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SPINAL LOCALIZATION

AS SHOWN BY A CASE OF ACUTE HEMORRHAGIC MENINGOMYELITIS, WITH SECONDARY SOFTENING AND CAVITY FORMATION.*

JAMES HENDRIE LLOYD, M.D.

Neurologist to the Philadelphia Hospital.
PHILADELPHIA.

The case which furnishes the text for this paper occurred recently in my service at the Philadelphia Hospital. It supplies some valuable data for spinal localization at the seventh and eighth dorsal segments. It also presents a unique example of rapidly fatal hemorrhagic meningomyelitis, with extensive secondary softening and cavity formation, originating in the lumbar cord and stopping abruptly in the eighth dorsal segment.

There is seldom seen, I believe, a case of acute myelitis of such distinct localizing value, nor one which presents such a rapidly fatal course from apparently slight beginnings. I report the case because, first, there still seems to be a demand for more accurate localization at and about the neighborhood of the umbilicus, and, second, because the sections are of such great interest from the pathologic standpoint, raising the question, as they do, of a possible acute syphilis of the cord.

Patient.—S. P., male, colored, aged 34, was admitted to the Philadelphia Hospital, April 4, 1907, with paralysis of motion and sensation below the waist.

History.—He gave a history of having been injured by being thrown from a trolley car in January, 1906, more than one year before. He was confined to bed for six weeks, and always had some pain in the back afterward, yet was able to attend to his work, that of a Pullman porter, for fully a year, until taken with his final illness. He had had syphilis some years before. Five weeks before his admission to the hospital he came home from work, feeling weak and ill. The next morning he had numbness in his legs, with some loss of power. This kept growing worse, until in less than a week he was paralyzed in both his legs. He also had a girdle pain about his waist, and lost entire control of his bowel and bladder. He was burned on one occasion on each foot by hot bottles because he did not feel them. A deep bed sore soon formed on the sacrum.

Examination.—This was his condition on admission. He was totally paralyzed in his lower limbs, and, of course, unable to stand. There were no contractures, and, what was especially noteworthy, his knee-jerks were abolished. The abdominal and cremasteric reflexes were also abolished, and there was no plantar extensor reflex. Below a line drawn around his waist at a little below the lower margins of the ribs, and therefore about two inches above the umbilicus, there was total abolition of all modes of sensation (Fig. 1). This uppermost line extended further up about one inch after his admission, until finally it was at about the lower costal borders. It was not a perfectly straight line, but extended upward a little in the

mid-line and was slightly higher in the back. The patient was unable to distinguish heat, cold, pain, or touch, below this line, or to tell the position of his lower limbs. He complained of pain at the upper level of the anesthesia, and his spine at the seventh and eighth dorsal vertebrae was extremely sensitive. There was dribbling of urine and incontinence of feces. The bed sore was intractable. About two weeks after admission the patient complained of pain and stiffness in his back, and difficulty of swallowing; also of some numbness in one arm, but these symptoms did not persist, and were possibly hysterical in type. There were no lesions found after death to account for them. A lumbar puncture was performed by Dr. Peterson, the resident physician, and about 15 c.c. of clear fluid withdrawn. This proved to be acellular and sterile. In view of the nature of the lesion determined postmortem, this acellular state of the cerebrospinal fluid is rather surprising. An x-ray picture was taken and showed slight haziness in the intervertebral space between the ninth and tenth dorsal vertebrae, more marked on the left side. The spaces between the tenth, eleventh, and twelfth dorsal vertebrae were also clouded, but equally on both sides. The appearance suggested a gross lesion, such as tumor or hemorrhage, rather than a myelitis, and is especially interesting in view of the fact that neither tumor nor extra-medullary hemorrhage was found at the autopsy.

Subsequent History.—The case progressed rapidly toward a fatal termination. The patient ran a febrile temperature, probably due to the bed sore and bladder infection. One very interesting symptom was sweating above, and dry skin below, the line of anesthesia. I have seen and described this before in grave transverse lesions of the cord.¹ An explanation of it has recently been attempted by Bruce, who has suggested that it is due to involvement of the intermedio-lateral tract, but this seems to me entirely theoretical. The patient's strength failed rapidly, the heart became irregular and slow; and for the last 24 hours he had distressing hiccough. He died May 4, about 9 weeks after the acute onset of his symptoms.

In a later statement the patient's wife told me that for at least three months before being taken with his final illness he had been suffering with pain and stiffness in the back, eccentric pain about the navel, difficulty in rising from a chair, and loss of sexual power. These symptoms probably indicated that during this precedent period he had been having slight leptomeningitis. As he had not been subjected to expert examination, it is impossible to state to what extent the disease had progressed, but it had not interfered with his work as a porter.

Microscopic Examination of the Cord.—The sections in this case were prepared with great care by Dr. C. D. Camp, assistant neurologist, on duty with me in the hospital. The spinal cord from the level of the eighth cervical segment downward was removed for examination and preserved in 10 per cent. formalin solution. Microscopic sections were made from the levels of the top of the fifth, fourth, and second lumbar segments; the middle and top of the first lumbar, twelfth, eleventh, tenth, ninth and eighth thoracic segments, and from the top of the first thoracic segment, and stained with the hemalum-acid fuchsin stain and the Weigert hematoxylin stain. Nerve cells were studied with the thionin stain. Sections were stained by the Marchi osmic acid method from the levels of the middle of the first and top of the seventh thoracic segments.

* Read in the Section on Nervous and Mental Diseases of the American Medical Association, at the Fifty-eighth Annual Session, held at Atlantic City, June, 1907.

1. A Case of Hematomyelia, Jour. of Nerv. and Mental Dis., February, 1906.

Summarized, a study of these sections showed that below the fourth lumbar segment there was no lesion. Beginning in the fourth lumbar segment and extending into the eleventh thoracic segment, there was an area of intense meningomyelitis, with the following characteristics: The pia, especially the posterior portion, was much thickened, apparently by a fibrous exudate rather than by proliferation of the tissue. There was an intense infiltration of cells, leucocytes and lymphocytes, about the blood vessels in the pia. The pial vessels were full of blood; all the walls of the smaller were thickened. The

Fig. 1.—Area of anesthesia in a case of meningomyelitis.

Fig. 2.—First lumbar segment in a case of meningomyelitis.

walls of the larger blood vessels did not show any proliferation of the intima. The posterior roots as they entered the cord were apparently constricted by the swollen meninges, and just outside of the meninges the nerve roots were swollen and the nerve fibers reduced to a granular debris. Not all the posterior roots were affected in this way; some were normal. Within the cord the blood vessels were congested, and there were numerous hemorrhages, especially in the gray matter; these were of recent occurrence, as shown by the partial preservation of the form and color of the red blood corpuscles. In the section from the second lumbar segment there was seen

an immense dilatation of the artery in the anterior fissure, with a hemorrhage in the posterior part of the anterior fissure, as large as the anterior horn at this level. The walls of the smaller blood vessels were much thickened, and they were surrounded by masses of cellular infiltration. Although the white matter stained deeply with the Weigert hematoxylin stain, the myelin had a disintegrated appearance and with the hemalum-acid fuchsin stain appeared granular and swollen. Axis cylinders were swollen or in many cases had entirely disappeared (Fig. 2).

Sections from the level of the top of the eleventh thoracic segment showed the pia congested, but only slightly thickened and with a moderate round-cell infiltration about the blood vessels. The blood vessels within the cord were congested, especially in the posterior and lateral columns. The myelin sheaths had the swollen and granular appearance that was noted in levels lower down in the cord. There were numerous spaces in the cord from which fibers had dropped out, and also numerous swollen axis cylinders in all parts of the section (Fig. 3).

At the level of the top of the tenth thoracic segment there was an area of softening in the gray matter of the posterior root, on the right side, just posterior to the position of the column of Clark. It consisted of granular debris and fatty granular cells. The pia at this level was normal and the blood vessels within the cord showed no thickening of their walls

Fig. 3.—Tenth thoracic segment; meningomyelitis.

or any evidence of inflammation. At the level of the middle of the ninth thoracic segment this area of softening was much larger and occupied the entire anterior two-thirds of the posterior columns. It was smaller and round at the top of the ninth thoracic segment and tapered to an end at the level of the middle of the eighth thoracic segment. Sections from the first thoracic segment showed some disintegration of the myelin in the columns of Goll, but otherwise the section appeared normal when stained by the hemalum-acid fuchsin and Weigert hematoxylin stains.

At the level of the seventh thoracic segment there was recent degeneration in the direct cerebellar tracts and in the columns of Goll and to a slight extent in the columns of Burdach. At the level of the first thoracic segment there was recent degeneration in the direct cerebellar tracts and in the columns of Goll.

The nerve cells in the anterior horns were normal at the level of the first thoracic segment. In the lumbar segments the nerve cells of the anterior horns had entirely disappeared. In the sections from the ninth thoracic segment (Figs. 4 and 5) the nerve cells of the anterior horns had undergone marked degenerative changes, with chromatolysis, displacement of the nucleus, and loss of processes. The cells of the intermediolateral tract show the same changes as the motor cells.

My explanation of these findings is as follows: In the four upper lumbar segments there was an intense meningomyelitis, with small hemorrhages in the substance of the cord. The thickened pia and thickened blood vessels suggest that the initial lesion was a syphilitic leptomeningitis at this level, and this is borne out by the clinical history, for the man had irritative symptoms for some months before the onset of the sudden acute exacerbation. The blood vessels, it is true, did not show the precise picture, as drawn by Heubner, of syphilitic endarteritis; in some places the intima was nearly destroyed, not thickened, the elastic tissue, which showed very plainly, being the innermost membrane left. The adventitia was much thickened. But it may be questioned whether Heubner and his followers are not rather too dogmatic in their statements about the appearance of syphilis of the blood vessels. The picture may vary more than they allow. For instance, there is a well-recognized periarteritis, as described by Kussmaul and others. In this form, as in my case, there is great thickening and infiltration of the outer coat. The pinching of the posterior roots by the thickened pia was very plainly shown, and suggests the idea of Nageotte, Redlich and

Fig. 4.—Ninth thoracic segment; meningomyelitis.

Obersteimer, who think they find in a similar condition the essential cause of locomotor ataxia of syphilitic origin.

In the first lumbar segment there was a large hemorrhage in the anterior fissure and a very large congested blood vessel from which apparently the blood had escaped. An area of softening and cavity formation began in the eleventh dorsal segment and extended upward. The substance of the cord was much disintegrated. This area of softening ultimately occupied a large space in the posterior columns and had broken down and formed a cavity visible to the naked eye. It ended abruptly in the eighth dorsal segment. This softening with cavity formation was probably secondary to the main lesion lower down, and occurred as the terminal process of the case.

Above the eighth segment the cord was practically normal, and the absence of ascending degeneration of any marked degree is in favor of the very recent and acute nature of the lesions.

The idea that the case was of syphilitic origin is borne out by the history of syphilis, by the exudative leptomeningitis, by the thickened arteries, and by the acute

hemorrhagic myelitis with secondary softening and cavity formation; for, as Oppenheim points out, this formation of cavity may happen in syphilis of the spinal cord.

Acute syphilis of the spinal cord is comparatively rare. The syphilitic process is usually slow and chronic. In the present case it would possibly be proper to say that there had been a slight chronic leptomeningitis lasting for some months and then an onset of fulminating symptoms, caused by an acute hemorrhagic myelitis with secondary softening and cavity formation, lasting but a few weeks. It is not probable that the trauma was of special significance, unless it was to furnish a starting point for syphilitic infection, as it is well known to do in some cases.

According to the view of Sottas, Dejerine and some others, a process of softening is very common as a secondary effect in spinal syphilis. The initial lesion

Fig. 5.—Ninth thoracic segment (higher level than in Fig. 4); meningomyelitis.

is vascular and is located especially in the membranes.

Cavity formation has also been described by Van Gieson in traumatic cases, and they are not unlike the one here presented. Van Gieson thought they were secondary to small hemorrhages. It has even been claimed that syringomyelia may be secondary to traumatic hemorrhage, and the resemblance in some ways is marked. But in the present case the cavity is in the white matter rather than in the gray matter, as in syringomyelia.

The localization in this case points to the eighth thoracic segment. The sections showed that the cavity ends in that segment rather abruptly and that there was only slight evidence of degeneration in the seventh segment. Sherrington² has shown in his experiments on monkeys and cats that there are what he calls "overlaps" in each area of skin innervated by the dorsal nerve roots. Thus each band-like area innervated by the eighth segment, for instance, has also sensory filaments from the seventh above and from the ninth below. In other words, there is no area supplied exclusively by

2. *Trans. Phila. Soc., Lond., 1893, vol. clxxxiv, pp. 341-763.*

any one segment (as Türck and others of the older physiologists taught), but every thoracic area is supplied by at least three segments, viz.: its own segment and the one above and one below. According to this view, a lesion located in the eighth thoracic segment would cause absolute anesthesia only in the lower half of its own skin area and impaired sensation in the upper half; this because the upper half is supplied from the seventh segment in part. If this holds true in this case, it tends to put the area supplied by the eighth segment in about the same location as given in the diagram of Head, who places the umbilicus at the lower margin of the ninth area. It also corresponds closely to the mappings out by Sherrington on the body of one of his monkeys.

It need not be insisted on here that the exact localization of the spinal sensory areas on the trunk is of great practical importance, both in neurology and in surgery. Hence the care taken to present this case.

DISCUSSION.

DR. WM. J. SULLIVAN, Lawrence, Mass., asked Dr. Lloyd whether in his opinion degenerative condition of the cord accompanying locomotor ataxia is due entirely to syphilitic poisoning? By some these changes are said to be due to syphilis in 85 per cent. of the cases, and by others in 100 per cent. of the cases, and that the history of syphilis is found in 100 per cent. of the cases.

DR. WILLIAM G. SPILLER, Philadelphia, referring to the level of the umbilicus and the sensory area said that some years ago he studied that question in fracture of the cord, and found it was in the tenth thoracic segment of the cord. I have seen in three cases the area of sensation was sharply limited in the trunk. Horsley makes mention of the same thing, and Sherrington's views would indicate that the edge should shade off gradually into the field of normal sensation.

DR. JAMES HENDRIE LLOYD, Philadelphia, closing, referred to the theory held by some that tabes is the result of leptomeningitis of the posterior nerve roots. He did not wish to say that he would commit himself to this theory, because he does not know how tabes is actually caused. He has gone over the subject very carefully recently, especially with reference to these syphilitic cases, and can not make out any absolutely satisfactory *modus operandi* in the case of tabes. It may be, he said, that it is merely an atrophy of the neurons of the posterior columns. In some cases of syphilitic leptomeningitis of the posterior aspect of the cord, there occurs a pinching of these posterior roots. That condition is very clearly shown in the present case. Dr. Lloyd sees a great many cases of myelitis, but the case which he presented is one that was of especial interest. The points in which it is of peculiar interest are (1) it seems to suggest the possibility of acute syphilis of the cord; (2) it shows secondary cavity formation very clearly; (3) it is sharply delimited in the eighth segment; therefore, it gives localizing data for the eighth segment of the thoracic portion of the spinal cord.

Serum Therapy.—R. Chiarolanza, in the *Policlinico*, June, 1907, reports the results of experiments with different anti-streptococcus and antipneumococcus sera in the corresponding infections. The experiments are described and tabulated; the results are stated substantially as follows: 1. Heterogeneous serum in small doses may exercise a preventive action in rabbits against microbes of slight pathogenic activity (e. g., mildly virulent streptococci). 2. The same serum in small dose has no preventive action against virulent streptococci and, also, if in larger doses, hastens the lethal outcome of the infection. 3. Antistreptococcic sera of different makers (Aronson, Tavel) have no preventive action against virulent streptococci, even when the dose is very small. 4. The specific heterogeneous serum against pneumonia (Pane) has a constant preventive action against pneumococcus infection, and this was observed when the infection was introduced after a traumatism (laparotomy) as well as when it was simply injected into the uninjured peritoneum.

FACIAL HEMIATROPHY.

STATISTICAL REVIEW OF ETIOLOGIC FACTORS AND PATHOGENESIS. REPORT OF A CASE WITH MORPHOLOGIC STIGMATA.*

THEOPHIL KLINGMANN, M.D.

ANN ARBOR, MICH.

It has been suggested that facial hemiatrophy is primarily caused by arrest of development during the growing period or toward the end of it. As far as I have been able to find, there are no typical cases recorded that support this view, and as a prominent feature in the case that is the object of this paper which I offer as an apology for introducing a subject that has been thoroughly and repeatedly discussed and is perhaps not of special interest.

Since Parry¹ first described facial hemiatrophy, particularly since the investigations of Romberg,² Lande³ and Eulenburg,⁴ the number of reported cases has been slowly increasing; nevertheless the disease is still comparatively infrequent; for this reason, and from the fact that our knowledge of nerve influence on nutritive processes is incomplete with but one satisfactory post-mortem examination,⁵ the etiology and pathology of the disorder can not be definitely disposed of.

Neither time nor space will permit of the consideration of the various theories on the causation of facial hemiatrophy, and it will suffice to refer to them in the discussion of this case. Any attempt at a physiologic interpretation of the symptoms will reveal the fact that the disease can not be limited to any one system. The trophic, vasomotor and sensory and motor fibers are involved. I have collected 83 typical cases which are divided into two distinct groups according to the predominance of certain clinical features.

1. Those cases in which the atrophy is complete without functional disturbance of the facial muscles, with little or no disturbance of cutaneous sensation and no vasomotor symptoms.

2. Those cases presenting, in addition to the clinical picture of the first division, vasomotor symptoms, flushing or paleness, temperature differences and secretory disturbances.

In this classification I have eliminated those cases of unilateral facial atrophy associated with facial paralysis, facial asymmetry associated with congenital wryneck, facial atrophy in poliomyelitis and hemiplegia of infants and adults⁶ and that following nuclear lesions.

Of the 83 cases collected, 57 belong to the first division and 26 to the second. Of the 57 cases in the first division, 38 of the patients were women, and the percentage was about the same in those of the second division. In 29 of the 83 cases the patients were under the age of 10 years, 37 between 10 and 20, and 17 between 20 and 30. There are two cases, one of a patient at the age of 75 and one at the age of 81 years reported by Touche,⁷ that are not included in this list. These cases were associated with other conditions due to a widespread cerebral degeneration. Another case was re-

* Read in the Section on Nervous and Mental Diseases of the American Medical Association, at the Fifty-eighth Annual Session, held at Atlantic City, June, 1907.

1. Collected writings, 1825, i, 478.

2. Klinische ergebnisse, Berlin, 1846, p. 75.

3. Essai sur l'aplasie lamineuse progressive celle de la face en particulier, Paris, 1870.

4. Lehrbuch der funktionellen nervenkrankheiten, 1871.

5. Virchow-Mendel: Neurologisches Centralblatt, No. 14, 1888.

6. Oran: Jour. Nerv. and Ment. Diseases, September and October, 1850.

7. Revue Neurologique, 1892.