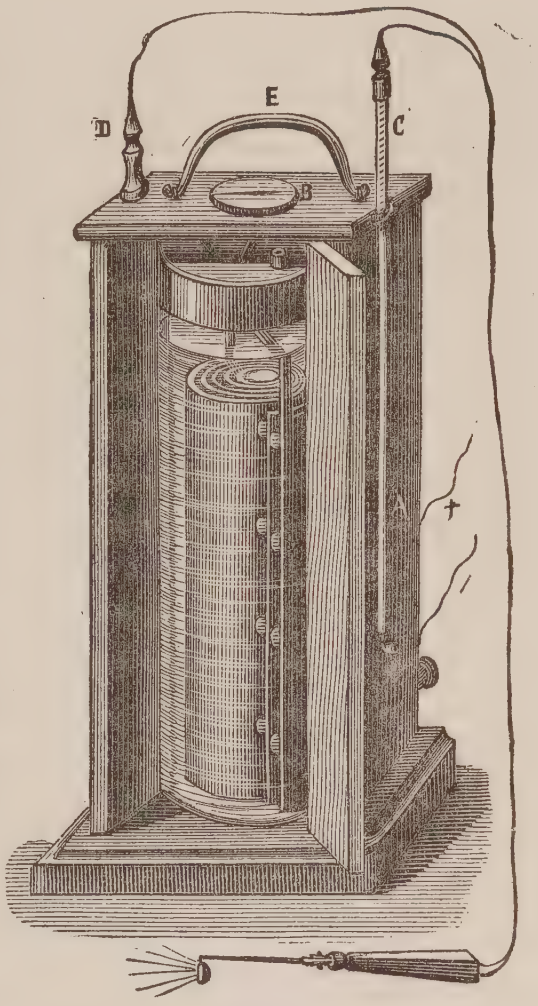

Fig. 1.

The accompanying illustration (Fig. 1) shows the whole apparatus. On the top of the box, in which Trouvé placed the Planté battery, which, henceforth, I shall speak of as the accumulator, there is a galvanometer, B, designed to indicate the activity of both the accumulator and the primary battery. It has a semicircular scale, with zero in the middle, divided into 90° on each side. When the apparatus, before being connected with a galvanic battery, is so placed that the needle points to zero—exactly north and south—the deviation when the correction is established, shows whether or not the battery is in proper working order. Trouvé advises that whenever the needle points to less than 30°, the battery should be examined and any deficiency corrected. On making use of the apparatus for heating the platinum, for either illumination or cautery, the galvanometer-needle deviates in the opposite direction.

On the side of the box, Trouvé has attached a simple rheostat, A C, which regulates the electrical illumination or cautery with the accuracy with which a graduated stop-cock would regulate the flame of a burning gas-jet issuing from a pipe. A rheostat, as is well known, is an instrument by which the resistance of any given circuit can be increased or diminished without opening the circuit. I have already mentioned that for the interposition of an artificial resistance, thin wire of German-silver should be used. Trouvé's rheostat consists of a tube, A, containing a hollow, close coil of fine German-silver wire, in which a graduated metallic rod, C, passes up and down. The more the rod is drawn out, the more of the coil is interposed in the circuit, consequently the greater is the resistance and the less the heating effect. This works so accurately and regularly, that the finest and shortest platinum-wire can be brought to a state of incandescence without melting; and the point up to which the rod must be pushed in, once determined for a particular effect on a given piece of wire, the same position of the rod invariably produces the same effect.

The end of the rod of the rheostat constitutes one of the poles of the accumulator, the other is a metallic projection at D, at the opposite side of the box. To these two poles, C and D, are attached the electrode-cords which go to the handle of the illuminating or cauterizing platinum-wire. In Fig. 1 is shown the manner in which the cords are attached to an illuminator, which lies at the bottom of the accumulator.

In order that cords for two handles may be attached to one and the same accumulator, Trouvé has furnished some with two rheostats and two opposite poles. This enables the operator to use one accumulator at the same time for both illumination and cautery.

I have here two accumulators, one with single and one with double poles, that have been in connection with a battery; and I shall presently exhibit to you their activity. That you may the better understand the construction, I show you an accumulator taken apart.

Here are two pieces of sheet-lead[1] rolled together in a spiral, but prevented from touching each other by strips of rubber about one-fourth of an inch thick. (They are well shown in the open box represented in Fig. 1.) They are immersed in a glass jar containing water acidulated with *ten per cent.* chemically pure sulphuric acid. Above the rolls the jar is tightly packed with a mass that prevents evaporation, and from the top project two poles, one connected with the outside end of the outside roll, and one with the inside end of the inside. The jar is placed into the accumulator-box, and the two poles of the rolls of lead are connected by conducting wires with copper-bands going to the galvanometer and poles of the accumulator. The box is provided with metallic connections for the reception of the battery-cords. (These are marked + and — on the right hand side of the box in Fig. 1.)

When connected with a battery, the water in the jar is decomposed; oxygen goes to one of the rolls of lead and produces a superoxide of lead, so that this roll becomes quite dark, in fact, brownish black; hydrogen is deposited on the other, and this remains metallic or looks bluish gray. When, after this has taken place, the two rolls are connected, a so-called "polarized current" results, and the electricity is sufficient to heat platinum to incandescence; in the meantime the superoxide of lead loses its oxygen, and the other roll its hydrogen, and a recomposition of the water occurs. The process of first superoxidizing and then deoxidizing the roll of lead, may be repeated over and over again. Not only need the contents of the jar never be changed, but the older they become the easier and better they will work. At first the accumulator requires either a powerful battery or connection with a feeble battery for a long time; but, after repeated use a comparatively weak battery, connected for a short time, suffices for longer continued heating effects. When the operator is hurried, it may be necessary to use a Bunsen battery for from half an hour to several hours; but even then the accumulator has great advan-

[1] Faure has recently claimed that to coat the lead with red-lead is a great improvement.

tage over the direct employment of the battery in convenience of transportation, ease of manipulation, and certainty of action. When the box is, so to speak, "full of electricity," you can take it under your arm or carry it by the handle into your operating room or to the bedside of your patient, without danger of

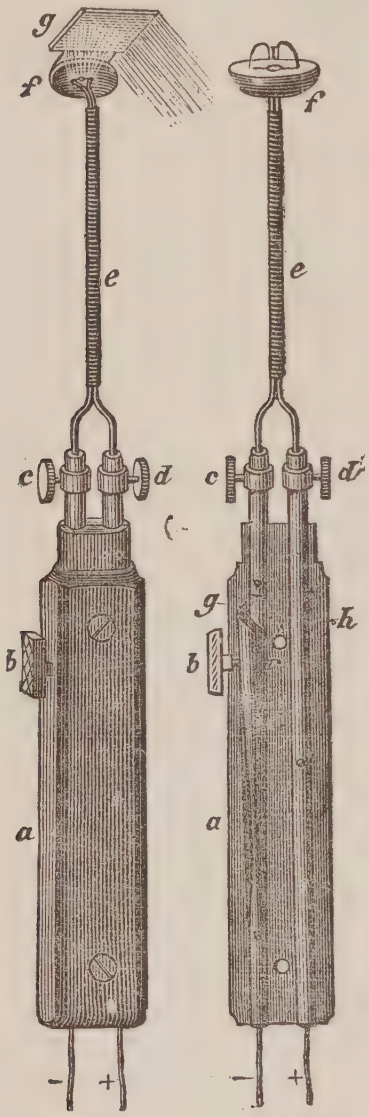

Fig. 2.

Fig. 3.

Fig. 4.

Fig. 5.

Fig. 6.

Figs. 2 and 3 show the handle, the latter in section. The electrode cords, marked + and —, are inserted into metallic cylinders, one of which, *a*, is cut at *g*, the connection being restored by pressing on the button, *b*. At *c* and *d* the illuminators or cauterizers are attached. In Fig. 2, a laryngoscopic mirror is shown in action.

Fig. 4 shows how the illuminating-wire, a little flattened in the middle, is attached.

Fig. 5 shows a pointed galvano-cauter, and Fig. 6 a flat galvano-cauter or knife.

spilling acid, and with the assurance that the apparatus will work until the "electricity is spent," *i.e.*, until the recomposition of water has taken place.

If you have from twelve hours to two or three days' time, any galvanic battery is applicable for the accumulator. I have made use of the battery of the burglar-alarm of my house; ordinarily I use the gravity battery of Trouvé-Callaud, a modification of Daniell's, which I leave connected nearly all the time with the accumulator, and thus have the latter always in readiness. I succeed with this small apparatus in heating a platinum wire loop, one-half to one millimetre thick, or an ordinary size galvano-cautery knife white hot, and can keep illuminating wire one-eighth of a millimetre thick, and one or two millimetres long, incandescent for from two to twenty minutes. The maximum effect claimed for Trouvé's apparatus is that it can keep the cautery knife incandescent for fifteen to twenty-five minutes, and the illuminating wire for two or three hours consecutively; and I have designed a large-sized accumulator, of which Messrs. John Reynders & Sons are the manufacturers, which is still more effective. I have long used this apparatus daily for galvano-cautery operations in the throat and nose. It has never disappointed me. I would not for a good deal do without it; and I recommend it most strongly.

As it is not my object this evening to dwell upon the electrodes for either illumination or cauterization—intending, with your permission, to present improved apparatus of this kind to the Academy on a future occasion—I shall merely show you the operation of the accumulator in heating to incandescence the reflector and cautery instruments of Trouvé. (See Figs. 2 to 6.)

LESIONS OF THE ORBITAL WALLS AND CONTENTS DUE TO SYPHILIS.

By CHARLES STEDMAN BULL, A.M., M.D.,

NEW YORK.

(Read October 20, 1881.)

Under the above head will be considered the syphilitic diseases of the bones forming the walls of the orbit and of the connective-tissue and adipose-tissue of the orbital cavity, in so far as the two may be connected. No reference will be made to syphilitic lesions of the eyeball or of the adnexa, which are contained in the cavity of the orbit. Disease of the bony walls of the orbit is not a very common manifestation of constitutional syphilis; though it is by no means rare. The lesions which occur here are 1st, a periostitis or osteo-periostitis, with or without sub-periosteal abscess; 2d, gummy tumor or syphiloma of the periosteum; 3d, periostosis, hyperostosis, or exostosis of one or more bones; and 4th, caries and necrosis, involving more or less of the entire thickness of the bony walls. Clinical observation would seem to afford ground for the belief that the bones of the orbit are not as frequently affected by syphilis as other parts of the bony skeleton, but the dead-house teaches a somewhat different story, and I am inclined to think that a more careful and minute examination of the patients in the venereal and surgical wards of our large hospitals would lead us to alter our opinion in regard to the frequency of the occurrence of the bony lesions in this region. Some of the symptoms are slight in severity and transient in duration, and often are not pronounced enough to attract the attention of any one but the patient. These lesions, according to most authorities, belong to the late stages of syphilitic infection; though the most recent investigations seem to point to the

existence of two forms of periosteal disease due to syphilis, which are to be distinguished from each other by the intensity of the process, and the period of constitutional infection at which they occur. It seems to be a recognized fact that the cases of syphilitic ostitis and osteo-periostitis developed during the early or secondary period of constitutional infection are much less severe than those observed later. The latter are accompanied not only by subperiosteal and osseous gummata, but also by dense ostitis and necrosis. In the late as in the early osseous symptoms of constitutional syphilis, the exciting cause of the bony lesion and of its location is generally found in contusions, repeated bruisings and slight injuries.

Simple syphilitic periostitis, or osteo-periostitis, occurring as an early or precocious bone lesion is limited to the surface of bone, to the periosteum and superficial layers. Under the periosteum, between it and the bone, is accumulated a large number of round cells, analogous, according to Cornil, to the cells of the embryonic medulla. At the same time the deep layers of the periosteum are inflamed and contain cells between the fibrous bundles. The neighboring connective tissue generally shows some inflammatory œdema, which accounts for the thickening and swelling observed between the skin and the bone. When the bone disease has lasted a long time, the round cells nearest the surface of the bone, beneath the periosteum, act like osteo-blasts during the period of ossification, and in fact become such. In other words, from a histological standpoint, simple osteo-periostitis of the bones of the orbit consists in the return of the cells between the bone and periosteum to the embryonic state. The varieties of new osseous products consequent on ostitis and periostitis, such as osteophytes, exostoses, enostoses, and eburnation, constitute the accidents common to all forms of syphilitic osteo-periostitis. The exuberant formation of the new osseous lamellæ may develop under the periosteum exostoses of varying size, and in the bone itself a parenchymatous hyperostosis and eburnation.

The gummy osteo-periostitis is a variety of rarefying ostitis, in which the subperiosteal embryonic tissue takes on the dis-

position observed in gummata. Gummata of bone are only an intense and limited osteo-periostitis, with destruction of the osseous lamellæ by a rarefying ostitis. Though common in the other cranial bones, they are rare in the bones of the orbit. When a thin section of such a gumma is examined under a high power objective, there is seen to be a tissue formed of very fine fibres, between which exist round cells with a nucleus and a small amount of protoplasm. These round cells, from 0.010 to 0.015 of a millimetre in diameter, are, according to Cornil and Ranvier, embryonic cells. Besides these cells there are other cells of a fusiform or irregular shape, and still others much smaller, apparently atrophied, measuring from 0.005 to 0.006 of a millimetre in a diameter, and almost entirely filled by their nuclei, and embedded in a finely granular matrix. What are called external gummata originate beneath the periosteum, and by pressure gradually detach it from the bone. They also press upon the underlying bone, enter its substance in the form of a cone, the bone becomes infiltrated and progressive rarefaction goes on up to a certain varying point. Then the gumma ceases to advance, undergoes lardaceous or fatty metamorphosis and finally disappears, leaving in its place a more or less extensive depression in the bone. Pathology teaches that the frontal bone is the region of predilection for these gummata. (See a short monograph by Moscovits, "De la Syphilis Tertiaire Crânienne," Paris, 1874.) When a small gumma of the orbital periosteum has formed a cavity in the bone and the inflammatory process is stopped, the new matter becomes caseous and atrophies; the peripheral osteo-periostitis heals, and there may be a partial reparation of bone. The cavity is not entirely filled, but the osteophytes developed beneath the periosteum end in a bony neoplasm at the margin of the loss of substance. If, however, the bone is completely perforated, the defect is not filled up by new bone tissue, but by a fibrous cicatrix.

A very interesting case of osteo-periostitis gummosa of the floor of the orbit has been described by Campana in the *Giornale Italiano delle Malattie Venerie e della Pelle*, anno vi.,

1871. The patient was a man, aged thirty years, who contracted a chancre in his twentieth year, which was followed by inguinal and cervical adenitis, alopecia, pre-sternal and pre-articular pains, with nocturnal exacerbations, ulcers of the fauces, and a pustular eruption on the face and trunk. During the past two years he had had a tumor upon the bridge of the nose, which extended to the inner canthus of the right eye, was at first hard, prominent and immovable, but after a few months it softened, the skin ulcerated and a quantity of stringy fluid was discharged. This remained open for two months, and soon after it closed a second appeared on the floor of the orbit near the outer canthus, which grew to the size of a filbert. Under anti-syphilitic treatment this softened and disappeared by absorption, leaving a slight depression in the bone.

One year later he began to complain of pain in the right orbit again, which spread to the superior maxilla, alveolar arch, and zygoma; there was slight failure of vision and some exophthalmus. A tumor could be felt on the floor of the orbit near the external canthus, which was painful on pressure. This was punctured and a small amount of sero-purulent fluid evacuated. A probe introduced showed caries, but no detached pieces of bone. The next day more pus was discharged and some detritus. The carious process extended to the malar bone, and a puncture made in the superior gingival fornix gave exit to pus, and carious bone was discovered opening into the orbital cavity.

The patient recovered entirely by proper treatment in about two months, and the eye was restored to its normal position.

The two essential signs of syphilitic orbital osteo-periostitis are pain and swelling, the former most intense at night and sometimes very violent. To these two objective symptoms is added a third, exophthalmus or protrusion of the eyeball beyond the plane of the orbit, if the bony lesion be extensive or situated deeply in the orbit. The neuralgia, due to a compression of a nerve-filament at some point in its passage through a bony canal, is a very common symptom. Osteo-periostitis of the bones of the skull produces a tumor, generally

broad and flattened. Subperiosteal gummata, extensive and thick, are generally accompanied by inflammation of the skin, long suppuration, necrosis of the portions of bone which remain imprisoned for a long time, and which eventually are cast off, leaving behind sometimes great losses of substance. The diagnosis of these gummata is not always easy. If the scalp is adherent at a point to the bone, periostitis is almost certainly present, and if a tumor is here present, it is certainly a gumma of the periosteum or a periostitis, according as the consistence of the tumor is elastic or bony. According to Hueter, the formation of a sequestrum rarely occurs here, owing to the slight tendency of the periosteum of the bones in this region to the new formation of bone. He admits that the caries in these cases is, however, generally due to the suppuration of syphilomata, and that they are tolerably frequent. His explanation of the process is as follows: The discolored and dead bone, due to the suppuration of a periosteal syphiloma, does not fall off as a sequestrum, but small granulations come from the diploe of the bone, perforate this dead cortical layer and push aside the latter, which necroses in small particles. The syphilitic tissue-proliferation does not, however, always end in suppuration, but sometimes forms a sclerosed bony substance, and then we have the syphilitic osteoma. These osteomata are generally smooth, flat, and more like hyperostoses than exostoses.

That these bony lesions are sometimes not recognized in the orbit during life and are only discovered at the autopsy, is a fact which we are all forced to admit. A single instance will suffice. In the *Archives de Médecine* for December, 1845, Hamilton reports the result of an autopsy on a patient who during life had had paralysis of some of the branches of the third cranial nerve. At the autopsy there was found caries of the orbital walls due to periostitis and periostosis in the vicinity of the apex of the orbit, which pressed upon the nerves in question and caused the paralysis.

Though these various lesions of the bony orbit are generally regarded as late manifestations of constitutional syphilis, yet

attention has been called to their by no means very rare occurrence as an early lesion, and this is particularly the case with periostitis of the orbit. Much careful study has been given to the early development of osseous lesions in syphilis, especially by Mauriac and Fournier, with reference to their relation to other constitutional symptoms. We know from Mauriac that epicranial periostitis may be one of the first manifestations of constitutional infection, occurring sometimes shortly after the appearance of the initial lesion, even before the appearance of any other constitutional symptom. This periostitis is confined almost exclusively to the periosteum, and whatever hyperæmic or inflammatory condition of the bone may be present, it is merely accessory and entirely secondary to the periostitis. In the adult these periosteal swellings tend to resolution spontaneously, and this can be markedly hastened by proper treatment. They rapidly disappear without leaving any trace. On the contrary, in the hereditary syphilis of children, this form of periostitis very soon assumes the purulent form of inflammation, and ends in the death of the bone. In acquired syphilis this early form of orbital periostitis is circumscribed, varies in duration between four and six weeks when left to itself, and disappears much sooner under proper anti-syphilitic treatment.

Mauriac admits that these precocious bony lesions are much more common and severe in hereditary syphilis than in acquired syphilis, and in warm latitudes than in temperate or cold latitudes. Such an early lesion in the bones of the skull aggravate the prognosis, of course, though the other constitutional symptoms may be slight. It bears no resemblance to the later periostitis, being much less indolent, lasting a shorter time, tending to spontaneous resolution and but little to destructive metamorphosis. Moreover, these cases of precocious periostitis are confined to the periosteum, are never followed by hyperostosis or exostosis, and never leave any trace of their presence. Large, hard frontal bosses, which may involve the entire supra-orbital margin and dip down deeply into the orbit, rapidly disappear under appropriate treatment.

Leaving the subject of the pathology of syphilitic osteo-

periostitis of the orbit with this brief and still unsatisfactory statement of our knowledge of it, we come naturally to a consideration of its *symptomatology.* Two forms of inflammation, acute and chronic, must be distinguished. The acute form is almost always a precocious lesion and the symptoms are apt to be severe. The patient complains of great pain in and around the eye, especially along the superior orbital margin, which is excessively sensitive to pressure, and this has been remarked even when the periostitis was deep-seated and did not involve the orbital margin. The eyelids are red and swollen, the ocular conjunctiva is injected and sometimes chemotic. This latter symptom is much more marked if the orbital cellular tissue is involved. There is more or less protrusion of the eyeball, according as the seat of the periostitis is deep in the orbit or near the orbital margin. The exophthalmus is rarely straight forward, but usually toward one side or downward, owing to the periostitis being confined to one wall or part of the orbit. The general constitutional signs of inflammation are usually severe in acute osteo-periostitis of the orbit, owing to the orbital cellular tissue becoming involved, and then the exophthalmus may be very marked. Another danger is loss of vision by pressure upon the optic nerve in the optic foramen or in the orbit, or loss of the eye by strangulation of the entire blood supply of the eyeball by the infiltrated orbital tissue. Optic neuritis followed by inflammatory atrophy of the optic nerve extending to the sheath of the nerve from the periosteum of the orbit and optic foramen; or simple atrophy of the optic nerve from pressure by the surrounding infiltrated tissues, are not very uncommon results of syphilitic orbital osteo-periostitis. When the orbital cellular tissue becomes inflamed, there is much more probability of the formation of pus in considerable quantity than when the periosteum alone is involved. The pain, which is usually very severe, may occur only periodically at certain times of the day. The attack may be ushered in by a chill, followed by high fever, and all the other symptoms are rapidly developed.

In the chronic form the inflammatory symptoms are far less pronounced, and the disease is more protracted and insidious in its course. The disease in the bone is often developed very slowly and gradually, and when the inflammation is deeply seated, it is easily overlooked. There may be little or no febrile excitement, but little pain, no protrusion of the eyeball until late in the course of the disease, and, in fact, scarcely any objective symptom, unless the periostitis is located near the orbital margin. Here the œdematous swelling of the surrounding parts, the decided thickening of the bone, and the presence of a hard, indistinctly fluctuating tumor which is painful and sensitive, all aid in the diagnosis. The chronic form is very often accompanied by the formation of a subperiosteal abscess, which sometimes strips up the periosteum from the bone for a long distance, and tends to end in caries and necrosis of the bone. Osteo-periostitis gummosa is always of this chronic type. The abscess once formed tends to open at some point, and this is usually outwardly through the conjunctiva or lid by one or several sinuses; but sometimes the purulent process has been so extensive that caries has occurred in several directions, and here the abscess may open into the nose, the frontal sinus, the maxillary sinus, or, gravest of all, into the cavity of the skull. If the abscess perforate through the conjunctiva, it is almost certain that the seat of the bone lesion is deep in the orbit, for if the margin of the bony orbit were involved, the opening would be in front of the tarso-orbital fascia, through the lid. If the tendency to suppuration is but slight, the periosteum may become very much thickened, and small nodules or periosteal growths may be developed, which may ossify and form true exostoses. These may exist between the periosteum and bone, or more rarely these nodules may grow in both directions, outward toward the orbit and inward toward the bone.

One danger which might seem to be imminent, very rarely occurs, and that is an extension of the periosteal inflammation to the meninges of the brain through the medium of the optic foramen. It is far more likely to induce meningeal inflamma-

tion when the roof of the orbit is the seat of the lesion, for here the bone is quite thin, and a carious process once started in this region may soon perforate the bone and open into the anterior fossa of the skull, leading to meningitis or abscess of the brain.

Though clinical observation teaches us that syphilitic periostitis of the orbit almost always ends in resolution, without permanent injury to the bony walls, if properly treated in the beginning, yet sometimes it leads to caries and necrosis of the bone. Where a sinus exists, the introduction of a probe proves the roughness of the bone, and this with the continued patency of the sinus proves that the disease has passed from periosteum to bone. Pieces of loose bone, no matter how small, are but rarely found, for though the bones are very thin, they are hard, and we are more likely to meet with a hole communicating with a neighboring cavity than with splinters of loose bone. Yet even this complication is uncommon, for the caries is almost always superficial, and the case ends in recovery after the carious bone has lost its roughness and the sinus closes. Caries and necrosis of the bones of the orbit occur most frequently at the margin of the orbit, or at the upper and inner corner of the roof. While most of these cases start with a periostitis or osteo-periostitis, the lesion may begin in some cases as a real ostitis, and involve the periosteum secondarily. These are probably the worst cases, for the lesion is usually situated deep in the orbit, has spread to the bones of the orbit from some other bone of the skull, and involves the orbital tissue, ending in prolonged suppuration. This has been known to spread from a syphilitic ozœna in the nose, extending to the ethmoid cells, thence to the orbital plate of the ethmoid, and finally to the orbital tissue. Here the process is a very chronic one, even under the most rational and persistent treatment, and results in extensive exfoliation of bone and considerable deformity. In these severe cases several sinuses may form in the lids or surrounding structures in different directions, through which pus and small fragments of exfoliated bone may be discharged for an indefinite period, and ending either in deeply

retracted cicatrices or in eversion of the eyelids. In some instances, where this extensive disease of the bones in the vicinity of the orbit has gone on to its natural termination, independent of any treatment, the adhesions between the bone, periosteum, and external soft parts, as for instance the eyelids, have been found so dense and firm that any operative attempt to separate them has been proved impossible. This syphilitic caries of the orbital margin, though usually a late manifestation of constitutional syphilis, has been known to occur among the precocious lesions. Thus Del Toro reports a case of primary caries of the superior orbital margin occurring in a patient five months after the contraction of the initial lesion and without any intervening constitutional symptom. The course of this carious disease of the bones here is always a chronic one, even when it is a precocious lesion, and though amenable to treatment, a rapid cure cannot be expected. A brief report of two or three cases of orbital osteo-periostitis and cellulitis ending in caries, may serve to illustrate the course of the disease and the destructive changes occasionally resulting therefrom.

The first case was that of a man aged forty-six, whom I first saw in June, 1875. He had contracted the initial lesion twelve years before, and had had numerous obstinate and destructive constitutional lesions. When I saw him, there was an extensive papular eruption on the face, neck, arms, and back. For about five weeks he had had a constant violent pain in the right orbit and temple, for which nothing seemed to do any good. There was marked exophthalmus downward and outward, the eye being on a plane with the bridge of the nose but much lower, and markedly limited in motion in all directions, especially upward and downward. There was great swelling of the lids and marked chemosis of the ocular conjunctiva. The pain was very severe and the slightest pressure on the orbital margin produced intense suffering. There was well-marked neuro-retinitis, the right eye presenting a typical picture of choked disk with hemorrhages, and vision was reduced to perception of light. Five years before he had had an attack of caries and necrosis of the upper portion of the frontal bone

and the adjacent part of the right parietal, which had lasted seven months, had extended to the external angle of the orbital margin and had left only the inner table of bone untouched. Prompt anti-syphilitic treatment was at once instituted, consisting in half-grain doses of calomel every hour until its constitutional effects began to appear, and then large doses of iron and quinine were administered. On the second day the mercury was discontinued, and then potass. iodide, grs. xxx., was administered three times a day. Leeches had previously been applied to the temple and a saline purge given. After five days signs of fluctuation appeared just inside the upper orbital margin, and a free incision gave exit to a small amount of very offensive pus. With the probe carious bone was discovered in the roof and inner wall of the orbit as far as could be reached. The wound was kept open and hot applications made. From time to time small particles of bone came away, and once a piece of sequestrum was removed by the forceps, which measured about 5 mm. long, and somewhat less in width, and proved to be part of the ethmoid bone. After a period of nearly three months the man was discharged cured of the attack of caries, but with total blindness and pronounced inflammatory atrophy of the optic nerve due to compression within the orbit. Two months later he had another attack of orbital osteo-periostitis and caries, ending in meningitis and death.

A second case was in a woman, aged thirty-two, in whom the date of the initial lesion was unknown. There had been a large ulcer on the right patella ten years before, which remained open six years. Subsequently she had ulcers on the fauces and pharynx, and at the same time dacryocystitis on the right side, with stricture of the nasal duct. This opened externally along the side of the nose and a fistula has remained ever since. Caries of the orbital margin next appeared, which involved the malar bone, the ascending process of the right superior maxilla and the floor, roof, and inner wall of the orbit so extensively that a probe could be passed into the maxillary sinus, ethmoid cells, superior nasal meatus and frontal sinus on the right side. There was also exten-

sive disease of the nose and an irido-cyclitis gummosa. While under treatment she developed symptoms resembling meningitis most strongly, but they lasted only a few days and then she began to improve. This patient eventually recovered, but only after about eighteen months' treatment.

The rapidity with which caries follows periostitis of the orbit, regarded as a late manifestation of syphilis, is well-shown in the following case: A gentleman, aged thirty-six, who had led a dissipated life from early youth, was first seen by me in November, 1874. He had contracted a chancre twelve years before, which was followed by lesions of the glands and skin of rather a mild character. Six months later he had double iritis, which lasted seven weeks, and left the vision in the left eye very defective. Ten days before he presented himself to me he was attacked by a severe pain along the margin of the right orbit, which rapidly extended to the malar prominence and zygoma, and thence to the articulation of the inferior maxilla and roof of the mouth. When I saw him these signs of orbital periostitis were extremely well marked. The exophthalmus and convergent squint were pronounced, all the tissues were very much swollen and very sensitive, there was great difficulty in opening the mouth, the lids were swollen and the eye pushed upward and inward. The periostitis evidently involved the floor and outer wall of the orbit, the malar bone and zygoma, and had spread to the superior maxilla and perhaps to the lower, and within a period of ten days. In spite of urgent and rapidly pushed anti-syphilitic treatment, the disease progressed, subperiosteal abscesses formed in the floor of the orbit and over the malar prominence, and opened, one through the conjunctival cul-de-sac, the other through the skin of the cheek, and the bone in both places was found roughened and diseased. In the course of a few days the caries in the floor of the orbit opened into the maxillary sinus, and that in the malar bone extended superficially in every direction. The carious process was finally stayed by large doses of potassium iodide, but not until the entire floor of the orbit and part of the outer wall

necrosed and either came away or was removed. It is not often found necessary to resort to operative interference in cases of caries and necroses due to syphilis; but in this case the caries was so extensive as to endanger the preservation of the eye, if not of life itself, and it seemed wise to enlarge the opening of the sinuses leading down to the seat of disease and remove what pieces of bone could be found loosened. In this way the entire orbital plate of the superior maxilla was removed in three pieces, and several small pieces were also removed from the orbital edge of the malar bone, and one from the external angle of the frontal bone. This patient subsequently made a good recovery.

Just how far it is justifiable to operate for the removal of carious bone in the orbit is a somewhat difficult matter to determine. Cases have occurred in which the carious process has involved the ethmoid bone and roof of the orbit and has opened into the ethmoid cells and into the cavity of the anterior fossa of the skull. In the latter case the condition of the patient is desperate, for the purulent process in the bone may extend directly to the meninges and kill the patient, or some loosened fragments of bone may set up meningitis by irritation of the meninges, or the carious process in the bone may develop a subdural or a cerebral abscess, even without perforation of the roof of the orbit, through the media of the fine foramina for the passage of the nutrient blood-vessels. It would seem safer and better surgery to remove all pieces of loosened bone, even from the roof of the orbit, through a free opening, thus doing away with one source of cerebral irritation and bringing about free drainage. If it is the ethmoid that is involved, and internal medication does not put a stop to the carious process, we should not hesitate to remove as much of the diseased bone as can be reached, even if we open into the ethmoid cells. Suppuration would almost inevitably be established here, even if the dead bone were not removed, and by removing the carious fragments, we do away with one source of continuous irritation, and also render easier the introduction of a drainage-tube or threads through the nose, which is a very necessary

procedure. The same thing holds true of the lachrymal bone, which, however, is not so frequently the seat of carious disease as the ethmoid, unless from chronic dacryo-cystitis and disease of the nasal duct. Extension of the carious disease from the nasal fossæ to the lachrymal and ethmoid bones is not an uncommon occurrence, but it seems better to consider the latter complications as more properly belonging to the category of diseases of the lachrymal apparatus, and this is particularly the case with the lachrymal bone.

Perhaps the most interesting cases of syphilitic orbital disease to the clinical observer are those patients who present the results of chronic hyperplastic bone disease, such as periostosis, hyperostosis, and exostosis, both on account of their rarity and of the possible resulting deformity. There seems to be still some doubt as to the pathogenesis of periostosis, pathologists being divided in opinion as to whether it is the natural result of a plastic periostitis, or whether it is a distinct pathological process in itself. It is certainly a rare process in the orbit, where periostitis syphilitica usually either yields to treatment and leaves no trace of its presence, or else ends in suppuration and caries. Periostosis here is probably a chronic periostitis which has ended in induration or sclerosis, forming a tumor more or less circumscribed along the orbital margin, and very rarely occurring in the deeper parts of the orbital cavity. Ricord believes in the existence of three kinds of periostosis, inflammatory, gummy, and plastic, of which the last is probably a stage of the first. He cites but one case of the gummy variety, occurring deep in the orbit on the nasal side, and which was probably nothing more than a periostitis with the formation of a sub-periosteal gumma. It is probable that the process is simply a thickening of the periosteum and that the term node would apply equally well to circumscribed periostoses of the orbit, as in other parts of the body. They never occur as precocious lesions of syphilis, but are late manifestations, the result of long-continued plastic inflammation, originating probably in the periosteum and confined to it, and only in isolated cases ending in ossification. They are gener-

ally sensitive to pressure and painful at certain periods of the day. If they happen to occur in the vicinity of the supra-orbital or infra-orbital foramina, there is more or less trifacial neuralgia all the time, which increases in severity as the periostosis spreads. Though rare under any circumstances, and almost always observed along the orbital margin, it is probable that they occur deep in the orbit, at or near the apex and around the optic foramen, oftener than we have supposed. It is probable that many of the cases of paralysis, partial or complete, of one or more of the extrinsic muscles of the eye, coming on somewhat gradually, are due to a periosteal node pressing on the muscle or its nerve-branch in its course or near its origin, producing at first paresis and then paralysis by direct pressure as it grows.

Such a node, growing from the periosteum at the extreme bottom of the orbit, might, if of any size, easily involve the origins of all the straight muscles of the eye, and this without any very great projection into the cavity of the orbit. Of course, in such an instance, the optic nerve would probably also be involved, and there would be atrophy of the nerve fibres, perhaps preceded by neuritis descendens. These cases, the writer believes, are not so very uncommon, and they offer a plausible explanation of the reason why so many cases of paralysis of the ocular muscles in syphilitic patients are not cured by well-directed anti-syphilitic treatment. The periosteal thickening goes on gradually involving the origin of the muscle or its motor-nerve branch, until the latter becomes atrophied from compression, and then, although in favorable cases the periostosis may be absorbed by treatment, the mischief has been done, and the paralysis is permanent. Another symptom which may be produced by periostosis deep in the orbit is exophthalmus. The form of periostitis involved in periostosis does not tend to spread, and hence is but little likely to involve the orbital tissue. Any projection of the eyeball is here due to the periostosis itself. Furthermore, there are no signs of acute inflammation, no constant pain in the orbit, and no sensitiveness to pressure along the orbital margin. On pressing the eye

backward pain is experienced, but the process may go on from the beginning without any pain, and the patient's attention may first be attracted by the exophthalmus, more or less limitation of motility of the eye, then diplopia or double vision, and finally impairment of vision.

Hyperostosis is the rarest of all affections of the bones of the orbits. It differs from periostosis in being a disease of the bones themselves primarily, and not of the periosteum, except secondarily. It was formerly supposed that it was the consequence of an arrest of inflammation in the bone before necrosis set in, but it is now known that the thickening of bones may go on for an indefinite period after the cessation of all symptoms of inflammation, and it is possible that hyperostosis may occur without any inflammatory symptoms. The process may affect the entire thickness of the bone, and in the orbit this would probably be the case. The excessive development of one or more of the bones of the orbit would produce very singular changes in the shape of this cavity, the most marked symptom being exophthalmus. There are very few cases of this sort in literature, and the writer has seen but two in the course of thirteen years; one of which was probably not due to syphilis. (See *New York Medical Journal*, November, 1879.) The other case was in a man, aged forty-nine, who had been syphilitic for seventeen years. The hyperostosis involved the adjoining portions of the frontal and malar bones on the right side. It had been of slow growth, painless, extended slightly backward from the margin of the orbit along the outer wall and floor of the orbit, and had pushed the eye toward the median line, and slightly forward. The latter was somewhat impeded in its motions, but not markedly so. The patient stated that the growth of the bone had extended over a period of about five years. His constitutional symptoms had been severe, and he had had repeated attacks of iritis in both eyes, with the development of a gumma of the ciliary body in the right eye, resulting in very defective vision. The treatment instituted seemed to have no effect in diminishing the hyperostosis, though there was apparently no increased thickening while the

patient was under observation, a period of eight months. This hyperostosis is a hyperplasia of bone, and is distinct from exostosis: it is of ivory hardness, and never yields to any constitutional treatment. It is more often due to some other cause than syphilis. Any operation for its removal is only justifiable when its mechanical presence, as a hindrance to the functions of the eye, demands it, and its removal is then best effected by a mechanical drill, such as the dentists use.

There remains the subject of orbital exostoses, due to syphilis, to be considered. Under this head is meant those outgrowths from the periosteum or bones of the orbital walls toward the orbital cavity. These exostoses differ from the swelling and projection of periostosis in size and shape, and somewhat also in location. Though occurring in all parts of the orbit, they are more frequently met with on the inner wall and near the margin of the orbit, than in the direction toward the apex of the cavity. They are usually smaller than a periostosis, with a narrower base, but project more into the cavity of the orbit. They do not differ in their growth and appearance from the exostoses due to other causes, are always covered by periosteum, and frequently do not involve the subjacent bone at all. They are more frequent than the periostoses and much more so than hyperostoses, in the orbit as elsewhere in the body. The pathology of these outgrowths is not always clear. They may develop in consequence of long-continued chronic periostitis, just as periostosis may be caused, and the two lesions may exist simultaneously in the same case, though the periostosis is almost always the earlier in appearance of the two. They may, however, occur alone, as direct outgrowths from beneath the periosteum, without any tendency to periostosis, and sometimes without any signs of periostitis, unless it be a circumscribed inflammation. If they are situated deeply in the orbit, the most marked symptom is exophthalmus. The os planum of the ethmoid bone is a favorite seat of exostoses, and there may be several, all small, growing from this bone into the orbit. Where they occur anteriorly and admit of digital examination, they are recognized as hard, smooth ele-

vations with circumscribed base, not usually painful on pressure, but generally causing pain from pressure upon the eyeball or upon the nerves within the orbit. Though usually slow in growth and a late manifestation of constitutional syphilis, they may advance rapidly in size and be accompanied by some of the signs of acute, localized inflammation. Several of these small exostoses have been known to appear on the inner wall or floor of the orbit, and after pursuing a short, isolated existence, have coalesced to form one large projection, which interfered seriously with the movements of the eyeball. The more deeply in the orbit these exostoses are situated, the more apt are they to escape attention, unless of so large a size as to cause protrusion of the eye from the orbit. When near the apex of the orbit their pressure upon the ciliary nerves, or the ophthalmic branch of the trifacial, causes deep-seated pain, which may be located in the cavity of the skull and excite suspicions of the presence of an intracranial tumor, or periostitis, or perhaps meningitis. If the pressure is so extensive or continuous as to interfere with the return circulation from the eyeball, the ophthalmoscope might reveal the presence of neuro-retinitis with choked disk, or an atrophic condition of the optic papilla, both of which conditions might go far toward strengthening the suspicion of serious intracranial trouble. The history of the patient's syphilitic lesions and the favorable results of a rigid anti-syphilitic treatment in the subsidence of the symptoms, would perhaps aid us in locating the disease within the orbit, though all the symptoms might have been caused by an exostosis at the base of the skull, except the exophthalmus. Of course the treatment of these cases should always be constitutional, at least at first. Though less favorable results are gained from medical treatment of exostoses than of periostitis, yet sometimes they disappear in the orbit very rapidly under the combined use of mercury and potassium iodide. For this reason a very careful examination should be made into the previous condition of the patient as to other syphilitic manifestations. Very often exostoses in the orbit will on careful examination be found to be accompanied by bone lesions else-

where in the body, or to have been preceded by a chronic periostitis with no very pronounced symptoms; the following case well illustrates this. A patient, Charles R——, aged forty-six, a blacksmith, presented himself with slight exophthalmus downward and outward, as well as forward, on the right side. There had been no pain of any kind for seven or eight months, but at that time he had complained of some deep-seated pain in the orbit, which, however, lasted only a few days, then disappeared and did not return. The exophthalmus was first noticed about two months before, but he paid very little attention to it until he began to see double. The diplopia was crossed, as might have been expected from the position of the eye. Vision was normal and there was no ophthalmoscopic evidence of disease. A careful examination with the finger and a stout probe revealed an exostosis of considerable size projecting from the orbital plate of the ethmoid bone, and another smaller one projecting downward and backward from the frontal bone, at the upper and inner angle of the orbit. This patient also had a number of dense, painful nodes along the crests of the tibiæ, and two somewhat sharp exostoses on the upper and outer surface of the right clavicle. The initial lesion had occurred eighteen years before, and he had had numerous constitutional lesions; the nodes on the tibiæ he had noticed for nearly two years. These orbital exostoses diminished very much in size under large doses of potassium iodide and the exophthalmus almost entirely disappeared, but the patient withdrew himself from observation before the treatment was concluded.

It sometimes becomes advisable and even necessary to resort to operative interference for the removal of these orbital exostoses, when internal treatment has failed in causing their disappearance by absorption. Generally a pair of strong bone-forceps will suffice to remove them, as they usually have but a narrow base; but sometimes it becomes necessary to employ the chisel and gouge. The following case is an example: A man aged sixty years, with posterior synechiæ and a pupillary membrane in one eye, from repeated attacks of iritis, presented himself for

treatment with exophthalmus of the other side and great pain deep in the orbit and along the lower margin. The protrusion of the eye was forward, outward, and a trifle upward. An examination showed a large exostosis on the inner wall of the orbit, near the margin, and two small ones, close together, on the floor of the orbit. The initial lesion had been contracted twenty years before. A course of rigid treatment was at once instituted, the potassium iodide being increased in amount rapidly until the patient took two drachms three times daily, which he bore very well. Under this and a full tonic course of treatment and diet, all acute symptoms subsided, and the exostoses on the floor of the orbit disappeared; but no apparent effect was produced on the large one attached to the inner wall, except that it appeared somewhat flatter and smoother. It was deemed best to remove it by an operation, and this was done through the skin of the upper lid, by detaching the inner half of the upper lid from the orbital margin, enlarging the opening into the orbit with the finger, and applying a strong pair of bone-pliers to the base of the exostosis. A slight rocking movement of the forceps brought away the growth and a part of the orbital plate of the ethmoid with it, and the ethmoid cells were laid open. A small rubber drainage-tube was introduced and brought out through the skin at the side of the nose. Somewhat free suppuration followed, but the wound eventually closed and the exophthalmus receded entirely, though the eye was somewhat limited in its motions laterally, and there remained a partial ptosis.

PYÆMIC PAROTITIS.

By CHARLES A. LEALE, M.D.

NEW YORK.

Synonyms.—Symptomatic parotitis or parotiditis; metastatic parotitis; suppurative parotitis; embolic parotitis; septic parotitis.

Pyæmic Parotitis.—That form of suppurative destruction of the parotid gland following diseases in other parts of the body, in contradistinction to the idiopathic contagious inflammation, or what is commonly known as mumps.

This form of parotid abscess, I believe, always is due to a solution of continuity of a membrane, generally at some distant part of the body. The ulcerative form of inflammation proceeds, until coagula are produced in the venous or lymphatic capillaries, ready at any time to become softened, broken into small fragments, or loosened *en masse*, filled with the bacteria which are always present in these indolent ulcers.

In regard to septicæmia and pyæmia, although we may have the two diseases combined, yet I consider them distinct, due to as different causes as scarlatina and variola. In the puerperal state, I have seen deaths from septicæmia alone, pyæmia alone, and scarlet fever alone; but in pyæmia I have only observed metastatic abscesses where some point of ulcerative suppuration previously existed in the body.

We have all probably seen the analogous condition where a slight suppuration under the nail of the big toe was followed by inflammation of the lymphatics of the groin, or where a minute ulcerating crack in the skin of the finger was followed by inflammation and suppuration of the axillary glands. The most rapid cure is by first treating the original source of infection at the toe or finger. At these points we generally find

that low grade of ulcerations, without much tendency to spread, and which, though small of themselves, continue to have their poison absorbed by the lymphatics, and frequently to be lodged in the glands of the groin or the axilla, when suppuration follows.

In pyæmic suppurative parotitis, such a source of infection can, I think, be found in ulcerations of a mucous or serous membrane, or of the cancellated tissue of bones. This would explain the occurrence of suppurative parotitis, during the course of typhus and typhoid fevers and dysentery, where ulceration of the mucous membrane of the bowels so often ensues, and following the ulcerations of peritonitis and cellulitis, and in ulcerations of the equally delicate tissue lining the Haversian canals, as we see in suppurative osteo-myelitis.

During the past week, I have observed this form of ulceration after the high grade of inflammation seen in the diphtheria of the posterior fauces, where, after the violence of the inflammation had subsided, the mucous membrane of the parts remaining red and congested, a small ulcer the size of a pea was left on the veil of the palate.

The original source of infection in suppurative parotitis is situated generally at some portion of the body impossible to treat locally. This would be one explanation why these metastatic abscesses of the parotid, liver, etc., present, frequently, such alarming symptoms, and so often prove fatal. The poison is continually increasing and entering the circulation at some hidden part, until, as a rule, death from general toxemia and exhaustion ensues. In those cases where recovery follows, active suppuration of the parenchymatous tissue of the gland is generally noted, or the coalescing of the minute multiple abscesses, whereby a free drainage of the purulent matter after an operation expedites the cure. Whereas, when the abscesses do not coalesce, but continue to present an indolent grade of inflammation, evidences are shown that the general nutritive functions of the body are impaired, and an unfavorable result may be expected.

Clinical investigation teaches that where the suppuration of

the parotid gland occurs during the course of diseases, where ulcerations follow violent inflammations, it is of septic origin; that materies morbi enter the circulation by the open mouth of the vein or lymphatic at the point of ulceration, and are arrested in the capillaries of the glandular tissue.

Case I.—Metastatic abscess of the parotid gland, with discharge of pus through an opening into the cartilage at the floor of the external auditory canal. Complete destruction, by suppuration, of the entire parotid gland—with the consequent deformity, a deep hollow beneath and under the angle of the jaw—following an attack of general peritonitis, suppurative pelvic cellulitis, and simultaneous with a lateral pharyngeal abscess. (Patient presented at the Academy on the evening of the reading of this paper.)

Mrs. C., aged forty-one, native of New York City, mother of one child, aged eleven years. Had very difficult labor; chloroform and forceps. Nursed the child for one year, when she became regular, and continued so for three months, when a slight constant flow for one month ensued, then a supposed miscarriage, which proved to be hydatids, about two quarts being removed by the attending physician. Profuse hemorrhage followed, leaving the uterus in a weakened condition, the patient never again conceiving. In 1878 she had an attack of idiopathic peritonitis, and again was taken ill July 10, 1880, with acute general peritonitis and pelvic cellulitis, caused by a chill while in the cellar. The peritoneal inflammation was very intense, and, when it subsided, pus began to form in the pelvic cellular tissue.

The propriety of making an opening into the vagina was now considered, but on account of the small quantity of pus, and the exceedingly grave symptoms, it was not considered justifiable to run the risk of the shock. All was done to sustain the patient—best of nourishment, nursing, and hygienic surroundings. She had a high fever, the temperature, on several occasions running above 106° F., notwithstanding the liberal use of water and antipyretic remedies. She remained without

much improvement until July 31, 1880, when fluctuation could be detected (deeply-seated) in the parotid gland. I made an incision with a tenotomy-knife through the skin, dense fascia, into the centre of the glandular tissue, before pus was reached, when only about a quarter of an ounce escaped, as the blunt-pointed director was pushed into the gland. The opening into the parotid gland was kept open with oakum, and discharged very sluggishly.

August 2d, pus was found to be flowing from the ear. After cleansing, and, when pressure was exerted on the parotid gland, pus could be seen exuding, anterior to the membrana tympani, through an opening into the floor of the cartilage. Only a small quantity of pus came from the ear, probably half an ounce daily. There were profound symptoms of disturbance at the base of the brain, low, muttering delirium, and slight opisthotonos, probably from serous effusion. The pupils were largely and equally dilated, and not sensitive to light. After the return to consciousness there was total blindness, continuing for more than twenty-four hours; a lighted candle, when placed within six inches of her face, could not be seen. An abatement of the cerebral symptoms in a few days followed the reappearance of those referrable to the pelvis, and a spontaneous opening and discharge of pus per vaginum gave great relief. The purulent matter for weeks flowed from the vagina, which was cleansed daily with a solution of the chloride of zinc. A lateral pharyngeal abscess appeared, and nearly caused death by suffocation. I was sent for, and, arriving just in time, immediately opened it with a gum-lancet, the long handle held over the point of the forefinger as a guide. The discharge of pus from the abscess in the pharynx was very thick, but did not continue long after the first evacuation, and never required a second operation for exit. The director could be pushed through the parotid opening, and its point felt with the finger in the throat, showing through and through drainage. The suppurating parotid gland continued to discharge through the incised wound for nearly three months, and continued from the ear for over a year.

The scalp at convalescence was covered with scaly dandruff, and nearly all the hair fell off the head. It was shaved three times, and poultices of flaxseed were applied for a month. During this time I removed eight sebaceous tumors from the scalp, from the size of a bean to that of a small walnut. By stimulating lotions, a magnificent growth of hair has been produced. Syr. ferri iodidi, ℥. xv., ter in die, was given for months. She can now see imperfectly, but hears nearly as distinctly as ever.

Case II.—Metastatic abscess of the parotid glands, with discharge from both ears; after perforating the floor of the cartilage, at its junction with the osseous portion of the temporal bone, the pus burrowing posteriorly over the superior surface of the osseous portion of the temporal bone, beyond the membrana tympani of each ear, followed by perforations, and exit of the pus through the external auditory canal. Complete deafness for three weeks, unconsciousness, formation of polypi protruding through ruptured membranes, profuse suppuration, lasting for seven months, followed by an excellent recovery, suppuration entirely ceasing, and hearing of both ears becoming good.

The treatment at first consisted in thorough cleansing, every two hours; the delicate application of pure nitric acid to the polypoid growths, gentle rubbing of the gland upward, syr. ferri iodidi, ten drops thrice daily, and the best hygienic surroundings at the sea-side.

Case III.—Abscess of the parotid gland in a young lady, aged fourteen years, appearing within three or four weeks after a very severe attack of typhoid fever. Abscess also formed in the cheek over the duct of Steno. Complete occlusion of the duct at its exit in the mouth. Operation under the lobe of the ear, establishing good drainage from the parotid abscess, and the abscess communicating with Steno's duct over the cheek. Long continuation of the fistulous opening, and long lasting adhesions of the cheek, followed by extensive enlargement of the thyroid gland, and successful subsequent treatment by iodine of the goitre.

Patient presented at the Academy on the evening of the reading of this paper.

Miss E. R., aged fourteen, native of, and residing in New York City, only child of parents in good health, during the autumn of 1869 had a very severe attack of typhoid fever, with enteric hemorrhage and great abdominal tension. She had a temporary abatement of the symptoms between the third and fourth weeks, after which alarming symptoms again appeared, viz., mental disturbance, suffused conjunctivæ, dry tongue covered with sordes, rapid pulse, feeble heart's action, hypostatic pulmonary congestion, abdominal distention and diarrhœa. At the beginning of the fifth week from the commencement of her illness the right parotid gland became tumefied, and at the same time the cerebral disturbances increased in severity. In a few days the cheek grew quite hard, and soon a circumscribed swelling could be detected at the apex of the socia parotidis, and directly over Steno's duct. The tumefaction of the integument, on becoming more defined, revealed fluctuation at this point, where nature was endeavoring to make a spontaneous opening. Pressure on this part showed complete occlusion of Steno's duct, and would not force any of the liquid through the ductus Stenonianus at its exit on the inner surface of the cheek, opposite the second molar tooth of the upper jaw. As the coverings of the fluctuating portion of the cheek at this point were rapidly giving way, the question arose how to prevent permanent deformity in this young lady, arising either from a salivary fistula or a lasting mass of cicatricial tissue, on a conspicuous part of the face. The inside of the cheek did not appear to offer any advantages for a good exit, as a probe could not be passed through the opening of the duct. I therefore decided to operate in the fossa, just beneath the lobule of the ear, between the temporal and posterior auricular arteries, and above the facial nerve, as it makes its exit beyond the border of the sterno-mastoid muscle. An incision was made, a quarter of an inch in length, through the skin and fascia, into the parotid gland, with a tenotomy knife, when thick, creamy pus followed. The opening was slightly en-

larged by tearing the deep-seated parts with the point of a firm director, and not by incision, in order to guard against wounding any of the numerous arteries and nerves in this region. It was now easily seen that pressure of the fluctuating point at Steno's duct on the cheek, caused the pus to flow in increased quantities from the wound behind the ear, and that the tumefaction of the cheek diminished. After the parotid abscess had been as much emptied as was thought desirable, by pressure in the direction toward the opening, a small sponge-tent was inserted into the incision to prevent closure, and warm-water dressing applied. The swelling in the cheek threatened to burst for nearly a week after the operation. At the end of four weeks the opening behind the ear was discharging thick, viscid fluid, making me fear a permanent salivary fistula at that point. The attack of typhoid fever was so severe that the patient kept her bed for two months, and was not able to leave her room for over thirteen weeks. About the end of that time the fistula closed, after the daily use of the compound tincture of iodine brushed into the opening. A small white, healthy looking cicatrix remains, which is shown to you on the patient this evening. The result of the suppuration at the cheek for nearly ten years was a dark blue spot, which appeared whenever the skin became chilled, greatly to the annoyance of the patient, and to this time is occasionally seen on a cold day, although twelve years have elapsed, thus still showing the results of adhesive inflammation and destruction of the cellular tissue surrounding that portion of Steno's duct.

One year after having typhoid fever, and the subsequent pyæmic parotitis, simultaneous enlargement of each of the lobes of the thyroid gland and its isthmus began, and increased to such an extent as to impair respiration by pressure on the trachea and caused a dry, irritating cough. The patient again came under my care, and wished a physical examination, fearing pulmonary trouble; but percussion and auscultation, revealed healthy heart and lungs, and the pressure from the goitre proved to be the cause of the cough, suffocative and oppressive feelings, and the increase of the quantity of blood to the brain from pres-

sure on the jugulars. In all other respects the young lady was enjoying good health.

August, 1871.—My notes state that the goitre has grown rapidly, and extends from a line beneath one ear to a corresponding line beneath the opposite ear, and that the circumference of the otherwise thin neck at this point was fourteen inches.

The treatment of the goitre by iodine was commenced. I gave internally tinct. iodin. co., ℥x., largely diluted with water, three times a day, after meals, and also directed iodine to be rubbed on externally, as much as could be borne without producing painful irritation. This treatment she continued faithfully for five months, until she and her friends supposed that the goitre had disappeared; but, on an examination, I found still remaining an enlargement of the gland, and noticed that it became more prominent at each menstrual period.

After continuing for ten months the treatment by iodine internally, the stomach could not tolerate the medicine, and the following was directed:

℞. Iodin..... ʒ iv.
Glycerin..... ℥ ij.

Misce. Sig.—Paint over the entire surface of the goitre at bedtime, then cover with a piece of oiled silk, and allow this to remain on all night; wash off in the morning.

October 4, 1872.—I examined her at my office, found her in excellent health, and, although there was a marked increase in the fat underlying the skin of the neck, and the size of the muscles, yet its circumference was only thirteen and one-fourth inches, or three-fourths of an inch less than it was when she had the thin neck, but enlarged goitre.

The iodine treatment was continued in all about three years. The young lady subsequently finished her course at school, remained healthy, is now married, has a fine large boy, and continues in the enjoyment of good health. On examination this evening, if you lift up the lobule of the ear, the little cicatrix

may be seen, and on pressing the cheek sideways, the remnants of adhesion may be noticed, also an enlargement of the thyroid gland, but only to such a degree as to add to the beauty and rotundity of the neck.

She is never ill, and her life is a pleasure to herself and a comfort to her relatives and friends. Her hearing and sight are normal.

Case IV.—Parotid abscess following suppurative nephritis in a man, aged forty-one years, native of Germany. This man, a grocer, had acute catarrhal nephritis, followed by an abscess of probably one of his kidneys, a large quantity of pus passing in his urine for several weeks, and at the same time he had violent uræmic convulsions; acute mania and facial paralysis followed.

In this instance a metastatic abscess of the left parotid destroyed about one-third of the gland. I made a small incision into it, evacuating at first only about half an ounce of pus. The suppuration was of a very indolent character, and a recurrent fungoid growth caused a constant oozing of blood mixed with the salivary secretion, and a permanent fistula was feared. As much as possible of the remaining broken-down glandular tissue was scraped out, and to stimulate the parts, nitric acid was gently applied, after which the wound slowly but completely healed.

The facial paralysis still remains, but after two summers' residence in the country he convalesced sufficiently to again resume his business to a cautious degree.

Ten years ago the following observations were made by me in this city:

Suppuration of the right side of the neck, in the region of the parotid, the result of general pyæmic poisoning of an infant by diseased cow's milk. Abscess extending to clavicle. Death from exhaustion.

An infant, aged ten months, almost in articulo mortis, was brought to my office hurriedly before its death, for the purpose of getting the certificate, and thereby avoiding a coroner's inquest.

This case was an exceedingly sad one, and showed how important it is to inspect all cows kept to supply milk. Such an examination would prove to be a benefit not only to the poor, but also to the rich, as the following history illustrates.

The father and mother of this child were young and healthy, but after the child was six months old it was found impossible for the mother to nurse it. She therefore went to a woman who, for her support, had two cows, the milk from which she sold at double the usual price of the milk of city milkmen, asserting that she supplied infants where physicians had recommended them to be fed on one cow's milk.

The mother told me that her infant was perfectly healthy so long as she was able to nurse it, and never gave her any trouble; was happy, and had a clear, rosy complexion; but that after losing her milk she did her utmost to give it attention, and had supposed that she was doing the best she could while she obtained all its milk from the same cow. On examination, I found the infant very much emaciated, and discovered that it had a number of small abscesses over different parts of the body, the scalp having four or five, and each contained about one drachm of pus. The abscess on the right side extended from the lobe of the ear to the clavicle, pointed just beneath the lobule of the ear, and bulged out over the upper border of the clavicle, beneath the insertion of the sterno-mastoid muscle. As usual in these cases, the mucous membrane lining the mouth was covered with aphthæ, and the tongue was parched and intensely red, pulse feeble, and temperature below the normal. The abscess of the parotid contained fully eight ounces of pus, and as the exhaustion was so extreme, and collapse already existed, I told the mother to take the infant home, apply artificial heat, keep it warm and as quiet as possible, give diluted stimulants, and if the child rallied I would operate in a few hours. The child, however, died before the end of twelve hours from pyæmia and exhaustion, having never gained sufficient strength to justify the operation.

In this instance the secondary abscess of the parotid with the pyæmia were the direct cause of death. I have seen a

number of instances where the pyæmic condition of infants, due to drinking milk from diseased cows, produced as many as twenty abscesses on the scalp, as well as a number on different parts of the body, yet recovery generally followed, as I always open these abscesses early, and as an internal remedy give five grains of finely powdered sulphur, ter in die, mixed with the food.

Two weeks before seeing this last-mentioned case I visited the only son of a wealthy family, and found that the child continued to have eczema appear about its mouth and nose, and other general symptoms of blood-poisoning, and of gastro-intestinal irritation. I had given the child medicine, and had used local applications, but the relief would be but temporary, for as soon as the treatment was discontinued the symptoms would reappear. I inquired again particularly in regard to the diet of this child, and found that it was fed on one cow's milk, as recommended to the mother by the child's nurse. I went to see the cow, as every physician ought to do under like circumstances, and found, in a sunken lot, a few boards nailed together and covered with old pieces of tin roofing, while inside one of the most loathsome sights presented itself. In a space of about twelve by fifteen feet were two cows in the filthiest condition imaginable, tramping in dung, urine, and mud, and tied side by side. The animals themselves were in a terribly filthy condition to take milk from, and they were milked three times a day. They were fed on swill collected by the woman's children, and the refuse from beer breweries, was eaten while in a heated, fermenting condition. The horns of these animals were rotten at the ends, the hoofs were elongated to twice their normal length, with turned-up toes, resulting from the long-continued maceration in the filth in which they were continually standing. Their skins were dry, covered with thick, crusty scales; they had lost at least half their hair, and the long hair at the ends of the tails had fallen off, giving them a peculiar stumpy appearance. These animals looked as though they were in the last stages of consumption, and were those from which the ignorant woman had

taken milk to supply, at double prices, several of the infants in the neighborhood.

These milk-venders went to the cattle market, purchased one or two cows, took them to miserable places, kept them until they got in the above-described condition, and when about to die from disease and exhaustion, sold them to the butcher for meat to be sold to the poor.

While speaking of this cause of inflammation of the parotid, and subsequent death from pyæmia, I gladly allude to the valuable report on the influence of milk in spreading zymotic disease in England, by Ernest Hart, Esq., where he submitted, in a tabular form, particulars of seventy-one recent epidemics due to infected milk. He enumerated three diseases as capable of being spread by milk, viz., typhoid fever, scarlet fever, and diphtheria, to which I can safely add, from a large number of observations in New York City, pyæmia and septicæmia, with innumerable forms of cutaneous diseases, and abscesses of the glandular organs.

I may add that the last mentioned child permanently recovered from all skin disease; and after the change to a healthful diet, its eczema all disappeared, and no further medication was necessary.

From personal observation of my own recorded cases, and the analysis of those related by others, I am led to believe that secondary suppuration of the parotid gland is best explained by the theory that the septic material has entered the circulation at points where the veins and lymphatics directly admit the decomposing poisoned substance, filled with the micrococcus septicus or the microsporon septicum of Klebs, which is conveyed to, and becomes colonized in, the capillaries of the parenchymatous tissue of the gland, where it exerts its destructive energy.

This condition, I think, explains the occurrence of suppuration of the parotid in osteo-myelitis and in ulceration of the intestinal mucous membrane or its glands. These metastatic abscesses I have never seen during even very profuse suppuration, where the protective pyogenic membrane remained unbroken,

or where the healthy reparative granulations continued. I have never seen a parotid abscess during the abundant suppuration of empyema, although I removed at one time half a gallon of most offensive putrid pus from the chest of a wounded officer. I have had a recovery after perforating gun-shot wounds through the small and large intestines, injuring the liver, causing an artificial anus behind and before, with continued oozing of bile from the anterior abdominal opening. Where gangrene of the omentum necessitated the removal of a large quantity, it left a gaping wound as large as the palm of the hand on the anterior abdominal walls, and at the back, where profuse putrid suppuration had taken place, yet no metastatic abscesses followed, as free drainage was continuously accomplished.

I have also reported, in the surgical history of the war, several instances of non-ulcerative necrosis of the pelvic bones, and abscesses of the pelvic cavity, and more recently have observed instances of profuse suppuration of the pelvic cellular tissue, lasting in one instance for over ten years; yet never in any of these mentioned cases did abscess of the parotid occur. If we do not find metastatic abscesses as a result in these cases, and see them follow diseases where ulcerations take place, have we not narrowed our field for search to the indolent ulceration, where a specific poisoning takes place? I distinctly remember a young, rosy-cheeked lieutenant with a penetrating gunshot wound of the ankle-joint, who at first refused amputation, and died of pyæmia. At the necropsy I found ulcerative osteo-myelitis of the tibia. The knee-joints, hip-joints, shoulder-joints, and wrist-joints contained thick creamy pus, and in the liver I found a large abscess. All other glands were free from pus.

I may also mention a case to the point, where, after complete primary healing of the stump after amputation of the arm, ulceration of the cancellated structure of the bone followed and progressed, producing metastatic abscesses, and ended in death.

This case is recorded by Dr. J. A. Lidell, in the "United States

Sanitary Commission Memoirs—Surgical," vol. i., in his excellent article on the relations of pyæmia to osteo-myelitis. He notes the " close relationship which exists between osteo-myelitis, or rather the suppurative form of that disease, and pyæmia. When inflammation of the medullary tissue of bone proves fatal, it does so in a large majority of instances by the induction of purulent infection. Numerous examples of pyæmia following suppurative osteo-myelitis of a traumatic origin are related." Pyæmia is the form of systemic poisoning which osteo-myelitis generally produces. The following case affords an illustration of the correctness of this statement. The report of it was contributed by Dr. Charles A. Leale, late Assistant Surgeon U. S. Volunteers.

Case XIII.—Amputation of arm for gunshot injury; acute pyæmia suddenly supervened after stump was healed; death; autopsy; suppurative osteo-myelitis of stump-bone; abscess of liver; other viscera sound.—William Collins, Captain Company G., Fifth New Hampshire Volunteers, aged thirty-five; wounded at Farmville, April 7, 1865, by a conical ball, which passed through right arm an inch above the elbow-joint, fracturing humerus. The limb was amputated in middle third, by flap method, at the Second Army Corps Depot Field Hospital, City Point, Va. Operator's name not known.

When admitted to Armory Square Hospital, April 16, 1865, the stump had entirely healed, and there was no sinus leading to the bone.

The general condition of the patient was very good; appetite good; walked about the ward since he was admitted.

April 23d.—Pyæmic symptoms noticed; has had several chills during the day; continued nausea, with an occasional spell of vomiting; at night was bathed in perspiration; gave quiniæ sulph. iron, stimulants, and beef-tea; during the day had him removed out of the ward and placed under the shade-trees in the garden. Careful examination did not reveal the abnormal condition of the end of the bone of the stump.

April 28th.—Diarrhœa commenced, which resisted the usual

treatment, and injections (astringent). He has become very anæmic; continued treatment. A few hours before death, a small opening was formed in end of stump, through which an oozing of black venous blood occurred.

April 29th.—He died.

The *autopsy* showed, after the end of the humerus had been sawn off and broken open, that its medullary canal contained circumscribed collections of pus, which had obviously been produced by the septic osteo-myelitis of the stump-bone.

The liver contained a large abscess filled with thick pus. The spleen, kidneys, and lungs were natural. No pus was found in the joints. There was a yellow color of the skin of the entire body. The necropsy was made by Dr. C. A. Leale, who sent a full report to the Surgeon-General's office."

The hospital gangrene, so prevalent at some localities during our late war, did not appear to be a provoking cause to produce suppuration of the parotid gland. In 1864 I was placed in charge of the ward where all the patients with hospital gangrene were sent, at the Elmira United States Army General Hospital, and yet neither then nor subsequently did I ever meet with a single instance of parotid abscess in a patient having gangrene.

Liebermeister, in his excellent article on the complications and sequelæ of typhoid fever, states that the occurrence of "suppurative parotitis used to be considered as a result of metastasis. According to Hoffman, it consists merely in an exaggeration of the changes that usually take place in this gland during typhoid fever, and bears the same relations to those changes that ulceration and perforation of the intestines do to infiltration of the intestinal follicles. Whereas the swelling of other glands, which is found in all cases of typhoid fever, even though it be accompanied with cell degeneration, commonly recedes without creating much disturbance, parotitis leads to more serious changes. Numerous points of suppuration and breaking down of tissue appear, involving both the glandular and the intermediate cellular structure; these isolated collec-

tions soon become confluent, and, in the worst cases, large portions of the gland are thus transformed into cavities, containing pus and torn fragments of altered tissue. The fact that this suppurative process is usually confined to the parotid, that it rarely invades the other salivary glands, and hardly ever the pancreas (though all of these are equally subject to the ordinary parenchymatous changes characteristic of the fever), is attributed by Hoffman to the close and tough texture of the fascia enclosing the parotid, whereby swelling of the confined gland, producing extreme pressure, more readily leads to severer inflammation. This destructive inflammation may also extend to neighboring parts; fistulous tracts may extend down the neck, or we may have thrombosis of the veins, periostitis, suppuration of the masseter or pterygoidei muscles, or, finally, diffuse purulent infection and septicæmia. The pus formed in these abscesses may find its way to the surface, or may open into the external auditory canal. The trifacial nerve, or some one of its branches, is sometimes involved in the destructive process, or it may be severed by incisions made to evacuate pus, in either case leaving paralysis of the muscles to which it is distributed.

"According to Hoffman, sixteen cases of suppurative parotitis were found at Basle among about one thousand six hundred typhoid fever patients, seven of the sixteen proving fatal. Parotitis without suppuration occurred three times. In fifteen cases the attack was confined to one side, nine times to the right and six to the left; in four it was double."

Liebermeister, in continuing, states that since the introduction of a systematic antipyretic treatment, the frequency of suppurative parotitis has greatly diminished. Whereas previously one case occurred to every hundred typhoid fever patients, since the year 1872 (inclusive) but two cases have occurred among one thousand one hundred such patients. Both these cases terminated fatally. This experience is well calculated to support our view that the changes commonly occurring in the parotid glands during typhoid fever, and whose excessive development produces suppurative parotitis, belong to the

class of parenchymatous degenerations that are brought about by an elevated temperature of the body.[1]

Niemeyer states that "symptomatic parotitis begins, according to the careful observations of Virchow, with decided hyperæmia, which causes the gland and interstitial substance to appear infiltrated and swelled. Changes in the gland-ducts soon begin; a tough filamentous, whitish or yellowish substance, which soon becomes purulent, collects in them. Even at the second or third day the microscope shows that it contains pus corpuscles. If the disease proceeds, the lobules of the gland soften and break down; this process begins within, so that at one time the lobules represent cavities filled with pus. Finally, the tunica propria is also destroyed, and the tissue begins to suppurate; this suppuration may extend rapidly and become a diffuse phlegmonous inflammation. In this case a large parotid abscess forms; more frequently the gland-tissue only is destroyed, and as the interstitial tissue remains intact, numerous small abscesses are formed. Occasionally, also, there are extensive destruction and gangrene of the gland-tissue and interstitial substance; the inflammation and suppuration may spread from its original seat in various directions, and cause dangerous results. It most frequently attacks the neighboring connective tissue, and the masticatory muscles lying even with the bones themselves. Where the disease is very severe, it occasionally passes from the bones to the membranes of the brain, and the brain itself, or to the internal and middle ear. This propagation of inflammation and suppuration to the cerebral membranes and the internal ear, may take place along the blood-vessels and nerve-sheaths as well as through the bones. Finally, in some cases parotitis induces phlebitis and thrombus of the neighboring veins, especially of the anterior and posterior facial and external jugular veins; the disintegration of these thrombi may cause embolism and septicæmia."

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[1] Liebermeister on Typhoid Fever: Ziemssen's Cyclopædia of the Practice of Medicine, vol. i., translated by Dr. W. Schauffler for Dr. Albert H. Buck's American Edition. Wm. Wood & Co. 1874.

Dr. Charles West states that the occurrence of suppuration in the neighborhood of this gland is a rare termination of the inflammation of the cynanche parotidæa, but he believes it is oftener met with in infants and young children than in those who are approaching the period of puberty. On the other hand, metastasis of the disease from the parotid to the mamma, the testicle, or the brain, all of which instances are recorded by different writers, appears to be rare in proportion to the tender age of the patient. (Fifth English Edition and Fourth American Edition.)

Dr. J. Lewis Smith, in speaking of idiopathic parotitis, states that the inflammation is specific, due to a materies morbi in the blood, and hence its decline after a fixed period. ("Diseases of Children," Second Edition, p. 261.)

Vogel considers as very uncertain the etiological connection of metastatic parotitis occurring in the course of typhus or scarlet fever, of variola, and of measles. Among other causes, especially for typhus fever, a mechanical occlusion of the ductus Stenonianus, as a result of the dryness of the mouth, must at any rate hold good.

Dr. R. C. M. Page, in the *Medical Record*, September 10, 1881, gives the histories of six fatal cases of metastatic parotitis, and regards it, especially when secondary to peritonitis, as certainly a very serious, if not a fatal, complication.

Professor Austin Flint states that "a rare but somewhat characteristic complication is inflammation of one or both of the parotid glands. It leads," he says, "to notable enlargement, and the appearance is like that of ordinary parotiditis or mumps. But, unlike the affection just named, in the great majority of cases suppuration takes place, and not infrequently more or less sloughing of the areolar tissue. This complication adds to the danger and retards convalescence. It may occur at any period of the febrile career, or during convalescence. It is not to be regarded as a critical event. The discharge of pus is sometimes into the *meatus auditorius*. This complication occurs in typhus as well as in typhoid fever. It occurred in five out of thirty cases of typhus and typhoid fever

which he recorded in the winter of 1849–50. Prior to that year he had never met with an example, and since that year," he says, "he has met with a very few examples only, in a much larger field of observation."

Professor A. C. Post, in the discussion of pyæmia before this Academy, gave the case of the late Dr. J. Kearny Rogers, where the condition which gave rise to the parotitis was due to ulcerations of the intestinal canal, and was followed by secondary abscesses in the liver. ("Bul. New York Academy of Medicine," Vol. I., ii., January, 1865.)

In pyæmia, pus may be found in almost any part of the body. Professor Fordyce Barker, in his work on "The Puerperal Diseases" (p. 421), gives an account of an autopsy made by his colleague, Professor James R. Wood, on a patient of Dr. Livingston, who died of pyæmia after miscarriage, when they estimated the amount of pus in the pericardium to be not less than twelve ounces.

Chronic pyæmic poisoning is a very common cause of many of the obscure diseases met with in general practice, and frequently never diagnosticated by the physician, who treats his patient as suffering from latent intermittent fever, gastric fever, typho-malarial fever, and even typhoid fever. These cases are frequently permitted to run an indefinite course, until metastatic abscesses in vital organs occur.

Surgeon J. J. Woodward, U. S. A., in his remarks on the "Morbid Anatomy of Acute Diarrhœa" ("Medical and Surgical History of the War of Rebellion," Medical volume, Part II., p. 314), mentions one case only of suppuration of parotid glands, observed by Assistant-Surgeon Harrison Allen, U. S. A. In this instance both sides were affected. This case had been regarded as one of typhoid fever, but after death only the characteristic evidences of catarrhal inflammation of the mucous membrane of the ileum and large intestine were observed, and "Peyer's patches were normal."

Doctor Woodward also states in his article on "Acute Dysentery" (*op. cit.*, p. 414), that "Finger and Trousseau have observed inflammation of the parotids in the advanced stages

of severe cases of acute dysentery, a fact which should not be overlooked in attempting to diagnosticate between dysentery and fever, when the case is first seen in its latter stages, without satisfactory previous history, as so often happens in hospital practice during times of war. Cases of this complication have been reported as follows:

"*A Case of Dysentery Complicated with Parotitis.* (*Virginia Med. Jour.*, vol. xii., p. 191, 1859.) G. G. Minor.—The patient was a boy, twelve years old, who died on the sixteenth day, of sporadic dysentery. The left parotid first enlarged two days before death; next day, the right. The swelling was so great as to render deglutition impossible. The boy had previously had mumps.

"In the minutes of the proceedings of the Buffalo Medical Association (*Buffalo Med. Jour.*, vol. x., p. 405, 1854–5) I find several cases, viz.: One reported by Dr. Strong: A lady, thirty years of age, had an apparently slight attack of dysentery, and appeared to convalesce on the fifth day, when parotitis made its appearance on one side (it is not stated which). Three or four days later the tumor discharged a thin, ichorous, purulent matter at the ear. Soon after the dysentery recommenced, was uncontrollable, and the patient died. Before death the opposite parotid had begun to enlarge. Dr. White said he had witnessed three similar cases: one in 1853, in which the dysentery was severe, and just as it subsided the left parotid enlarged. No suppuration took place, nor did the dysentery return, but the patient died. He had seen, in 1849, a young girl, and, in 1852, a child of seven years die under similar circumstances. In all these cases the tumor is spoken of as having 'a peculiar stony hardness.' All had taken acetate of lead, and Dr. White suggested that this might be the cause, as 'lead, more than any other drug, affects the secretion of saliva, checking it and making the mouth dry.' Dr. Wyckoff had seen a similar enlargement in the submaxillary gland, after a mild case of dysentery, in a boy who had not taken lead. He recovered."

At the last meeting of the Obstetrical Section of this Acad-

emy, I gave the history of a case of suppurative pelvic cellulitis where the discharge of pus was through the rectum, above the internal sphincter ani muscle, and where I made through and through drainage. This patient presented many of the symptoms of pyæmia, and although this offensive collection had existed for years, there was no formation of metastatic abscesses. Also, several years ago, in the presence of Professor Fordyce Barker, Professor William T. Lusk, and several other physicians, I made a large opening at the utero-vaginal junction, entered into an old pelvic abscess, and irrigated by a large double tube, yet in this case no metastasis of pus occurred.

After the occurrence of osteo-myelitis in a part easily reached, can we, by amputating the diseased bone or gouging out the ulcerating cancellated tissue, cure our patient after metastatic abscess have formed? is a very important question. And, in answer, I may state that I have endeavored to remove this diseased bone by amputation and otherwise, and, although, going far above the part showing evidence of ostitis, yet never have I seen any good result follow.

I have not been able to find any recorded instance of an operation on a parotid abscess through the mouth by the orifice of Steno's duct, although Professor John C. Dalton had obtained the parotid saliva of the human subject in a state of purity by introducing directly into the orifice of Steno's duct a silver canula $\frac{1}{20}$ to $\frac{1}{25}$ of an inch in diameter. The other extremity of the canula projecting from the mouth, between the lips, and the saliva collected as it runs from the open orifice.[1]

The discharge of pus by an opening through the floor of the external auditory canal has been mentioned by a number of writers, and is probably due to destructive pressure. Anatomy teaches that on the floor of the external auditory canal, midway between the membrana tympani, are openings of the ceruminous glands, the most internal of which form a curved line,

[1] Treatise on Human Physiology, by Professor John C. Dalton, M.D. Fifth Edition.

which corresponds with the beginning of the osseous portion of the external meatus.

According to Professor Austin Flint, Jr., "the walls of the external meatus are partly cartilaginous and fibrous, and partly bony. The cartilaginous and fibrous portion occupies a little less than half of the entire length, and consists of a continuation of the cartilage of the pinna with fibrous tissue. About the lower two-thirds of this portion of the canal is cartilaginous, the upper third being fibrous. The rest of the tube is osseous, and is a little longer and narrower than the cartilaginous portion. Around the inner extremity of the canal, with the exception of its superior portion, is a narrow groove, which receives the greater portion of the margin of the membrana tympani."[1]

Professor Erichsen, in his work on "The Science and Art of Surgery," states that "excision of the parotid itself is occasionally spoken of, but is very rarely, if ever, done. I believe that in most, if not all, the cases in which it is stated that complete removal of the gland has been accomplished, tumors overlying and compressing it, have been mistaken for it. It is evident that a diseased parotid could not be removed without the division of the external carotid and the portio dura."

Extirpation of the Parotid Gland.—Dr. Valentine Mott, in the *American Journal of Medical Sciences*, vol. x., pp. 17–20, 1832, gives an account of a formidable operation, where he removed the parotid in consequence of melanotic disease of the gland. The patient was twenty-one years old, and, fearing scirrhus, Dr. Mott decided to operate. The carotid was first ligated, and before the melanotic mass was removed, the facial nerve, or portio dura, was divided and the side of the face paralyzed. "The operation lasted one hour, and the patient lost, perhaps, a pint of blood."

The wound in seventeen days healed, excepting at the point opposite the ear, which had every appearance of a reproduction of the disease. Thirty days after the operation tumors

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[1] See Fig. 10, Physiology of Man, by Professor Austin Flint, Jr., vol. v., p. 155, from Sappey, Traité d'Anatomie, tome iii., p. 803. Paris, 1871.

appeared on the scalp, and fungus of the wound increased. On the fifty-fourth day he died.

Dr. A. C. Post has successfully performed the operation for the removal of the parotid.

Dr. Joel Foster kindly sent the following letter, giving the account of the first successful operation for the removal of the parotid supposed to be on record, at which he assisted over fifty-five years ago.

265 Fourth Avenue.

Dear Dr. Leale:

In compliance with your request, I send you a condensed report of the successful removal of the parotid gland, the first, I believe, on record.

On February 27, 1826, the late Dr. George McClellan, of Philadelphia, in the presence of the late Dr. Nathan R. Smith, then professor of anatomy in the Jefferson College, and several students, including myself, removed the entire left parotid gland from my room-mate, the late Dr. John Graham, then a fellow-student of Jefferson Medical College, subsequently a Fellow of the New York Academy of Medicine.

An unsuccessful attempt had been made by an eminent surgeon of Dublin, several years previous. The gland was very much enlarged and indurated. There was a large cicatrix over it, the result of the former operation. Two curvilinear incisions were made, from a little above the zygoma downward, to meet about two and one-half inches below the angle of the jaw, including the old cicatrix. The integument was reflected from the surface of the tumor, a dissection was made down to the zygoma and masseter muscle before, and to the cartilaginous tube of the ear and mastoid process behind. The posterior belly of the digastric muscle was divided. The trunk of the external carotid artery was insulated just as it entered the tumor, together with the descending veins which accompany it. They were torn from the body of the tumor with the thumb and fingers, instead of cutting them. An instantaneous gush of blood ensued, which soon ceased, without using a ligature, in consequence of the contraction of the lacerated extremity of the artery. After painful and repeated efforts at wrenching, aided by the occasional use of the knife to divide the strong bands of condensed cellular substance, and the fibres of the styloid muscle, which adhered to the tumor, the whole mass was then everted above the ramus of the jaw and mastoid process.

The trunk of the portio-dura nerve, very much enlarged, was seen emerging from under the mastoid process, and mounting over the posterior margin of the tumor to enter its substance. The great tension of this exquisitely sensitive nerve caused convulsions—the pain was instantly relieved by dividing the nerve; and, after separating the tumor from the zygoma, the operation

was completed. The main trunk of the temporal artery having been divided, a ligature was applied, the only one used during the operation.

The cavity of the wound was fully four and one-half inches deep, being larger at the bottom than at the surface. The walls of the pharynx were largely exposed, and no portion of the gland, sound or morbid, was left. The recovery was slow; the wound had to be reopened to remove the coagula. Granulations slowly filled the cavity, leaving a deep hollow behind the jaw, and permanent paralysis of the muscles of that side of the face, but the countenance, while in repose, was not much deformed. The patient lived many years, a successful physician in this city, and at last died with typhoid fever while under my care. The tumor is now in the Museum of the Jefferson Medical College. The trunk of the portio-dura, about an inch in length, enters the posterior margin of the preparation.

The above is an abstract published in the *American Medical Review*, August, 1826, with my recollections of the case, which are still very vivid, as I remained constantly at his bed-side for two weeks after the operation, and subsequently through the remainder of his life I was his bosom friend.

Yours truly,

JOEL FOSTER, M.D.

CONCLUSION.

Pyæmic parotitis is preceded by the ulcerations following inflammations, whereby the terminal veins or lymphatics are opened, and the poison is directly admitted into the general systemic circulation. It may be of idiopathic or traumatic origin. It always indicates a depraved constitutional state when the recuperative powers are nearly exhausted. Pyæmic parotitis, following the enteric fevers, is symptomatic of a low grade of ulceration of the intestinal glands or mucous membrane, and must be looked upon as a grave, complicating condition.

Pyæmic parotitis in osteo-myelitis is almost, if not always, fatal. The same can be said when it occurs in the course of ulcerative pelvic cellulitis

Pyæmic parotitis causes death through general toxæmia, pressure on the brain, suffocation from internal pressure on the throat, or exhaustion.

Spontaneous openings may occur: 1st, beneath the angle of the jaw; 2d, under the lobule of the ear; 3d, on the cheek, over the duct of Steno; 4th, through the floor of the external

auditory canal; 5th, into the middle ear and perforate the membrana tympani; 6th, to the base of the brain; 7th, into the throat.

Treatment of Pyæmic Parotitis.—Eliminate as much of the poison as possible from the blood by producing free evacuations from the bowels, active diuresis and diaphoresis. Prevent exhaustion by the most nutritious and easily assimilated diet, and alcoholic stimulants largely diluted, and as medicines to restore the vigor of the constitution: 1st, comes quinine in moderate doses; 2d, iron; and 3d, iodine. Watch closely the tumefaction of the parotid gland, and so soon as fluctuation shows disintegration of the glandular tissue, choose your point for opening, which will generally be under the angle of the jaw, or beneath the lobule of the ear, where fluctuation is most decided. Make a small incision with a narrow-bladed tenotomy knife directly into the pus cavity, and slightly enlarge by tearing the coverings upward and downward to about a quarter of an inch in extent, after which it can easily be enlarged by means of a sponge-tent. This careful procedure is absolutely necessary if we wish to prevent profuse hemorrhage, or injury to the facial nerve.

If symptoms of suffocation occur, careful examination of the throat is to be made, where fluctuation may be expected to be felt, by passing the finger back and below the tonsils. An abscess in this location I have found to be easily opened with the small, rounded blade of a gum-lancet, having a long handle, the small round blade being pressed directly into the abscess, as counter-pressure is made with the other hand over the parotid. Through and through drainage can now be easily accomplished by pushing the probe through the integumentary opening and parotid gland and to the opening into the throat, when it can easily be withdrawn with the silk suture following. The two ends of the silk can be fastened with a piece of adhesive plaster on the cheek, and easily be kept in the most convenient place. Thorough irrigation can now be accomplished.

THE EARLY DIAGNOSIS OF CHRONIC BRIGHT'S DISEASE.[1]

By T. A. McBRIDE, M.D.,

NEW YORK.

Mr. President and Fellows of the Academy:

I invite your attention this evening to the means at our command for the early diagnosis of chronic Bright's disease of primary origin.[2]

I shall limit myself to the signs and symptoms which are generally accepted as belonging to the contracted or granular kidney, or, as it is also known, the atrophic kidney of chronic diffuse nephritis. It must be remembered, however, that it is not always possible to differentiate the contracted kidney from some cases of certain other varieties of chronic diffuse nephritis, particularly the large white kidney, and very occasionally the same is true of some rare cases of chronic parenchymatous nephritis.

I shall first present in detail the symptoms and signs of chronic Bright's disease, and then present some reasons for thinking that a functional, forming or initial stage of this disease may exist, characterized by certain signs.

A. Symptoms and Signs of Chronic Bright's Disease.

1.—*Symptoms :*

a. Headache, usually persistent, { Frontal, Vertical, Occipital.

[1] Read November 3, 1881.

[2] The diagnosis of chronic Bright's disease secondary to other diseases is not considered in this essay, although most of the symptoms and signs are common to both.

b. Deafness.

c. Amblyopia.

d. Dyspnœa,
- Spasmodic (pseudo-asthmatic).
- Cheyne-Stokes respiration.
- On exertion (heart disease, etc.).
- From emphysema and chronic bronchitis.
- From hydrothorax, œdema of lung, etc.

e. Dyspepsia, especially with great flatulency.

f. Morning nausea and vomiting.

g. Abdominal colic, usually coming on at night with pains about the umbilicus and with or without diarrhœa.

h. Diarrhœa, dysentery, and constipation.

i. Pains in the lower extremities, with or without cramps; also coldness and numbness of extremities.

j. Trifacial neuralgia.

k. Insomnia.

l. Vertigo.

m. Frequency in urination, especially at night.

n. Palpitations, pain over precordia, attacks of angina pectoris more or less severe.

o. Pain in lumbar region.

p. General muscular pains and lassitude.

q. Epistaxis.

r. Anxiety, restlessness, mental confusion and pantophobia.

2.—*Signs:*

a. Great pallor of skin and mucous membranes; dryness of hair and baldness of scalp. Early appearance of gray hair.

b. Tortuous temporal arteries, often with very prominent pulsation.

c. The so-called staring eye of Bright's disease with a pearly conjunctiva.

d. Subconjunctival œdema, which is best appreciated by turning down the lower eyelid and with a piece of blotting-paper absorb the moisture which is upon the

conjunctiva, and then the subconjunctival œdema will be very apparent.

e. Peculiar odor of breath.

f. Emaciation, and in some rare cases polysarkia.

g. Slight œdema of the legs is often mentioned as being present, but this will be found in many persons, especially in those who are stout, and pitting of the skin is often apparent over the tibiæ.

h. URINE.—The odor of the urine. Bravais states that in chronic Bright's disease the urine is free from all odor peculiar to urine, and that this inodorous condition is not affected by the ingestion of turpentine, asparagus, and other substances capable of producing a strong odor in the urine. I question whether this holds true in all cases.

Frothing of the urine when passed is often observed when the urine contains albumen. This is, however, observed when the urine is of high specific gravity, when it contains biliary matter, etc.

QUANTITY.—The amount of urine passed in the twenty-four hours is a factor of some importance in the diagnosis of the presence of chronic inflammation of the kidney, and also in the differential diagnosis of the varieties of chronic nephritis. It may be stated as a rule that the longer the disease has continued the greater is the quantity of urine excreted. It would be of assistance in diagnosis to find that a much smaller amount than normal was being excreted, and likewise if too much was being passed.

In chronic parenchymatous nephritis the amount passed is usually below the normal. It may be as low as ten to twenty ounces in the twenty-four hours, and often for a considerable portion of the disease as much may be passed as by one in full health. Again, a small amount may be passed for some days and then a normal amount may be passed for a while, and this may be succeeded by a smaller amount. The longer the disease lasts and the more the pathological changes tend to pass into those characteristic of chronic diffuse nephritis,

especially of the small variety, the greater becomes the quantity of urine and the less frequent are the reversions to a small quantity.[1]

In chronic diffuse nephritis, especially in the large white kidney, the quantity may be small at first and then return to the normal amount. It may at the end be suppressed. The longer, however, that the patient lives the greater becomes the amount of urine secreted.[2]

In the atrophic form of chronic diffuse nephritis Bartels[3] well describes the variations in the amount of urine. He says: ". . . urine is occasionally temporarily excreted which is in no way to be distinguished from that secreted by a healthy kidney. . . . The genuine process of contraction of the kidney is associated with polyuria, but neither does this symptom at once become prominent at the very beginning of the malady, nor does it persist absolutely to the very end of the case. In fact, in the progress of the disease it may entirely subside for a longer or shorter interval of time, whenever from any debilitating influence the heart's propulsive power is diminished or affected for a longer or shorter period. Indeed, under such conditions we find the urinary secretions of these patients falls back to an abnormally small amount."

Grainger Stewart thinks that in the early stages of the small kidney the urine is not increased in quantity.

Dickinson[4] believes that in the small variety an increased quantity of the urine appears early. Saundby[5] is also of that opinion. One should always bear in mind that any cause leading to a diminution of arterial tension, any obstruction to the circulation through the lung, or through the right heart, any severe diarrhœa, dysentery or pyrexia, would tend likewise to reduce the amount of urine passed.

It is therefore very important to save the entire quantity of urine passed by the patient in twenty-four hours, including

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[1] Delafield-Roberts' report of lectures, 1880. [2] Delafield, loc. cit.

[3] Ziemssen's Cyclopædia, American edition, vol. xvi., pp. 431–433.

[4] Diseases of Kidney. Part II. Albuminuria.

[5] Birmingham Medical Review, April, 1881.

even the urine passed at the time of defecation. The necessity of saving the whole amount is rarely insisted upon by the physician, and it is often difficult or impossible to persuade the patient that this is of the greatest importance.

Specific Gravity.—In chronic parenchymatous nephritis the specific gravity does not vary much from the normal. It may range from 1020 to 1030 or from 1010 to 1020. In chronic diffuse nephritis the specific gravity is, as a rule, below the normal. It is usually from 1010 to 1015. In the large white kidney there occur often exceptions to this statement, and the urine may be of the normal specific gravity and even higher. In the atrophic or contracted kidney the specific gravity accords with the rule, and is usually much below the normal, and yet in some cases the specific gravity may be normal for weeks and months. Bartels[1] states that, as a rule, in advanced cases of kidney disease, especially in the atrophic or cirrhotic kidney, "the specific gravity remains below the normal. Thus for months, and even years, examining the water daily, I have found the specific gravity of the urine of such patients excessively low and fluctuating only within very narrow limits, varying, for example, between 1004 and 1012." He refers also to the presence of a low specific gravity of the urine of the cirrhotic kidney at what he thinks is the beginning of the disease, and that then the increase in quantity of the urine is not great. He states, however, that even in advanced cases of the small kidney, circumstances may arise wherein the urine has no longer a low specific gravity, and these conditions are very much like those which have been mentioned as diminishing the quantity of urine passed. A high specific gravity may supervene after an attack of uræmic coma, or of convulsions, or of delirium with high temperature, or of some one of the inflammations so frequently intercurrent, especially when attended with considerable pyrexia. Again, failure of the heart's vigor, diminution of quantity of drink, excessive sweating or diarrhœa will produce it. Bartels[2] also

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[1] Ziemssen, vol. xv., p. 434, American edition. [2] Loc. cit., p. 435.

states that "should the process of contraction, however, have advanced up to a certain extreme limit, it appears impossible for the urine, under any circumstances, to attain such a concentration and specific gravity as it ought normally to present. Numberless observations, made by me upon patients who remained under my care up to the end of their lives, convinced me that in extreme cases of this nature, even although the amount of urine passed per diem fell to a few hundred cubic centimetres only, the specific gravity could not rise any longer over 1009 or 1011." One may say then, as was said of the quantity of urine, that the longer the disease has existed, the lower will be the specific gravity, and that in the estimation of this in any stage of the disease it is necessary to consider whether any of the conditions have been present which sometimes are known to influence the degree of the specific gravity.

Color of the Urine.—This is usually pale, unless some of the conditions are present which favor an increase in specific gravity or diminution in the quantity passed. If, also, there is present any functional or organic disease of the liver, the color of the urine may be more or less heightened.

Albumen.—The tests for albumen, endorsed by general use, are several.

The most common test is that of heat and nitric acid, the method of which is so familiar that I will not dwell upon it, but I must call attention to the suggestions of Dr. Munn[1] for the appreciation of small quantities of albumen which usually are not detected. He advises that the test-tube be placed before a black background, and that, by means of a curtain, the eyes be protected from the light which illumines the test-tube from above. The eye of the observer, thus protected from the light, looks against the test-tube in front of the black background while the light obtained from above falls on the test-tube and the background of black.

The test, however, which seems to me to be free from all possible errors is the following:[2] Take a conical test-glass and

[1] Medical Record, March 29, 1879, p. 297.

[2] Heller's test, Nebauer and Vogel, Trans., Am. Ed., 1879, p. 96.

fill it two-thirds full of nitric acid, C.P. Then if the urine has a specific gravity above 1010, and especially if the specimen has a high color, dilute it from two to ten times its bulk with distilled water, and add this diluted urine very carefully to the acid in the test-glass, so that it will flow out upon its surface and not mingle with it. If you then comply with Dr. Munn's suggestion you will observe a stratum of albumen at the point where the urine comes in contact with the acid, and this will be more evident in the test-glass than in the test-tube, by reason of a greater thickness or depth of the stratum of albumen than you can obtain in a test-tube. By the great dilution of the urine the precipitation of urea as nitrate of urea, and the precipitation of the urates is prevented, and these precipitates are not so infrequently mistaken for albumen. Again, in this way we diminish the density of the precipitate of the coloring matter, which is sometimes so great as to obscure the presence of albumen. I have often observed that the presence of albumen is overlooked in the turbid urine which is obtained from women, and likewise from cases in which there is present some inflammation in the urinary tract, also in urine which has stood for some days. The cloudiness of the urine obscures the presence of albumen when the usual tests have been applied. It is true that often some albumen is present by reason of the urine containing more or less pus, but there may be present a much greater amount of albumen than can be explained by the quantity of purulent matter present. To detect albumen under these circumstances one should make use of the Hoffman and Ultzman method as described by Dr. Tyson.[1] "Boil a portion of the urine with an equal bulk of officinal liquor potassæ and filter. If still not quite clear add a few drops of the magnesian fluid." (The magnesian fluid is made by dissolving magnesium sulphate and pure ammonium chloride, each one part in eight parts of distilled water, and adding one part of liquor ammoniæ.—Foot-note in Tyson's book, p. 54.) "Warm again and filter. To the clear fluid thus obtained

[1] A Treatise on Bright's Disease and Diabetes, p. 54. Philadelphia, 1881.

Heller's acid test may be applied, or, after careful acidulation, the heat test. In this manner very small quantities of albumen may be recognized in urine which would not otherwise permit its detection." In some few cases it should be remembered that the cloud of albumen does not appear until a few minutes after the tests have been made.[1]

So much for the tests for albumen.

The presence of albumen in the urine is a factor of a certain degree of importance in the diagnosis of chronic Bright's disease, but is not by itself of the great value once supposed. In connection with other signs it is deserving of considerable weight as assisting in arriving at the diagnosis. To ascertain whether albumen is present or not in a patient supposed to have chronic Bright's disease, it is very necessary to be careful and exhaustive in one's efforts. A specimen of urine obtained from the whole amount passed in the twenty-four hours should be first examined, then the urine should be tested which is passed on rising in the morning, and also that which has been passed after each meal and on going to bed at night. It was after reading Murchison,[2] that I began to examine specimens passed on going to bed, and I have, as he records, found at this time of the twenty-four hours that many cases of lithæmia only have albumen in their urine. Albumen has, however, been found to be present in the urine under circumstances in which the patient is enjoying perfect health. This fact has been referred to in medical journals by Sir William Gull, Moxon, George Johnson, Leube, Ultzman, Bamberger, Clement Dukes, Rooke, Saundby, Munn, and others.[3] Dr. George Johnson and Clement Dukes believe that albumen appearing in the urine in a person apparently healthy is always pathological and never physiological, and that sooner or later the evidences of Bright's disease will appear. The albumen in the urine in the cases

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[1] Dr. Wm. Carter : Clinical Reports on Renal and Urinary Diseases. London, 1878.

[2] Clinical Lectures on Diseases of the Liver, p. 573. New York, 1877.

[3] Dr. F. P. Kinnicut in a paper read before the Academy, December 15, 1881. Transient Albuminuria.

reported has almost always been associated with deposits of oxalate of lime and uric acid, and as no mention that I can recall has been made of the heredity or condition of blood tension, I should hesitate to agree with the opinion of some of the writers that the presence of the albumen had not a graver significance than they were disposed to admit. It may be that the presence of the albumen is due in these cases to some disturbance of the vaso-motor system, by which dilatation and consequent retardation of the blood-flow occurs, and that the albumen then escapes into the urine; but may not this be an indication that the kidney is the locus minoris resistantiæ of the system, and if in the case a heredity predisposing to renal disease be ascertained, and a condition of more or less increased arterial tension be ascertained, would not the transient albuminuria become a symptom of importance in diagnosis and prognosis?

In chronic parenchymatous nephritis albumen is usually present. The amount varies from time to time. For days and weeks there will be from fifty to eighty per cent. of albumen in a test-tube of urine, and then for some time only moderate quantities will be present. In chronic diffuse nephritis it is present in varying quantities, from a little to a great deal. The urine of the large white kidney contains albumen very constantly, and most commonly in large quantities. In the granular or small kidney the urine often contains no albumen at all or only a slight cloud occasionally. In the last days before death a large quantity may be present. It may be of some interest to mention that when, in cases of sudden death, the urine has been withdrawn *post-mortem*, to aid in arriving at the cause of death, often the urine will be found to contain albumen, and this may be so in cases in which no albumen had ever been discovered in the urine during life, and in cases in which the kidneys were perfectly healthy to the closest scrutiny.[1]

Tube-casts.—The presence of casts of the tubules of the kidney in the urine is not at present considered as a sign of

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[1] Carter, loc. cit. Appendix, note A.

such importance and value as regards the pathological condition of the kidney, as was once thought. It is an unsettled question whether casts, or, at least, any number of them, are derived from that portion of the kidney beyond the "down-looping" tubules of Henle, that is, from the labyrinth of the kidney where the true convoluted tubes and the Malpighian bodies are situated. The tube-casts are apparently derived from that portion of the tubules which extends from the termination of the "down-looping" portion of the tubules of the kidney to the point of termination of the tubules in the papillæ in the pelvis of the kidney. The "cast matter" present in the tubules of the labyrinth of the kidney may, by considerable pressure from behind and the consumption of much time, be forced through the "down-looping tubes" of Henle, which are elastic, and finally appear in the urine. Casts from the labyrinth which escape in this way must be very few; moreover, it would seem most likely that the tube-casts, after passing into the larger "union tubes" and into the larger straight tubes, would partake of the shape of the tubes through which they had last passed. The significance of the granular casts found in the urine of the granular kidney seems to me this, that they are made up of the disintegration of the "cast matter" formed in the tubules of the labyrinth, which has been slowly and gradually forced down and through the narrowest portion of the tubules, receiving finally the shape or impress of the straight tubules. In this connection one should bear in mind another point, that though fatty casts may sometimes indicate the presence of extensive fatty changes in the kidney, yet when one considers that a considerable time must elapse for the passage of "cast matter" from the labyrinth if it does occur, it would not be at all improbable for fatty changes to occur during the probable long transit. When convoluted casts are found in the urine it is most likely that their tortuous shape has been imparted by their passage through the intermediate or connecting tubules of Schweiger-Seidel. Charcot[1] states

[1] Maladies des Vires Biliaires et des Reins, p. 288, Paris, 1877.

that the clinical importance of casts has been much exaggerated, and that casts found in the convoluted tubules can but very rarely, if at all, as I have said, reach the urine.

Reginald Southey[1] holds the same view, and remarks, ". . . that it was pretty generally accepted that the larger casts, and those which present most distinctly cellular elements in their interiors, were derived direct from the tortuous tubules, and this, indeed, before even the minute anatomy of the organs or the intricate course of the tubuli uriniferi were established."

Of the value of tube-casts in the diagnosis of chronic Bright's disease one can only say that their absence does not indicate the non-existence of chronic nephritis; that the presence of a few granular casts, or a few fatty casts, may indicate the arrival of some disintegrated "cast matter" from the labyrinth; that a great abundance of granular or fatty casts may make it probable that the disease of the kidney is more general, and that the process has extended to the pelvic portion of the kidney, that is, to the straight tubules.

Hyaline casts are found in health;[2] especially also in urine containing deposits of oxalate of lime and crystals of uric acid; likewise in urine in cases of jaundice. The casts found in waxy kidney cannot be considered in this paper, since I have not considered the symptoms and signs of any of the forms of secondary chronic Bright's disease.

Deposits in the Urine.—In addition to tube-casts certain other deposits are found in the urine in chronic Bright's disease, especially those of crystals of oxalate of lime and uric acid. Sometimes the crystals mentioned undergo a change in form when mucin, peptones, or albumen are present. If familiar with these changes in form, one's suspicion may be

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[1] Some Points on the Minute Anatomy of the Kidney. Medico-Chirurg. Transactions, vol. lxi., 1878, pp. 201–213.

[2] It is even now a question with pathologists whether the presence of hyaline casts in the urine of persons apparently in health, does not point to the beginning of structural changes in the kidney or of functional troubles likely to terminate in organic disease.

aroused, and further examination of the urine may reveal the special colloid matter. Attention has been especially called to these changes by Dr. W. M. Ord.[1]

FIG. 1.—From Dr. Ord's paper, Medico-Chirurg. Report, vol. lviii. A. Uric acid crystals from albuminous urine of sp. gr. 1027 and upward (Dr. Donkin, Obs.). B. Uric acid crystals from albuminous urine of sp. gr. 1015 to 1021 (Dr. Donkin, Obs.). C. Uric acid crystals from albuminous urine of sp. gr. 1015 to 1021 (Dr. Ord, Obs.). D. Uric acid crystals from albuminous urine of sp. gr. 1009 to 1015 (Drs. Donkin and Ord, Obs.).

[1] St. Thomas's Hospital Reports, vol. ii., 1871, pp. 1–23; Transactions Medico-Chirurg. Society, vol. lviii., pp. 165–182; British Medical Journal, June 2 and 9, 1877, pp. 641–672 and 701–703,

I have not referred to the diminution of the amount of urea found in the urine as a means of diagnosis, since[1] the amount of the daily excretion is irregular, and since the number of considerations to be regarded, in respect to diet, exercise, etc., make it impossible to give the time for such an inquiry to each patient.

i. Increased Arterial Tension.—This sign is at the present time regarded by many as one of the most constant and valuable of all the signs of Bright's disease. It is present both in the acute and chronic forms, but is most constant in the latter.

To appreciate the presence of high arterial tension requires considerable experience and practice, especially when one has not the aid of instruments of precision, the sphygmograph and sphygmometer—particularly the former. Some years ago, when reading the "Handbook for the Physiological Laboratory," by Burdon-Sanderson, I was much impressed with a statement that he there made,[2] namely, that the arterial pressure in the mean is remarkably constant—almost as constant, indeed, as the temperature of the body; that is, as long as the individual remains in a natural state. Until within the last few years I was disposed to look upon this as an exaggerated statement. Since, however, I have been using a sphygmograph which can be very accurately placed over the artery and to which weights can be so added that the pressure employed can be accurately estimated, I have been very much inclined to accept Dr. Burdon Sanderson's statement. I shall refer to this when I shall describe the use of the sphygmograph, preferring to consider first the means at our command for the appreciation of high tension without the use of any instrument.

In extreme cases of high arterial tension the temporal and radial arteries are tortuous, and being full of blood they leap from their beds, apparently, at each pulsation, producing a

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[1] Since this essay was read, Senator's monograph (Die Albuminurie imgesunden und Kranken Zustande, Berlin, 1882) has appeared, and in this he calls attention to the not unusual appearance of peptones in the urine.

[2] Handbook for the Physiological Laboratory. Edited by Sanderson. Vol. i., p. 218. Philadelphia, 1873.

pulsation somewhat resembling that of aortic regurgitation, only that the arteries in the interval do not disappear. The radial artery in marked cases feels very much like the tendon of the flexor carpi radialis adjoining it, or like the vas deferens. Sibson[1] well describes the method for the appreciation of the condition of the radial artery in high tension. "You do not press the fingers into the artery so as to compress it, but you glide them backward and forward, and from side to side, of the artery, and you endeavor, if the case be one with extreme arterial degeneration, to gently poise it up and lift it from its bed. You thus feel whether it is continually hard, whether the vessel is pulsating or not. You notice whether you can feel and count the pulse by simply placing the tips of four fingers with gentle firmness, without the least compression, over the artery. If the vessel softens between the strokes of the heart and hardens during or just after, then you know that the case is not one of great arterial tension. . . . In the first or highest degree of tension the artery feels like a cord or tendon. In the second degree it is like an india-rubber tube distended with fluid. In the third degree it resembles a rather finer india-rubber tube." There are cases, however, in which neither of the three varieties mentioned by Sibson are to be found, and in which a very considerable degree of pressure must be exercised before the artery can be compressed. The appreciation of cases of this nature is especially important, since it is very unlikely that any changes have as yet taken place in the walls of the vessels, and sometimes one cannot feel convinced as to the degree of high tension in these cases until sphygmographic observations have been made. In compressing the radial artery, the compression should be made where the artery is closest to the surface of the lower end of the radius; and while this is done with the fingers of one hand, palpation of the distal portion of the artery should be made with the other, since if the compression and palpation be made

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[1] Two Harveian Lectures on Bright's Disease and its Treatment. British Medical Journal, January 6 *et al.*, 1877, p. 34.

with fingers of the same hand, it is very difficult to say when the distal pulsation ceases. In a case where the walls of the vessels have not been thickened, and there is no increased arterial tension—the conditions observed in health—the pulse, the radial artery should not be appreciable in the interval of pulsation.

In some cases it at first would seem impossible to decide whether the radial artery was simply over-distended with blood or that the walls were the seat of extreme arterial degeneration. Dr. Broadbent has suggested a simple test by which the exact condition may be ascertained, and that is by the inhalation of nitrite of amyl, and then, if the artery has been simply over-distended, the excess of blood having escaped by the arterioles and capillaries, the vessel will be found soft and easily compressed: if, however, the walls of the vessels have undergone degeneration, little change has taken place. In most cases, however, in which only endarteritis deformans is present, the walls of the vessel are quite easily compressed if not calcified. When high arterial tension coexists with endarteritis deformans the pulsation in the vessels is often unusually distinct, and, as I have mentioned, may simulate the pulsation observed in aortic regurgitation.

The qualities of the pulse in high tension are described as *long*, *persistent*, and *hard* or *incompressible* (Mahomed). The pulse is *long* by reason of the prolonged systole, which is due to the heart's overcoming the resistance presented by the overfilled vessels. It gives the finger resting on the pulse a labored pushing sensation. The pulse is *persistent* because always full, and is recognizable in diastole as well as in systole. Mahomed states that a pulse of large volume with the artery relaxed and the blood flowing freely through the capillaries and arterioles, and soft, has with a languidly acting heart some qualities of persistence which might be confounded with a pulse of high tension, but it is so soft and easily compressed that the mistake is not easily made.[1]

The pulse is *hard*, the arterial system is overfilled with

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[1] Mahomed: Guy's Hosp. Rep., 1879, p. 372.

blood, and the hypertrophied left ventricle forcing more blood into the vessels gives the pulse a peculiar hard, taut feeling to the fingers. This very hard quality is not apparent in all cases, but with high tension there is always present *incompressibility.* When the heart begins to fail the pulsation may seem very feeble, but pressure upon the artery will still reveal the incompressibility and over-fulness of the vessel.

A beat or pulsation " is perceived over the enlarged ascending aorta to the right of the upper portion of the sternum, and mainly in the second space in a few rare cases of acute Bright's disease on the one hand, and of granular kidney in its advanced stage on the other." [1] This is best felt by pressing over this region with that part of the palm of the hand that is nearest the fingers, and it is well, in order to well appreciate it, also to press this hand very firmly against the second interspace by placing the other hand over it. This pulsation is produced by the widening of the artery in consequence of the arch of the aorta being somewhat displaced downward, and the ascending portion of the arch forward and to the right. This is what Sibson states, and I have sometimes thought that dilatation of the arch is more common than is usually thought, since rarely at post-mortem is the size of the aorta measured, and either this is the case or the arch of the aorta is often also displaced upward and forward—pulsation in the supra-sternal notch occurring frequently.

Marked accentuation of the second aortic sound is one of the most constant signs of high arterial tension before failure of the heart occurs. This sound is heard with greatest intensity over the second right intercostal space, but it is also heard over the left third and fourth intercostal spaces near the sternum, over the conus arteriosus. It may be more intense over this region than at right second intercostal space, it may be as intense and it may be not so loud. The presence of the loud aortic second sound over the aorta is often accompanied with a feeble muffled first sound to the right of the upper portion of

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[1] Sibson, loc. cit., p. 34.

the sternum, or this may be even absent. This is due to the fact that "the arteries are already overfilled when the left ventricle forces its blood into them, and the fluid already in the aorta prevents the ventricle from giving a sudden distention and shock to the walls of the aorta. In cases of aortic regurgitation and aneurism, when the aorta is flaccid, a loud shock is often heard with the first sound, owing to the hammer-like action of the suddenly expanding waves of fluid which strike against the wall of the artery."[1]

Mahomed remarks that when the accentuated aortic second sound has a tympanitic quality it usually indicates that dilatation of the ascending aorta is present.

Sibson also states that in many cases of acute Bright's disease a doubling of the second sound of the heart over the region of the conus arteriosus may be discovered. This occurs less often over the region of the pulmonary artery, and less still over that of the ascending aorta. This certainly must be of more frequent occurrence in the acute than in chronic Bright's disease.

As regards the first sound of the heart, certain peculiarities have been observed of more or less importance by various observers.

Potain has especially called attention to what he terms the *bruit de galop*, which is very frequently overlooked. A soft murmur precedes the first sound—is presystolic, but does not resemble, except in the time of occurrence, in any way the true mitral presystolic murmur which accompanies mitral stenosis.

According to Sibson,[2] doubling of the first sound not infrequently occurs in many cases, and is more or less limited to the region of the septum, or a little within the mammary line, and from one to two inches below the level of the nipple. Sometimes it is easy to discover, and again often difficult. "When the doubling is markedly accentuated and extensive, it is usually doubled over the whole of the right ventricle, where it may be especially emphasized; also over the epigastric space, that

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[1] Sibson, loc. cit.

[2] Loc. cit., p. 35.

being the space at which the heart is most uncovered and is nearest to the surface. It is lost over the left ventricle and its apex, being usually inaudible to the left of the nipple line; and this, under the circumstances, is natural, for the left ventricle is large and extensive, while the right ventricle is relatively small, so that the sounds from the former are readily transmitted through the latter."[1]

"When the doubling of the first sound is diffused over the right ventricle, the first of the two first sounds is heard over and to the left of the region of the sternum and ensiform cartilage, and the second of the two sounds is louder over and just to the left of the septum, thus demonstrating that the second of the two first sounds is that made by the ending of the systole of the left ventricle."[2] This Sibson believes to be very frequent, but "more difficult than easy to discover," and as a rule he would say that the second sound would be found single, and the first double.

Hypertrophy of the heart, and especially hypertrophy of the left ventricle, is one of the most valuable signs of the presence of chronic Bright's disease. It is very likely that increased arterial tension precedes it and is the direct cause of it.

Its constancy may be judged of by the following statistics: Bamberger[3] found hypertrophy of the heart in 344 out of 807 cases of primary chronic Bright's disease, or 42 per cent. Reginald Southey, in the "Lumleian Lectures on Bright's Disease," *British Medical Journal*, agrees with Bamberger in its very frequent occurrence. Traube states that he has found hypertrophy of the left ventricle in 93 cases out of 100 of chronic Bright's disease. Grainger Stewart has recorded that out of 68 cases of chronic Bright's disease, hypertrophy was decidedly marked in 31 cases at the post-mortem examinations, and this statement is exclusive of those in which the hypertrophy was associated with valvular disease or adhesion of the pericardium. The analysis of 250 cases of granular kidney, drawn from St. George's Hospital books, gives 48 per cent. as

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[1] Loc. cit., p. 36. [2] Lancet, January 13, 1877.
[3] Volkmann's Sammlung klinischer Vorträge, No. 17.

the proportion of cardiac enlargement. Bartels[1] states: "Here I must distinctively state that I have never failed to observe the objective signs of hypertrophy of the left ventricle in any of my cases of genuine contracted kidney, or to confirm the fact in every post-mortem made upon their bodies, and therefore I can never hold that a diagnosis of this renal disease is made certain when no enlargement of the left ventricle is recognizable. Another circumstance—one which must not pass unheeded—is that the degree of hypertrophy of the heart, and therefore its compensating power or capacity, will vary in different cases in accordance with the general state of the patient's nutrition and the external conditions to which he is subject, and that, as a matter of course, the same factor will materially influence the course and issue of individual cases of the disease."[2]

Galabin[3] records that in 79 cases of granular kidney, hypertrophy of the left ventricle was found in 53 cases, 24 of which were in women. Hypertrophy of the left ventricle may be considered then the rule; but if it be asked why it is not always found post-mortem, or always made out during life, the answer will be:

First.—The hypertrophy of the left ventricle or its compensatory power will vary in different cases in accordance with the general state of the patient's nutrition and the external conditions to which he is subject.

Dr. George Johnson presents, in explanation of the absence of hypertrophy, "that in states of general malnutrition which include a watery condition of the blood, or a general diminution of its mass, neither can any hypertrophy take place, nor need dilatation of its cavities take place."[4] Bamberger[5] explains

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[1] Ziemssen, American edition, vol. xv., p. 419.

[2] Loc. cit., p. 466.

[3] Thesis, Cambridge: On the Connection of Bright's Disease with the Changes in the Vascular System. London: Smith, Elder & Co., 1873.

[4] Lancet, January 9, 1875.

[5] Ueber Morbus Brightii und seine Beziehungen zu anderen Krankheiten. Volkmann's Sammlung klinischer Vorträge, No. 178 (Medical Times and Gazette, January 3 and 10, 1880).

in much the same way the absence of hypertrophy in many cases of chronic Bright's disease, which occur secondary to other diseases. In cases of secondary Bright's disease, hypertrophy of the heart occurs in 3.3 per cent. against 42.6 per cent. in chronic Bright's disease of primary origin.

Second.—Moxon[1] says: "When a heart is larger than it should be, but its cavity is of natural size, its possessor is in certain dangers in consequence of this, but *he does not feel those dangers, and his enlarged heart yields no symptoms.* This state is almost peculiar to Bright's granular kidney, and I accept it as the chief cause of the fact that seventy-five per cent. of the cerebral apoplexies with effusion of blood from bursting of the vessels, which came under my observation, were due to granular kidney. The heart's increased power is exerted in the vessels instead of being diffused on hydrostatic principles over an enlarged interior, as is the case when the cavity is widened. But the heart may reach twenty ounces in weight, and yet, if its cavity is not enlarged, it may give no sign at all, as we have proved on several occasions in cases where, knowing the presence of granular kidney in persons with apoplexy, we have examined for evidences of cardiac hypertrophy, and found none at all, whereas the post-mortem directly after showed hearts weighing from seventeen to twenty ounces."

Excluding cases with concomitant valvular disease, endocarditis, aneurism, or emphysema, Bamberger states "that hypertrophy of the heart occurs in every period of the disease, but with greater absolute as well as relative frequency, the longer the disease lasts;" so that, in accounting for the absence of hypertrophy, the element of time must also be considered.

The determination of high arterial tension by instruments of precision.—The Sphygmometer (Fig. 2). This instrument, as represented in Fig. 2, was invented by C. Handfield Jones, and it is intended to measure the compressibility of the pulse by means of a spiral spring carrying a needle which registers

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[1] Clinical Lecture on the Relievable Aspects of Heart Disease, Lancet, January 12, 1878, p. 42.

upon a scale the amount of pressure expressed in grammes necessary to produce occlusion of the radial artery, or of any other artery which lies superficially where it can be compressed against bone. Instruments for this purpose have been proposed by others, but the sphygmometer I have referred to is the one in most general use at present. The instrument is used in the following manner: The narrow oblong extremity is placed over the radial artery—for example, where it passes over the radius most superficially, and where the artery can be directly compressed against the bone. Pressure is then made until no pulsation can be observed over the distal portion of the vessel. The sources of error in the instrument are the slipping of the oblong end from the artery, the uncertainty of the spring in recording pressure, and occasionally the great freedom of the return circulation.

Fig. 2.

The Sphygmograph.—After daily use of the sphygmograph for four years and more, I am prepared to say that I regard it as an instrument of very great value. While making this assertion, I must add, that of all the instruments of precision, this one requires the greatest experience and the most careful application to make the deductions from its use of any value. It is certainly not a "plaything," as some have declared, but an instrument of the greatest value when employed with especial care, and this must be apparent when one considers that by no other instrument can we calculate, although relatively, the amount of blood contained in the arterial system. It is true that by the appreciation of the signs of the pulse, etc., that I have just referred to, a high degree of increased arterial tension can be appreciated, but there are cases which do not present marked evidences of increased blood tension by the signs given, although it is present, and in such cases the increased arterial tension can only be demonstrated by the sphygmograph; and what is said of high tension may also be affirmed of low arterial tension. In the sphygmograph,

also, one has an instrument by which arterial degeneration can be made out before the grosser changes appear which permit of diagnosis by palpation.

The sphygmographs in common use in this country are those

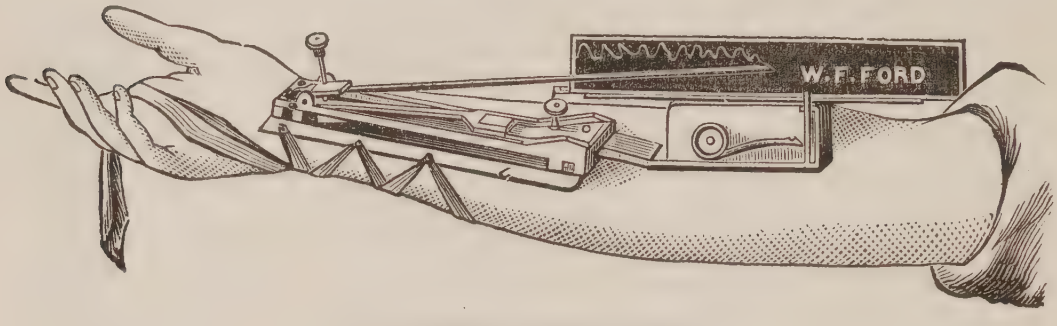

FIG. 3.—Marey's Sphygmograph.

of Marey, Mahomed, and Pond. Recently the sphygmograph of Dr. Dudgeon of Birmingham has been introduced, which combines certain features of the Pond and Mahomed instrument. In all of the instruments that I have mentioned thus far, a

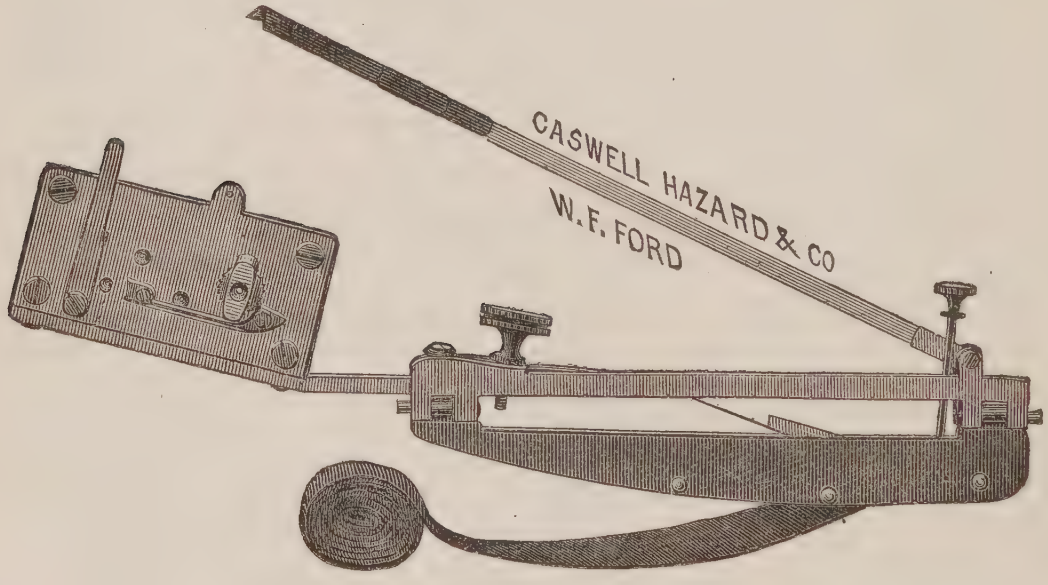

FIG. 4.—Mahomed's Sphygmograph.

spring is used in transmitting the movements of the artery to the writing lever (in the Pond instrument the rubber diaphragm serves as a spring). Figures 3, 4, 5 and 6 represent respectively the instruments mentioned.

I myself am not disposed to regard the instruments provided

with a spring with much favor, since it is very difficult, if not impossible, to accurately measure the amount of pressure employed. In the instruments provided with a spring, the pressure applied to the artery to produce the greatest amplitude in the pulse, tracing or to obliterate the artery varies much in instruments of the same kind, and I think often varies in the same instrument. The instrument, in my opinion, which at the present date seems to have been so constructed as to meet the necessary indications and to give the most correct tracings of the changes in the artery in systole and diastole is a modification of the Brondel sphygmograph (Fig. 7). The instrument is supported by two

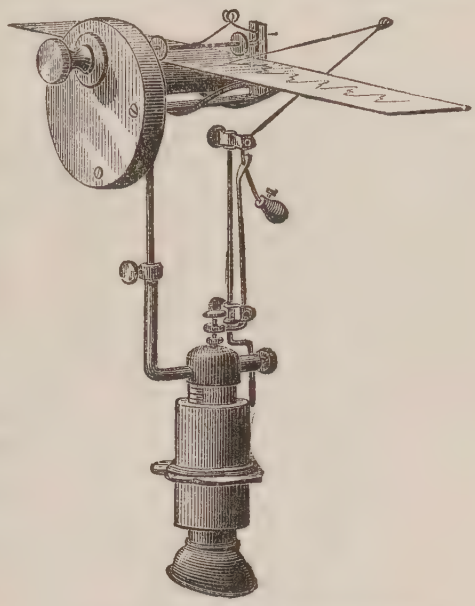

Fig. 5.—Pond's Sphygmograph.

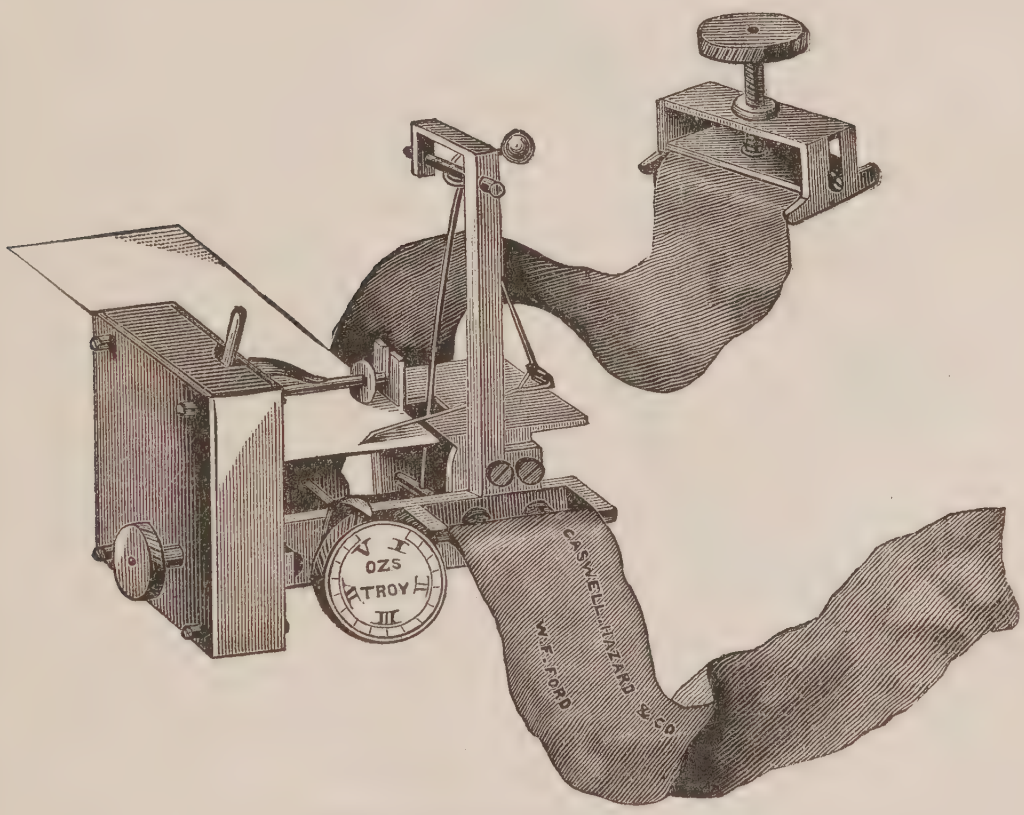

Fig. 6.—Dudgeon Sphygmograph.

parallel brass bars, and in this way when placed upon the arm no pressure can be made upon the artery by the instrument. The movements of the vessel are communicated to a lever of brass of 15 grammes weight. To this lever is attached an arm on which may be suspended gramme weights —from five grammes to two hundred and fifty-five. In applying this instrument, the course of the radial artery is marked out upon the skin over it by a lead-pencil, and the button of the lever, which is narrow, is placed upon this line at the point where the radial artery rests upon the lower end of the radius. The sphygmograph is then fastened in place by straps, and one must be careful in tightening the straps not to draw the skin with the marked line away from the artery. If the artery is superficially situated, pulsating movements of the lever will at once be apparent. If the artery is deeply situated, a varying amount of weight must be suspended on the arm of the instrument to develop the pulse. If the artery is atheromatous, and no high tension is present, the addition

Fig. 7.—Brondel's Sphygmograph Modified.

of weights is necessary to bring out the pulsation of the vessel.

The amount of weight necessary to obtain the greatest amplitude of the pulse-wave varies from 40 to 80 grammes, rarely so high as the latter figure. I refer, of course, to the pulse in health with normal tension, the artery superficially situated.

When the artery is deeply situated a weight of 30 to 40 grammes is necessary to develop the pulsation, not the greatest amplitude, and if no pulsation at all appears on the addition of 50 grammes, you may rest assured that the button of the instrument is not over the artery. I have observed thus far that even in the weakest pulse to which I have had the opportunity to apply this instrument, that when deeply situated 30 to 40 grammes will develop a pulsation which is, however, extinguished on the addition of a few grammes more weight.

If the radial artery be atheromatous—and bear in mind there are degrees of this, and I have had difficulty in being able to obtain cases presenting a moderate amount of this change without high tension—it will be necessary to use weights of from 60 to 100 grammes to develop pulsation, but the pulsation when developed is extinguished soon by the addition of more weight, that is from 120 to 160. The characteristic as far as pressure goes is the great amount of weight to develop the pulse, and the speedy obliteration of the pulsation by the addition of only a little more weight.

In the pulse of high tension, pulsation may appear as soon as the instrument is set, but if deeply seated, weights will be required. It will be always found that the greatest amplitude of the pulse is to be obtained under weights varying from 100 to 300 grammes, and what is specially characteristic is that when the greatest amplitude is obtained but little apparent change takes place in the pulse-trace, while quite an amount of weight is added—that is, if the greatest amplitude be developed at 160 grammes weight, there will be but little change in the amplitude of the pulse-trace after 40 to 60 grammes have been added.

I now ask your attention to the pulse-trace or sphygmogram

obtained by these instruments. The traces obtained by the Mare , Mahomed, Pond, and Dudgeon sphygmographs resemble one another so closely that one explanation of the parts of the trace of a normal pulse will suffice for all.

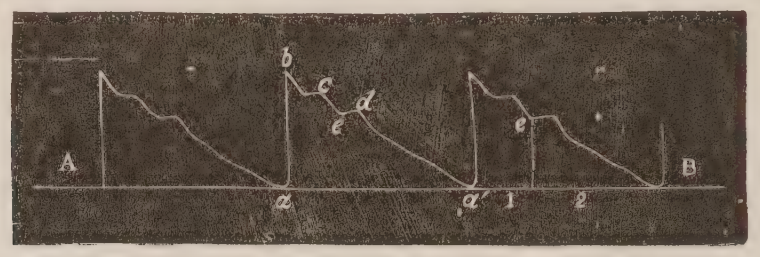

FIG. 8.—Line of ascent, up-stroke or percussion stroke=*a* to *b*. Apex=*b*. Line of descent=*b* to *a'* ; *d* =aortic or dicrotic wave ; *e*=aortic notch ; *c*=tidal wave. A B=base or respiratory line. 1= Systolic portion of the tracing, *i.e.*, with reference to the systole and diastole of the ventricle, not of the artery. 2=Diastolic portion of the tracing.

In the Pond[1] and Dudgeon sphygmographs the percussion waves are usually higher and the dicrotic waves more highly developed than in the Marey and Mahomed tracings, and there are often also secondary waves. One defect in the Pond instrument which has been referred to by Professor Robert Edes[2] is that the base of the instrument is so broad that the pressure is applied not upon the artery, but upon a portion of the lower end of the radius and the tendon of the flexor carpi radialis, and movement consequently of the lever can only be obtained from that portion of the vessel which happens to rise above the level of points on which the base of the instrument rests.

It has certainly seemed to me that since I have been using an instrument in which the amount of pressure was applied directly by weights, I have obtained more constant results than before in observations on the same patient, and that I have also been able to compare the tracings in different patients and find some constant appearances, while heretofore the tracings obtained by the other instruments in which the degree of pressure had not been accurately estimated would present the most variable appearance. The body of the original

[1] The Pond instrument has been very much improved since the above was written, and I think will now prove a good instrument.

[2] Bost. Med. and Surg. Jour., May 19, 1881.

Brondel instrument rests upon the surface of the forearm, and when tightly strapped in place may exercise some compression on the radial artery. The tracings obtained by the original instrument present secondary waves and exaggerated percussion and dicrotic waves, especially when the heart contracts brusquely. I discovered some time ago that, by having the

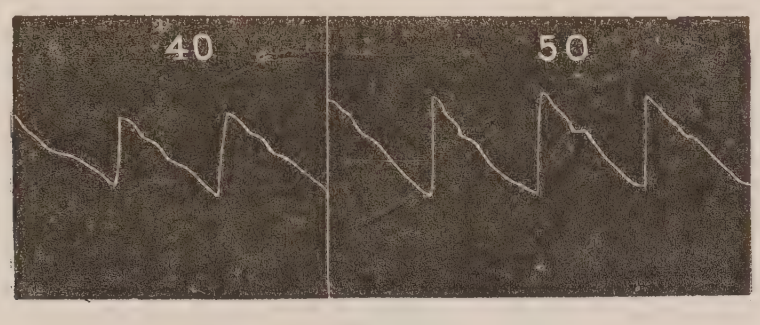

Fig. 9.

body of the instrument rest upon parallel bars, there was no possibility of compressing the artery, and furthermore I found that the inertia of the writing lever was almost entirely prevented, and that no exaggerated percussion or dicrotic waves were present, and likewise no secondary waves or oscillations. The great value of the instrument is the accurate measurement

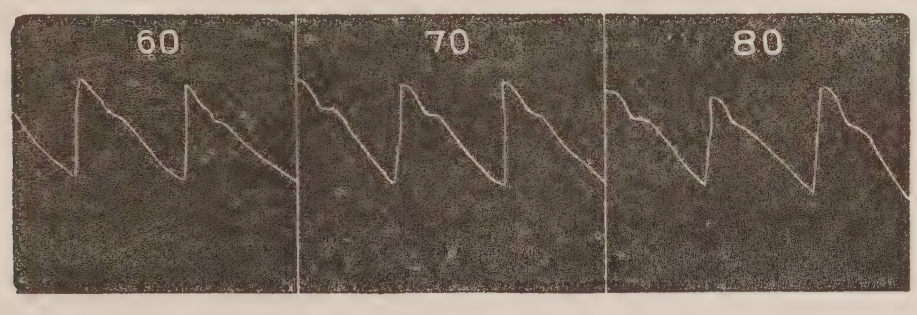

Fig. 10.

of the degree of weight necessary to produce the greatest amplitude of the pulse-trace, and also to occlude the arteries.

The normal pulse-trace obtained by the Brondel sphygmograph is represented in Figs. 9 and 10. The greatest amplitude, it will be observed, is obtained with weights of 50 and 60 grammes. The addition of more weight flattens the apex and diminishes the height of the percussion wave.

I now invite your attention to the interpretation of the pulse-traces which indicate high tension. Figures 11 and 12 are traces of high tension obtained by Mahomed's instrument, and what is said of the signs of high tension in these traces applies equally to the traces obtained by the other instruments, including Brondel's.

1. It is a sign of high tension when the tidal wave, or a part

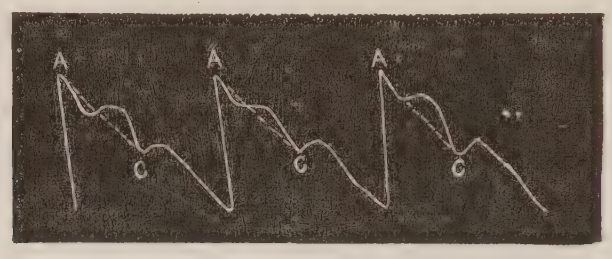

Fig. 11.

of it, is above a line drawn from the aortic notch C to the summit of the percussion wave A, Fig. 11.

2. The greater the height of the aortic notch C from the respiratory line D D, the higher the tension, Fig. 12.

3. The greater the length of the systole—the distance A to D, as compared with the length of the diastole—the distance

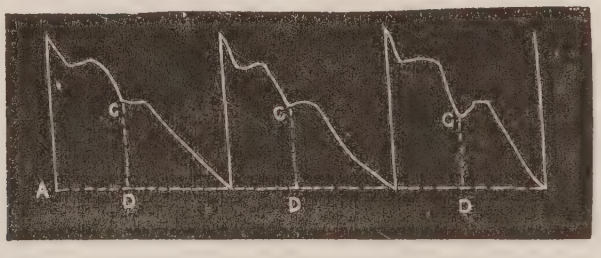

Fig. 12.

D to E, the greater the tension, as a rule; but in some slowly contracting hearts the systole may be much greater in proportion to the diastole than it should be, and yet the tension be low.

4. Effacement of aortic notch indicates high tension.

5. Slanting of percussion stroke with effacement of aortic notch.

6. Rounding of top of the percussion trace indicates very high tension, with slow, strong contraction of the heart, or it

may indicate failing heart with high tension, or simply a feeble heart. The amount of pressure applied will distinguish the two former from the latter condition.

7. The amount of pressure required to produce the greatest amplitude of the pulse-trace, and the continuance of this amplitude with only slight change in size, while the weight or pressure is increased by several ounces or a number of grammes, indicates high tension.

8. The amount of pressure necessary to occlude the artery, so that no pulsation is perceptible, is likewise an indication of degree of tension.

Certain anomalies in the sphygmographic traces or sphygmograms.—1. When the heart dilates, as it often does under great arterial tension, or when the hypertrophied heart is beginning to undergo degeneration, traces are sometimes obtained which greatly resemble the traces of low tension. This error will not be committed if it be remembered that the arterial system is overfilled with blood, and that the trace resembling a low-tension trace is due to the difficulty with which the weakened heart empties its contents into the overfilled vessels. One can easily see that under these circumstances the pulsation will be apparently weak. If, however, pressure enough is employed to give the trace of the greatest amplitude, it will be found, although the amplitude of the trace is not great, yet the amount of pressure necessary to obtain it was very considerable. If one also ascertains how much pressure is necessary to obliterate the artery, the high tension will be still more apparent. The pulse under the circumstances is described as small or large, long and labored.

2. In weak hearts, or hearts contracting slowly, the systole may be prolonged, and the pulse-trace will resemble very closely that of high tension. In cases of this kind the degree of pressure alone enables us to appreciate the error.

4. In mitral regurgitation, which not infrequently appears to result from dilatation of the left ventricle producing insufficiency of the mitral orifice, low-tension sphygmograms are frequently obtained. It is well in these cases to observe care-

fully the length of the ventricular systole, and to compare the length of the systole with that in others having the same pulse-rate or frequency. In mitral insufficiency from endocarditis the arterial vessels are empty, as a rule. When the insufficiency is due to the long presence of high tension, they are full and turgid if the condition of high tension is kept up.[1]

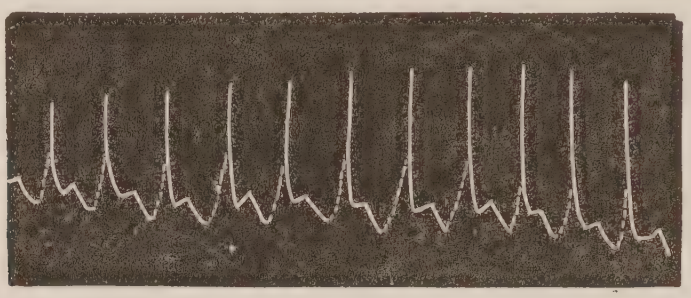

Fig. 13.

5. Aortic regurgitation results very often from the dilatation of the aorta or from *aortitis deformans*—two conditions due to increased arterial tension, and the cause of the insufficiency can be appreciated if the high tension is sustained and present at the time that the traces are taken.[2]

Fig. 13 is the tracing from a case in which the insufficiency

Fig. 14.

is due to an antecedent attack of endocarditis. Fig. 14 is the trace of aortic insufficiency resulting from aortitis deformans. Mahomed has called attention to this, and the traces which I have submitted, though obtained by Pond's sphygmograph, correspond to those presented by Mahomed in his paper.

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[1] Mahomed; Clinical Aspects of Chronic Bright's Disease. Guy's Hospital Reports, vol. xxiv., 1879.

[2] Ibid.

7. An extremely shallow percussion trace and a sustained tidal wave are due to a feebly acting heart with or without overfulness of vessels, which fact must be determined by the amount of pressure used.

8. C. Handfield Jones[1] calls attention "to pulse-traces resembling high where the percussion wave is sloping gradual, and low where the descent of the trace is greater. This indicates slow, languid contraction of the ventricle, and a similar mode of resilience on the part of the arteries." The degree of pressure employed would determine in these cases the presence or absence of high tension.

While believing that increased arterial tension is one of the most constant and important symptoms of Bright's disease, one must not expect to find it always. It is absent, as a rule, in the following conditions, and one should bear them constantly in mind:

A. In pyrexia. As Anstie and Wolf long ago stated, when the temperature of the body exceeds 102.5°, the pulse is decidedly dicrotic; usually full dicrotism is present.

B. Great loss of blood, as perhaps by epistaxis.

C. Contraction or obliteration of many pulmonary vessels, as occurs in emphysema.

D. In certain forms of valvular disease, as in mitral stenosis, also in aortic and mitral regurgitation, when the heart is undergoing degeneration and the backward leakage is great.

E. Paresis of vaso-motor centre from drugs, poisons, etc., or in injuries to the spinal cord.

j. Endarteritis chronica.—This becomes a sign of considerable importance in the diagnosis of chronic Bright's disease. It is generally admitted that changes in the walls of the small arteries throughout the body is a common feature in this disease. Drs. George Johnson, Gull and Sutton have especially called attention to this, Dr. Johnson maintaining that the increase in the thickening of arterioles is due to hypertrophy of the muscular walls of the small arteries; and Drs. Gull and Sutton, on

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[1] Lancet, March 2, 1880.

the other hand, that it is due to some fibroid change, which they term arterio-capillary-fibrosis. Still another change takes place in the small arteries in chronic Bright's disease,[1] namely, *endarteritis obliterans*—this is found principally in the kidney and in the vessels supplying the brain, and some accidents in the course of Bright's disease are to be explained by the presence of this pathological condition.

The presence of thickened arteries—I refer particularly to the radials and temporals now—has been so constant in all of the cases of chronic Bright's disease that I have seen, that I have thought that its existence in a patient should receive careful recognition, and especially in a disease in which the diagnosis must in a great measure depend upon the collocation of a number of symptoms.[2]

Endarteritis chronica is especially frequent in males. It is rarely found before forty according to Quincke,[3] although very many cases have been observed under forty. It is common to old age, and yet is sometimes absent in persons from eighty to one hundred years of age. Guéneau de Mussy [4] considers that it may occur just as frequently and as severely before forty-five as afterward. The causes assigned for it are chronic alcoholism, lead-poisoning, gout, rheumatism, syphilis and Bright's disease. It occurs also in some cases of anæmia, starvation, and great exposure. It is also produced by high arterial tension. Its importance as a symptom rises in value when one appreciates the fact that most of the supposed causes of endarteritis chronica are also considered causes of chronic inflammation of the kidneys to a greater or less extent, since it is only with limitations that one can exclude from the causes of kidney disease alcoholism, rheumatism, syphilis, and great

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[1] Likewise in syphilis and gout.

[2] I have not alluded to the ophthalmoscopic appearances of the fundus oculi in chronic Bright's disease or of the inferential importance that the detection of a retinal hemorrhage might have, only because the limits necessary to this essay will not permit of it.

[3] Ziemssen's Cyclopædia, vol. vi., p. 340, Am. Ed.

[4] Quoted by Quincke.

anæmia, since they are so very frequently associated with all varieties of chronic nephritis.

Charcot[1] has recognized the importance of this as an evidence of the presence of chronic Bright's disease, and he considers this condition more fully than most of the writers on kidney diseases.

I have been unable to find any mention made of the condition of the walls of the arteries of the extremities in cases of chronic Bright's disease. The frequent presence of apparent thickening of the coats of the temporal and radial arteries in these cases during life leads me to think that if a careful examination was made of these arteries more frequently *post-mortem*, that endarteritis would be found more commonly than is now supposed. There are also many reasons for thinking that the arteries of the extremities would be more likely to undergo the changes peculiar to endarteritis than the arteries of the viscera and the internal organs.

I have yet to meet with a case of chronic Bright's disease, which had existed for any length of time, without careful examination having disclosed the presence of endarteritis chronica. I think, therefore, that if one finds this condition present, when other causes bearing directly upon its causation can be excluded, one must accept its presence as a factor of some importance in connection with other evidence of the presence of chronic Bright's disease.

k. Coma.—The diagnosis of uræmic coma is made by the history if this can be obtained, and by the signs and symptoms of the disease which may be present. It is stated by Bourneville and other French writers that in uræmic coma a low temperature is present. In the cases that I have observed the rectal temperature has always been quite high. It may be that in uræmic coma occurring in the aged, or in the forms of chronic Bright's disease which are secondary to other diseases, the temperature will be found low.

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[1] Leçons sur les Maladies du Foie, des Vories Biliares et du Reins, p. 322. Paris, 1877.

It is necessary that something should be said in regard to the differentiation of uræmic coma from coma occurring in patients having chronic Bright's disease when the coma is not uræmic but due to some one of the accidents which may occur in course of the disease, and which result from some of the other pathological changes which are found in cases of chronic inflammation of the kidneys.

1. Coma in cerebral hemorrhage. The recognition of this is important since cerebral hemorrhage occurs so often in chronic Bright's disease that there is danger of the true condition of affairs not being discovered. In coma in cerebral hemorrhage the rectal temperature pursues usually a peculiar course. For the first half hour to two hours the temperature is low and then rises, and if the patient is about to die goes up very high. If the patient does not die in the course of one to two days the temperature oscillates between 100° to 102° for two to four days, provided no complications exist; this is the stationary period, and then reaches the normal if the patient is to recover. If at the end of the stationary period a very high rise of temperature occur, it indicates, usually, approaching death. In uræmic coma the temperature is usually high, and has, I believe, no characteristic course, and the duration of coma is also not so great. In coma in cerebral hemorrhage, if the lesion be hemispherical there may be present conjugate deviation of the eyes with rotation of the head away from the paralyzed side. There may be abolition of all of the superficial reflexes, and exaggeration of patella tendon reflex and other deep reflexes on the paralyzed side, if there is paralysis as is usual, while on the opposite side the superficial reflexes are preserved and the deep are normal. Perfect flaccidity may be present, but if the hemorrhage be ventricular or meningeal, spasmodic movements or rigidity may be present.

2. Coma in cerebral *ramolissement* from thrombosis or *endarteritis obliterans.* It is likely that, in most cases in which endarteritis obliterans may be present in the arteries supplying the brain, the changes will occur in small arteries, and that coma will not be a common symptom; still it may

occur, and I only refer to it here to call attention to the necessity of bearing in mind this pathological condition when investigating a case of chronic Bright's disease with cerebral symptoms.

If endarteritis chronica be of the very common occurrence in chronic nephritis that I take it to be, thrombosis should not be of infrequent occurrence in this disease when the heart's action begins to fail. I have been unable to consult any statistics in which the complete pathological changes in the body had been given, on which to base a statement of its frequency. In the diagnosis of coma from this cause, the previous history of the patient, the mode of onset, the presence of hemiplegia with abolition of superficial and presence of deep reflexes on the paralyzed side, etc., with the rectal temperature, will assist in the differential diagnosis from uræmic coma. In cerebral softening the fall of temperature observed in cerebral hemorrhage does not as a rule take place—the temperature in rectum ranging from 99.5° to 100.5°. After the first two hours the temperature rises quickly to 102° to 104°, but again quickly descends to the normal, and afterward exhibits irregular exacerbations for a few days. Again, death may take place in softening when the rise of temperature has been small, 102° to 104°, and again it may go very high.

l. Convulsions.—The differential diagnosis from epileptic convulsions will depend upon the detection of the signs of renal disease, but in two cases that I have under observation, true epilepsy of some years' duration exists with chronic Bright's disease. The rectal temperature in the uræmic convulsions which I have witnessed has always been high, above 102° at least, whereas in a single epileptic convulsion the temperature is always below 101°. If, however, the status epilepticus occurs, in which one convulsion succeeds another, and other signs supervene peculiar to this condition, a very high temperature is present. Another sign of some importance in the differentiation of epileptic and uræmic convulsions is the condition of the pupils, which are dilated and mobile in the

intervals of the attacks.[1] The rectal temperature is of great aid in the differentiation of hysterical convulsions occurring in one having chronic Bright's disease—the temperature not being elevated above 100.5° or 101°, and in the uræmic being high.

m. Delirium and other head symptoms.—I have at present two patients under observation in whom at times the following symptoms appear, ushered in first by some stupor or tendency to somnolency; delirium not active, appearing at first only on awakening from sleep, then it becomes more continuous, and sleeplessness occurs; great pain in the head may be present or not; twitchings of the extremities may be present or not; in one case temporary aphasia occurs during the attack: in both hallucinations of sight, hearing and taste are present. These symptoms are accompanied with a decided febrile movement ranging from 101° to 103·5°. The urine diminishes in quantity, has a higher specific gravity than is common, and is loaded with albumen. In one obstinate constipation prevails, and in the other diarrhœa with profuse evacuations. The pulse is frequent and easily compressed. Both patients have had several attacks from which they have entirely recovered. I can also recall several cases seen in Bellevue Hospital, the histories of which I have not been able to obtain, in which delirium and most of the other symptoms that I have mentioned prevailed, and the patients died, as it was supposed, of "subacute meningitis." At the autopsy small kidneys were found and more or less serum in the ventricles and subarachnoid spaces, but no signs of meningitis. The diagnosis in such cases must depend upon the signs of renal disease present, and upon the absence of certain causes, symptoms and signs of meningitis, and often the diagnosis cannot be made.

n. ASSISTANCE AFFORDED BY INTERCURRENT AFFECTIONS.

a. *Emphysema* occurs in a very considerable number of cases of chronic Bright's disease, and the discovery of this condition should have its weight in assisting in the diagnosis or in ex-

[1] L. C. Gray: American Journal of Medical Science, October, 1880.

citing one's attention to a more thorough examination of the case.

b. *Pneumonia* is very common. In Bamberger's statistics 22 per cent. of primary chronic Bright's disease were complicated with it. "It generally is of the croupous or lobar kind, and here again the elderly people whom it kills are often only found to be the subjects of granular kidney" (interstitial or chronic diffuse nephritis, contracted or cirrhotic kidney), "in the post-mortem theatre."[1] The occurrence of a pneumonia, then, in a patient over forty years of age, should be regarded with a suspicion of the presence of chronic Bright's disease.

c. *Cerebral hemorrhage.* Wilks and Moxon assert that in 75 per cent. of chronic Bright's disease this occurs. They also state that the hemorrhage in these cases is very great, and that it often occurs in persons who, up to the time of this accident, have been supposed to be in perfect health. Bamberger agrees with them, and also calls attention to the great size of the hemorrhages, and to the frequency with which the renal disease escapes observation during life.

d. *Chronic meningitis*[2] appears to occur very frequently with chronic Bright's disease when the latter has existed for some time, more often indeed than in connection with any other disease.

e. *Pericarditis.* When this is present unassociated with rheumatism, pneumonia, pleurisy, or the exanthemata, it is almost invariably associated with chronic inflammation of the kidneys.

f. *Peritonitis.* When this occurs without some local disease to account for it, without uterine or puerperal causes, or the exanthemata, or a pleurisy, it most likely, instead of being idiopathic, is due to chronic Bright's disease.[2]

g. *Pleurisy*, especially when double, is encountered most often in phthisis, tuberculosis, or chronic Bright's disease.

h. *Cirrhosis of the liver* is quite often accompanied by the small, atrophic, granular or cirrhotic kidney.

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[1] Bamberger, loc. cit.

[2] Delafield, loc. cit.

i. The presence of aortic and mitral regurgitation occurring singly or together without a previous history of rheumatism; or of the occurrence of any of the exanthemata, or of typhus or typhoid or severe malarial fevers; of pleurisy or pneumonia; of alcoholism or syphilis; or of physical strain—warrants the assumption that the lesions and the signs significant of them may have been produced by high tension continued for some time, and taken in connection with other signs which are likely to be present, the signs of valvular insufficiency may have their place in enabling us to arrive at the diagnosis of chronic Bright's disease when the indications of its presence are obscure. It should be also remembered in support of this proposition that aortic disease is most constant in those "turned forty" and upward; that mitral regurgitation, although common through life, is met most frequently in my experience after forty. Mitral stenosis is an infrequent lesion except where there exists a direct history of some one of the causes of valvular disease.

o. Age.—The age of the patient is of some aid in arriving at the diagnosis of chronic nephritis. According to Bamberger's statistics the small kidney is found most frequently between the ages of forty and seventy. Dickinson[1] states that "After thirty and toward the approach of forty it becomes more common, and at about fifty obtains its greatest mortality. It however is very destructive for the whole time between forty and sixty, after which, though productive of a much smaller number of deaths, it continues to number its victims to the extreme limits of life." A few cases are reported of having occurred early in life.

p. Heredity.—I think that there can be no question but that a heredity is present in many of the cases of Bright's disease, and I have been surprised, in inquiring into the histories of parents and grandparents of patients sufficiently intelligent, how often this occurs. Dickinson[2] gives details of an ancient house in England in which it was very evident through three genera-

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[1] Loc. cit., p. 376.

[2] Loc. cit., p. 378.

tions, and possibly in many more. Delafield[1] states: "There seems to be a disposition in certain families toward the development of the disease. I do not know that you can say that the disease is, strictly speaking, a hereditary one, but you will very often find in the same family a very considerable number of examples of it. In a family consisting of a number of brothers and sisters, if one suffers from the disease, you will very often find that other brothers and sisters of the same family suffer from it, although you can hardly say it is transmitted from father to son. The disease also seems to have a certain relationship to chronic pulmonary phthisis, that is, in some families in which phthisis is developed, other members of that family who do not suffer from phthisis will suffer from chronic diffuse nephritis."

Tyson[2] mentions a remarkable instance of hereditary transmission which had recently come under his notice, in which eleven members of one family were affected with chronic Bright's disease.

q. The residence of the patient in city or country.—Statistics force this upon one's attention. If any one should become familiar with the health of a district situated at a considerable distance from any large city, he will be astonished at the infrequency of chronic Bright's disease, which occurs frequently in cities. I have had opportunities for knowing quite well the health of a rural district not in the vicinity of any large city, and I know that very few deaths have occurred in that place from chronic kidney disease for the past ten years, and it has been a very exceptional thing to even see a case of chronic diffuse nephritis. The explanation of this freedom from chronic Bright's disease seems to me to be explained by the mode of life of the people.

I have now presented to you the means at our disposal for the early diagnosis of chronic Bright's disease, and, to recapitulate, they are as follows:

1. The subjective symptoms of chronic Bright's disease.

[1] Roberts' Notes on Lectures of Delafield, 1881, p. 292.

[2] Bright's Disease and Diabetes, p. 166. Philadelphia, 1881.

2. The objective signs of chronic Bright's disease.

3. The diseases appearing as complications or as sequences of chronic Bright's disease.

4. Heredity.

5. Age.

6. Life residence of the patient, whether urban or rural.

I shall now ask your attention to the reports of some six cases, from which I think some conclusions may be deduced that will enable the diagnosis of the most common form of chronic Bright's disease—the small, atrophic or granular kidney—to be made in its functional, forming, or initial stage. I shall not claim that the signs by which this diagnosis can be made are always to be found preceding every case of the atrophic or granular kidney, but I do believe that in the small kidney, so commonly encountered in cities, the conditions that I shall mention almost invariably precede the appearance of the symptoms and signs which are considered diagnostic of the small kidney.

The histories of the six cases I have referred to are as follows:

Case I.—J——, first seen by me in 1874. Heredity of gout and Bright's disease. Was fifty-four years of age when first seen. For three years monthly examinations of his urine were made. Until within the last year of his life the specific gravity of his urine was always high—1030 to 1035—and no albumen was at this period ever found. Abundant deposits of ordinary cayenne pepper crystals of uric acid were present. No casts and no sugar. In the last year of his life the specific gravity of the urine dropped to 1010 and continued at about this degree. Occasionally small quantities of albumen were present. At this time the uric acid crystals changed from the halbert shape to the cask-like shape. The amount of urine passed daily in the first two years of the time that he was under my care, when I could prevail upon him to save it, was rarely more than a quart. Twice in the last year of his life he passed at one time sixty-five ounces, and at another eighty ounces. During the entire period of three years there were

always present the signs of hypertrophy of the left ventricle and a pulse of high tension, and tortuous, thickened radial and temporal arteries. About eight months before death an aortic regurgitant murmur was discovered for the first time, but this was inconstant, though when heard very distinct. He died very suddenly one day, with symptoms of angina pectoris, before I could reach his bedside, although I was in the vicinity at the time of the attack. Acute dilatation of the heart was probably the cause of death. No autopsy.

Case II.—A dentist, aged forty-four years, came under observation in 1875. Much troubled then with dyspepsia and inability to lie on left side.[1] During a period of two and a half years his urine invariably was of a high specific gravity, sometimes 1035 to 1040, containing no sugar and no albumen, with abundant precipitate of uric acid crystals, form not mentioned in the notes. During the years 1878 and 1879 he complained often of considerable pain over the precordia with sense of suffocation. In the fall of 1879 had a very severe attack of bronchitis, and then for the first time albumen appeared in his urine with some few granular casts, but the specific gravity, although much lower, 1020–1025, did not fall to 1010–1015 until after the attack of bronchitis had terminated. He then passed large quantities of urine, at one time ninety-two ounces in the twenty-four hours. Examination in the spring of 1879 revealed the presence of hypertrophy of the left ventricle mitral regurgitation, and thickening of the walls of the radial and temporal arteries. High arterial tension was present throughout his case. Patient left town in spring of 1880, and died in a few months in coma. No autopsy.

Case III.—M——, came to me in the spring of 1876, with urine of high specific gravity, 1030–1035. The amount passed by him daily, at this period, averaged forty ounces. Uric acid cayenne pepper crystals, shape not recorded, were almost in-

[1] This has been an early symptom accompanying increased arterial tension. I have found it very frequently when there were no signs of any heart disease by physical examination. It seems to me to be connected with the beginning of hypertrophy or dilatation.

variably present. I was unable at any time to say that he had hypertrophy of the left ventricle. It was very difficult to obtain traces of his pulse, since the radials were very deeply situated. There was always present, however, high arterial tension, and the accompanying trace taken from the temporal artery very well shows that.

A peculiarity in this case was the presence of Cheyne-Stokes respiration for eighteen months before his death.

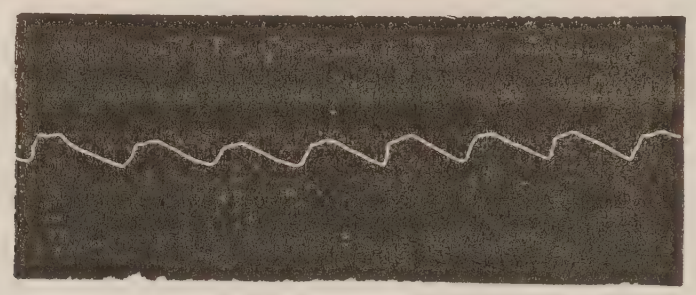

Fig. 15.—Trace from temporal artery of M——.

Some time during the year 1878 the urine assumed a lower specific gravity; contained a trace of albumen; occasionally granular casts, and the quantity of the urine increased to eighty ounces, and even more on some days. In December of 1879 this gentleman died of a very extensive cerebral hemorrhage.

At the autopsy the kidneys were small and very characteristic specimens of the granular kidney; hypertrophy of the left ventricle was found, the walls of that ventricle being an inch in thickness. Endarteritis chronica was extensively present, and the Cheyne-Stokes respiration of long duration was explained by the very atheromatous condition of the arteries supplying the medulla.

Case IV.—D——, aged thirty-five or thirty-six years, and has been under my observation for five years. He came to me in 1876 with symptoms of chronic gastric catarrh. At this time he was passing urine of high specific gravity, 1030 and upward, containing no sugar and no albumen, but an abundant deposit of uric acid crystals, and at times of oxalate of lime. He was very anæmic, and insisted upon it that he was suffering from malarial poisoning, which was not the case. The amount

of urine passed in this period of his case was never estimated. No heredity could be obtained. In 1878 I lost sight of him for three months, and when he returned I found that the urine presented a low specific gravity, never above 1018 and usually below 1015. Albumen was found at this time in the latter part of the day only, after meals. The quantity of urine passed was very much increased, and in the year 1879 he would often pass from eighty to one hundred ounces of urine in twenty-four hours. The radial and temporal arteries were thickened, and the blood tension high. I never have been able, however, to discover any hypertrophy of the left ventricle, and perhaps its absence may be explained to a certain degree by his profound anæmia. He is still under observation.

Case V.—L——, aged fifty-five years at time of death. Came under my observation in fall of 1879. Had led a very active life. His urine was always of high specific gravity, 1030 and upward. Contained uric acid crystals occasionally. No albumen. Frequent examinations of the urine were made during the spring and fall of 1880, and the urine always presented the same appearances as mentioned. No further examinations were made until in the spring of 1881, when he had an attack of gout. The urine then presented the same appearances mentioned. Soon after this, syncopal attacks appeared, which I discovered to be due to excessively high arterial tension. At this time albumen was first discovered in the urine; the specific gravity varied from 1010 to 1015, and a few granular casts were found. In the progress of this case from the spring of 1881 to the spring of 1882, time of death, the specific gravity of the urine varied much, sometimes as high as 1030, and again as low as 1010. The quantity of urine was sometimes much increased and sometimes diminished. The patient died in an attack of *angina pectoris.*

At the autopsy were found kidneys presenting the lesions of chronic diffuse nephritis in which contraction had not begun. The arteries, small and large, were generally atheromatous.

Case VI.—H—— has been under my observation since 1873.

Frequent examinations of his urine have been made in that time—at least one every two or three months. The urine has always been of a high specific gravity (1030 and upward), containing uric acid cayenne pepper crystals, but no albumen, no sugar, and no casts. In 1876 and 1877, when I began to make frequent use of the sphygmograph, I found that his pulse presented the signs of high tension. It was at this time that he began to complain of inability to lie on his left side at night. Frequent examinations showed no valvular disease of the heart. In 1878 I could appreciate hypertrophy of the left ventricle, and by the end of that year a mitral regurgitant murmur could be heard at times, not being always present, but when present could be heard behind as well as in front. In the spring of 1879, before his departure for Europe, albumen could be found at times in his urine, likewise granular casts. At the present time he presents many of the signs of chronic Bright's disease. The arterial tension is always high. There is present hypertrophy of the left ventricle, with a mitral murmur more or less constant. He passes large quantities of urine, as a rule, with low specific gravity, and with albumen at times, and occasionally a few granular casts. Sometimes the quantity of urine greatly diminishes, and the specific gravity rises to 1020–1030. He is anæmic, has subconjunctival œdema, flatulent dyspepsia, insomnia, etc., and in all probability has chronic Bright's disease—the small kidney. He is fifty-one years of age.

Besides the cases which I have related, I have at least six more with histories almost as complete as No. 6, now under observation, and in which, although the symptoms and signs usually considered as diagnostic of the small kidney have not, thus far, "come out" or declared themselves, yet I feel very positive that before long they will be found to be present. The points in the above cases to which I especially wish to direct your attention are as follows:

a. The persistently high specific gravity of the urine—not a high specific gravity of one specimen of urine, but of the whole amount passed, or of specimens taken at different times of the

day—which precedes the appearance of the low specific gravity so common to chronic Bright's disease. It will be found that it is extremely difficult to reduce this by any remedies.

b. The very frequent presence of the large crystals of uric acid, and more occasionally of crystals of oxalate of lime.[1]

c. The persistent presence of high arterial tension.

d. The early discovery of the beginning hypertrophy of the left ventricle.

e. The occurrence of the murmurs denoting valvular insufficiency, aortic and mitral regurgitation, which apparently are the result of the high arterial tension; the aortic regurgitation being due to an aortitis deformans (sometimes an aortic direct murmur occurs with this, and is due to the same cause), the mitral regurgitation being due to the dilatation of the left ventricle.

f. The early detection of endarteritis chronica. It appeared to me, after having studied the above conditions, that one might be allowed to infer that there was a forming or initial stage of chronic Bright's disease, and that one might predict the ultimate appearance of the signs and symptoms of chronic inflammation of the kidneys in patients presenting the conditions I have given, and that, if one would take into consideration the heredity, the age of the patient, and the mode of life of the patient, a presumptive diagnosis of a "functional," forming or initial stage of chronic Bright's disease might be made.

I was not aware, at the time that this thought occurred to me, that any one else had recognized or interpreted these signs in this way; indeed, when I began this essay I did not know that anything had been done by others in this direction. I there-

[1] Since Aufrecht has called attention to the significance of hyaline casts, I have looked over my book of urinary examinations and find mention made of the frequent appearance of hyaline casts in urines of high specific gravity, and in urines containing the crystals of uric acid and oxalate of lime. Unfortunately the test of acetic acid was not applied, but I think since the hyaline casts were almost invariably found, that they may prove to be of some assistance in diagnosis.

fore ask your attention to what has been written on this subject by others.

Dr. Murchison[1] after referring to his own opinion, quotes from Dr. George Johnson in support of his views: "Again, we find that functional derangement of the liver resulting in lithæmia, with dyspeptic symptoms such as those which I have described, is a common cause of the contracted, granular, or gouty kidney. Our colleague Dr. George Johnson, one of the greatest living authorities on diseases of the kidneys, thus writes respecting the causes of this form of Bright's disease: 'It is often associated with the gouty diathesis, as one of its synonyms indicates; and it is of common occurrence in persons who eat and drink to excess, or who, not being intemperate in food or drink, suffer from certain forms of dyspepsia, without the complication of gouty paroxysms.' And further on, in the lecture from which I have just quoted, he observes: 'Dyspepsia is frequently associated with this form of disease, sometimes as a cause, sometimes as a consequence. You may often learn that a patient of strictly temperate habits has for months or years suffered from pain or uneasiness after food, flatulent distention of the stomach or bowels, habitual looseness or irregularity of the bowels, constipation and diarrhœa alternately. With this, there is often turbidity of the urine, which is high-colored, excessively acid, and deposits urates abundantly. After a time, the urine, which had been scanty, becomes more copious, of pale color, of low specific gravity, and is found to contain albumen and granular casts. In such a case probably *renal degeneration is a consequence of the long-continued elimination of products of faulty digestion through the kidneys.* I have seen this sequence of events so frequently, that I have no doubt as to their causative relationship. Dyspeptic symptoms, such as I have described, and consequent renal degeneration, are in some cases excited or greatly aggravated by habitual excess of alcohol.'"[2]

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[1] Clinical Lectures on Diseases of the Liver, p. 573. Second Edition. New York, 1877.

[2] British Medical Journal, 1873, vol. i., pp. 161, 191.

Dr. Parkes[1] called attention to the fact that the antecedents of chronic Bright's disease are often those which arise from disturbances of the function of the stomach and liver, and that dyspeptic symptoms often appear before the renal.

Dr. Robert Saundby, a most excellent authority, in an article entitled "The Functional Stage of the Granular Kidney,"[2] writes: "Bright's disease, like other diseases, originates in a functional disturbance, but in an essentially chronic disturbance which takes half the lifetime in many cases to effect the structural changes which are capable of anatomical demonstration. This disturbance is primarily in the digestive organs, and results in the imperfect oxidation of nitrogenous material, a function which recent observations have shown to be chiefly located in the liver. This imperfectly oxidized material circulating in the blood has a directly stimulating effect upon the kidneys (Heidenhain), maintaining in them a constant state of functional hyperæmia, and, by increasing the general capillary resistance, raises the systemic blood-pressure so as to keep up a permanent strain on the organs of circulation. These conditions are necessarily aggravated if, as so often happens, the bowels are sluggish. Such a state of things is immediately characterized by dyspepsia, constipation, lassitude, headache, muscular pains, coldness of the extremities, tendency to pharyngeal or tracheal catarrh, and, later on, results in cardiac dilatation and hypertrophy, dyspnœa, chronic bronchitis, chronic gastric catarrh, arterio-capillary fibrosis, aortic valvulitis, interstitial nephritis: finally, coming death by rupture of small vessel or failure of the heart or kidneys, or an intercurrent attack of an inflammatory kind to which the tissues in such cases seem especially prone."

It is apropos of the remarks of Saundby to quote those of Bamberger[3] in regard to the causation of secondary Bright's disease: "The poisons manufactured in the system itself are probably more important factors in the production of chronic

[1] Med. Times and Gaz., April 22, 1854.

[2] Birmingham Medical Review, p. 377, April, 1880.

[3] Loc. cit.

Bright's disease than those external to it. They are the products of self-infection caused by the absorption of organic material destroyed by suppuration or gangrene, and of the lowly organized, rapidly proliferating bodies which cling closely to it. In this form we have scrofula, tuberculosis, and phthisis, caries and necrosis of bones, suppurative inflammation of the joints, skin, subcutaneous connective tissue, ulcerating cancers, and typhoid ulcers of the bowels, etc. More than a third of all of the secondary forms of chronic Bright's disease may probably be referred to it."

From the data presented in this communication I think that the following conclusions may be permitted:

1. That the diagnosis of chronic Bright's disease can be made with much certainty if a thorough examination be made of all the symptoms and signs presented; that a very considerable aid is afforded in the diagnosis by the condition of arterial tension and by certain conditions of the heart and blood-vessels, by the quantity of the urine passed and its specific gravity, and by the disturbances in nutrition and complications which may supervene.

2. That it is very probable that the diagnosis of the presence of chronic Bright's disease can be made when the ordinary well-known conditions of the urine common to chronic Bright's disease are not present or have not appeared, and when no evidences of œdema, anasarca, or uræmia can be found. In such cases the urine is of a high specific gravity, 1020 to 1036, contains abundant deposits persistently of oxalate of lime and uric acid crystals modified or not with hyaline casts, and much granular matter. Albumen may be entirely absent or only occasionally found. In these cases increased arterial tension is present with accentuation of the aortic second sound and perhaps beginning hypertrophy of the left ventricle, and not infrequently aortic murmurs or a mitral regurgitant may be heard. Associated invariably with these signs we find nasopharyngeal, bronchial, or gastric catarrh. Emphysema is frequently met with, and bronchitis, pneumonia, and cerebral hemorrhage are of very common occurrence. I have been much

surprised to find how frequently heredity exists, and this may assist somewhat in diagnosis.

3. That it would seem that we are justified in claiming that a diathesis or tendency to chronic Bright's disease may be made out. The evidences in support of such a claim are as follows: Heredity; the presence, as a rule, of urine of a higher specific gravity than the normal; the frequent presence of crystals of oxalate of lime and uric acid in the urine; frequent appearance of hyaline casts with granular matter; the occasional appearance of albumen in the urine for a few days; the presence of high arterial tension from time to time, and changes in the heart in consequence of the increased arterial tension; the occurrence of non-pharyngeal, bronchial, gastric, or intestinal catarrh, and of emphysema.

Observations to demonstrate a tendency to chronic Bright's disease must, of course, be carried on for a very considerable period of time.

ON SPONTANEOUS VERSION AND EVOLUTION IN SHOULDER AND ARM PRESENTATION, ETC.

By I. E. TAYLOR, M.D.

(Read April 7, 1881.)

Mr. President and Fellows of the New York Academy of Medicine:

The paper I propose to ask your attention to this evening was read before the Section of Obstetrics of the Academy, November 26, 1880. By a resolution of the Section I was requested to present it for your consideration. Accepting the compliment of the Section, I most cheerfully comply with their wishes.

The experience obtained from the cases I shall relate will assist to elucidate the object in view.

I offer first a few remarks on the spontaneous evolution and version the fœtus undergoes in shoulder and arm presentation, and the management of such cases without the sacrifice of the child.

The two principal objects I have in view are, *First:* To consider the *retractive* power of the uterus, and consequent *recession* of the child, as aiding or producing, whether in the uterus or in the pelvic cavity, the partial or complete version or evolution of the fœtus, and the natural delivery of the child.

Second: To suggest a method of treatment which will avoid mutilation of the infant, especially in those cases where the shoulder with the arm protruding is low down in the excavation of the pelvis, or external to it, when spontaneous evolution could not, under any circumstances, take place, and where traction on the arm or attempts at version could not succeed.

Case I.—*Generally contracted pelvis—Premature labor instituted at eight months—Cephalic presentation—Complete*

spontaneous evolution or version of the child—Natural delivery.—On February 15, 1880, I was requested to visit Mrs. S——, who was eight months pregnant; third pregnancy; aged thirty-five. Her first confinement terminated unfavorably; convulsions existed, and the child was sacrificed, version having been performed. The second pregnancy: premature labor was produced at six and three-quarter months; child lived only a few days. Acquainted with the defect of the pelvis, premature labor was induced at twelve o'clock. At half-past twelve labor had fairly commenced, and at five P.M. the os uteri was dilated fully three-fourths of its diameter. Membranes were intact, and the bag of water full and large. Membranes ruptured; a considerable discharge of water followed, and continued to flow for more than an hour. Head presenting, though previous to the commencement of labor it was lying on the left iliac fossa. The labor progressed till 8 P. M. As there had been no progress made since the rupture of the membranes, and the head was well adapted to the superior strait, and from the extreme desire of the parents to have a living child I decided to effect the delivery, or rather aid it by the forceps. My friend, Dr. Burchard, joined me and gave the anæsthetic. Forceps easily applied, and gentle traction made during a pain to engage the head in the superior strait. Believing that this object had been accomplished, the forceps were removed. As no advance was made in ten or fifteen minutes, they were reapplied. After the application, and before any effort at traction was made, an examination recognized the forearm of the child along side of the head. With this change I decided to produce version. After anointing my hand with the intention of introducing it into the vagina, while assisting external version with the other, I was surprised to feel no head within the reach of my finger. Dr. Burchard also recognized the fact, and immediately afterward a foot was touched, and the head was at the fundus of the uterus. The complete evolution, or *version in utero*, was almost instantaneous. In a few minutes the child was delivered living, requiring, however, prompt attention; weight, eight and one-half

pounds. There was no external manipulation by the hand, nor was the hand in the vagina. The evolution or version was quick and decided.

Case II.—*Contraction at the superior strait of one and three-fourths inch—Cæsarean section to be performed—Spontaneous version or evolution occurring while on the operating-table—Partial cephalic presentation—Craniotomy and cranioclasm.*—This was an Italian dwarf, twenty years of age, having a deformity of the pelvis in the superior strait of one and three-fourths inch. Patient in Bellevue Hospital. March, 1861, it had been decided by consultation with my colleagues that Cæsarean section should be performed. At 8 A. M. an examination revealed the head of the child partly presenting. Liquor amnii evacuated at 10 A. M., the hour for the operation. Having been examined by several of the gentlemen present, the head was found to be in the same position as at 8 A. M. When, just before proceeding with the incision of the abdomen, a vaginal examination was made, the leg of the child was found lying across the entrance of the superior strait. This was immediately seized and brought down, and the child was delivered by craniotomy and cranioclasm instead of the Cæsarean section. In this case the spontaneous evolution or version was accomplished imperceptibly, while preparing for the abdominal delivery.

Case III.—*Tedious labor—Cephalic presentation—Application of the forceps at the superior strait—By the retraction of the uterus the head of the child was freed from the forceps and spontaneous evolution or version was recognized as having taken place.*—In consultation with Dr. Oliver White, April, 1865. Patient aged thirty-six. Labor had existed for several hours. Head presentation, first position, and at the superior strait. Waters evacuated for fully ten hours. Pains appeared efficient though short. It was decided to overcome the difficulty, if possible, by the instruments. Application easy. The instruments appeared to be well and properly adjusted on the

head of the child. Slight traction was made for some minutes during the contraction. After a contraction the forceps were noticed as having lost their grip, and though retained in the hand and held gently without any compression upon them, they came away. I was much astonished at this circumstance, as I do not recall an instance, under similar circumstances, where the forceps had slipped after their application. In this instance they were clearly removed from their purchase by the retractive power of the uterus after a contraction. On examination, a foot and the breech were found to occupy the superior strait instead of the head. Shortly afterward the child was delivered living.

Case IV.—*Tedious labor—Arm protruding externally—shoulder in the cavity of the pelvis—Spontaneous evolution or version of the child—Child dead.*—During the spring of 1861, I was sent for to visit a patient at Bellevue Hospital with shoulder presentation, arm external. On seeing the patient I recognized the arm protruding and the shoulder as filling the cavity of the pelvis and within a short distance of the perineum. This condition of affairs had existed for over an hour. As the shoulder was not external, I decided to try and accomplish the delivery of the child, if possible, while under an anæsthetic, by version. My house physician was requested to notify the house staff to be present. On examination, just as he was going to invite the staff, I found a partial evolution or version taking place, and requested him to remain. In a few minutes the breech was found presenting, and during a slight contraction was engaged in the pelvis. In a few moments more the child was delivered still-born. Its weight was about six and one-half pounds.

It might seem almost a useless task to enter upon or even suggest a different explanation than that which has been rendered of these rare and important cases of spontaneous evolution of the fœtus where the shoulder is in the pelvic cavity, arm protruding, or external to it.

It is more than a century since Denman first called the attention of the profession to a "new order of events" in cases

of this nature, and he gave the name of spontaneous evolution to the important change which the fœtal ovoid made from being a shoulder presentation, arm external, the shoulder either jutting into the superior strait, or in the pelvic cavity, and even pressing or resting low down on the perineum. The title which Denman gave to the class of cases that he witnessed and the rationale he gave was called in question by Douglas, of Dublin, in 1810 (though the fact had long been known and acknowledged before Dr. Douglas and others denied that such a method of delivery as Denman advanced ever existed), who claimed it was at variance with the true mechanism of these cases. At the present day there still exists a difference of opinion respecting the meaning of the expression of spontaneous evolution, and its proper or correct application to either Denman's or Douglas' rationale of what these individually saw of the cases under their charge.

Dr. Douglas, in his celebrated essay "On the Real Process of Spontaneous Evolution of the Fœtus," published in 1811—forty years after Denman's first recorded cases in 1772—and republished in 1844, created after so long an interval of time considerable sensation and discussion, and changed materially the views and opinions which Denman had given to the medical profession. It was then generally believed that the explanation of Douglas was correct, and that he had shown and proved that the views of Denman were wrong. Douglas considered that Denman's explication was not only wrong, but "purely theoretical and hypothetical," and frankly admitted that he "could not comprehend the solution of Denman's explanation, and that if he believed in them he should despair of such an event taking place as a spontaneous evolution."

Since the published essay of Douglas, a more extensive experience and close study of these cases has demonstrated that Douglas, as well as others accepting his explanation, had not understood as clearly as they should have done the views of Denman respecting the description of the cases he saw and considered in comparison with the cases which Douglas and others had witnessed.

To appreciate the great difference of opinion respecting the class of cases referred to by these authorities, and to estimate properly the explanations which they have rendered, it may be well to present an illustration from each of them, as showing the rationale of their cases. I select the first case of Denman in 1772: "The arm of the child was presenting. The two physicians in attendance attempted to turn, and extract by the feet, but the pains were so strong as to prevent the introduction of the hand into the uterus. I found the arm much swollen and pushed through the external parts in such a manner that the shoulder merely reached the perineum. The woman struggled violently with the pains, and during their continuance I perceived the shoulder of the child to descend. Concluding that the child was small and would pass doubled through the pelvis, I desired one of the gentlemen to sit down and receive it. I remained, however, at the bedside till the child was expelled, and I was very much surprised to find that the breech and inferior extremities were expelled first, and before the head, as if the case had been originally a presentation of the inferior extremities."

In this case of Denman's, it is evident that the shoulder of the child was external to the uterus and inside the pelvis, and resting on the perineum internally. In explanation of this occurrence, Denman says: "The body does not come doubled, but the breech is the part first delivered, and the head last."

In selecting the expression or title of "spontaneous evolution" to explain his meaning of the *act*, or change of position of the child, Denman says: "To some it appeared objectionable, but I could not fix upon one better to explain my meaning. I only intended to say that the 'series of events' terminating in an evolution of the child, were wholly independent of the practitioner, and not that this was produced from any impulse or exertion of the body moved." It was this change of position of the child, as the shoulder was in or occupying the pelvic cavity, and not external or outside of the pelvic cavity, arm protruding, and the child delivered by the breech first, that Douglas endeavored to controvert, and to prove that it was er-

roneous, judging from the cases which he himself had witnessed. Of the seven cases recorded by Douglas, there is no complete relation like Denman's by which we have a clear understanding of them, and we have to rest solely on his rationale or explication.

As an instance, "An arm protruded, the evolution took place while I was deliberating on the employment of means to moderate the violent action of the uterus before I proceeded to turn."

Case 3, "Arm protruding. In about two hours, and five hours only from the commencement of the labor, the evolution was complete."

Case 4 is the most important of all his cases, in 1814. This was a twin case. The second twin, "arm protruded. On examination I found so much of the chest of the child engaged in the pelvis, and likewise the action of the uterus so powerful that I made no attempt to turn. The evolution was effected in one hour." With this experience, Douglas says "he could not comprehend how successive repetitions of the same propelling power, in contractions of the uterus, which forced the child in this situation, should subsequently at any period produce a counter-effect, causing the shoulder to retreat or recede into the uterus." "The fact is, the shoulder and thorax thus low and impacted, instead of receding into the uterus, are at each successive pain forced still lower, until the ribs of that side corresponding with the protruding arm press on the perineum and cause it to assume the same form as it would by the pressure of the forehead in a natural labor. At this period not only the entire arm, but the shoulder can be perceived externally, as the clavicle is laying under the arch of the pubes. By further uterine contraction the ribs are forced more forward, appearing at the *os* externum as the vertex would in a natural labor." It is evident Douglas did not believe that recession of the child could take place in these kind of cases. Nor do I see how he could, and I must therefore coincide with him; as by the continuous and successive contractions of the uterus there was no time allotted the child to

recede, either by its own movement or by the relaxation of the uterus.

Denman, in response to the rationale, or explication of the cases which Douglas saw, in his letter, October 10, 1814, remarks:

"In your practice I presume you have met with many instances of it, but it may be supposed that one case would not be sufficient authority for forming an axiom, and determining the general question. The fact is a distinct question; the manner of the evolution is another. For the former I am not answerable, but I certainly have remained responsible for the explanation of the manner. If there be an error in the explanation, others may also err in their opinions."

It is clear that the views and opinions of Douglas had not changed Denman's opinions on the subject, but appeared rather to establish them more firmly in his mind.

In the first published statement of Denman's, 1772, on the subject of cases of shoulder presentation, and the child coming doubled similar to Douglas, he remarks: "He has seen premature and small children often expelled in a doubled-up state, whatever might be the original presentation, when the pelvis was well formed, but that case is a different one to that we are discussing."

From Denman's remarks, and his reasons for the term adopted to express his understanding of the process of delivery, he evidently believed in two entirely different classes of cases—one delivered by a spontaneous or natural evolution, the other delivered naturally by expulsion, though doubled up. Douglas believed only in one method of evolution. No one, I presume, will undertake to gainsay the truthfulness which Douglas gave of the cases which he himself saw. His description has not been more clearly given, though others have attempted to do so. Nor, on the other hand, can the fidelity of the description of the cases which Denman witnessed be called in question. Douglas, nevertheless, informs us "that no practitioner will again have the opportunity of witnessing a process such as Denman has described." By this

remark of Douglas it must be apparent there can be no spontaneous evolution if the child's shoulder is in the cavity of the pelvis, and here is the issue I shall endeavor to controvert, both by cases of eminent authorities and my own.

By some of the Continental obstetricians the title which Denman gave to his cases, though differing so materially from Douglas, which Douglas yet accepted for the explanation of his own, is still applied to such cases as Douglas recognized, and are spoken of as cases of spontaneous evolution. From the explanation which Denman gave of his cases, shoulder inside of the pelvis, he applied the name of spontaneous evolution; but, he says, he was in doubt about the correctness of the title. Douglas himself had no doubt, for he says, "I wish to retain the appellation of spontaneous evolution, both because the fact is known by the form, and because it appears to me more suitable than that of expulsion."

It is somewhat singular that Murphy, Leischman, Hodge, and others consider that Douglas spoke of his cases as cases of spontaneous expulsion, while he decidedly disclaims it, but says that the fact is known by the form, Evolution.

Velpeau, writing as late as 1852 on this subject, informs us that the nature and mechanism of spontaneous evolution are still badly understood. Chailly, on the same subject about the same time, asserts that evolution is better understood than spontaneous version.

Different interpretations have been given to the word evolution respecting the manner or method, how the child is delivered in these cases. Little agreement exists also, simple as it may appear, as to the proper application of the word.

Velpeau considered Douglas' cases as "a simple unfolding of the body of the child past the presenting part." Playfair "that the fœtus is expelled by long squeezing through the pelvis without the original part being withdrawn." Radford, "that they are cases of doubling up and expulsion of the fœtus." He does not accept Denman's meaning or description of an evolution of the child when in the pelvic cavity.

C. Clay wrote a long article on the subject, as editor of the

Obstetric Record, Great Britain, Vol. I., announcing the singular opinion, "that no such circumstance as an evolution ever took place either in the pelvis or uterus, except when the accoucheur turns the child by his own hand. In such a case the body of the child performs an evolution; with such an exception, evolution never occurs, the strict meaning of such a term being a turning round of the fœtus. The term evolution is incorrect." Clay was a strong advocate of Douglas' view. Barnes applies the term to both Denman's and Douglas' cases, but he believes that Denman's cases were cases of partial version, and if so they were not cases of expulsion as Douglas' were. He does not accord to Douglas any credit. Palmer's "Medical Pentaglott Dictionary" defines evolution, in obstetrics, as simply "a movement supposed to be performed by the human fœtus a short time previous to its expulsion. It has no relation to expulsion. It also means a gliding or sliding upward." We all know by our own experience that expulsion means a driving out, a delivery of the fœtus through the vulva, by the contractions of the uterus, without a change of its original position, "line after line," requiring considerable force and sometimes violent action to accomplish it—in this class of cases of shoulder presentation, Douglas calls it, "a prodigious, continuous action." Douglas and others consider the fœtus as a passive body and contributing in no degree to its own liberation." Self-evolution of the fœtus in utero we now recognize, even with the negation of Douglas and others, whether cephalic or shoulder presentations, as a frequent occurrence of nature before and after labor has been instituted. There is nothing strange or extraordinary or singular in the evolution of the fœtus in the uterus, complete or partial. It is a natural version, and it is as natural a phenomenon as spontaneous expulsion is a natural delivery of the child during strong contractions of the uterus. Spontaneous evolution of shoulder presentation, the arm being external, in the pelvis, or even resting low down on the perineum, are simply only partial versions of the child in that position.

Self-evolution or version of the child may occur at any time

after the sixth month, and when the accoucheur is present at the bedside during the labor.

Cephalic has become breech, and breech has become cephalic, and this may occur several times during gestation. Schroeder has found such changes as 6 out of 214—1 to 36. Tredinat as often as 5 out of 60—1 to 12.

The fact of the evolution or version is one question, and, as Denman says, the manner is another.

There is not one of us who has not noticed, when examining females for the purpose of clinical information and instruction, how easily extreme version can be performed if the child is living. Sometimes it is done by only a few touches of the fingers on both sides of the abdomen, and even after the evacuation of the waters.

Chailly, as well as Clay and others, deny that the fœtus in utero can undergo an evolution or version from its original position after the loss of the waters. The cases I have adduced attest the incorrectness of that view, as well as the testimony of others (see Cases Nos. I. and II.). Spontaneous or natural version is generally believed to take place when there is a large quantity of water, where the fœtus is not large, where the uterus is more oblique, and where the pelvis is badly formed.

Two methods have been recognized as explaining the fact of the evolution in the delivery of the child in the kind of cases we are considering, viz.: general and irregular contraction; and to which I shall presume to add a third, viz.: retraction of the uterus, recession following, then evolution. The first is that which Denman gave, and which was approved by Velpeau and adopted by others as explaining "how" the evolution may take place. Denman's explanation is that "by the long-continued action of the uterus the body of the child is brought into such a compact state as to receive the full force of each returning action. The body being too large in its doubled up state to pass through the pelvis, the uterus pressing upon its inferior extremities, which are the only parts capable of being moved, they are forced gradually lower, making room as they are pressed down for the re-

ception of some other part into the cavity of the uterus, the body turning on its own axis, the breech of the child is expelled as in an original presentation of that part." Velpeau, respecting uterine contraction producing the evolution, or rather partial version, remarks, "when the womb contracts, if the fœtal ovoid is well placed, it becomes compressed equally in every direction; but if, on the contrary, it is in a deviated position its extremities suffer almost alone the whole effort of the contraction, and either the head or the breech will be brought to the superior strait, or into the pelvis." Caseaux suggested a different explanation, and believed that the irregularity of the contraction of the uterus is not wholly foreign to such an effort as spontaneous evolution or version. It is what the Germans have described as partial contraction of the womb, the organ appears to contract over a limited part of its extent, the remainder remaining entirely inert. In other words, the uterus contracts on one side and is relaxed on the other.

Caseaux is credited with this view of the method, but he candidly admits that on his part it is purely theoretical, and that he has never witnessed an instance of that kind.

I fail to grasp the exact meaning of the explanation, as it is difficult to conceive how one-half of the longitudinal muscular fibres of the uterus can be in action, as they are the chief motive power of the uterus, while the other half is in a state of rest.

From the anatomical arrangement of the muscular structure of the uterus it is easy to understand when the circular fibres are contracted, the longitudinal may be at rest, and *vice versa*, although I do not and cannot deny it, for I have never witnessed it. I conceive, however, that an entirely different explanation may be offered of how the evolution or version may be or is effected sometimes, and as a case or two have come recently to my attention as bearing on the subject, I shall refer to them. They may be instructive, as elucidating one of the methods under consideration, although in both cases the shoulder of the child was at the superior strait.

The following cases will illustrate the opinion of Caseaux

respecting the unequal contractions of the uterus producing the evolution after the evacuation of the waters. Very few of us may have seen an instance of the kind:

A woman, aged twenty-eight; fourth pregnancy; full-sized pelvis; at full time; labor commenced by slight pains. By noon next day the pains had assumed an expulsive character; membranes ruptured in the presence of a midwife, who detected an abnormal presentation. Dr. Genuiel, on being summoned, found the left arm swollen and hanging from the vulva; fœtal head in the right iliac fossa; os tightly contracted around the shoulders. The hand could not be introduced into the uterus. Decided not to attempt version at that time, and resolved to attempt embryotomy. At the end of half an hour, having made his preparations, he had his hand on the uterus, and was astonished to find that on the left side there were strong contractions, while upon the right there were none. Thinking, therefore, as the pelvis was large, spontaneous version or evolution might be accomplished, he left the case to nature. By 4 P.M., four hours after, the shoulder began to recede, and by 4.30 P.M. the breech was presenting. The contraction then became uniform, increased in vigor, and at ten minutes to five o'clock a dead child was expelled. The author believes that the powerful contractions on the left side of the uterus forced the breech down, while the relaxation on the right side allowed the head lying in the right iliac fossa to recede.[1]

M. Tredinat (*Lyons Medicale*, December 7, 1879): Primipara. Pains began in the evening; next morning at 2 A.M., membranes ruptured; at 8 A.M., admitted into the Maternity. Examined and found shoulder presentation, back anterior; the os as big as a florin. Preparations made for version. While waiting, a powerful uterine contraction affected the left half of uterus, right remaining flaccid. Head felt through the parietes, and slightly raised. Another similar contraction, but passing more to the right, and with this the upward move-

[1] Annales de Gynecologie, June, 1876.

ment was more pronounced, and on examination the axilla was recognized; in six or seven minutes more, contractions continuing, the breech presented. One hour after, child delivered living. It was noticed that most of the uterine contractions began at the left and passed to the right side of the uterus.

No two classes of obstetrical cases bearing the same title and yet so widely different as to the position of the shoulder, one in the pelvic cavity and the other external to it, and the manner and method of delivery, as those of Denman and Douglas could exist. They are spoken and written of as though they were the same, and the same order of events transpiring. The differential diagnosis is very marked and distinct between them.

In Denman's cases the shoulder, with arm protruding, was in the pelvic cavity, and in some cases so low as to press upon the perineum.

In Douglas', the shoulder, with arm protruding, was without, or external to the pelvic cavity, and so far external as to be found under the symphysis pubes, and the chest on the perineum.

In Denman's, there was a distinct change of position of the child, the shoulder receding from the pelvic cavity and the breech descending.

In Douglas', the shoulder being external to the pelvis, remained, and did not change its original position till its delivery.

In Denman's, the breech was expelled first, with the feet, next the abdomen, then the chest, and finally both shoulders, the head following.

In Douglas', the shoulder and arm, then the chest and ribs, next the abdomen, then the breech; after the delivery of the breech, then the other shoulder, and finally the head.

In Denman's, while the breech was external the shoulders were at the superior strait.

In Douglas', the breech was at the superior strait while the shoulder was external and under the pubes.

This comparison demonstrates the great contrast which exists between these two classes of obstetric cases.

In Denman's case, spontaneous evolution or partial version could be accomplished naturally, as well as artificially sometimes. With the class described by Douglas, no natural or physical power could possibly affect or alter the position, and the child must be delivered by strong, powerful, and continuous action of the uterus, obeying the laws of natural delivery, doubled up as the fœtus is, and going through the same process or mechanism of delivery, as flexion, descent, rotation, and expulsion.

In truth, the class of cases which Douglas saw were the natural delivery of shoulder presentation. Should the delivery not be accomplished, the child must be sacrificed.

If the natural delivery of a shoulder presentation, shoulder and arm external, is to be called a spontaneous evolution of the fœtus, or an unfolding of the fœtus without any change of position of the child from its original position, then every case of breech presentation with the breech external going through the various changes of the mechanism of delivery might justly be called spontaneous evolution.

I refer to a case taken from the *Obstetrical Record*, of Great Britain, Vol. I. It was cited by Clay as establishing Douglas' view, while, on the contrary, it bears no relation to that class, as the shoulder was in the pelvic cavity. It illustrates more clearly the views of retraction and recession. It is these kind of cases which are spoken of or published as similar to Douglas', but they can claim no relation to them whatever. Dr. Winterbottom's: "By a vaginal examination, the arm presenting externally, and on passing the finger, I felt what was considered to be the shoulder. I decided to turn; on passing my hand to change the position of the child, I recognized that part which I had taken for the shoulder to be the breech obliquely placed and which had partially entered the pelvis. A strong contraction occurred which pushed down the breech, during which event the arm had receded into the uterus; in a few minutes the child was expelled."

A more important case, however, is that which Barlow, the celebrated surgeon, writing as far back as 1792 on this subject,

recites, which is so clear and explicit on the retractive power of the uterus and recession that I will quote his case:

"Patient at full term of gestation, thirty-six hours after labor had commenced. On examination I found the arm of the fœtus presenting, and the hand pushed out of the os externum and very much swollen; the waters evacuated early; pain strong and frequent. I made an attempt to introduce my hand, but failed. A second attempt, but failed again. The action of the uterus was such as to forbid any further attempts. The shoulder of the child now became forcibly pressed upon the perineum. The woman continued in this misery till the following evening, when the pains of the uterus were so great that I dreaded a rupture of the uterus. On examination I was agreeably surprised to find the presenting arm of the child retreating, and the breech supplied the place of the shoulder, and was expelled in a few minutes by the efforts of the uterus."

This case of Barlow's is identical with those of Denman and my own—by the change of position or evolution, "the child turning on its own axis."

It is conceded that Schœnider anticipated Denman in the recognition of the fact, but he gave no explanation of the evolution.

Preceding all authorities on this subject, we have the two most remarkable, historical, and memorable events of this nature, which are recorded in the Book of Genesis, chapters xxv. and xxxviii. The first recorded case of twins was that of Rebecca and her sons Esau and Jacob. The second that of Tamar and her children Zarah and Pharez. In Rebecca's case, "when Esau came out first, red all over with hair as a garment, his brother Jacob followed, and with his hand took hold of Esau's heel, and came out." In Tamar's case, "when Zarah came forth the midwife bound a scarlet thread around his arm, saying, 'Thou camest forth first,' and afterward it came to pass that he drew back his arm, and Pharez came out, when the midwife said, 'How hast thou broke forth? the breach be upon thee.'"

Both these scriptural instances not only verify the division

referred to, but they illustrate the difference existing between spontaneous evolution, recession and retraction, and spontaneous expulsion solely. The fourth case of Douglas, as related of twins, applies with some force to the similarity of the manner of the birth of Jacob, and the quickness of delivery by expulsion alone. I know of but one authority, Dr. Kelly, of Manchester, Eng., who referred these cases, especially Denman's, for an explanation of the manner by which the evolution was produced, to the relaxation of the uterus. He makes no reference to the retractive power of the uterus as the agent by which the recession of the fœtus from its original position is produced by turning on its own axis. Kelly was the only one during the heated discussion which agitated the profession at that time who dissented from the general opinion that the evolution was produced by contraction of the uterus in these cases similar to Denman's.

I now reach the important question in cases of this nature, and I will let Barlow put the simple question: "How the recession of the fœtus in utero or pelvis was affected in his case;" and he answers, "I will not attempt the explanation, but I recognize the fact that nature has accomplished it." One of the objects of my paper has been and is the solution from the experience I have, and the cases adduced in verification of the fact and opinions entertained. Burns says: "That it is an impossibility for the shoulder to mount up into the contracting uterus, even if it is in the pelvic cavity partially or wholly; and if this is impossible, how much more so could it be when the shoulder is protruded externally." Every one of us must coincide with this view, that no change of position or evolution of the fœtus could take place *during* the contraction, and if *relaxation* does not follow, and the pains are continuous, or with very slight intervals, the child must be delivered as in natural labor, whether in the pelvic cavity or not. Should relaxation take place, however, as in ordinary labor at longer intervals, then the natural retraction of the uterus occurring, the shoulder of the fœtus will be, or is changed from its fixation or location, and a partial evolution or version can take place.

Contraction following, the part of the child which is the highest is forced lower down, while the other will ascend after the contraction ceases during a relaxation. The retraction may be so strong and great and effective, as in the cases I have related, that the fœtus is immediately turned on its own axis, and a complete evolution or version may be made. The recession or evolution of the fœtus is assisted by the child's own movements after the retraction. Retraction is as natural an act of the uterus as contraction is to deliver the child. It is not, then, a miraculous act or an "impossibility" for cases of shoulder presentation, arm external and as low as the perineum, to change as here described.

All of us who have given any attention to the subject of ordinary cases of labor know how well and perfectly Nature performs her work in some cases of cephalic presentation. The head may be low, and even resting on the perineum, and after contraction relaxation follows; retraction occurs and the head is lifted up or recedes nearly or to the superior strait, and when contraction ensues it is driven down to the position it formerly occupied, and still farther, and by this process of retraction and contraction, it becomes arrested and is finally expelled. To such an extent, as F. Ramsbottom says, is this recession sometimes carried, "it may give those not well acquainted with the process an idea that the child's body has passed partly into the abdominal cavity." We need not, therefore, be astonished at the recession of the child when a shoulder presentation exists, arm external, when in the pelvis, to realize a change of position of the child by version or evolution any more than expulsion of it by contraction. Recession alone cannot take place without the motive power of retraction of the uterus. Recession is the respondent or the sequence of retraction. Expulsion is the respondent or sequence of contraction of the uterus. Retraction effects or produces the version or evolution of the child in those cases within the pelvis; contraction, the direct expulsion or natural delivery of the child without change of position.

It is impossible not to admire the beneficent wisdom and

power which nature adopts and adapts, even if it is one of the minutest character, to the exigencies of gestation and labor.

Treatment.—It is considered by many authors, as well as by some of the profession, that the views and opinions of Denman and Douglas respecting the treatment in their cases tend to produce a passive conduct on the part of the practitioner and that experience does not warrant such a course. This remark would more appropriately apply to Denman, but not to Douglas. Should the distinction I have given be admitted, and I do not see how it could not, under the illustration presented—then there are two distinct classes of shoulder presentations, arm protruding: one with the shoulder in the pelvic cavity and even as low as the perineum, the other with the shoulder external to os externum and the arm under the pubic arch.

Accepting this distinction, a different line of treatment must be adopted for each. With the first, version may be attempted in that manner which may be considered advisable with a prospect of success. With the second, shoulder external, version cannot be entertained. It is totally impracticable, and if attempted would place the patient in greater jeopardy. Douglas discountenanced it, as it could not be attempted in the kind of cases he witnessed.

Denman, in reference to the first class of cases, uses this language, "It is possible, when we have conducted ourselves in the expectation that the evolution could be made, that the pains may fall off, or be unequal to the effort, and we may be disappointed. It might then be apprehended that the difficulty would be increased. But though the evolution was not perfected, I have not found that consequence. The child not expelled has been brought into such a state that afterward I could pass my hand with ease, and bring down the feet, though in an attempt to do this in the beginning of the case, I had failed." If we have from the nature of the case decided not to wait, then there is nothing left but to resort to the destruction of the child, either by amputation of the arm or shoulder, or both, hoping then that evolution may ensue, or by evisceration or de-

capitation. A few instances have been related where the child was born living after amputation of the arm. As a general rule the child is dead, and any of these operations may be proceeded with at once when that fact is recognized. Delay must still be exercised if the child is living, though the labor may have existed for several hours; and more especially at this day than formerly, as there is such a great abhorrence of embryotomy. Dewees has said we must keep no truce with the child. The first of all the operations which naturally suggests itself is amputation of the protruded arm, either to facilitate the version if necessary, or to have a natural evolution occur, or to eviscerate the thorax. Pénard, of Rochefort, 1879, adopts it, and considers it a substitute for embryotomy. It is, however, condemned by others.

Pinard, of Paris, 1879, agreeing with this view, considers, and very naturally, that such a proceeding should never be thought of while there is the least spark of life. In cases where the child is dead it has been unjustly condemned.

In the cases where the shoulder is external, and the acromion fixed against the lower border of the symphysis (Douglas class of cases), the arm should be amputated, and sometimes the projection of the scapula destroyed, when the trunk will readily recede and the lower extremity come down. "This is, then, virtually evolution or version, and could only be produced by retraction of uterus, although the shoulder was external after the obstruction had been removed. Pajot and Stoltz perform version in cases in which it was previously thought impossible after simply amputating the arm. They were, however, cases only partially engaged in the pelvic cavity. In the *Annales de Gynecologie*, December, 1879, *Obstetrical Journal*, Great Britain, 1880, Hodge acknowledges he had never seen a case, and with many others, recommends to remove the arm and shoulder, including the whole scapula and even the clavicle. The thorax should then be opened, and if necessary the side of the abdomen, the crotchet then passed into the body and fixed on the pelvis of the infant, and traction made from the breech downward.

Dr. Alexander H. Stevens, of this city, as far back as 1819, as Dr. Francis in his comments on the case says, "adopted a judicious practice and suggests a similar course."

Dr. Stevens first removed the protruded arm, then opened the chest, removed the contents and tried to bring down the body with the blunt hook, but ineffectually. Then he made an incision along the spine high up, down to the hips, introduced a blunt hook, and extracted the child, breech foremost. The delivery occupied two hours.

It will be noticed from the opinions cited, and the treatment advised and executed, that these severe, destructive, and laborious operations have been principally and chiefly on the upper half of the body of the child.

It has been remarked before that it was almost an impossibility to return the shoulder of the child when external to the pelvic cavity, and it appears equally as difficult to effect the delivery of the child when the arm has been amputated and evisceration performed.

Douglas never performed any of these operations, for all his cases were delivered naturally. He enters his decided protest against the doctrine, as recommended by some, of fixing the instrument at random on any part of the fœtus which happens to be the nearest; because if the physician fix it on any of the superior ribs, he might pull with Herculean force, and yet not be able to bring down the body, unless he separate the head or decapitate it. In all those cases reported as delivered naturally nothing is said respecting the rupture of the perineum, which it is admitted is excessively distended by the lower part of the child's back, and it seems almost impossible it could escape laceration. It is clear to my mind that the principle which has been enunciated, that the operation should be commenced on the upper half of the body of the child, is incorrect, and that it has not been properly understood. The chief object, and in fact the only one, has been to lessen the size of the child, in the hope that it could be easily delivered. The evidence is decidedly opposed to such a principle.

The great difficulty which does exist, after one-half of the

upper or superior part of the child is delivered externally, is from the lower end of the scapula, or middle of the spine, to the pelvis or the breech, which is distending the perineum so extensively, to the extent of fully four to five inches.

Burns says, although he had not seen a case, that if we consider that a line drawn from the side of the neck to the end of the thorax, when the shoulder is at the vulva, is $4\frac{1}{2}$ inches, the difficulty must be, and is, to get down the body of the child from the end or lower part of the thorax to the breech. This, then, is the greatest length of the substance which is to pass or to be born.

The non-delivery of the lower portion of the body of the child, therefore, has necessitated the various methods for destroying the child.

To overcome, and avoid this great destruction of the child, I advise the division of the perineum to the extent of $3\frac{1}{2}$ to 4 inches laterally, when the child will be delivered very soon without sacrificing it. Another important advantage is that if the child is recognized as being alive, and the shoulder just jutting from the vulva, the delivery may be prompt and successful. Surgical attention for the restoration of the perineum, can be given to the mother as soon as convenient after the birth of the child. This course of practice I have before recommended in face cases, chin posteriorly, while the face was in the pelvic cavity, to avoid the sacrificial act in that class of cases ("Journal New York Medical Association," 1869). I will illustrate my suggestion by the recital of a case of doubled fœtus, united at the thorax and abdomen, which occurred under my notice, May, 1860. The autopsy on the children was made by my friend, Professor J. W. Gouley.

I refer to this case, as it was the lower part of the body of the child, which it was utterly impossible to deliver unless it was mutilated. It presented a similar external appearance of the back of the child as in the shoulder cases we have been considering. Before I relate my case, I shall offer one similar of shoulder presentation, where both of the shoulders were ex-

ternal, instead of one, the head of the child resting against the internal pubic arch.

It is an observation of Peu, and which Nannoni in his work on "Delivery," 1785, says "modern accoucheurs do not appear to have understood when they attempt to turn into ridicule the operation of this author." Both arms, together with the shoulders, reached the vulva; violent traction alone brought down the fore part of the neck, head remaining internal. A fillet was passed over the back near the breech. Traction was made, and the breech descended and the woman recovered. Traction, as Nannoni says, here took the place of the uterine effort, and the child was born as in "spontaneous evolution." De la Motte also illustrates this point respecting the lower part of the child being the first to be delivered, instead of the upper.

In his case, "both of the arms and the shoulders were external, head internal, the two arms and the thorax advanced toward the vulva. The practitioner made unusual efforts to bring down the feet; the limbs were, however, fractured, and after strong efforts by De la Motte, the breech descended first and the child was born."

In May, 1860, I was requested to visit Mrs. E——, whom I was engaged to attend in her confinement. She was exceedingly large in form; extremities very œdematous. I saw her at 7 A.M., in consequence of the evacuation of an immense discharge of water. On examination, the os uteri was found fully dilated, cephalic presentation, and at the entrance of the superior strait. Pains every ten minutes. 9 A.M.: No advance of the head. Presuming that there was some special cause which I was not conversant with, I decided to apply the forceps to aid the delivery. Auscultation recognized a strong fœtal pulsation; palpation and manipulation, a large child. My friend, Dr. I. O. Stone, was requested to join me, and give the anæsthetic. At 10 A.M., no change; forceps applied; contractions more active; head brought in half an hour as low as the perineum. By strong traction the head was delivered—child dead. A towel was placed around the neck, and with considerable force the shoulders were delivered and the arms.

It appeared impossible to deliver the child any farther. The hand could not be introduced into the vagina. The head, shoulders, and arms were now external, the back of the child from the middle of the dorsal region was distending the perineum enormously. Not knowing the cause which prevented it, the question respecting evisceration was entertained to accomplish the delivery. I decided, however, to divide the perineum laterally for over three inches, and if the child could not be delivered, then evisceration. The division was made during a pain, and almost immediately the breech came down; with a finger in the anus the child was tilted out. As soon as the breech of the child was delivered, the feet of a second presented; seizing them, this child was delivered in a few moments. There was but one cord. The children weighed fifteen and one-half pounds. If you will look at the drawing I present to you, it will be easy to comprehend the cause of the difficulty in the delivery. While the head of the child was at the superior strait, and on the pubes internally, the other was external, in front of the pubes. As the whole back of the child presented externally, the same as in the double shoulder cases I have cited, it had to be delivered in the same manner, breech first. The principal obstruction, or barrier, therefore, which prevents the child being delivered, in these cases especially, where the shoulder and arm are external, is the greatly distended perineum, and when this part is set free, the principle I have advocated, as to the lower part of the body of the child instead of the upper, will, I believe, be verified as true and successful.

By this simple operation in these cases, we may avert the sacrificial act of the child, spare the mother, and avoid the unpleasant and distasteful operation; and when performed early, according to the nature of the case, we may have in some instances the gratification of delivering a living infant. Denman was censured bitterly for advising delay, under the expectation that an evolution might or would ensue, rather than proceed to turn the child. We have heard his reasons why he he did so. We must not forget that it is an easy matter, even

when our experience is or has been limited, to suggest the operation of version, but the circumstances by which that operation may be surrounded, will, or may preclude any attempt at its accomplishment with success. "The fact is one thi g, the evolution another," whether spontaneously or artificially performed. Denman was cognizant of thirty cases and only one living child. From this striking disproportion of successful cases, a medical gentleman of London stated before the London Medical Society "that Denman had done more mischief by his article on spontaneous evolution than good in all his works."

My remarks in this paper, as you will perceive, gentlemen, are to sustain the correctness and truthfulness, as I believe, of Denman's rationale respecting the manner of the evolution "of a child turning on its own axis" in the excavation of the pelvis when the shoulder presents, and to show that in this class of cases such a movement does and can take place. There is, therefore, no more "impossibility" that the shoulder should ascend and the breech come down, than in those cases referred to by Fichet where the shoulder is in the pelvic excavation, and where the arm is pulled upon with some force, the breech ascends and the head descends through the pelvis. I have not seen a case representing Douglas' except the one I have related of double fœtus; nor have I any similar to Fichet. I have, however, witnessed an instance, in the case of a lady with amplitude of the pelvis, where the child in her first labor presented face, and was delivered by one strong, continuous pain through the whole depth of the pelvis in that position. In the second, it was a shoulder presentation, back anterior, and this child was delivered with the head flexed on the chest, after three or four severe and strong pains.

The weight of the child was about six and one-half pounds. Evolution or version of the child, whether in the pelvic cavity or in utero, is not, therefore, confined to shoulder presentations alone, for it occurs, as we have seen, when other parts of the fœtus present, though it may be doubled up.

"The why and how," as Barlow asks, I have wished to ex-

plain by another and a different process than that known to the profession at the present time, and called the general and irregular contraction of the uterus. From a critical examination of the views of most prominent authors who have written on this subject, and from my own experience and investigation, I present these conclusions, with what success I leave to your judgment to decide.

SOME CLINICAL OBSERVATIONS ON DIABETES MELLITUS, WITH CASES.

BY A. A. SMITH, M.D.

(Read before the Academy, February 16, 1882.)

In the past three years, four cases of "Diabetes Mellitus" have come under the observation, jointly, of Dr. Barker and myself.

During September, 1879, three patients presented themselves for treatment, all giving very similar histories. Two came in one week, and one the following week.

Case I.—Mr. X——, aged sixty, had been suffering for months from mental depression, disturbed sleep, indigestion, great restlessness, inability to concentrate his mind on his work; his profession being that of a lawyer. He had suffered greatly all summer from the heat, and although he had gone out of town to his country place, July 1st, he had not been able to recruit as he had formerly been able to do. He easily became tired, which was not dyspnœa, but muscular fatigue. From being a large, robust-looking man, weighing two hundred pounds, he had become reduced in weight to one hundred and sixty-five pounds. He had noticed considerable thirst, but attributed it to the heat of summer, and yet he noticed he did not perspire much. He observed no particular increase in the quantity of the urine. He was obliged to rise at night to urinate, but he had done that as a matter of habit for many years. He had no increase of appetite; on the contrary, his appetite was less than usual, and sometimes food was loathsome to him. He came for treatment because of the disturbances of digestion, the inability to do his work well, and the mental depression. He feared he was breaking down, and that his working days were over. He had led a very active and laborious professional

life, confining himself closely to his practice, and doing his work with a certain amount of anxiety. He had become quite irritable and nervous. He had a dread that something terrible was about to happen.

He has been a fairly good liver, taking wine at his meals, almost always champagne in considerable quantity and of the sweet variety, rarely ever drinking spirits of any kind. His appetite was moderate; he was a sufferer at times from hepatic and gastric dyspepsia; this dyspepsia had existed for many years. He had had no special mental strain, certainly not more than usual; had never had any injury nor severe sudden shock to the nervous system. He had never had malaria in any of its manifestations. So far as known, he had no hereditary tendency to disease.

An examination of the urine revealed a specific gravity of 1034; a considerable quantity of sugar, as shown by Fehling's test; no albumen; negative microscopical appearances.

At the end of three days the examination showed exactly the same as above, and the quantity sixty-five ounces in twenty-four hours. He was put at once on the same treatment as the other two cases which I shall refer to later on.

Case II.—Mr. J——, aged forty-nine, residing in the country, leading a literary life, presented himself for treatment the same week as Mr. X——.

He had been suffering all his life from attacks of indigestion, but they had been more frequent during the summer. He had suffered greatly from mental depression for some weeks. He had always been a hard student, delighting in abstruse subjects, and giving very close attention to subjects requiring keen, analytical thought, such as political economy and finance. He had been a sufferer from headaches from his childhood, and during the year preceding his visit for advice, he had suffered more than usual. He was inclined always to take a very gloomy view of everything; indeed, he was somewhat of a pessimist. Thinking to improve his digestion, he had gone through a process of dieting, partially giving up meat and resorting more freely to a farinaceous diet. But on this diet he became

worse. He was always excessively fond of sweets. He noticed that his thirst was very great, his skin dry, his urine increased in quantity, as he supposed because of the increase of thirst. He was obliged to get up at night several times to empty his bladder. The increase in the quantity of the urine led him to estimate it, and he found the quantity was from two hundred to two hundred and thirty ounces in twenty-four hours. He thought he had some febrile movement at times, and was supposed to have malaria, but antimalarial treatment had not given relief. He complained of great weakness. He had lost in four months about thirty pounds. He had been a moderately good liver, taking wine occasionally but not regularly. He had never had gout, rheumatism, or malaria.

An examination of the urine revealed a specific gravity of 1036; sugar in abundance, and the quantity in twenty-four hours two hundred and twenty ounces; negative microscopical appearances.

Case III.—Mr. Z——, aged forty-six, a very eminent artist in this city, came under observation the next week after the other two. He had complained for some weeks of great weakness, mental depression to a marked degree, loss of appetite, inability to sleep as well as usual. He could not do his work with any pleasure; he became irritable, from having been a most amiable man. He had been accustomed to take long walks, but found he was unable to do so because of shortness of breath and muscular weakness. He noticed great thirst during the summer, and a considerable increase in the quantity of urine. He, like the others, attributed the thirst to the heat of summer, and the increased quantity of urine to the increased quantity of fluid taken. He was a good liver, but not always taking wine at meals. He had lost about twenty-five pounds in weight in a few months. He had suffered some pains in his legs and joints, which were attributed to gout. Since then he has had a distinct attack of gout. He had always worked hard at his profession, was ambitious, and did his work with anxiety. He had never had any severe illness, had

had no sudden, severe shock to the nervous system, and had never met with a serious accident. He had been a moderate eater, but, like the other two, he was excessively fond of sweets, and had been so all his life. He had never suffered from headaches. He had no trouble with vision; no skin trouble. He had no hereditary tendency to disease of any kind, so far as he knew.

An examination of the urine showed a specific gravity of 1035; sugar in considerable quantity, and the quantity voided in twenty-four hours about eighty ounces only.

The treatment in each of these three cases was precisely the same.

They were given codeine in quarter-grain doses, three times daily, gradually increased to one grain three times daily. This was the largest quantity given. They were also given tincture of the chloride of iron, twenty drops three times daily, and this was not increased. They were also given a laxative pill, consisting of aloes, nux vomica, rhubarb, and extract hyoscyamus, sufficient to relieve constipation, which was a prominent symptom; water, as much as desired, and a diet of the following. This list may be found in the different works on the subject. A written list was given to each patient.

Vegetables.—Tomatoes, celery, cabbage, lettuce, cucumbers, pickles, spinach, radishes, mushrooms, cauliflower, asparagus, truffles, oyster-plant, onions, water-cresses, olives, tea and coffee, gluten bread (New York Health Food Co.), gluten flour, almond-flour bread.

Meats.—Fats, oils, cream, butter, poultry, fish, eggs, cheese.

Brandy, whiskey, claret, Burgundy, very dry sherry, the acid Rhine Valley wines, lemons. Nuts of all kinds.

Patients tire very quickly of the bread, and soon learn "bread is the staff of life" indeed. Since the "New York Health Food Co." have prepared what they call a "gluten flour," which has the starchy matter to a great extent extracted, there is less objection to bread made from this.

There was a gradual diminution in the quantity of sugar; the specific gravity gradually came down; the unfavorable

symptoms disappeared, and by January 1, 1880, the sugar had entirely disappeared from the urine; the specific gravity was respectively 1022, 1020, and 1018, and the patients expressed themselves as feeling entirely well. The treatment was kept up for three months longer and then discontinued, except that the tincture of the chloride of iron has been continued most of the time since. I have examined the urine of these three patients within a year, and there is no evidence whatever of any difficulty. It is over two years since I have found any sugar in the urine. During the past two years they have been engaged in their work the same as before coming under observation, and are apparently entirely well.

There are some points of interest which I desire to emphasize; and *first*, as bearing on the etiology of the disease. These three men were all hard-working men mentally, men of ambition, who did their work with a certain amount of worry and anxiety. All had attained eminence in their different lines of work; all were very comfortable pecuniarily; all were sufferers from disturbances of digestion, and all were suffering, at the time they came under observation, from great mental depression. They had all led sedentary lives, lived quite freely, and took very little muscular exercise. As so much has been said of late in reference to disturbance of the nervous system as playing an important part in the causation of the disease so far as we can draw conclusions in any case clinically, the histories of these cases seem to point to disturbance of the nervous system as at least connected with the causation of the disease. Then, as to disturbances of the digestive system in connection with the etiology. It will be observed in the histories related that all had been sufferers from hepatic and gastric dyspepsia for many years, and although this was somewhat aggravated for a few months previous to applying for relief, it was not markedly so. Years ago diabetes mellitus was regarded as a kidney disease; later still, a blood disease; then a disease due to some interference with the glycogenic function of the liver; and still later, as originating in the nervous system. It is a fact easy of demonstration that sugar can be produced in the urine by even a very slight pricking with

a needle the floor of the fourth ventricle. It is also well known that cases of diabetes mellitus have occurred immediately after severe sudden shocks, whether of anger, fright, or accident. Some of the most severe and persistent forms of chronic dyspepsia the physician is called on to treat, in cities at least, are in persons who are laboring under severe mental strain, whose nervous systems are constantly under great pressure. There seems to be in such cases a direct connection between the condition of the nervous system and the condition of the digestion, for as soon as the strain is removed the digestion becomes good. It will be noticed that all these patients had always been excessively fond of sweets, an important fact in connection with the question of diet in producing the disease. Diabetes is quite common in Italy, and it is attributed to the fact that the Italians eat quite largely of sweets. Whether the excessive fondness for sweets shown by the three patients had anything to do with the production of the disease or not, it is a singular coincidence, if it be only a coincidence. It is thought by some observers that a diet consisting largely of farinaceous or starchy food conduces to diabetes. In this way is explained the great prevalence of the disease in the country portions of England and in Thuringia. But diabetes is not markedly prevalent among the Irish, who live largely on a diet of potatoes, nor among the Scotch, who live largely on oatmeal.

The chain in these three cases would seem to have been continuous strain on the nervous system over a number of years, disturbance of gastric and hepatic digestion, excessive fondness for sweets, and then sugar in the urine. Just what part disturbance of the pancreatic digestion plays in the etiology of the disease has been much discussed, and is interesting but very obscure. Some diabetics emaciate very rapidly. It is well known that any disease which affects the pancreas in such a way as to interfere with the secretion from it, whether it be by pressure on the duct or by destruction of its secreting cells, produces very rapid emaciation. Is it not probable that in those cases of diabetes in which emaciation occurs very rapidly

there is interference with the functions of the pancreas, either as cause or effect?

In December, 1881, a lady who has been a patient of Dr. Barker's for twenty years sent for me, complaining of great thirst, inability to sleep well on account of being called up at night so frequently to empty her bladder. I had attended her for various ailments during the past eight years. In the summer of 1872 she fell and met with a severe injury, which confined her to the house three months. In 1874 I attended her during an attack of typhoid fever. She has had several not severe attacks of rheumatic gout. During the past three years she has suffered very much from attacks of dyspepsia, usually intestinal flatulence, but frequently hepatic and gastric disturbances. She has had during the past year severe pains in the right leg below the knee, and what she calls cramps in the right foot, sometimes so severe as to cause her to jump out of bed if she happens to be lying down at the time. She has been a sufferer for forty years from very severe headaches, which at times have produced for a short period semi-unconsciousness, suggesting "petit-mal epilepsy." She is a large person, sixty-three years of age, has suffered all her married life from great anxiety on account of domestic trials, and during the past two years her anxiety has been greatly added to by a special domestic trial. During these two years her sleep has been much broken, frequently getting for days together only two or three hours' sleep in the twenty-four hours. She had lost about twenty pounds in weight in a short time. She estimated the quantity of urine at about one gallon and a half in twenty-four hours, and she thought she had been passing as much as that each twenty-four hours for ten days. She had never had malaria; had taken wine very rarely; she had the gouty tendency. She had lost a relative (not a near one) with diabetes.

Examination of the urine showed a specific gravity of 1034; sugar in abundance; quantity in twenty-four hours two hundred ounces.

She was put on exactly the same treatment as the others, ex-

cept that five drops of tincture of belladonna were added to each dose of the codeine, because of the headache and drowsiness produced by it, and she was not given the tincture chloride of iron. January 20, 1882, she had been under treatment three weeks, and had followed out instructions as to diet and medicines only moderately well. An examination of the urine showed a specific gravity of 1028; small quantity of sugar, and the quantity passed in twenty-four hours just one-half—one hundred ounces. Certainly a remarkable result. With this favorable result of the examination of the urine she expressed herself as feeling better than she had in a year. She felt so much better that neither her family nor myself could induce her to go on with the diet and medicine. February 1st she slipped while getting into a carriage, and although she bruised the ankle some, the skin was broken in only one place, and that very small; but it refused to heal, and the leg and ankle swelling considerably, I took occasion to frighten her into resuming the treatment by telling her just what disease she was suffering from, and assuring her that the only chance she had of the leg recovering was to resume the medicine and diet as prescribed before, in which there was probably more truth than fiction. Not until February 12th, however, could I induce her to do it. February 12th, she having been without treatment twenty-three days, the urine on examination showed a specific gravity of 1032; the quantity in twenty-four hours one hundred and fifty ounces; sugar again in abundance, and all her old unpleasant symptoms returning. This case is still under observation, and judging by the results in the other cases, and by the good results of treatment in her case so far as it was followed, I feel justified in making a favorable prognosis, provided I can induce her to follow up the treatment.

In the spring of 1873 a gentleman came to Dr. Barker, recommended to him by a physician in a small town near Boston. He had had charge of the transportation of a large railway and steamboat company, and in this connection had had great responsibilities. He was naturally of a nervous temperament and quite excitable. The physician who recommended him

had discovered diabetes, and had told him his fate was sealed, as there was nothing to be done for him. Naturally this did not tend to give him much courage, and when he came to New York he was a pitiable sight indeed. He was a man forty years of age, of large frame, very pale and thin; he had weighed one hundred and eighty pounds, but had lost fifty pounds; he was extremely weak. It was with difficulty he walked into the office. He was very excitable and utterly hopeless in regard to his case. In fact, before he left home he had arranged his business affairs preparatory to his probable death while away. I examined his urine and was struck at once with the specific gravity, 1044; sugar in great abundance, and the quantity in twenty-four hours from two hundred to two hundred and twenty ounces.

He was at once put on the diet, codeine and iron, and was urged to drink claret, which he did in large quantities. He was encouraged to hope for recovery. Improvement began at once, and, although not as rapid as in the other cases, in eighteen months he had gained thirty-five pounds in weight, sugar had entirely disappeared from his urine, the specific gravity was 1022, and he considered himself a well man. I saw him in the summer of 1875, and he was then apparently entirely well, and had resumed his business as actively as ever. I saw him again in 1877, and he told me he was more actively engaged in business than he had ever been, and was not only entirely well, but capable of doing more work than he had ever been able to do before. Dr. Barker has seen him within a year, and found him in excellent physical condition.

Taking the results in these four—yes, I might say five cases—we ought to be encouraged to take a more cheerful view of diabetes mellitus than has been done heretofore. One patient has remained entirely free for seven years, three others for two years, and the fifth one is still under observation. Opium was first used in the second century in the treatment of diabetes, or at least what we suppose was diabetes ("a heavy urine, sweet to the taste"). Codeine was suggested first by Pavy, and he reports some successful cases. He used codeine rather

than other preparations of opium, because of a less tendency to produce disturbances of digestion and drowsiness. Codeine has some tendency to produce disturbances of digestion, and yet in all these five cases the disturbances of digestion which had existed in all (in some more than in others) were relieved immediately the use of codeine was begun, an important fact, it seems to me, as bearing on the etiology of the disease. If the digestion were primarily at fault, it would seem that codeine ought to aggravate the disease. On the other hand, the codeine being a distinct and direct sedative to the nervous system, and particularly to the pneumogastric nerve, it would seem to add another argument to those already adduced to show that the disease has its origin in the nervous system. The position taken by some observers that codeine only diminishes the quantity of the urine and the sugar, and has no effect on the causes of the disease, does not seem tenable. The mistake is often made of ceasing treatment too early. The codeine and diet were kept up in these cases months after the sugar had become normal.

Either we have been fortunate in getting cases very early in the disease, and so amenable to treatment, or the treatment has been specially effective. I have no doubt that many cases of diabetes mellitus go unrecognized, and frequently it is possible they do not seek advice until the disease has advanced sufficiently far to produce organic changes somewhere, which prevents any treatment being successful. In two of these cases I am quite certain the diagnosis would have remained obscure for some time longer if we had not followed the routine of examining the urine of almost every patient. The result of the examination was, in fact, quite a surprise in these two cases. It illustrates again the importance of examining the urine of every patient, and frequently. A distinguished physician of London examines the urine of every patient who consults him, and declines to prescribe until this examination is made. While an *interne* in the Bellevue Hospital, I was in the habit of examining the urine of every patient, and very frequently gained valuable information in regard to diagnosis which would otherwise have escaped observation.

I have remarked only upon those points suggested by the histories of these cases, and have purposely avoided many points of interest in connection with the subject of diabetes.

To sum up the points in these cases:

First.—All occurred in cases of disturbance of the nervous system. In none was there injury nor sudden shock to the nervous system, but continuous mental strain. They were not hospital cases, but occurred in private practice and among people exceptionally well to do.

Second.—All had disturbance of hepatic and gastric digestion.

Third.—None had albuminuria nor malaria, nor were addicted to the use of alcohol; two had gouty tendency, three had none, none had rheumatism.

There was not great emaciation in four, it was great in one. None ever had any trouble of vision referable to the diabetic condition.

None ever had any skin troubles, either during the progress of the disease or as sequel.

Hereditary tendency was traceable in only one, and that very remote.

There was in none any increase of appetite.

These cases, it seems to me, illustrate the importance of selecting a plan of treatment and following it up persistently. Unless care in the examination of the urine is shown, one may be deceived.

Case VI.—During the spring of 1881 a lady, the wife of an eminent judge in a neighboring city, came to Dr. Barker for treatment. She came, she said, to be treated for diabetes, as her physician had told her she was suffering from the disease. She brought a specimen of her urine with her.

The urine was examined by Dr. Griswold, and I append his report:

"The urine was found to be acid, *pale*, and very clear. On testing the specific gravity, I was surprised to see the urinometer floated up some distance above the highest figures of the

scale, which did not go beyond 1060. The specific gravity was found to be 1080 by another instrument of wider range. On applying Fehling's test, no glucose was found to be present. Moore's test (boiling with caustic potash) and the bismuth test were then employed, but without indicating the presence of glucose.

"The absurdly high specific gravity, together with the absence of glucose, and the fact that the patient wished to suggest that there was sugar in the urine and that she had diabetes, created a suspicion that she might be simulating diabetes by adding sugar from the breakfast-table, and was overdoing it. Accordingly, I tasted the urine and found it distinctly sweet. I then took two drachms of the urine in a test-tube, added a few drops of sulphuric acid, boiled a few seconds, and then let it stand five minutes. This would change the *cane-sugar* into *glucose* if any were present. I then tested for glucose by Fehling's test, and found abundant evidence of its presence. It was therefore plain that the high specific gravity was the result of her having added a little too much sugar.

"Since that time this same patient's urine has been found by a physician in Brooklyn to contain glucose. She has evidently studied the subject more carefully in the interval."

I have asked Dr. Griswold to give me the history of a case now under his care, which illustrates some important points in connection with the subject of diabetes mellitus.

"Mr. S——, a stout, healthy man, aged sixty, weight two hundred and ten pounds, height 5 feet 6 inches, complained to me one day of disturbance of vision. Examination of his urine revealed no albumen or other abnormality; the specific gravity was only 1020. As an extreme precaution I examined for sugar, in view of the fact that eye troubles occur in ten per cent. of all cases of diabetes mellitus. To my surprise I found sugar present, eight grains to the ounce. Further questioning revealed no symptom in the case which could be referred to the glycosuria. The patient has now been under observation for eight months. During that time repeated examinations of

the urine have been made, and show that sugar is always present in the urine, varying from twelve grains to three grains per ounce. He has no increase in the quantity of the urine, and his eye troubles disappeared after a dose of calomel. He is so comfortable that it is impossible to alarm him sufficiently to induce him to take medicine or adhere to an anti-diabetic diet."

This case illustrates the fact that sugar does not always imply a high specific gravity. It is not always safe to say, because a specimen of urine does not show a specific gravity above 1026, it is unnecessary to examine for sugar. It also illustrates the amount of tolerance which a patient can show to the disturbances in the system of such a character as to produce glycosuria. There may, then, be glycosuria without any increase in the quantity of urine, a normal specific gravity, and no special symptoms pointing to the disease.

PERSISTENT RECURRING REFLEX SPASM OF THE BLADDER DURING A PERIOD OF OVER TWENTY YEARS, RESULTING IN THICKENING OF ITS WALLS, DILATATION OF THE URETERS AND HYDRONEPHROSIS—DEATH FROM URÆMIA.

By FESSENDEN N. OTIS, M.D.

Mr. President and Fellows of the Academy:

As introductory to the subject of persistently recurring spasm of the bladder, referred to in the announcement for this evening, I desire to cite a few instances, in certain leading features similar to it, and illustrative of some of the possible sources of error in the diagnosis and treatment of urinary difficulties.

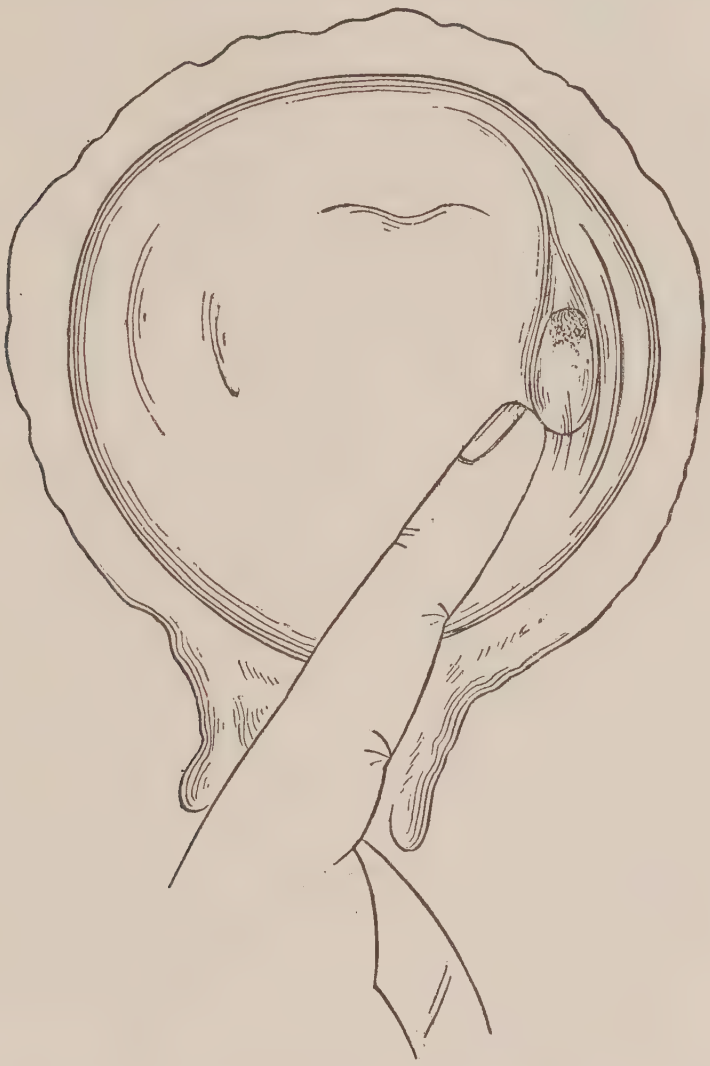
Mr. C——, a miner, forty-one years of age, from Scranton, was sent to me by Dr. M. J. Williams, of Hyde Park, Pa., March 2, 1882, with a history of urinary troubles, dating back to a gonorrhœa some eighteen or nineteen years previous. This gave him much trouble for several months; then, after an interval of some years, he suffered with other troubles, which were considered due to the passage of urinary calculi, and he subsequently passed three small specimens per urethram. Again free from any special urinary trouble for several years, he began once more to be afflicted with pains in the region of the kidneys, frequent urination, and finally inflammation of the bladder and retention of urine. He was treated for stricture by dilatation, and, finally, by divulsion, without benefit. He suffered greatly from frequent and painful urination, with pain in the glans penis and great irritation in the rectum. He was repeatedly examined for stone in the bladder, but none was ever detected. For ten months previous, his sufferings had been constant and intense, passing his urine every few minutes during the day and several times during the night, the stream shutting off suddenly, as if something had dropped down at a certain point in his urination, and closed the vesical orifice. Repeated ex-

aminations of the bladder failed to detect either the presence of stone or a polypoid growth. The patient was subjected to all the usual and some unusual procedures for disengaging any possible hidden or sacculated stone. The prostate was slightly enlarged, chiefly on the right side, and this was the only abnormal point ascertained about the genito-urinary apparatus.

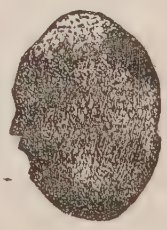
In view of the history of the case pointing to probable stone in the bladder, and the symptoms distinctly indicating what seemed to be a movable obstacle to urination, such as a polypoid growth within the bladder or an encysted or otherwise hidden stone, an exploratory operation was determined upon. On Friday, March 10, 1882, the patient was etherized, and the median section for stone was performed. Through the opening thus secured, the prostate and bladder were explored with the forefinger of either hand successively, but without finding anything abnormal. The examination was repeated by my associate, Dr. Bangs, and also by Dr. Williams, of Pennsylvania, who was present and assisting in the operation, and with like negative results. The bladder was then carefully explored with sounds, but no stone was detected. Just as we were in despair of achieving any beneficial result from the operation, I again introduced my left forefinger to the utmost limit, gaining, perhaps, by urgent pressure, a quarter of an inch in the depth of exploration, when a soft, apparently pendulous body, about the size of an ordinary bean, was felt by the tip end of the finger on the left side of the bladder. The conditions thus appreciated are represented by the accompanying diagram on opposite page.

A pair of narrow forceps was then introduced, and the supposed tumor was seized in about one-half inch grasp, and twisted gently entirely around. As it still held, the forceps were disengaged and the finger again introduced. The tumor could no longer be felt in its former locality, but, on searching the floor of the bladder, it was again detected, and on introduction of a pair of duck-billed forceps, what appeared to be a firm clot of blood was removed. This, on examination proved to contain a stone about three-fourths of an inch in length by one-half inch in

breadth and three-eighths in thickness. The surface of the apex was smooth and light-colored for about one-fourth of an



inch, while the remainder was dark-colored and rough, apparently marking the encysted or sacculated portion, as may be seen in the specimen which is here presented, as well as in the accompanying photograph.



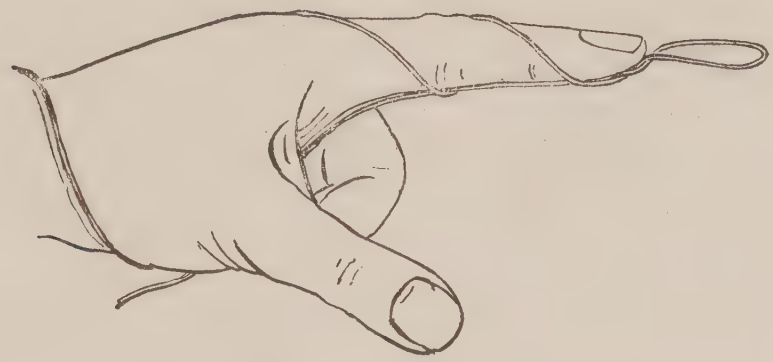
After thorough irrigation a large, soft catheter was introduced into the bladder through the perineal opening and left in, the proximal extremity of the catheter being fastened into a male urinal. The patient did not experience a single unfavorable result. All pain and difficulty about the bladder was removed. The urine flowed freely into the catheter. There was no rise of

pulse or temperature. The catheter was removed on the third day. On the fourth the patient began to urinate through the urethra. On the fifth he no longer passed any urine through the wound. A No. 34 French sound was passed every other day through the urethra and well into the bladder. By the ninth day the wound of operation was healed, the urine was free from pus as a sediment, every symptom of former troubles had disappeared, and on the morning of the tenth day he left for his home in Scranton, apparently well in every respect.

Encysted calculi are fortunately rare. Several interesting examples are cited in Sir Henry Thompson's recent edition (1880) of his work on "Practical Lithotomy and Lithotrity," page 93 et sequitur. "A calculus," he says, "may be altogether contained in a cyst in the bladder, a small part of its surface only being exposed at the mouth of the cyst." "I had the opportunity," he further says, "of watching at University College Hospital one example of this, which was once, and only once, struck with a sound during life, although the sounding was repeatedly performed. . . . At death the condition described was found, and it was then obvious that the chance of striking such a calculus was exceedingly remote, and that no operation could have removed it." In the present case a distinct click was appreciated during exploration of the bladder on two separate occasions, in just the locality where the stone was subsequently found, but as this could not be repeated, it was referred to some accidental sound outside the bladder. The difficulty of thoroughly exploring the bladder with the unaided finger became painfully apparent in this case, where failure and success in diagnosis was seen to have depended upon but one-fourth of an inch in the length of the finger. This fact suggested the desirability of some ready method by which the finger might be efficiently supplemented under similar circumstances. The accompanying contrivance for lengthening the right forefinger may be readily constructed of ordinary copper wire, and will, I think, answer the desired purpose.

But for the rare fortune of finally discovering this encysted

stone, in all probability the trouble would have been relegated to the list of obscure reflex nervous disorders dependent upon urethral, spinal, or rectal causes, until a post-mortem examination should have revealed the true nature of the difficulty. It must not be forgotten, however, that symptoms almost, if not perfectly identical with those of stone in the bladder, in certain cases, may arise from reflex causes quite independent of the presence of stone in the bladder, free or encysted.



It may not be irrelevant to our purpose here to cite a single instance in point, which was published by me in the *Hospital Gazette* of June 22, 1879, but which has now the added interest of nearly three years' subsequent experience:

Mr. W——, sixty-four years of age, came under my observation December 25, 1876, with the following letter from his family physician: "Mr. W —— is suffering from enlarged prostate gland and the symptoms which usually accompany that condition of things, and his trouble has been coming on for some time past —difficulty in passing urine, pain, and straining, requiring use of catheter. Treatment has been: use of catheter, warm hip-baths, suppositories of opium and belladonna, laxatives, infus. buchu, mur. tr. iron, as the symptoms from time to time indicated, with regulation of diet, etc." From the patient I gleaned the following: Never had gonorrhœa. First trouble of urinary apparatus was an attack of dysuria March, 1875, without any apparent cause, except, perhaps, drinking largely of carbonic acid water; lasted nearly a day, and passed off without treatment. Second, four months after, similar to first; quite well in the interval. Again free for a month, when

urinations became gradually more frequent during day, and obliged him to rise four or five times during the night; walking gave him relief. Finally had a retention of urine, lasting, with much suffering, for twelve hours. Introduction of catheter resisted. Dr. Stephen Smith (visiting physician to Bellevue and St. Vincent's Hospitals), who was called in consultation, passed a catheter and drew off the urine. From this time, catheter used three times in twenty-four hours. No urine passed voluntarily; great urgency and frequent agonizing pain before passing catheter; great straining, involving diaphragm and abdominal muscles. This condition continued up to the date mentioned, December 25, 1876.

Examination of prostate by me shows but *slight, if any enlargement.* Ordinary catheter passes in without force. Urine drawn is thick with pus and mucus.

Examination of penis: Circumference, $3\frac{3}{4}$ inches; meatus, 32; size of urethra, 36 mm. from meatus to bulbo-membranous junction, as shown by urethrometer. Quiet and infus. triticum repens prescribed. January 2, careful examination made for stone; none found. Bladder irrigated with solution of borax twice a day. Examination of several specimens of urine showed nothing but catarrhal elements. No abnormal condition could be detected about the neck of bladder, and yet the patient could pass no urine voluntarily, and as soon as he made the effort, tenesmus of the vesical neck came on, which gave great distress.

Passing urine every two hours through catheter, which he has been taught to introduce. Having seen cases of somewhat similar character, and unable to find any cause for the trouble, except a spasmodic one, *I introduced with great care, bearing in mind the importance of such a procedure in a man of his age, and suffering with disease of the bladder,* a No. 32 solid steel sound, without force, through the entire urethra. I then followed it quickly with No. 34, in order to over-distend the membranous urethra, which I believed to be the seat of the trouble. A few minutes after, Mr. W—— was seized with his accustomed desire to urinate, rushed into an adjoining closet

and introduced his catheter as usual. Returning somewhat hurriedly to resume conversation thus suddenly broken off, in two or three minutes he again felt desire to urinate, and believing that his bladder had been emptied, simply took up the chamber, without any idea of urinating, when, to his infinite astonishment and delight, he passed with perfect ease over a gill of urine. This was the first passed voluntarily since first relieved of his retention by Dr. Stephen Smith. From this time Mr. W—— passed his urine *without the aid of a catheter*, on an average of every two hours for the next four days, introducing the catheter only night and morning for the purposes of irrigation. Great and rapid improvement in health and entire freedom from straining and tenesmus.

January 4th.—To carry out the treatment by *over-distention*, more fully, I incised the meatus to 36 mm., the pre-ascertained normal calibre of the urethra, and passed a No. 36 solid steel sound with complete ease through the entire urethra and well into the bladder.

From that time the recovery from cystitis was rapid, and urine was passed voluntarily and in full stream up to October 18th (over nine months), when the patient called to say that he had remained quite well up to two weeks previously, not having in the interval to rise during the entire night to urinate; but that, since then, having taken cold by sitting on a cold stone, his urine had presented some sediment, and his urination was with increased frequency. The only treatment (aside from infus. triticum repens), was by introduction of a No. 33 solid sound.

October 19th.—Mr. W—— called to say that the irritation at neck of bladder, and referred to end of penis, disappeared at once on introduction of the sound the day previous. Intervals of urination increased to between three and four hours, rising only once during the night. Recovery from the vesical catarrh, which was but slight, was complete within the week, and Mr. W., who is still under my observation in a general way, has been entirely well of his urinary trouble from that date to the present, over six years.

In this case, in the absence of any prostatic enlargement or discovery of any polypoid growth, the evidences of hidden stone were most marked until the passage of a full-sized sound, which promptly demonstrated the reflex nature of the difficulty.

I beg leave now to present this case of

PERSISTENTLY RECURRING SPASM OF THE BLADDER DURING A PERIOD OF OVER TWENTY YEARS, RESULTING IN THICKENING OF ITS WALLS, DILATATION OF THE URETERS, AND HYDRONEPHROSIS — DEATH FROM URÆMIA.

Mr. Z——, fifty-seven years of age, had suffered from frequent, difficult, and more or less painful urination for over twenty years. His earliest trouble with the genito-urinary apparatus was an acute urethritis, which soon merging into a chronic form lasted for some years, during which he was treated for urethral stricture by several surgeons. Subsequently he came under the care of a distinguished physician of New York City. At this time he was suffering from frequent micturition and other troubles of the genito-urinary apparatus, which suggested the possible presence of stone in the bladder, a search for which proving unsuccessful, the patient was referred to an eminent surgeon (this was in 1860), who also failed to find any calculus, and treated him for some time by local and general measures for his cystitis, with varying success. The frequent and painful urination continued, however, and he came under the care of various surgeons, and physicians, regular and irregular. He was at one time under the care of Sir Henry Thompson of London, who also examined him for stone in the bladder, but found none. M. Civiale, of Paris, also made a most exhaustive examination with the same negative result. The patient, in relating his experience with M. Civiale said: "I was unfortunate enough to arrive in Paris just after an important personage who had suffered from symptoms of stone, and had been examined by many surgeons without detecting any stone, had been referred to M. Civiale. After a long and careful search Civiale found and removed a very small, rough calculus. With

this success fresh in his mind, he examined my bladder with such thoroughness that I was confined to my bed for six weeks after—but he found no stone." His frequent and painful urination continued unrelieved, and his urine, at times bloody and always with more or less pus, was passed every hour, or oftener, for several years. Treatment in great variety had been used under the advice of eminent surgeons and medical men of every school. Patent medicines, medicinal waters, and spiritualistic agencies had all been tried without avail. Everything, he said, that had ever been attempted for his relief, instrumental, local, or medicinal, had signally failed, and for the few years past he had been under the general care of Dr. M——. His sufferings increased to such an extent, that surgical aid seemed imperatively called for. He was referred to a surgeon eminent in genito-urinary matters, who also failing to find any stone to account for the cystitis, treated it by repeated washings and the occasional passage of a No. 26 sound into the bladder; which latter procedure, the patient stated, was the only thing that had ever been done for him that appeared to do him any good.

After a time his sufferings increased and became complicated with malarious symptoms. He was then greatly debilitated, and in almost constant suffering with his urinary difficulty, when I was called to see him. His general appearance was that of a man suffering from malignant disease. His urination, which occurred regularly at intervals of about fifteen minutes, was one continued agonizing spasm for about two minutes at each act. The spasm he said was less frequent and less severe at night. He complained also of severe pain in the region of the left kidney. This, he said, he had had from time to time for a long period, and surgeons had universally attributed it to the presence of a stone in the kidney. Frequent examinations of his urine had failed to detect any organic disease of the kidney. A large quantity of epithelium from the bladder had, at one time, suggested the possibility of epithelioma of the bladder; cancer had also been suggested. The pains during micturition were always referred to the region of the neck of

the bladder, pubis, and perineum. He had never had any pain in the glans penis, nor was his trouble aggravated by motion, or in a carriage. He suffered only during urination. He had long worn a urinal. Diet, chiefly of milk. On December 30, 1881, I was called in consultation by Dr. Lewis Fisher, the family physician, and obtained the following particulars of the case. On December 31st, a careful general examination failed to discover any evidences of organic disease in the thoracic or abdominal regions. The penis showed a circumference of three and three-fourths inches and a urethral orifice of 25 French. Examination with the urethrometer showed a normal calibre of 37 French from bulbo-membranous junction to within three-fourths of an inch of the urethral orifice, where the canal suddenly narrowed to 26 French, registering the same to the orifice, where it was 25 French. The repeated examinations of the bladder (some quite recent) by distinguished surgeons, together with the entire absence of pain in the glans penis, or any sudden stoppage of urine during the act of urination, satisfied me that if there was any stone in the bladder it was encysted, and probably would not be discovered by the use of the sound. The case seemed to me one where the spasm of the bladder, occurring with great frequency and severity through such a long period of time—then over twenty years—must have ended his existence long before if due to organic disease of the spine or kidneys, and no evidence of any organic disease being present. I suggested the possibility of the difficulty being in a measure, if not wholly, of reflex origin, and due to the irritation of the contracted and thickened urethral orifice, and proposed to test the truth of it by dividing the meatus so as to make it correspond completely with the remaining portion of the canal. To this the patient finally consented.

Operation, Sunday, January 1st.—Patient brought under the influence of ether by Dr. Bangs. I then made a division of meatus and tissues extending three-fourths of an inch back, from 25 mm. to 38 mm. and passed a No. 37 solid sound well into the bladder without the least force. Following this, there was absolute incontinence, the urine passing away without pain, and

almost without consciousness. This condition of things continued without especial change until Wednesday, the 4th inst., when he had some slight power to retain his urine, and for the first time, a twinge of pain. He remarked that the second night after the operation he had the best night's sleep he had had for ten years. His habitual hypodermic dose of morphia had been omitted up to Wednesday, when, in the early evening, an attack of the kidney colic (left side) which he had previously suffered from came on, and with such violence that Dr. Fisher was sent for, and administered ten drops of morphia hypodermically. A comfortable night's rest resulted. On Thursday morning, the patient appeared in good condition, passing urine without pain. Thursday night he had another attack of pain in the kidney requiring another hypodermic injection. Another comfortable night. The next day (Friday) he felt miserably, little or no appetite, consciousness of desire to urinate every half hour, some slight control and some pain, *now, and for the first time in the history of the case, referred especially to the end of the penis.* Never before had any pain at the head of the penis. The pain, previous to operation, was always and solely referred to the neck of the bladder. Took no morphia, staid in bed all day. Last night, suffered from pain in head of penis whenever he attempted to urinate, but, when he checked the effort, the urine would flow without pain. In all, had about six attacks of the pain referred to during the night. To-day, Saturday, January 7th, feels weak; no appetite; urinates about every half hour. Now, at 1.40, has not passed urine for forty minutes. Urine under better control. While dressing this morning he had two or three slight urinations during the hour, with quite sharp spasms of pain in the head of the penis. Since then the spasms of pain have been less in degree and frequency. Pus and mucus, which heavily loaded the urine at the date of the operation, have distinctly and steadily decreased up to the present time. Held urine for fifty minutes and then urinated voluntarily without pain, but very slowly. After this, passed No. 37 bulb through the meatus only (and this simply to keep it patent), but with less pain than

anticipated and less than at any time before. The bladder, which was washed out with warm salt water twice a day before operation, has not been touched since that date. Advise to have this resumed to-day. Milk diet.

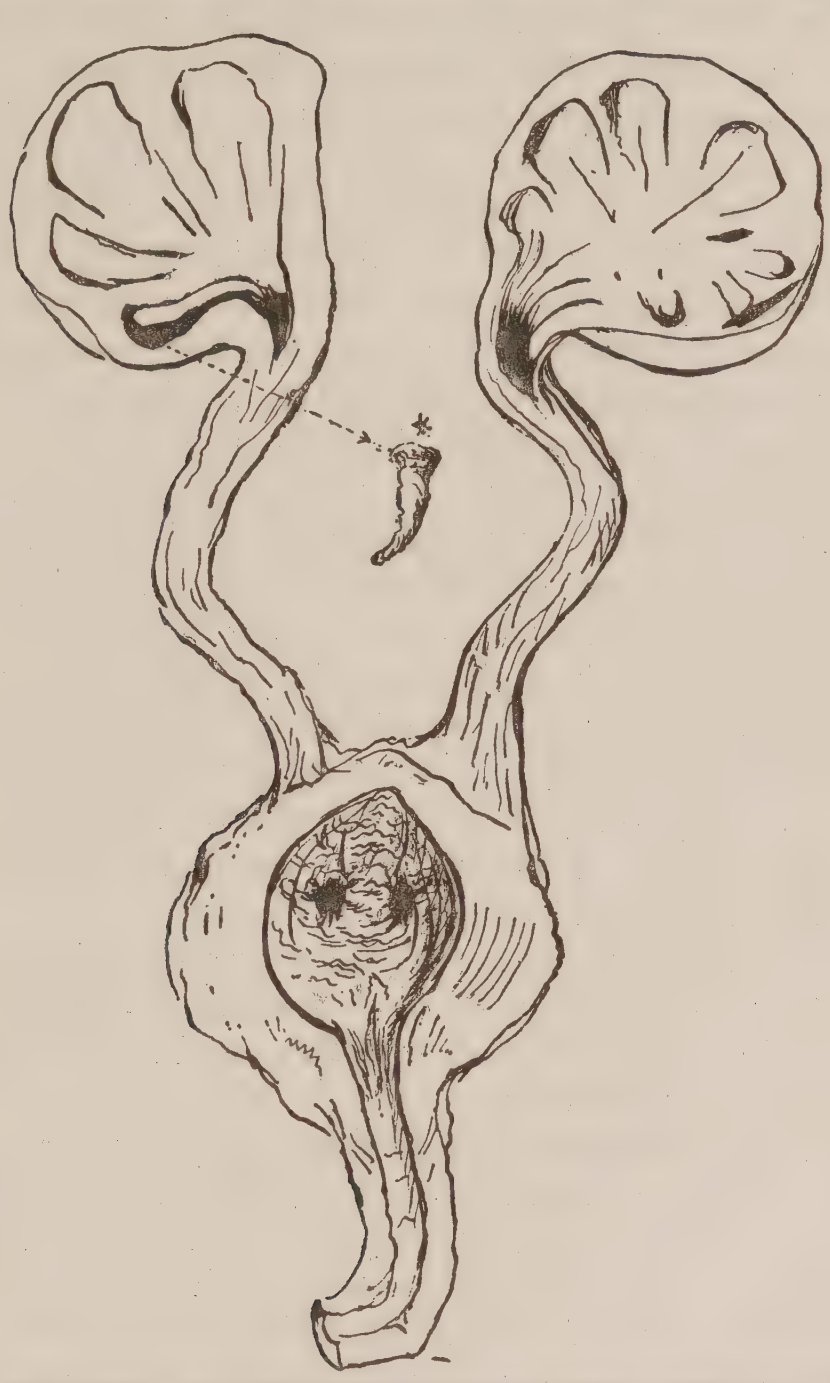
January 8th.—Had a poor night. Frequent and severe spasms extending from head of the penis to the bladder, was greatly weakened by them; occurring twenty or twenty-five times during the night. These lasted two to three minutes, and were always excited by the effort to pass water, passing only half an ounce at a time. Yesterday, washed out the bladder about 5 P. M., with no special effort. This pain in the head of the penis is quite new, and was not felt at any time before the operation, and not until five days after, when it came on suddenly. Much of the increased nervousness appears to be due to reaction from morphine. Spasms have been less for the last three hours, and has once passed water without spasm. Has taken no morphia since night before last, when he had ten drops by hypodermic injection. Now takes an injection of six drops. Says that effect of this will last to make him comfortable until to-morrow.

January 9th.—Has had a better night, only half a dozen spasms. This morning apparently under the influence of morphia, most probably uræmia, as he had only five drops last evening. Urine quite thick with pus, and has passed a full pint since 7.30 this A. M. Complains of great exhaustion, but is evidently so uræmic that not much can be inferred from what he says. At 2.50, he had a severe paroxysm, about one and a half minutes pain in bladder. At 3.15, another. At 4.30 Dr. Fisher gave him rectal injection of peptonized beef. He continued in somnolent condition, but less profound. About 10 P. M. Dr. M—— called. Frequency of urinations as follows, 10.5, 11, and 11.45, the last being accompanied by great pain.

January 10th.—Urinated with great pain every hour, sometimes oftener. After pain stopped, dropped off to sleep, but could be roused easily. Attempted to give beef enema, but he refused to have it.

Died uræmic on the 11th.

Autopsy by Dr. Welch.—"By request only the abdominal organs were examined. KIDNEYS.—Both kidneys are enlarged.



Outline sketch, from photograph of post-mortem specimen in case of Mr. Z.

The fibrous capsule is adherent to the surface of the organs. The cortical substance presents a grayish, nearly uniform ap-

* Calculus found in the bladder and fitting in calyx of right kidney.

pearance, with little trace of the normal markings. The pyramids are in a great part encroached upon by the dilated calyces. No abscesses are present in the kidneys. The pelvis and calyx of each kidney are greatly dilated, and contain turbid, ammoniacal urine. The ureters are likewise dilated, so that their calibre nearly equals that of the small intestine. The walls of the ureters are thickened. No obstruction to the passage of urine exists either in the pelvis of the kidneys or in the ureters.

"BLADDER.—The wall of the bladder is thickened to about four times its normal diameter. This thickening affects all of the coats of the bladder, but especially the muscular tissue. The mucous membrane of the bladder is thickened, and presents in many places, especially about the base, slightly elevated, grayish, discolored patches, such as are seen in the so-called diphtheritic cystitis. The capacity of the bladder is about that of the normal organ. Its contents are ammoniacal, purulent urine, and a small calculus. This calculus is about an inch in length and conical in shape, resembling in form somewhat a canine tooth. Such a calculus might have been formed in one of the dilated renal calyces. The calculus is apparently of recent formation, being very friable and composed wholly of phosphates, without a nucleus of uric acid or oxalate of lime, as was shown by chemical examination.

"URETHRA AND PROSTATE.—The prostate was of about normal size and had not occasioned any obstruction so far as could be detected. The calibre of the urethra seemed normal, presenting no evidences of stricture.

"SPLEEN.—The spleen is somewhat enlarged and surrounded by firm, fibrous adhesions. The liver, stomach, and intestines present no noticeable change. The microscopical examination of the kidney shows a marked new growth of fibrillated connective tissue, which is infiltrated with lymphoid cells. The uriniferous tubes are, in places, compressed and atrophied, in places dilated, in places filled with fatty epithelium.

"*Diagnosis.*—Chronic cystitis with dilatation of the ureters, hydronephrosis and chronic interstitial nephritis. The cause of the cystitis is not apparent."

We have, then, here conditions which are not explained by anything found in the kidneys, ureters, or bladder. The cause of the hypertrophy of the bladder walls, the dilatation of the ureters, the dilatation of the kidney, and the interstitial nephritis would have been plain had a mechanical obstruction to the flow of urine been discovered, but none was apparent: it was certainly not in the kidney, ureters, or bladder. At every period in the progress of the case, sounds passed easily into the bladder. There was therefore no apparent mechanical obstacle in the urethra, and yet the conditions were such as to demand a mechanical obstruction to the exit of urine from the bladder to account for their existence. It appears to me that the only possible solution of the difficulty is through the claim of a persistently recurring spasmodic closure of the orifice of the bladder, *as a result of irritation reflected from some point in the urethra.* The examination of the urethra on December 31st showed a normal urethral calibre of 38 mm., except at the orifice, where this and one-half inch of the canal was contracted to 25 French, thus showing an obstruction, practically a stricture, of 13 mm. In this connection it is interesting to recall the fact that in the very early history of the case there was a persistent urethral discharge, and that he was treated for urethral stricture by several surgeons.

The possible influence of a contracted meatus urinarius in producing, in certain cases, disturbance more or less grave throughout the urinary tract has long been known, although not generally appreciated. M. Civiale in his "Traité Pratique des Maladies Genito-Urinæus," second edition, Paris, 1850, at page 160 says: "That which has struck me most forcibly in dividing a meatus, often only slightly contracted, is the sudden and complete change effected in the general condition of the patient. The constriction, which seemed hardly to impede the flow of urine, is no sooner divided than all morbid symptoms vanish—the urethral walls, which were rigid, hard, and inelastic, immediately recover their normal condition. The bougie, which at first passed only with difficulty and pain, slips into the bladder with ease and in five or six days the slight incision

at the meatus heals perfectly, and the patient finds himself in a state so satisfactory that it would be incredible but for the fact that the instances are again and again repeated; an effect so prompt, through means of which the significance is plain, shows that the slightest obstruction in the urethra is able to produce the gravest symptoms, local and general." Again, in 1858, Sir Henry Thompson, in the second edition of his work on "Stricture of the Urethra," page 249, says: "I have given complete relief to distressing symptoms of very long continuance, *the cause of which was not suspected*, by dividing an external meatus, which nevertheless admitted a No. 6 English catheter. I have met," he further remarks, "with three marked examples of a similar kind, in which the very simple operation necessary was followed by a complete disappearance of urinary difficulties, which had long been regarded as of an extremely obscure character." In 1874 a paper was read by me before the New York Academy of Medicine, on "Reflex Irritations throughout the Genito-Urinary Tract, resulting from Contraction of the Urethra, at or near the Meatus Urinarius, Congenital or Acquired." In this paper, nineteen cases of this kind were cited, in one of which, Case XVII. (page 26 of monograph on "Reflex Irritation," published by McDivitt, Campbell & Co., and republished in the Charleston *Medical Journal and Review*, of July, 1874), in which frequent micturition of ten years' standing, complicated finally by a grave and prolonged cystitis; this was promptly relieved by the division of a meatus, which readily admitted the passage of a 23 French bulbous sound. Further proof of the capacity of anterior urethral contractions to induce spasm of the urethra and bladder may be seen in my work on "Stricture of Male Urethra," second edition, Putnam's Sons, New York, 1880, page 301 *et seq.*, and in articles on "Urethrismus, or Chronic Spasmodic Stricture," in the *Hospital Gazette* of April 19, 1879, and June 28, 1879. In all the cases reported by Civiale, Sir Henry Thompson, and myself, immediate relief followed division of the contraction.

In the case of Mr. Z——, the subject of this report, relief to the

spasm of the bladder supervened immediately upon the complete division of the contraction at the meatus urinarius, followed by the introduction of a sound corresponding in size with the normal calibre of the urethra, as previously determined by measurement with the urethrometer. How much of this relief was due to the passage of the instrument, may be a question, but not the least more force was used in its passage than in that of an ordinary sound or catheter through a healthy urethra, and not the least pain was subsequently felt which could be considered a consequence of this procedure. The only result which was in any way different from other cases where a similar operation had been performed was the complete incontinence which followed, and which continued until the fourth day, when slight power of retention was also accompanied by a slight twinge of pain. The recurrence of the spasm of the bladder soon after, as a distinct effect of the sudden advent of the calculus, renders the estimate as to the permanence of relief to the spasm through the operation entirely conjectural. It still remains, however, that the cessation of painful and frequently recurring spasm of many years' standing was immediate and complete, as a result of the operation, and practically so continued until an added mechanical irritation within the bladder reinstated it. The proof that the spasms which came on on the fifth day after the operation procedure were due to the sudden presence of the calculus were, first, that it was for the first time in the history of the case accompanied by a well-defined pain in the glans penis, which persisted until the termination of the case; second, that the calculus was recent, as indicated by its great friability, and its shape contra-indicated its origin within the bladder, but distinctly pointed to one of the calyces of the left kidney, into which it was subsequently shown to fit in the most perfect manner. In the absence, then, of any other means explaining the years of suffering endured by Mr. Z——, of accounting for the post-mortem conditions presented in his case, it appears to me reasonable to claim that the difficulty was of reflex origin, dependent chiefly, if not wholly, upon the contracted meatus urinarius, and I think we

are warranted in believing that, if this condition and its possible effects had been appreciated at an earlier period in his life, years of agonizing suffering would have been avoided, and that his life might have been saved through an operation, in a surgical point of view, of the most insignificant possible character. In considering the salient features of the foregoing cases, which I believe to be typical, it must, I think, be admitted that symptomatic evidences of organic or idiopathic disease of the bladder and prostate, as well as of stone or other adventitious material in the bladder, should be received with the distinct understanding that such evidences are possibly due entirely, or in part, to sources of irritation quite outside of the organs apparently the subject of disease.

CASES BEARING ON THE DIAGNOSIS AND LOCALIZATION OF CEREBRAL DISEASES AND THEIR DIFFICULTIES.

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(Read before the Academy, November, 1882.)

I have thought that it would be more profitable if I should at this meeting present before you results and cases bearing upon the subject announced on the card. Had I taken the subject of cerebral localization alone, I should feel, except in so far as I could contribute cases bearing upon this subject, that I was uselessly occupying your time. The numerous works which have appeared have culled the available cases, and presented them in shades varying somewhat with the author's views on the subject. The work of Ferrier, the published lectures of our fellow, Dr. Seguin, the numerous journal articles, and the translation of Dr. Charcot's work in English; the works of Grasset, of Charcot and Pitres, and of Boyes, etc., in French, and of Nothnagel and of Exner, give information up to the time of their issue.

Amongst the diseases which most frequently present difficulties, I would first allude to the fevers. Head and front in this class stands typhus fever, on account of the marked cerebral symptoms usually attendant on it. Yet there is none about which it is more necessary to arrive at a correct conclusion, owing to the liability to the spread of the disease by contagion. The writer knows of cases, both during the outbreak of the last and the present winter, in which a certificate of death from cerebritis, alcoholism, etc., should have been written typhus fever. The result was that the disease was spread to relatives and friends, and the mistake discovered when an investigation as to the mode of origin in one of these latter had been conducted. The converse was also true, that cases of cerebral disease, encephalitis, and meningitis were supposed to

have typhus fever. The difficulty in deciding between these conditions is dependent upon the exaggeration of some of the factors of the fever, the delirium, stupor, stiffness at the back of the neck, and slowing or irregularity of the pulse. The stiffness at the back of the neck more particularly misleads, if somewhat prominent. With reference to this point, I saw, in particular, one lad and a group in one family. In the lad's case this was a pretty marked feature; he was delirious, somewhat stupid, pulse frequent, not irregular, and an ophthalmoscopic examination showed no apparent change in the disc or vessels. Yet there was no eruption, and there was but a single temperature record. The probabilities, save the stiffness of neck, were in favor but not decisive of fever. I resorted then, as I have on other occasions, to an additional means of decision—an investigation of the boy's surroundings. The report received was that a member of the family had died of a fever, and that others were sick at the time with the symptoms and eruption of typhus.

This study of causation is in not a few instances of marked value in deciding doubtful cases at an early period; and I can commend it to you. There is still another, which, together with this, will throw light on a single case of a group sick, the others having recovered, or died, providing that this one have symptoms rendering a diagnosis doubtful. It consists in a use of the duration of the disease in those recovered. Hence, if several members of a family have had a fever, or supposed brain inflammation, and the duration should have been in each twelve to fourteen days, then the probabilities are decidedly in favor of typhus; for tubercular meningitis would not attack several members of a family in a short period of time, nor could we expect recoveries; hence the only probable condition would be cerebro-spinal meningitis, and the duration in difficult cases of this disease is apt to vary greatly. There is one condition, however, which simulates both disease of the brain and typhus, from the fact of the participation of the brain in the process in many cases. This is acute ulcerative endocarditis. We have in this, as you know, the development of soft

masses on the heart-valves, which, as a rule, are ulcerated, and then the little fragments from these pass as emboli to the different organs, giving rise in the skin to petechiæ from the obstruction of small arteries. According to the site, size of artery, and number of emboli in the brain will a varying picture be produced. In such cases as have no heart murmur, from the disease being situated on the auricular aspect of mitrals, or from weakness of the heart's action, without defined paralysis, the presence of petechiæ must naturally excite a suspicion of typhus, if the latter disease is, or has been prevailing in the place or neighborhood. Should, however, petechiæ be present on the body, and a definite paralysis occur, as of an arm, or hemiplegia, or these have proceeded, or should aphasia occur, then a suspicion of the presence of this disease, or of multiple embolism from some other source, would at once be awakened. Last autumn I met with a patient presenting these features.

The history was that he had fallen from his wagon and been brought to the hospital. Nothing was known of his past history or surroundings. The spleen was somewhat enlarged, the rash was petechial, he was in a stupor, and had fever. The left arm raised dropped readily to side. The temperature was 104° F.; pulse, 120. On the next day he remained in the same condition. Thermometer, 104¼°, A.M.; 103° P.M.; pulse, 140; respiration, 40. He died at 12.45 A.M. of the third day after my seeing him. The duration of the disease was possibly ten days. The autopsy disclosed two distinct conditions: The heart showed the aortic valves covered with a soft mass of vegetations, with slight erosion of their surfaces. In addition, pericarditis existed. The middle cerebral artery of the right side, outer branch, contained embolus. The spleen and kidneys contained numerous infarctions. In addition to the lesion on the right side of the brain, there was discovered a cyst in the anterior lobe of the left side, reaching to, but not involving, the posterior fourth of the first and second convolutions of the frontal lobe. The dura was adherent to the pia over its site, and it was filled by a plexus of convoluted vessels

above and by clear serum below. It occupied particularly the middle portion of the second frontal convolution, and projected downward to the corpus callosum, covering the anterior horn of the lateral ventricle. After discovering this, an additional inquiry resulted in disclosing that the patient had been subject to epileptic seizures, following an injury received in the left frontal region several years before. A cicatrix existed over the outer part of the left eyebrow, reaching to the bone. But there was no evidence that the bone had been fractured or perforated. In attempting a diagnosis with the meagre facts at first possessed, and in the absence of a heart murmur, taking the fever and the petechial eruption into account, I advised that the patient be quarantined. Some will question the possibility of the disease having been typhus fever, to which the endocarditis was superadded. I can only say that the existence of this disease dates within the last few years, so far as concerns its recognition in the category of special disease conditions. I have seen illustrations of it years ago in connection with puerperal fever. In this disease it is, as in the case above given, the cause for the development of hemiplegias and paralysis, if such occur. In the work of your president will be found an illustrative case, which occurred in 1872, at Bellevue Hospital, under the head of cerebral embolism.

To return to the diagnosis: I considered the possibility of the fall from his wagon having been due to the weakness of fever, and that the paralysis, or better, paresis, might mean that as a result of such fall he had a clot on the surface of the brain; also that this, together with the fever, had probably caused the stupor. I should again err on the safe side in quarantining such a patient. I might add that in so far as the absence of the spread of the disease would be considered evidence against typhus, we had that as an argument in favor of the endocarditis being due to some unknown septic cause, perhaps gaining entrance to the body through the influence of his work, as driver of an ash cart. As regards the cyst on the left side, this was evidently an old affair, as the condition of the veins and its own character proved. I allude to it as another

illustration, if one were needed, to show the absence of paralysis from lesion of the anterior lobe at that point. It very possibly stood in causative relation to the epileptic convulsions.

Typhoid fever rarely poses under the form of cerebral disease, owing to its longer forming stage, and the usual tympanitic abdomen, and diarrhœa. Yet, at times, patients seen late in the disease, without diarrhœa or eruption, may deceive a physician; or, on the other hand, the typhoid state, which precedes death in certain chronic brain diseases, as tumor, or in the multiple embolism of ulcerative endocarditis, or in tubercular meningitis, may mislead, if no careful history is obtained. I have known the headache, pain in the back and limbs, with some stiffness of the neck-muscles, associated with early delirium, give rise to inquiry as to possible meningitis, the bowels being constipated; and broncho-pneumonia, under such circumstances, to still further increase the doubt, by raising the suspicion of tubercular trouble in the lung. Moreover, I have known of instances in which, on tracing the origin of typhoid fever in an institution, cases of death were found which had been considered to be due to meningitis, but which from their arrangement with reference to the other cases made it probable that they were illustrations of this possible error; and in one of such groups typhoid fever occurred among the inmates of the house to which the invalid had gone.

I have on several occasions, finding the muscles very tender, sore and stiff, thought of the possibility of trichinæ, and if on inquiry the patient admitted eating raw ham or pork, and was free from delirium or marked stupor, as happened on two occasions, whilst waiting for the dilatation of the pupil I have taken out a small piece of the deltoid for microscopic examination, in order to exclude this as an accompanying disease, should the facts warrant a diagnosis of basilar meningitis. I will allude to one of these cases when speaking of Bright's disease. The freedom from delirium manifested by some individuals in the earlier stages of basilar meningitis is remarkable, though not surprising, when the freedom of the convexity is considered, and the absence at such time of marked pressure

effects. The examination of the muscle can be performed so easily that no objection need be entertained to it in appropriate cases.

Malarial diseases, fortunately, are not often mistaken for cerebral, though the converse is not true. For as regards those with paroxysmal headache, especially if conjoined with fever, a mistake is not unlikely to happen. Tumors, particularly, are apt to be attended with cephalalgia, and I have known it to be as periodic as a tertian. Later I shall narrate an illustrative instance bearing on another subject. The earlier stages of cerebral abscess, or of a pachymeningitis suppurativa externa, leading to abscess, or to septo-meningitis suppurativa, may be attended by both headache and hectic type of fever, which, as I had occasion to note this summer, may so simulate a malarial as to render a decision dependent upon a careful survey of all symptoms, and upon the presence of an otitis, an injury of the skull with bare bone, or of some other lesion exposing the bone to the influence of the atmosphere and germs floating in it. I have twice known so simple an operation as rhinoplastic, the skin being taken from the forehead, to follow the above course. Unfortunately, we meet cases of a severer type of malarial fever, so far as our city is concerned, usually coming from more tropical climates, which may lead in the comatose and convulsive types to the opinion that a cerebral disease exists. It is not impossible to have a coincidence of the two diseases; and the malarial may produce such a periodicity in the symptoms that the question may be raised which disease should be given the prominence in the treatment. I saw such a case last week, at Bellevue Hospital, in a man without a history, semi-unconscious, with marked rigidity of the back of the neck and some stiffness of the other muscles, but with a temperature of 104° F. on one day, 99° F. at the end of twelve hours, and 104° F. at the end of twenty-four hours. I could discover nothing else, on ophthalmoscopic examination, than swollen retinal veins and capillary congestion of the disc. The autopsy, two days later, showed basilar meningitis and the lesions of malarial fever. I might add that in

this case I examined a drop of blood from the finger and one from the spleen, but without finding true pigment. I have found, in my notes of autopsies, a previous instance of this same combination, cerebro-spinal meningitis with the pigmented liver and spleen, indicative of malarial fever. Where malarial fever is abundant and severe, it is probable that such combinations would not be found uncommon.

Pneumonia and pleurisy have been in numerous instances mistaken for meningitis and cerebral diseases. The former leads to this mistake much more frequently than the latter. It arises from the same condition as previously mentioned, an undue prominence of muscular pain and stiffness in the cervical region, together with a reaction of the nervous system to the fever out of proportion to what ordinarily obtains. The error is not nearly so likely to obtain in adults as in children, with their delicate nervous organizations. It is aided by the absence of the physical signs of pneumonia, which not infrequently begins in the centre of the lung and takes several days before it reaches the surface. I have, in former years, seen as many as three cases of this error in a single fortnight. He who desires to give a positive opinion on a first visit will not infrequently be misled in these and similar conditions. Moreover, it is not alone the central pneumonias which deceive, but those which begin in that portion of the lung high in the axillary region, or at the posterior summit of the lung. On the other hand, meningitis may coexist with pneumonia, or different cerebral diseases be complicated by its hypostatic or lobular types. The history and order of symptoms must be used in deciding such cases.

Bright's disease of the kidneys, more especially the small contracted type, in various ways tends to mislead the physician. It is a frequent accompaniment of cerebral hemorrhage, so that the rule of diagnosis sometimes given, that albumen and casts in the urine are to be allowed considerable importance in coming to a conclusion as to the cause of an apoplectic state, must be accepted in a guarded manner. I attach less weight to them than other considerations. These take into ac-

count the age of the patient, the probable type of kidney disease, whether associated with hypertrophied heart and arterial changes, and far more important, the presence or absence of paralysis, monoplegic, hemiplegic, or general. I have known this finding of albumen to mislead in different cases, where the extravasated blood was beneath the skull, on the surface of the brain from pachymeningitis hemorrhagica, or from rupture of an aneurism of one of the arteries, or of a vein going to one of the sinuses. Whilst at times we may have a transient paralysis following the convulsions of Bright's disease, and a recovery takes place in a manner to render it improbable that extravasation of blood had occurred; yet such occurrence necessarily must arouse a strong suspicion of hemorrhage or of some other morbid process to which the paralysis is connected in the relation of cause and effect.

Should there be any such method in the convulsion as is usually due to surface irritation of the brain, repeated convulsions beginning at one point, extending to one side, and limited there, and if such convulsion should be unattended with loss of consciousness, it would naturally lead to the opinion, if acute in its development, of hemorrhage in the cortex at its motor portion. If paresis or paralysis of the convulsed side were found in the time between the convulsive seizures, it would tend to confirm the diagnosis. I saw such a case recently at Bellevue Hospital in a female with a history of having become unconscious, of having a fever reaching 104° F., subsequently remitting, then convulsive seizures in the left side of the face, arm, and leg, with a paresis of these parts in the intervals between the paroxysms. These seizures lasted for two days. During the convulsions she was able to recognize and even to say a few words. Later she became entirely comatose. Her urine was scanty, albuminous, and contained casts. The autopsy showed considerable fluid blood and bloody serum in arachnoid sac. There was a firm blood clot adherent to the inner surface of the dura mater over the middle of the brain on the right side corresponding to the situation of the lower two-thirds of the vertical convolutions and the anterior extremity

of the temperosphenoidal lobe. There was only a thin layer over the upper third of the vertical convolutions.

The kidneys showed chronic diffuse nephritis.

The following case will illustrate a little different phase:

C. R——, aged thirty-six, was brought to the hospital unconscious. Those coming with him said that he had stayed at an hotel down town, had been a hard drinker, and had fallen in his room in the morning; then coming down-stairs, he had again fallen, striking his head against the bar; immediately after had convulsions, and that after these he became and had continued unconscious. When observed he was in coma, without stertor, pulse 80, regular, insensible to skin irritation, general paralysis, pupils equally dilated, not acting to light. The urine, drawn off by a catheter, was found loaded with albumen. Later, stertor developed, the pulse became frequent, and death occurred fourteen hours after the convulsion.

The autopsy showed a small, superficial clot on the left side in the arachnoid sac, and a much larger clot on the right side, compressing the brain. There was a clot in the longitudinal fissure also. His kidneys presented the appearance of chronic nephritis.

Of all the varied cases which I have met none has impressed me more strongly than the following, concerning the uncertainty of diagnosis:

Young man, sixteen years of age; had at first been sent to the insane pavilion owing to a wild delirium, during which he used very profane language. There he stated that he had had two convulsions, one several months, the other ten days before his admission, and that he had been treated for kidney trouble. Later, he became less delirious, so that in response to questions he seemed simply to fail in proper regard for his surroundings, being alternately profane and facetious in his response to questions. His marked symptoms were pain in the muscles on pressure out of proportion to cutaneous tenderness, marked rigidity of the muscles at the back of the neck, pupils contracted responsive to light, marked choked disc appearance in each eye, the œdematous infiltration being so great as to allow

the disc to be made out only by the course of the vessels. The hearing was good. The power was equal in the muscles on the two sides of the body, and there was no evidence of paralysis. The urine was retained. Later, he became unconscious, and on the next day was in that condition, with well-marked Cheyne-Stokes respiration, of seven-second pauses and six to eight respiratory acts between the pauses. One quart of urine was drawn from the bladder by catheter.

The autopsy showed that the spinal cord was normal, except that the cerebro-spinal fluid was stained reddish by blood-coloring matter, as was the pia mater of the cord posteriorly. The brain showed a clot of blood, three inches in length, by two in width, and one inch in thickness, in the lateral ventricle of left side, anterior horn and body, and a bloody serum in the other ventricles in quantity greater than normal. The hemorrhage came from a small rupture in the wall of the lateral ventricle at the anterior extremity of the tænia semicircularis. A transverse rupture, about three-quarters of an inch long, existed. The surrounding tissue was slightly blood-stained. In the centre of this little rent was a small miliary aneurism, from which the blood had come.

The heart showed left ventricular hypertrophy. The kidneys, though apparently large, owed their size to hydro-nephrosis. The structure was not only compressed, but of the small granular variety in structure. The pelvis showed a chronic inflammation, and contained calculi of mixed blood-clot and phosphatic nature. There was no stricture of the urethra nor any calculus in the bladder.

The phenomena observed during life seemed to warrant the diagnosis of basilar or cerebro-spinal meningitis in addition to any kidney trouble. I must confess to having been surprised at a ventricular hemorrhage instead, considering the age. It may be questioned whether the retinal appearances were due to the ventricular hemorrhage, or dependent upon the kidney disease, and of anterior date to the hemorrhage. As sight was not markedly impaired at the time of examination, his history could throw no light upon that point. The phenomena of

rigidity, etc., were to be referred to the irritation of the ventricles, and to the increased fluid in the cerebro-spinal canal.

It should be stated that the arteries of the brain did not appear diseased, so far as the circle of Willis and pia were concerned.

Fearing that if I continue to speak in the main of the results of *post-mortem* examinations, you may feel that the majority of the cases observed pass under that crucial test, I desire to enter the following case, because at first the optic nerve appearances led to the supposition of Bright's disease. The history was as follows:

Male, twenty-two years of age, without any family or personal tendency to disease. His habits had been good, and he was free from venereal disease. His first symptoms consisted of impaired eyesight, and the ophthalmoscope revealed appearances to the examiner which led him to suppose the disease due to Bright's disease. The whole subsequent course has, however, refuted that idea, and more than thirty examinations were made of the urine with negative results.

His eyesight improved, independent of any treatment, to such an extent that he was able to read for and pass a difficult examination. Shortly after it again became so impaired that he was unable to read type, but at the end of ten months from his first attack it again improved, until at the end of a year he was able to read. He enjoyed perfect physical health until one year and eight months from the inception of the disease; for during the spring he again passed a difficult examination, as he had done before. At the end of the time mentioned he was seized with a violent headache, frontal on each side, and with nausea and vomiting. This condition lasted for two weeks, and the complaints were most marked after sleep. At the end of this time he awoke from sleep delirious, remaining so for twenty-four hours. The delirium disappeared after free purgation. The headache, however, still continued, with vomiting, for five days. After the application of six leeches to frontal region and use of blisters, he was comfortable for four or five days. His pulse was 40 to 45, temperature normal, and secre-

tions healthy. Then headache and vomiting returned for several days, relieved by similar measures. This paroxysmal headache continued for four or five weeks, with two or three days of remissions and of attacks. Then the intervals increased, and the attacks grew shorter and milder. During this period he had several times sensations as if he were going to sleep. At times he had also noticed a sensation as if he had a hard, rough substance of the size of a pigeon's egg principally at the tips of the fingers, now in the mouth, cheeks, often across the forehead, rarely below the level of the chest. He had no mental impairment until one year and nine months from the beginning of the complaint. I first saw him at this time. He was emaciated, weak, and listless, lying on the sofa on account of headache. He was obliged to walk with two persons supporting him, as his legs tended to give way under him. He presented no difference on the two sides; all of the cranial nerves except the optic reacted well, and were free from lesion. He understood and answered questions only a little indifferently; he occasionally forgot the names for colors, yellow and violet. His eyes were bright, pupil reflex to light good. The sensation was perfect. The urine was voided slowly. In walking he had no vertigo or tendency to rotation, simply a tendency, unless supported, to sit down. The sight was much impaired, and the optic nerve presented the swollen or choked disc appearance. I heard at the end of two months that he grew worse, the mind becoming gradually more and more blunted, so that he simply recognized people while by him, but then lost all thought of them. He also had for some days a tremor in the right arm. After passing a couple of weeks in this state, he became much better in all respects, eyesight included, and was able to read, walk, etc., and gained flesh rapidly. He stated that during those two weeks everything appeared a dream, and that he had no recollection of it. For several months he remained much the same, save occasional attacks of headache and vomiting, and for a time had a tottering, unsteady gait. To make a long story short, he recovered, with complete loss of eyesight. At the end of three years he

was able to act as a telegraph operator. I think that his mental and motor symptoms were due to ventricular distension, brought about by the lesion which produced the optic neuritis. I at first feared a tumor. The freedom from other symptoms than those affecting the sight would, as both eyes participated, put the lesion, perhaps, in the corpora quadrigemina, and then the secondary motor and mental phenomena be due to some cause which produced, in consequence of pressure or inflammation, dropsy of the ventricles.

The question of locating cerebral disease and of deciding upon its nature must ever be a matter of interest to the physician. I have had several cases which are possessed of enough interest to present them, notwithstanding the numbers of similar nature which are accumulating in the literature of this subject. Before doing so I would draw attention to certain points which it is always necessary to bear in mind when attempting this problem. At first we are obliged to decide whether it is probable that the disease is of itself inducing the symptoms, or whether these are partly or mainly due to the irritation or softening induced by the tumor, etc. This is at times of practical value, as a surgical operation may depend in some measure for the site of its performance upon the answer. As an illustration, I will suppose that a man has been injured in a given portion of the head, it may be anterior or posterior to the motor tract, but that after a time motor symptoms become added in the form of hemiparesis or monoplegia. The symptoms being supposed to indicate an abscess, is this seated beneath the site of the wound, and are the symptoms due to spreading inflammation reaching the tract in question? Such an answer can only be given by a careful consideration of all the facts. The evident existence of symptoms of trouble in the brain before the motor symptoms occurred would point toward an outside process, increasing till it had involved the area presiding over motor functions. When a foreign body has entered the brain and remained without motor symptoms until after extraction, as in the case recently occurring in the practice of, and published by Dr. Noyes, then the probabilities would be

strong that the site occupied by the foreign body would form the centre of the new disturbance. The same explanation has to be adopted for motor symptoms added to those already existing, which, perhaps, have for a long time indicated a cerebral tumor. In the next place the question will arise whether the morbid process is diffused, as in meningitis, or local, and the symptoms present indicate the extent of the lesion. We will find that meningitis, though general, may produce effects which give it the aspect of a local process in some respects. Somewhat similar is the possibility, in a given case, that there may be several centres of disturbance, as in multiple embolism, tumors, and abscesses. Then, again, in so far as the decision of the value of a given case in settling the functions of a definite area of cerebral cortex is concerned, the requirements must be very precise. But we may add cases which, though not sufficient for this purpose, increase somewhat the weight of these more precise cases. For the first purpose cases are needed which are limited in area, stationary in character, and not passing to any considerable extent beyond the limits of the gray matter. Some of the cases, in fact many, which have been used are open to objections on these scores. Then, again, as regards others which seem to come closer to the requirements, the question may be raised, as about some of Exner's series, whether though so localized by the appearance of the softened patch, vessel obstruction may not have produced some results beyond those due to the softened patch. I might briefly indicate the methods which have been employed in deciding the question of special areas in the cerebral cortex having definite functions.

We may discard at present the localization of the speech centre, or rather, as some prefer to say, the emissive centre, in contradistinction to the perceptive centres, auditory and optic, which date back to the publications of Gall, Bouilland, Dax, and Bevan. The impetus was given by the experiments on animals by Hitzig and Ferrier, the electrical irritation of certain portions being followed by motions in certain parts of the body. Then followed the attempt to prove that a lesion in a

given site induced symptoms differing from those due to a lesion situated elsewhere, and hence the accumulation of two sets of cases—the positive, proving irritation or paralysis due to a lesion situated in a given portion of the so-called motor portion, and the negative, in which lesions situated elsewhere failed to produce such results. The same method is used with relation to surface areas of sensation, sight, and hearing. In the next place cases, though few in number, are found, which show that these so-called motor areas underwent atrophy if the arm or leg over whose functions they presided had been lost for a long time. So again other few are met which exhibit, along with the destruction of these motor areas and subjacent fibres, descending degeneration descending into the internal capsule and pyramidal tract, as in the case of the destruction of the internal capsule.

As regards these series of proofs I desire to record the following:

The first was that of a man who was under my observation for two years. He was only able to say two words, *ja* and *nein*, the German for *yes* and *no;* yet he was bright and intelligent. He had no paralysis of arm, leg, face, or tongue. He was simply affected with aphasia and agraphia, the latter to a less extent than the former, though not to any such extent of difference as to serve him in giving me any idea of his past life or how he came to be affected as he was. He had on the left side of his head, in the position of the inferior and anterior position of the parietal bone, a triangular depression capable of admitting the end of the little finger. When asked how this had been brought about, he expressed himself by gestures, about which there could be no doubt. He at first imitated a man charging a hole drilled in a rock, the lighting of a match and the application of it, then the too sudden discharge, and the falling of something on his head. After this he would imitate his being rendered insensible. He made no improvement as far as his aphasia was concerned.

The next case is that of Patrick Quigley, aged forty-seven, a native of the United States. He was well until February

7th, when he was struck with a billiard cue in the left fronto-temporal region. He fell and lay for about twenty minutes motionless and unconscious. On coming to himself he had considerable pain and swelling at the seat of the blow, and found that he could not speak, and that on swallowing water it flowed out of his lips. There was no loss of power in the arms or legs. When seen three days later, there was a swelling at the left temple, but no wound. He had severe pain in the head. There was a paresis of the muscles of the right side of tongue and of face below the eye (facial paralysis of cerebral type). There was no paralysis of the right arm or leg, nor of the opposite side. He was unable to repeat his name, to name an object shown him, and repeat what was said to him. On attempting to write his name, or some other simple and familiar word, he made some of the letters right, but either omitted, transposed, or substituted in the case of others, and manifested marked signs of annoyance at this failure. I, moreover, tested him as to his perception of words, and found that he understood the difference between the right and wrong pronunciation of the word.

The treatment consisted in quiet, a blister to the back of the neck, cold at the point of injury, laxatives and the use of iodide of potassium. He made a comparatively rapid recovery, so that at the end of eight days he was able to speak almost any word, though he did it with considerable stammering, effort, and hesitation. The headache, vertigo, and facial paralysis also gradually improved, so that he was discharged at the end of six weeks without any paralysis of the face or tongue, with scarcely noticeable aphasia or agraphia. He, however, said that he forgot the name and spelling of some words, and that at the time of his illness part of his trouble was due to the fact that he could not make his tongue or hand say or write what he wished, and another to a forgetfulness of the right word or letter.

Cornelius Keating, thirteen years of age. The father died of cancer of the stomach, at fifty-two years of age, but beyond this the family history, as far as known, was free from hereditary taint.

The boy stated that a little time before his illness he was struck in the head with a stone. Then he had a fall down some ten steps at school, coming on head; then, on getting up, he ran against a door with his head. A month later, while running, he tripped, fell, and struck his head against a curb-stone, receiving a black eye. Two weeks after this he began to complain of severe headaches, and his mother noticed that his left leg was a little weaker than the right. He could not lift it so well, and dragged it a little. The left arm also, according to the boy, was a little affected, and that before the leg.

The mother says also that at times she thought that there was a little squint in the left eye. The headaches continued, occurring three times a week and lasting most of the day. With these he would vomit what he ate. Involuntary starts have also occurred in the shoulder, arm, and hand, muscles jerking the arm up. These occur from once to several times a week. About four months after the beginning of these symptoms his sight began to fail, until at the end of six weeks he had become almost blind. No convulsions had affected him, nor had he any trouble with the bladder or bowels. I examined him about six months after the disease had begun, with the following results: The boy was of medium size for his age, his left pupil was dilated, not reacting to light, and he was unable even to perceive light in this eye. His right pupil was dilated, reacted sluggishly to light, and he could perceive light impression on a large object. He had double optic neuritis, marked choked disc, with swollen veins.

The hearing in the left ear was only one-half that of the right. The sense of smell was impaired in the right nostril, but only for fragrance, as alcohol was perceived on both sides, though a perfume distinguishable on the left readily could not be told on the right.

The movements of the left arm were slow, stiff, uncertain. The dynamometer gave 30° on the left side, 50° on the right. The movements of the leg were slow, and of the same peculiarity as the arm, being weaker than its fellow. There was no

difference of tactile sensibility on the two sides, nor on face. He had a pain in the left leg, when he extended it, of a stretching character. This examination was made August, 1877.

The boy died April 21, 1878, at his home. The friends stated that he had continued in the same condition, with headache, vomiting, blindness, left hemiplegia, and intense pain in the right leg. Three days before death he became partially comatose with fever.

The bones of the skull were thinned, and presented spaces where absorption had occurred in parietal of right side, and also in lambdoidal suture.

The dura mater was firmly applied to skull, and on its interior showed the markings for convolutions. On section of the dura mater the brain projected, as if the space had been previously too small. The arachnoid was dry. The convolutions were flattened against the skull on both sides. A portion of a tumor appeared in the right posterior central convolution, near the longitudinal fissure, while the central half of this convolution was cyst-like from softening. A tumor of lobular structure, light grayish color, comparatively firm, occupied the white matter of the right hemisphere in the following portion. It reached in the longitudinal fissure from the corpus callosum to surface, appearing at the point indicated above in the posterior central convolution. On section it was found to have the size of a small orange. It reached forward as far as the first and second convolutions (frontal), backward into the precuneus. It slightly involved posterior fibres of corpus callosum, and reached as far backward as this.

The convolutions especially affected were the anterior and posterior central convolutions, the latter especially, except its lower portion, being pretty well destroyed, the posterior part of the gyrus fornicatus, the posterior part of the first frontal, the paracentral lobule and the anterior portion of the precuneus. The brain around the tumor was markedly softened, and this softening affected the corona radiata fibres and those in the posterior part of the corpus callosum. The lateral ventricles contained an increased quantity of clear serum, the left

being the larger. There was no other growth or lesion of brain. The right optic nerve was smaller than the left.

The tumor was a glioma, with round and branched cells.

The softening was probably an event of the few days previous to death, during which he had fever.

The following case shows that pressure on the surface of one side, particularly on the paracentral lobule and its neighborhood, may cause hemiparesis and convulsions:

P. M——, male, aged twenty years, was admitted to Bellevue Hospital, having received an injury on his head three weeks before. The wound, which had nearly healed, showed a fistulous opening leading to bare bone. It was situated on the right side over the portion of frontal bone at the point where the hair begins, and near the median line. He had paresis of the left side, arm, and leg, the latter being the most affected. The only voluntary power which he could exert was to grasp with the fingers when told. Flexion was more powerful than extension. He could slightly flex forearm, but could not extend it. He had no ability to move foot or leg when told to do so. There was no apparent paresis of face or tongue, but the sensation of the affected side was blunted. The next day he became almost comatose, and later developed convulsions. During two which I witnessed the paralyzed side was the site of predominant rigid spasm, the body being arched to that site. During the continuance he winked on touching the conjunctiva of the right side. After the convulsion the paresis continued as before, but he did not talk, though exerting some movements when told, and whispering some words. I considered that the decision lay between abscess and meningitis, with predominant affection of the paracentral lobule in consequence of the greater implication of the leg and foot than of the arm and hand. Professor Alexander Mott performed the operation of trephining, and exploration was made for pus beneath dura with hypodermic syringe needle. There was a suppurative pachymeningitis beneath bare bone. The exploration only succeeded, though the needle was introduced directly downward and then backward toward paracentral lobule, in obtain-

ing a few drops of thick dark blood. It seemed almost useless to make any further operation, as the convulsions pointed toward meningitis as the more probable cause; yet a second exploration was made and also failed. A convulsion during operation showed pleurosthotonos first on one and then on the other side.

The autopsy showed suppurative pachymeningitis interna, the pus being in sac between arachnoid and dura, particularly that portion between falx and paracentral lobule. The whole surface of falx and of arachnoid over longitudinal fissure convolutions was covered with pus, yet the greatest was at the point indicated, and it had here made a marked depression on the inner aspect of the paracentral lobule. This pus was held by falx, lobule, and some fibrin contained in exudation. These lesions were on the right side. A thin layer of pus was also present on the convexity of this and the opposite side, and on the falx on that side. There was a beginning leptomeningitis over the first frontal and over the upper extremities of the anterior and posterior central convolutions. In the anterior lobe medullary portion of first frontal convolution there was a dark clot of blood, posterior to site of injury and about two inches long by one inch wide. Its walls were in the main smooth, though above and to the outer side there were punctate extravasations.

The points of interest in this are the following:

The proximal paresis of the arm was greater than the distal; the paresis of the leg absolute, and hence greater than of arm. Again, that the lesions situated at the upper portion of central convolutions, as in so many other cases, produced paralysis of limbs, not of face or tongue. The purulent pachy- and beginning leptomeningitis show that no operation would have benefited patient.

The patient was under the care of Prof. Mott and Dr. Bull.

A. C——, aged forty-five years; Ireland; domestic.

The patient was brought to Bellevue Hospital August 10, 1874, with the history that she had been thrown out of a window on the 6th, in a drunken brawl. She had a scalp wound

one inch long over the left parietal bone, not exposing it, and a fracture of the olecranon, with a small communicating cut.

When admitted the patient was stupid, answered questions in monosyllables, had no apparent paralysis, pulse feeble.

She gradually grew more stupid, with increasing rapidity of pulse, passed urine involuntarily, failed to answer questions, but would protrude tongue when told. She died at the end of four days. The autopsy showed that the patient had chronic diffuse nephritis, an hypertrophied and dilated heart, and somewhat calcified mitral valves. The brain showed some œdema of pia mater, and slight distension of lateral ventricles with clear serum. There was a tumor about the size of a pullet's egg situated in the left middle fossa of the skull, pressing on the anterior extremity of temporo-sphenoidal lobe, and on the left optic nerve. The tumor grew from the dura mater, and was a spindle cell sarcoma. A number of years ago I made an examination of an old woman, an inmate of the almshouse, in the anterior lobe of whose brain there was a tumor the size of a small orange, a fibrous glioma with mucoid softened centre, around the outer part of which the brain was softened. This tumor was in the white mater of the frontal lobe, anterior to the central convolutions. It was affirmed that she had not been paralyzed on either side, but that while walking she had a fall, became unconscious, and died of a supposed cerebral hemorrhage.

Male.—This patient had been affected with cirrhosis during life, and had died from its results. He had also a paralysis of the sixth and of the ninth or hypoglossal nerves on the left side.

The autopsy showed a pachymeningitis of slight amount on convexity. It, however, stretched to the base, and at the point of emergence of the ninth and sixth nerves compressed these. The sixth on the left side was also more firmly adherent to its fellow from its point of emergence through dura till its entrance in sphenoidal fissure. There was partial atrophy of the left half of tongue, with a granular or granulo-fatty state of muscle fibres or simple atrophy.

The new membrane on the dura was made of connective tissue, and contained capillaries and pigment. The liver was markedly cirrhotic, the spleen enlarged, and the muscles of the body contained a few encapsulated trichinæ.

I publish this case because at times it is claimed that these nerves are not implicated in this process.

I have presented to the Pathological Society the brain of H. G——, in which there was a tumor in much the same situation as in the case of the boy, with identical symptoms, hemiparesis of arm and leg, advancing to complete hemiplegia of these parts. Also, as bearing on a point raised by Professor Dalton of the cause of recovery in destruction of the motor area—that of a boy with congenital heart disease, the aorta coming from the right and the pulmonary artery from the left ventricle. There was a thrombus in the right ventricle, and the emboli from this led to the destruction of the brain supplied by the anterior and middle cerebral arteries on the left side, so that these parts were represented by a pultaceous mass. Dr. Castle, who brought the brain to me, said that this child had at first been completely hemiplegic and aphasic, but later had recovered somewhat, so that it could manage to walk and use its hand and arm somewhat, as well as to say "Mamma" and "Papa." He also said that it had so far improved as to produce the impress of reflex paralysis on a neurologist who saw it, and that he desired to circumcise the boy in the hope of a cure.

ON EXCISION OF THE CHANCRE AS A MEANS OF ABORTING SYPHILIS.

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(Read November 16, 1882.)

Is it possible, by excision of the initial lesion, to destroy the syphilitic virus and prevent infection of the general system? Such is the question which, at the present time, is engaging the serious attention of syphilographers both in this country and Europe.

Perhaps no problem connected with syphilis has been found so difficult of definite solution as the prophylactic value of destruction of the chancre. From the time of Jean de Vigo (1503), who recommended cauterization, until the present, the idea that the syphilitic virus might be entirely suppressed, or the severity of its constitutional effects modified, by destruction of the primary sore, has been received with more or less favor by the profession. John Hunter, who regarded the chancre as a purely local affection, was accustomed to cauterize or extirpate it. This was also the practice of Benjamin Bell, who refers to this mode of treatment as the one generally employed at his time. Ricord was for many years a most enthusiastic advocate for the early destruction of the chancre, either by caustics or excision, and we may regard it as the almost universal practice until a comparatively recent date. Within a few years past, however, we find that professional sentiment has undergone a complete change in this regard. The principles upon which the abortive treatment was based have been generally recognized as founded in error; it has ceased to be recommended in standard text-books, and its practice virtually abandoned.

In 1877, however, a communication published by Auspitz and Unna, reciting many cases in which they had excised the initial lesion with the result of preventing constitutional manifestations, gave a fresh impulse to the examination of this question. Since then numerous excisions have been made by different experimenters, with results more or less favorable, the bearing of which upon the question at issue we propose to consider in this article.

In Germany, where the so-called abortive method of treatment was revived, we find that the current of professional opinion is largely in its favor. Among its partisans are numbered some of the most prominent specialists, such as Auspitz, Unna, Kölliker, and others—men whose position in the medical world invests their opinion with the weight of high authority. In France it has by no means received the same prompt recognition and favor; its advocates are fewer in number and urge its claims with less pretension. In England the operation seems to have been practically ignored, or its results have not been of so brilliant a character as to justify their publication; at least we find no mention made of them in the current British journals. In this country excision does not seem to have been often practised. Some of our specialists have expressed themselves guardedly in its favor in certain selected cases, but the general sentiment seems to have been adverse to its adoption as a means of cure.

Before entering into an examination of the arguments urged for and against this method of treatment, let us state the position and claims of the partisans of excision. The operative procedure may first be briefly described. The chancre is firmly grasped with a pair of forceps or tenaculum and uplifted so as to put the parts on the stretch. The indurated mass is then removed by a clean sweep of the knife, with an area of healthy tissue immediately surrounding it. Formerly curved scissors (Hüter's) were used, but they have been laid aside for the knife. The wound is then sutured and covered with carbolized dressings, and a compressive bandage applied to prevent swelling. Usually there is but little hemorrhage, and the wound

often heals by first intention. The loss of tissue rarely occasions much deformity or interference with the function of the part. Not infrequently a subsequent induration occurs in the cicatrix, which is accepted by most operators as an indication for its removal by a second excision.

While admitting that the rationale of the operation involves nothing new in principle, the excisionists claim that they effect thoroughly and completely with the knife what was imperfectly done by caustics and other means; asserting that it is difficult, if not impossible, to penetrate to the bottom of the indurated mass and radically destroy certain chancres, even with powerful caustics or the hot iron.

They claim that, in a large proportion of cases, excision of the chancre effects a radical cure of syphilis; a cure attested by the fact that the most careful surveillance, continued through a period of months or even years, fails to detect the slightest constitutional manifestation of the disease; a cure rendered certain, beyond all possibility of doubt, by the patient's susceptibility to a fresh inoculation of the syphilitic virus, as frequently demonstrated by experiment.

They claim, further, that, even where a complete destruction of the virus is not effected by excision of the chancre which contains it, it becomes attenuated, emasculated, deprived of its potentiality for mischief, so that the resulting syphilis will be of a milder type, as shown by the delay in the appearance of secondary symptoms and their general character of benignity.

Another advantage claimed for excision of the chancre is that, even though its abortive effects be negative, even though it fails to arrest the diffusion of the poison through the system, still it is to be recommended as a local adjuvant; it acts advantageously by converting a hard unhealthy sore into a simple wound, which often heals by first intention.

The indications for excision are not precised with clearness, but are variously stated by the different operators. Obviously enough, the earlier the excision is done the greater the chances of the patient's escape from subsequent infection. By Auspitz,

Kölliker, Hüter, and others, the induration of the chancre, even consecutive adenitis, is not regarded as a contra-indication of the operation. They assert that it may be undertaken, with hope of success, at any time previous to the appearance of secondary symptoms. Some of its more enthusiastic advocates recommend excision of the involved glands as well as the initial sclerosis.

From a theoretical standpoint, the whole question hinges upon the determination of the nature of the primary sore. If, as is contended for by the partisans of excision, the syphilitic virus remains localized at its point of entrance during the period of the first incubation (its primary effect being limited to the development of the chancre, from which the system does not become infected until after the second incubation), then it is quite possible, by removing the depôt of the virus, to prevent constitutional contamination.

If, on the other hand, the initial lesion be the local expression of a constitutional condition, the evidence of an already accomplished infection which follows immediately the inoculation of the virus, then it is manifestly impossible, by excision or other local means, to arrest or modify its effects upon the general system.

In endeavoring to appreciate the pathological character of the initial lesion, we are embarrassed by our absolute ignorance of the nature of the virus which produces it. Whether the infectious principle be a pathological ferment, a micro-organism, or some other undemonstrated specific entity, we can only conjecture. It cannot be isolated and examined by anatomical or chemical tests; the most minute microscopic analysis fails to detect its presence in the fluids which contain it; our knowledge is altogether limited to its effects upon the organism.

As regards the physiology of syphilitic infection, much has been written and many theories broached. We will not consider at length the mode of penetration of the virus into the economy, whether through the intermediary of the lymphatic vessels, as has generally been claimed, or whether by gradual

implication of the blood-vessels contiguous to its point of entrance, as has been asserted by Auspitz and Unna. We are concerned with the celerity rather than the course of its passage.

To bring the points of issue prominently into relief, let us consider the ordinary evolution of syphilis. It may be stated:

That the first appreciable phenomenon which results from the inoculation of the syphilitic virus is always manifested at the point of infection after a period more or less prolonged, on the average from three to four weeks (period of first incubation).

That this primary lesion constitutes for a time the sole sign, the unique expression of the disease.

That after a certain lapse of time, during which the contiguous lymphatic glands undergo an indolent enlargement (period of second incubation), there is an eruption of the so-called secondary symptoms, which may be generalized over the entire body.

Now, the question arises, At what particular time does generalization of the virus take place; at the moment of inoculation, with the apparition of the chancre, with the engorgement of the lymphatic glands, or immediately preceding the explosion of general symptoms? At the first glance, it would seem that the theory upon which the practice of excision is based was the correct one. Judged by its visible manifestations, and the regular order of their succession, the syphilitic virus remains latent, more or less inactive, for a time, at its point of entrance. It is powerless to proceed further until it has gathered strength by repose and germination. Its first effect is irritation of the cells in immediate proximity, resulting in the hyperplasia which constitutes the initial sclerosis. This proliferation of cells continues affecting, first, the *adventitia*, then the *intima* of the contiguous vessels. By gradual implication of the vessels, the poison proceeds until it reaches the nearest lymphatic glands; here, after another period of repose and multiplication of cells, it gains an added potentiality which enables it to invade the entire organism. This concep-

tion of the evolution of the disease regards the chancre as the depôt of the virus, within which for a certain time it is encapsuled, so to speak, and from which, as a source, infection propagates itself to the entire system. To excise the chancre, then, is to suppress the source of contagion.

The theory that the action of the virus is primarily limited to its point of insertion is further supported, it is claimed, by analogy. Tuberculosis, for example, has been referred to as having many points of similarity with syphilis, and the well-known experiment of Conheim has been cited, who introduced into the anterior chamber of the eye of a rabbit a drop of tuberculous matter, and watched the result. Absolutely no change occurred until the eighth day, when he observed a congestion, with subsequent formation of tissue, analogous to that of tubercle. Tuberculous masses were afterward found in the lungs and other organs. In this case general infection did not take place until after an incubation of eight days. Cancer, it is claimed, shows, in its primary development as a local disease and subsequent invasion of the system, a resemblance to the evolution of syphilis.

The theory of immediate infection through the lymphatics is objected to by Auspitz, on the ground that it involves the assumption that the virus first traverses the lymphatic system, enters the general circulation, then returns to develop the characteristic lesion at its point of entrance, and then slowly, for the second time, makes its way along the lymphatic vessels, producing irritation, engorgement, and other changes. If the virus be generally diffused through the system, why should it always first manifest its presence at the point of inoculation and not at another portion of the integument? Again, if the disease be a constitutional one from the first, why await the appearance of the inevitable secondary symptoms before commencing the administration of specific treatment?

Finally, the partisans of excision justify their practice upon clinical experiment, the results of which will be examined later.

Opposed to the theory upon which the practice of excision

is based, there are many facts drawn from analogy, from experiment, and from clinical experience.

So far as our knowledge of the mode of action of the *contagia* of other diseases, the acute exanthemata for example, may throw light upon the operation of the syphilitic virus, it tends to confirm the assumption that the infection takes place from the moment the virus gains access to the system. In these diseases, although infection immediately follows exposure, there is at first no derangement of the general health, indicated either by local or general symptoms, and the effects only declare themselves after a period of incubation, more or less prolonged, according to the nature of the specific poison and the reaction of the organism.

The vaccine virus affects the system in a manner analogous to that of the syphilitic virus. They both produce lesions at the point of inoculation, which appear after a certain period of incubation. These lesions, the matured pustule and the initial sclerosis, each represent the acme of development of the morbid process producing them, and should, in one case as in the other, be considered evidence of the complete saturation of the system. In regard to the vaccine virus, it may be considered as definitely settled by the experiments of Bosquet and Steinbrenner that, from the moment of its inoculation, it is rapidly absorbed and general infection begins. These experiments are supplemented by those of Aime Martin (1863), who vaccinated seven children, and cauterized the punctures at intervals ranging from one to twenty hours after the inoculation. Although the cauterization prevented the development of typical pustules, it none the less failed to arrest the diffusion of the virus through the system, as shown by insusceptibility to revaccination. Monneret vaccinated fourteen cases, and revaccinated them from the first to the eighth day afterward. The second operation failed to produce vaccinal pustules when performed later than the second day. These experiments would seem to show conclusively that "receptivity is extinguished even before the appearance of any eruptive accident." Analogically, we might infer the same for syphilis. Whether the syphilitic virus

permeates the system so completely, before the appearance of the chancre, as to exhaust its susceptibility to a fresh inoculation, is not pertinent to our present inquiry; and, besides, is of no practical value, as we have to deal with the disease only after the development of the primitive accident. The important fact that the chancre is not inoculable upon the person bearing it has been abundantly proven by numerous experiments. Among the hundreds and thousands of cases in which auto-inoculation has been attempted, the successes recorded have been so insignificantly few that they cannot be held to invalidate the proposition that the chancre is, as a rule, non auto-inoculable.

Experiments with the virus of other diseases might be referred to, illustrating the instantaneity of absorption. Renault d'Alfert inoculated twenty-two sheep with the sheep-pox poison, and although the wounds were thoroughly cauterized within from five to thirty minutes after inoculation, the animals were all attacked with the disease. The same experimenter inoculated thirteen horses with the poison of glanders. The wounds were all cauterized within short intervals, varying from one to twenty-four hours afterward, yet they all died from the disease.

Glanders has certain analogies with syphilis. The so-called horse-chancre manifests itself at the point of inoculation after a period of incubation. There is a subsequent swelling of the nearest lymphatic glands, and later, the characteristic nodules become generalized.

Cauterization of the bite of a mad dog, though done immediately, and with all possible thoroughness, does not afford protection against hydrophobia. Other examples might be adduced, all tending to prove the utter uselessness of attempting to arrest the diffusion of a poison when once it has gained entrance to the circulation.

Turning now from the teachings of analogy and deductions drawn from experiments with animal poisons, let us examine the results of clinical experience. We have seen from a glance at the literature of syphilis that the theory of the local nature of the chancre was formerly generally accepted by the pro-

fession, and, as a logical sequence, its destruction by caustics or other means was the classic treatment. Benjamin Bell, who wrote in 1793, says: "The practice of healing chancres by the internal use of mercury originated from an opinion that venereal sores proceeded from the constitution being infected, and, were this the case, there might be cause to consider it well founded; but now that we know that chancres are always *local*, and that they are the *source* of whatever matter enters the system, it is obvious that the more speedily they can with propriety be healed, the less will be the risk of the constitution being injured." John Hunter may be quoted to much the same effect. Ricord says: "If chancre be at first a local affection, as Hunter asserts, and as observation and experience prove it to be, we must be consistent, and treat it as everybody treats the bite of a mad dog, that is, destroy the local disease as soon as possible." Again he says: "The primary influence of the infecting chancre being limited to the region which it affects, general infection is not an immediate and instantaneous result; it is an accident consecutive to the development of the chancre, and which requires a certain time to produce itself. In destroying a chancre at its débût, a chancre which is about to become indurated, you can at the same stroke exhaust the source of constitutional infection. . . ." "Excision may effect what cauterization does; it is, in fact, an excellent method which suppresses the chancre at a single stroke." He declares that "of all the chancres which I have seen cauterized or I myself have cauterized from the first to the fourth day of contagion, not one has been followed by symptoms characteristic of constitutional infection." Sigmund's testimony upon this point is equally emphatic. He says: "The observations of more than a thousand cases in a period of eleven years has shown me that never have secondary accidents declared themselves when the chancre had been completely destroyed within the first four days."

Statements so strong and positive, coming from the leading representatives of the French and German schools, are in startling contrast with the results of treatment at the present day.

But from the advanced standpoint of our knowledge of the normal evolution of syphilis, we know that these claims are based upon a wrong interpretation of clinical facts. Inoculative experiments, as well as clinical observation, prove conclusively that a period of incubation, on the average twenty days, always intervenes between the reception of the virus and the appearance of the initial lesion, and when Ricord speaks of cauterizing a chancre from the first to the fourth day after contagion, he utters an absurdity, for the obvious reason that the chancre has no existence at that time.

It will be granted that evidence drawn from clinical experience before the distinction between chancre and chancroid was recognized is without value, since the destruction of a venereal sore and the subsequent immunity of the patient from syphilis do not necessarily stand in the relation of cause and effect. If the sore happened to be a chancroid, constitutional manifestations would not have followed in any case; hence the exemption from syphilis would argue, not the efficacy of the treatment, but the non-infectious character of the sore. Even after Basserau had enunciated his dualistic theory, and indicated with precision the characteristics which differentiated the two forms of sore, we find that Ricord still advocated the abortive treatment, but with a limitation as to its applicability. He had modified his former views as to the chancre being the source of infection, and taught that the treatment, to be effective, must be applied before induration takes place. He says: "From the moment that induration is produced, the disease is acquired, and from that time you may cauterize or you may excise the chancre—you only destroy a symptom without preventing the diathesis. It is, therefore, less appropriate to regard the induration as the origin of the syphilis than to consider it a consequence; it is less a cause than an effect."

The rule of treatment laid down by Ricord, it may be remarked, is coupled with a condition which is equivalent, practically, to a rejection of all abortive attempts, except in those rare cases where confrontation is possible. All syphilographers recognize the fact that there are absolutely no distinctive signs

which would enable the most skilful diagnostician to pronounce upon the syphilitic character of a venereal sore before induration takes place. Fournier says "the incipient chancre is the most insignificant of erosions, an erosion without distinctive character, without special physiognomy, absolutely deficient in every sign sufficient to differentiate it from a common erosion. . . . When one has cauterized an incipient chancre, one never knows certainly what he cauterized." He concludes that "cauterization, destruction of the chancre as a preventive, abortive means against syphilis, is purely illusory. To suppress the chancre is not to suppress the diathesis. The diathesis is acquired when the chancre is produced, and the chancre in definitive is only the first phenomenon of confirmed syphilis." Even when it is possible to determine the syphilitic character of a sore upon its first appearance by confrontation, the facts of clinical experience teach us that its immediate destruction would offer no guarantee against the subsequent development of constitutional symptoms.

Attempts to neutralize the effect of the syphilitic virus by disinfecting washes applied immediately after contact have proven notoriously unsuccessful. In this connection we may quote a case reported by Jullien of a physician whose finger, upon which there was a minute abrasion, came in contact with the chancre of a patient he was examining. "The moment Dr. X—— perceived it, he had recourse to the most careful and thorough washing, and endeavored to remove from the wound everything which had been deposited on its surface. It was in vain; the progress of the virus had already commenced, and infection followed its course. This fact, which the scientific position of its author places above all doubt, establishes categorically, it seems to us, that absorption begins at the same time that the act of contagion takes place."

Berkley Hill cauterized with fuming nitric acid a torn frænum within twelve hours after intercourse; nevertheless an indurated sore appeared twenty-eight days later, followed by general symptoms. Langston Parker reports a case where a chancre was effectually destroyed within one hour after its

appearance, yet a more severe attack of syphilis followed in due time. Diday applied caustic to a chancre within six hours after its appearance, the sore healed kindly, but general syphilis followed after the classic delay. But why multiply instances? Medical literature abounds in cases where the most energetic destructive treatment, from the moment the chancre appeared, utterly failed to prevent or even retard the outbreak of constitutional symptoms.

From the foregoing considerations, and others which might be cited, the large majority of syphilograpers have come to believe that it is contrary to the teachings of analogy, contrary to the facts of experiment, that a virus introduced beneath the integument should remain for several weeks isolated in the tissues without mingling with the blood or lymph ; while careful clinical observation, based upon a clearer comprehension of the laws of infection and the relations of the chancre to general syphilis, has culminated in the conviction that destructive cauterization is absolutely useless as an abortive measure.

Upon these points there is a remarkable unanimity of sentiment among authorities, as reference to standard text-books on Venereal will show. Cazenave, Rollet, Langlebert, Diday, Fournier, Jullien, Bärensprung, Lewin, Zeissl, H. Lee, Bumstead, Keyes, and others, all testify to the same effect. Finally, upon this point may be quoted the latest utterance of the eminent syphilographer, whose genius has solved so many of the difficult problems connected with syphilis, and whose name is identified with every important advance made in the progress of this specialty during the last half-century. In an oral communication to Leloir (*Annales de Dermatologie et de Syphiligraphie,* 1881), Ricord declared that " he was completely converted from the practice of cauterizing or excising chancres. He regarded the destruction of the infecting chancre as absolutely useless at whatever epoch of its existence it may be. As soon as it has appeared, before its appearance even, syphilis exists. In vain you might amputate the patient's penis on the appearance of the infecting chancre, syphilis would none the less declare itself."

From this survey of the subject, we see that the verdict of enlightened experience condemns the cauterization of the chancre as a prophylactic measure, and so far as clinical experience can settle a question, it may be considered settled by the overwhelming testimony of careful and competent observers. But on applying this evidence against the practice of excision, we are confronted with the assertion that the cases are not parallel, that extirpation of a chancre with the knife is radically different from its destruction by caustics. While failing to appreciate the force of this argument, which seems to be based upon a distinction of means rather than upon a difference of effect, we will now examine the published results of excision as bearing upon the question at issue.

Obviously enough, this examination can embrace only a small proportion of all the cases in which excision has been performed, as we have seen that it was a recognized procedure among the older specialists. But these earlier records, even were they available, would be without value, since, as before pointed out, testimony as to the results of abortive treatment before the line of separation between chancre and chancroid was rigidly drawn is untrustworthy. Coming now to recent times, we find that Humphry and Neale, in 1864, strongly recommended excision of the chancre, but they fail to publish results which would enable us to appreciate its value from a statistical standpoint. The tabulated list which follows includes all cases reported in the various medical publications accessible to me since 1867.

Operator.	Authority.	No. of cases.	Successes.
Hüter	(Berliner klin. Wochenschrift, 1867)	8	2
Langenbeck	(Idem, 1867)	2	1
Coulson	(Treatise on Syphilis, London, 1869)	1	0
Thiry	(Presse Médicale Belge, 1870)	1	0
Kuzlinski	(Thèse de Greifswald, 1874)	1	1
Caspary	(Vierteljahresschrift f. Dermat. u. Syph., 1876)	3	0
Auspitz u. Unna	(Idem, 1877)	33	14
Kölliker	(Centralblatt für Chirurg., 1878)	8	3
Prospelow	(Moscow Med. Gaz., 1878)	3	3
Rydigier	(Leczience Chirurg., 1879)	3	2

Operator.	Authority.	No. of cases.	Successes.
Pick	(Arch. f. Ex. Pathol. u. Pharm., 1879)	1+?	1
Folinea	(Il Morgagni, Tome XXI., 1879)	19	8
Klink	(Medycyna Varsovie, 1879)	5	0
Chadzynski	(Annales de Dermat. et Syph., 1880)	30	7
Zeissl	(Wiener Med. Presse, 1880)	5	0
Mauriac	(Annales de Dermat. et de Syph., 1881)	11	0
Quinquard	(Idem, 1881)	3	0
Terillon	(Idem, 1881)	1	0
Gibier	(Idem, 1881)	2	0
Jullien	(Trans. Internat. Med. Congress, 1881)	5	1
Rasori	(Giorno Ital., 1881)	1	0
Bifani	(Giorno Ital. dell. Scienze Med., 1882)	3	2
Spillman	(Annales de Dermat. et de Syph., 1882)	8	2
Bumm	(Vierteljahresschrift f. Derm. u. Syph., 1882)	27	5
Beevan	(Md. Med. Journ., 1882)	8+?	8
Bumstead and Taylor	(On Venereal Diseases, 1880)	15	0
Otis	(oral communication, 1882)	15	0
		222	60

To sum up, there are claimed 60 successes in 222 cases, or about twenty-five per cent. By success is understood the non-appearance of secondary accidents during a period of observation continued from four months to one or more years.

It may be remarked that the force of the foregoing statistics is materially weakened by a number of qualifying conditions. We cannot, of course, take up these cases *seriatim* and analyze them, stating the grounds of their authenticity and pointing out possibilities of error which would impair their value or lead us to reject them altogether, but we may, in general terms, indicate certain objections.

In the first place, it is to be borne in mind that a number of the experimenters are *unicists*, and believing as they do in the etiological identity of chancre and chancroid, it cannot be supposed that the sores were differentiated with that absolute exactness which would allow the results to serve as a criterion of the prophylactic value of excision. The same objections which were urged against the validity of the earlier results claimed by Ricord and others for cauterization would apply in this case.

In the next place, we will observe that many of the cases should be rejected on account of the doubtful character of the diagnosis. Confrontation, the most important element of certainty in diagnosis, was employed but seldom. Among the cases reported as successful in which excision was performed eight, nine, and ten days after exposure to contagion, the correctness of the diagnosis is open to serious objection. The appearance of the chancre at that epoch, though not impossible, is altogether exceptional and opposed to what we know of its normal evolution. Even admitting its existence at that time, all syphilographers will testify to the exceeding difficulty, if not the absolute impossibility, of pronouncing upon its syphilitic character before induration and engorgement of the inguinal glands takes place. Even then the diagnosis is not certain; the induration may be inflammatory, and the glands sympathetically swollen. It is a fact verified in the experience of all of us who have much to do with syphilis, that oftentimes a lesion presenting the typical induration and other characteristic signs of chancre, though subjected to only the simplest local treatment, is not followed by general syphilis. So frequently do such mistakes occur that most authorities recommend the postponement of specific treatment until the diagnosis is confirmed by the appearance of secondary symptoms.

Another source of error which depreciates the value of the statistics is the insufficient observation to which the patients were subjected after the operation. In many of the cases, the observation was continued only four or five months—a period manifestly too short to justify a conclusion of the patient's exemption from syphilis. Again, many of the patients were seen only monthly, or at longer intervals, while in some of the cases the claim of success was based upon the patient's statement, twelve or eighteen months later, that he had been free from syphilis. Any one familiar with the evolution of secondary accidents, their sometimes insignificant character, their susceptibility to pass unperceived by the patient, and the tendency of the disease to manifest itself in cyclical explosions,

with intervals of repose and entire freedom from all symptoms, will at once appreciate the possibilities of error in such conclusions.

In a number of cases excision was performed from twelve to forty-eight hours after the appearance of the chancre; the general average was from seven to nine days. Now it would naturally be supposed that the immediateness of the operation is the prime condition of success; yet in Rasori's case of excision of a chancre situated upon the prepuce, twelve hours after its appearance; in Gibier's and Mauriac's cases, forty-eight hours later, and in others where excision was done from the second to the fourth day, general syphilis followed in due time.

Another case which may be set aside as not proven is that, though excision may fail to prevent constitutional syphilis, yet by removing the mass of infected cells contained in the chancre the virus is attenuated and the severity of the disease modified. There is no evidence that numerical diminution of the germs of an infectious disease will deprive it of its power of thoroughly impregnating the system. Experience proves that the mildness or severity of the disease is a matter of individual constitution; that the *quality* of the syphilis is dependent upon the reaction of the organism rather than the *quantity* of virus implanted. Besides, these observations are restricted to a period which comprises only secondary accidents, and even admitting that these were mitigated in severity, yet we know that the initial benignity of a syphilis confers no guarantee against the malignancy of tertiary manifestations. As Fournier has pointed out, the worst possible forms of tertiary syphilis, with tendency to localization in the brain and other central organs, are characterized by the exceeding mildness of secondary accidents.

A final objection to the practice of excision from a surgical standpoint: We know that the chancre is a lesion essentially benign, occasioning little pain or other subjective symptoms, self-limited, with a tendency to spontaneous resorption, and healing, generally, without cicatrix. It is opposed to the principles of conservative surgery to intervene with an opera-

tion which involves hemorrhage, loss of tissue, deformity, and an indelible cicatrix, in order to effect that which is surely and painlessly accomplished by the unaided forces of nature.

From this study of the subject we conclude—

First.—That the facts of clinical experience, as well as deductions from analogy and experiment, are opposed to the theory of the local nature of chancre upon which the practice of excision is based.

Second.—That the practice of excision of the chancre, as a means of aborting syphilis, is condemned by its clinical results when these results are weighed in the balance of discriminating judgment, due regard being had to the possibilities of error.

Third.—That these sources of error are comprehended under doubtful diagnosis, insufficient observation, both as regards time and method, and *post hoc* conclusions.

Fourth.—That in cases where secondary accidents fail to appear after excision, there is no positive evidence that it had an abortive influence, since experience proves that sores with all the typical signs of infecting chancre are sometimes not followed by constitutional syphilis.

Fifth.—That there is no evidence that excision of the chancre attenuates the syphilitic virus and modifies the intensity of general symptoms, since the benignity or malignancy of syphilis is a matter of individual constitution.

Sixth.—That it cannot be recommended as a local adjuvant, since it is opposed to principles of sound surgery to remove, by an operation involving loss of tissue and an indelible cicatrix, an accident which always disappears by a process of spontaneous resorption, leaving, as a rule, no posthumous evidence of its existence.

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