Riggs (C. E.)

THE SPINAL CORD IN A CASE OF PERNICIOUS ANÆMIA.

READ AT THE ANNUAL MEETING OF THE AMERICAN NEUROLOGICAL ASSOCIATION, JUNE 2, 1896.

BY

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C. 1.

C. 2.

C. 3.

C. 4.

C. 5.

C. 6.

C. 7.

C. 8.

D. 1.

D. 2.

D. 3.

D. 4.

NOTE.

On page 1, line 2, read Dr. Gottfried Stamm.

Through an oversight, the credit which should have been given in the text to Dr. L. B. Wilson, for his efficient services in preparing the plates, was omitted.

Owing to my absence from home, and consequent inability to correct the proofs, the technique of the preparation of the specimens was inadvertently included in the following article.

In addition to the references already made, I desire to call attention to Dr. James Hendrie Lloyd's "The Spinal Cord in Pernicious Anæmia" (Journal of Nervous and Mental Disease, April, 1896,) and Dr. James Taylor's (M. R. C. P., London,) "On Nervous Symptoms and Morbid Changes in the Spinal Cord in Certain Cases of Profound Anæmia" (Volume 78, Medico-Chirurgical Transactions).

Robinson (B.)

The extra peritoneal treatment of the stump in abdominal hysterectomy for uterine myomata.

8°. New York, 1896.

Repr. from: Am. Gynaec. + Obst. J., N. Y., 1896.

Robinson (Byron)

The extra peritoneal treatment of the stump in abdominal hysterectomy for uterine myomata.

3 pp. 8°. New York, 1896.

Repr. from: Am. Gynaec. & Obst. J., N.Y., 1896.

THE SPINAL CORD IN A CASE OF PERNICIOUS ANÆMIA.

MRS. B., aged forty-five, was referred to me in September, 1895, by Dr. Goetfried Starm. She was paraplegic and exceedingly anæmic; the paraplegia was, however, not complete; she could move her limbs, although unable to walk or stand. Her disease seemed to date from some nervous shock three years previous, when one of her children had been injured at school and died from the effects; she had never been the same person since. Two years before I saw her she had been previously sick with an ill-defined trouble, which, I am informed by Dr. E. J. Abbott, her physician at the time, presented many of the clinical symptoms, but none of the physical signs, of an acute tuberculosis. In July, 1895, the loss of power in the legs was first observed. Her temperature during her illness fluctuated between normal and 100° F.

After her death I was told that at one time she was supposed to have suffered from Bright's disease. Examination of the urine, however, while she was under my care at the hospital was entirely negative. She complained especially at night of a sensation as if, as she expressed it, her bones were going through her flesh.

Examination showed that tactile sense was markedly diminished over both lower extremities and over the trunk as high as the ensiform cartilage, being most marked over the area supplied by the anterior crural nerve. Temperature sense was normal, while that of pain was diminished. She complained of distressing paræsthesia, especially in the area of lessened tactile sensibility; it was present in the upper extremities as well. Rectus clonus was marked in both legs; patellar reflexes were exaggerated. At my first examination I succeeded in obtaining ankle clonus; later it became barely appreciable. There was no ataxia either of the extremities or of the trunk; no lightning pains.

Superficial reflexes: Plantar diminished; gluteal diminished, abdominal epigastric, dorso-lumbar absent; interscapular present.

On October 21 a blood-count was made by Dr. Charles L. Greene which showed 2,264,000 cells per cubic millimetre, representing a count of eighty squares. Hæmoglobin thirty per cent., adherent poikilocytes,

microcytes, and megalocytes. On November 27 another blood-count was made, showing 1,340,000 cells per cubic millimetre. The post-mortem examination, by Dr. A. W. Dunning, was made about six hours after death. Subject was found extremely white and but little emaciated; calvarium normal in thickness and easily removed; meninges not unusually adherent. On removal of the cerebellum there was found a necrotic area the size of the end of the little finger, located upon the posterior surface of the petrous portion of the right temporal bone, just below the attachment of the tentorium cerebelli. The forceps were thrust into this aperture to the depth of three-quarters of an inch in a forward and outward direction into the mastoid process. The spinal cord was removed by dividing the pedicles, thus exposing the spinal canal anteriorly. Within this canal in the mid-dorsal region was found a considerable extravasation of blood, together with a quantity of apparently serous effusion. The thorax was filled with serum; the pericardium was distended, and the abdomen also contained a large quantity of serum. The blood-vessels were everywhere empty and colorless. The heart was pale and flabby, with a small clot of blood in each ventricle; liver was enlarged; the spleen was dark and also enlarged. The pancreas was enlarged and extremely fibrous; the intestines were thin-walled and pale; the lungs normal in size and apparently healthy.

MICROSCOPICAL EXAMINATION.

The upper three segments of the spinal cord were attached to the brain when received in the laboratory, and, together with the brain, were hardened in four per cent. formalin.

In hardening, these three segments were somewhat flattened dorso-ventrally by the weight of the brain partially resting on them. The remainder of the cord was hardened in Müller's fluid and alcohol. This portion was slightly twisted at the level of the D. 6.

(All the segments were then separated, embedded in celloidin, soaked *en bloc* in copper acetate solution, cut, stained in Weigert's hæmatoxylin, decolorized in potassium ferricyanide and sodium biborate solution, cleared in clove oil, and mounted in xylol-balsam.)

On examination with low power the following degenerations were observed:

1. *Anterior Pyramidal Tract.*—The area of degeneration was greatest at the level of the C. 6, much less at the level of the C. 5, and showed only as a trace on the right side in the C. 4. Below the D. 7 it became gradually reduced in size till it finally disappeared as a trace on the right side at the level of the D. 8.

2. *Direct Cerebellar Tract.*—The area of degeneration appeared as a trace at the level of the D. 10, gradually increased in extent to the level of the D. 4, and continued thence without materially changing in size to the C. 1. The area was greatest on the left side throughout the whole extent of the cord.

D. 5.

D. 6.

D. 7.

D. 8.

D. 9.

D. 10.

D. 11.

D. 12.

L. 1.

L. 2.

L. 3.

L. 4.

L. 5.

3. *Cross Pyramidal Tract.*—The area of degeneration appeared as a trace in the C. 2, gradually increased in extent to the level of the D. 8, and decreased from there to the L. 3, where it disappeared. It was greatest in the left side throughout.

4. *Column of Lissauer.*—The area of degeneration extended from the C. 1 to the D. 6, and was greatest at the C. 6.

5. *Column of Burdach.*—The area of degeneration extended from the C. 1 to the L. 3, being greatest at the level of the D. 6. The degeneration was most marked at the median and posterior margins of the column.

5(*a*). *Posterior External Field of Posterior Column.*—The area of degeneration extended from the C. 1 to the D. 7, being greatest at the C. 5.

6. *Column of Goll.*—The area of degeneration extended from the C. 1 to the end of the column, being greatest at the D. 6 and the C. 3. From the C. 4 downward the posterior median aspect of the column was less degenerated than the other portions, and from the L. 2 downward the area in the right side was wider than on the left.

All other columns were normal throughout their entire extent.

Examination of the degenerated areas with high power showed axis cylinders and medullary sheaths in various stages of disintegration. Many of them were granular and others fatty. The degenerated columns, especially in the mid-dorsal region, were shrunken and contained an excess of connective tissue.

The gray matter appeared normal throughout.

The arteries wherever present showed a thickening of the endothelium, and here and there an inward projecting growth of new connective tissue.

The Liver.—(The liver was hardened in alcohol, embedded in celloidin, cut, stained in lithium carmine, treated with potassium ferrocyanide and acid alcohol, cleared in clove oil, and mounted in balsam.)

Examination showed areas of fatty infiltration and degeneration, and an enormous deposit of hæmosiderin and hæmatoidin, both in the liver cells and in their trabeculæ. The pigment was mostly in the centre zone, but extended in some lobules through the middle zone, even lining the walls of the capillaries.

Spleen.—The spleen was treated as the liver. The only pathological change observed was a scattered deposit of the same pigments found in the liver.

Arteries.—(Portions of arteries from the circle of Willis were hardened in alcohol, embedded in celloidin, cut, stained in lithium carmine, cleared in xylol, and mounted in balsam.)

Examination showed thickening of the middle coat.

No apparent relation existed between the areas of degeneration and the mid-dorsal clot noted at the post-mortem. The points of origin of degeneration were widely separated, some lying in the upper cervical and others in the lower lumbar. None of the columns, except possibly the left direct

cerebellar and the right direct pyramidal, had all their fibres involved, supposing their sectional areas to be correctly figured in the standard atlases.

Taken as a whole, it would seem that the scleroses were certainly not systemic in their origin, but probably were of vascular origin.

This view is held by Nonne, and quoted by Marie, in the *Revue Neurologique* for November, 1895.

Nonne examined cords in seventeen cases of pernicious anæmia, in seven of which the findings were negative; in three the lesions were clearly defined in the neighborhood of vessels, and in the last seven there were medullary lesions of pronounced type. The first two of these groups showed no distinctive clinical symptoms; not even in all the cases of the third group were they present, and when found consisted mainly of the loss of the knee-jerk, a slight ataxia, and some troubles of sensation.

Nonne concludes further that the pathological characteristics are small patches of degeneration, beginning in the cervical cord, showing predilection for the root-zones of the posterior columns, as well as for the anterior and lateral columns.

The findings in this case would seem to bear out Nonne's conclusions.

Osler also quotes Lichtheim, Morris Lewis, and Burr as reporting cases of sclerosis of the posterior column. He further notes the constant and peculiar distribution of hæmosiderin in the liver.

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BY

HENRY W. CATTELL, A.M., M.D.

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