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RUPTURE OF THE SPINAL CORD IN DYSTOCIA

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Lesions of the central nervous system in children born by forcipal extraction or some other obstetrical manipulation have often been described; cerebral lesions of that kind are in fact well known. Also descriptions of lesions of the spinal cord, though certainly less generally known, are occasionally met with in literature dating from 1870, when Parrot for the first time mentioned rupture of the cord in consequence of difficult birth.

Still the case, which I intend to discuss in this paper, is unique, as it deals with an almost total rupture of the spinal cord, while the child survived the trauma for nearly nine years.¹ Seeing that the

¹ As far as I know, four months is the longest survival in analogous cases, as recorded in literature (*Lawetschek*, 1911).

case also offers some remarkable neurological peculiarities, its description may be sufficiently justified.

Historia Morbi.—D. P., a child of the feminine sex, was born on January 26, 1910. The first child of the mother, being in transverse presentation, was born by version and extraction; the second child was born by the same manipulation; the third and the fourth child were born spontaneously, but the fifth, our patient, being in transverse position, was again born by version and extraction. The doctor, when summoned, stated a first cross-presentation (dorso-anterior) and a prolapse of the umbilical cord and of one arm; the dilatation of the os externum was 7 cm. The child lived; version and extraction were performed, and by Müller's method, the after-coming shoulders were delivered. The head offered some difficulties, the child was asphyctic and was revived with some difficulty. The method used was not stated.

Three days post partum, the mother noticed three ulcers on the child's rump, which grew rapidly larger and deeper. Soon afterwards she observed that the child moved its legs only feebly. According to the mother, the child had never been seen to move its lower legs, feet or toes, but she is sure that the child could flex the thighs on both sides, though only weakly. She had not tested the sensibility, but the child, when 1½ years old, had burned its lower leg seriously, without feeling any pain. Though it grew up prosperously (except for the lower extremities, of course) and appeared to be very intelligent, it continued to show a complete incontinentia alvi et urinæ.

July 2, 1912: On account of the paralysis of the legs and the decubitus, the child was brought to hospital on September 11, 1912.

Of the status præsens, I have found the following notes: The head and the arms are quite normal. Intellect also normal.

Motility.—The legs are spastic and paretic, both can be moved very weakly (unfortunately it was not noted what muscles still functioned). Walking and standing quite impossible.

Reflexes.—The knee and ankle-jerks of both sides are greatly exaggerated. The phenomenon of Babinski is positive on both sides.

Sensibility.—The tactile sense does not seem to have been examined (perhaps the child was too young to give correct answers).

Pain sense not completely lost in the legs, but much decreased.

Thermic sense lost in the legs and the gluteal region.

Deep sensibility not examined.

Large and deep *bedsore* on the sacrum.

During its stay in the hospital, the child had a spontaneous frac-

ture of the right femur-diaphysis (excellent consolidation with luxuriant callus-formation, which is afterwards resorbed).

No visible or palpable abnormalities of the vertebral column; neither on the Röntgen-photograph.

August 26, 1914: Of the polyclinical examination on August 26, 1914, I only found the note: status unchanged.

July 2, 1917: On July 2, 1917, the little patient was again admitted to the hospital.

Status Præsens.—Head, arms, thorax and abdomen, back and vertebral column quite normal, but for a lordosis of its lower part; the Röntgen photo again does not show any abnormality.

Abdominal reflexes present on both sides.

Lower Extremities.—Both legs are completely paralytic, muscular contracture in hip and knee-joint; all passive movements in the ankle-joints, however, are amply possible; the musculature of the lower leg is atonic.

Reflexes.—Knee-jerks are diminished and the ankle-jerks are abolished. The symptom of Babinski has disappeared on both sides.

Electric Reactions.—Complete abolition of electric excitability on both sides, but for a slow contraction of the m. tibialis anterior and of the m. m. peronei as a response to the galvanic current.

Sensibility.—Tactile sense probably not quite lost in the upper legs; the lower legs are totally anesthetic. Pain sense perhaps not quite absent in the upper legs, but also here, it is at least greatly diminished. An abscess was incised a little above Poupart's ligament without the patient's feeling any pain; when it was over, she asked whether the operation had already begun.

Lower legs in any case quite analgetic. Thermic sense and deep sensibility not examined.

Decubitus at the sacrum very large and deep.

Incontinentia alvi et urinæ, cystitis.

September 27, 1918: The child was again admitted to the hospital; bedsores and cystitis had grown considerably worse. Same neurological status præsens, only this time the knee-jerks were found absent as well as the ankle-jerks.

The patient died on October 8, 1918, from pyemia.

The diagnosis was a lesion of the spinal cord, probably in consequence of dystocia. The absence of any vertebral abnormality and the long survival of the child after such a serious lesion of the cord, made the diagnosis somewhat dubious.

Résumé of the Historia Morbi.—Summing up the principal facts of the historic morbi, we note: a child was born by version and ex-

traction, the birth was very difficult. This child always suffered from bedsores; from total paralysis of the lower legs and from incomplete paralysis of the upper legs, of which it is certain that the m. m. iliopsoas were able to contract during the first years; from total anesthesia, analgesia and thermanesthesia of the lower legs and from, at least, an almost total loss of sensibility of the upper legs; probably the pain-sense had been abolished up to the thoracic segments; finally, from complete incontinentia alvi et urinæ.

Post-Mortem Examination.—The following points in the report of the autopsy are important: the decubitus has penetrated the sacrum, the vertebral column shows no abnormalities, neither can any trace of an old luxation or fracture of the spine be found after a careful examination.

Outside the dura no peculiarities.

When the dura was opened caudo-orally, the scissors came to a point of resistance on the level of the seventh thoracic vertebra. In a transverse section, it appeared that the meninges were fused here and surrounded the very small medulla as a thick solid fibrous ring. The spinal cord and the cerebrum were then taken out and put in formol 10 per cent., for microscopical examination.

Microscopical Examination.—But for a part of the medulla cervicalis, the whole spinal cord was examined in serial sections, alternatively stained after van Gieson and Weigert-Pal.

Examination of the Place of Lesion.—Fig. 1 shows a section at the level of the greatest lesion. It appears that nearly the whole spinal cord has disappeared (this is even more apparent in other sections) and that it has been replaced by connective tissue, consisting of a network of fibers, between which small isles of glia (fibers and darkly stained cells) are found. At the right lateral border of the cord lies a small strand of glia without nerve fibers and at the left border, a larger strand of glia, in which still occur a very few nerve fibers at the extreme periphery. The examination of serial sections (in both directions) makes it clear, that these nerve fibers chiefly belong to short (intra-medullary) tracts.

The only remaining tract belongs to the right anterior column, directly adjacent to the antero-median fissure, and, on tracing it further in serial sections (in a caudal and oral direction), it appears that it corresponds to the ventral part of the direct (ventral) pyramidal tract; it has an oval form and it is surrounded by a glia-ring.

A part of the section of Fig. 1 is reproduced greatly magnified in Fig. 2, showing the small tract in question and the connective tissue, which has replaced the nervous tissue.

FIG. 1. Section at about the level of the greatest lesion. Hyperplastic leptomeninges. Of the spinal cord, only a small tract to the right of the antero-median fissure has remained intact. At the border of the cord only a few nerve-fibers, belonging to the left posterior column and the adjacent part of the lateral column are spared. (Paraffin; van Gieson stain.)

FIG. 2. Showing a part of Fig. 1 (containing the intact ventral pyramidal tract), greatly magnified.

Over a distance of about $\frac{3}{4}$ cm. the aspect remains the same, then, following the serial sections orally and caudally, the form of the medulla again becomes recognizable; the pia is still hypertrophied, the spinal cord itself remains nearly totally degenerated. The connective tissue is found most at the places where the roots (especially the anterior ones) leave and enter the cord. This makes it seem as if the spinal cord had been torn asunder by pulling at the roots.

I shall afterwards return to this question.

In anticipation, I want to refer, however, to Fig. 7, showing a section, orally to the lesion, which demonstrates this fact.

Descending Degeneration.—Examining now the serial sections in a caudal direction, we see the posterior column's first redress.

FIG. 3. Section, caudally of the greatest lesion. Weigert-Pal's stain.

In Fig. 3, the place of the normal cord is still for the greater part occupied by connective tissue, glia and degenerated nerve fibers. The posterior columns already contain more myelinic nerve fibers, the border of the left lateral column contains only a few of them (having a more ventral position than in the sections of greatest lesion) and the right one still less. The small round tract which we found running intact throughout the place of lesion is distinctly visible, stained dark by Weigert-Pal's method; it will henceforth be called the ventral pyramidal tract. I shall afterwards discuss whether this assumption is correct. A few medullated fibers appear in the anterior column on the other side.

In Fig. 4, the form of the medulla is nearly restored, the posterior columns are stained dark by Weigert-Pal's method, but for some lighter spots, probably containing (short) descending fibers. Of the

remaining part of the cord, only the right ventral pyramidal tract is normal, the lateral columns contain only the few myelinic fibers,

Posterior columns.

Connective tissue, surrounding the anterior root-fibers, Fissura mediana anterior. Ventral pyramidal tract.

FIG. 4. Section, caudally of Fig. 3. Descending degeneration. Intact ventral pyramidal tract. Weigert-Pal.

mentioned above, the left anterior column still less. The grey figure is not yet restored, in van Gieson sections of this level, not a single ganglion cell can be detected. The whole spinal cord is much too small and remains so until the lumbo-sacral enlargement.

Small field of degeneration in posterior columns.

Ventral pyramidal tract.

FIG. 5. Section of about the first lumbar segment or the last thoracic segment. Weigert-Pal.

Fig. 5 reproduces a section of about the last thoracic or the first lumbar segment. The spinal cord is still too small, but its structure

is normal again. With the exception of a slight degeneration in their left half, the posterior columns are also normal.

The figure of grey substance is well visible again, the ganglion cells of the horns and of the small columns of Clarke have the usual form, number and tincture. The figure shows the intact direct pyramidal tract clearly. The fundamental tracts round the anterior horns begin to be restored. The rest of the cord is degenerated, with the exception of the above-mentioned medullated nerve-fibers at the border. Also in van Gieson's preparations, no axis-cylinders are visible in the lateral columns of the cord, except for these peripheral fibers. The fibers correspond with the position of the tractus spino-

FIG. 6. Sacral segment. Degeneration of the lateral pyramidal tracts. Weigert-Pal.

cerebellaris dorsalis (Flechsig), directly lateral of the pyramidal tracts. We may therefore state that the lateral pyramidal tracts themselves have been completely abolished.

The aspect soon changes, when we come to the lumbo-sacral enlargement. Tracing the descending degeneration in a caudal direction, I found the intact right ventral pyramidal tract still present in the upper lumbar segments. Its corresponding part of the left anterior column, however, is no longer completely degenerated but already contains many medullated fibers. In many sections, one gets

the impression that the fibers of the ventral pyramidal tract run partly along the ventral median fissure to the commissura anterior, partly reach the anterior horn of the same side in a direct way. I found it impossible, however, to trace the fibers of the right ventral pyramidal tract as far as their termination with any certainty. Suffice it to state, that in the lower lumbar segments the difference between the two anterior columns has ceased to exist and that in the whole section only a very small field of degeneration is found at the ventral periphery of the cord on both sides, exactly lateral of the above-described ventral pyramidal tract, except, of course, for the degeneration of the direct pyramidal tracts in the lateral columns.

The sacral segments no longer show any abnormalities (Fig. 6) but for this degeneration of the lateral pyramidal tracts, which has now reached the border of the cord. It is a remarkable fact, which I shall have to discuss afterwards, that the degeneration of the ventral pyramidal tract ends already in the middle of the lumbar segments, whereas the degeneration of the lateral tracts can be traced up to the lowest sacral segments, where they are gradually lost sight of. The rest of the sacral segments and the filum terminale do not offer any peculiarities.

Ascending Degeneration.—Now, examining the ascending degeneration from the section of Fig. 1 onwards, we may note first of all that the leptomeninges remain hypertrophic over a considerable distance. The pia is equally hypertrophied on all sides, the arachnoidea chiefly over its dorsal half, where it lies as a thick semi-circle of connective tissue against the dura mater. Of the cord itself, the central parts are the first to be restored, but the peripheral parts, especially those surrounding the anterior root fibers, remain replaced by connective tissue over a much greater distance. First of all, the central canal reappears, which soon again assumes the usual form, with its lining membrane of ependyma—it is clear that it was only secondarily affected at the level of the greatest lesion, there is no sign of a former hematoma in or around the central canal.

The first ganglion cells (totally absent in the sections of greatest lesion) which can be recognized are those of Clarke's columns—afterwards, in more oral sections, also those of the anterior horns.

In the section of Fig. 7, the ganglion cells are still totally absent. The hypertrophy of the meninges is clearly visible, like the quite normal central canal. The right ventral pyramidal tract, though faintly visible in the photograph, is very distinct in the slides; it is the only tract not affected by the lesion.

In Fig. 8, the same section is reproduced, greatly magnified, in

order to demonstrate the tract in question better. At the left border of the cord, the strand of nervous tissue already mentioned (Fig. 1),

FIG. 7. Section orally to the segment of lesion. See the connective tissue round the root-fibers; the ventral pyramidal tract; the intact central canal. Van Gieson stain. Paraffin preparation.

can easily be recognized, it is more or less separated from the rest of the medulla by a strand of connective tissue; some intact nerve

FIG. 8. Part of the section of Fig. 7, greatly magnified. See the ventral pyramidal tract and the central canal. Van Gieson stain.

fibers are visible in it. They are still less numerous than in the section of Fig. 1, consequently part of those fibers (mentioned on page

4) are ascending fibers, belonging perhaps to the direct cerebellar tract, they are partly short intramedullary fibers, for some of them have disappeared in more oral sections. At the right border of the cord, only a very few scattered medullated nerve fibers are present.

The medulla spinalis as a whole remains too small up to the cervical swelling. Examining the serial sections in an oral direction, we see the anterior columns and the inner part of the lateral column gradually grow normal again; the columns of Burdach, directly adjacent to the posterior horns, though less intensively stained than normally in the first segments following the segment of the lesion, yet attain their normal size and tincture rather soon; they include the wedge-shaped field of degeneration, corresponding to the columns of Goll.

Ventral pyramidal tract.

FIG. 9. Section of the lower thoracic segments. Degeneration of Goll's columns and of the periphery of the lateral columns. Weigert-Pal.

In the lateral columns we find a degeneration of the peripheral parts, which is broadest near the posterior horns and ends in a point at about the anterior root-fibers. Whereas the degeneration of Goll's column is very sharply outlined, the degeneration of the ascending tracts of the lateral columns is rather diffuse (Fig. 9). Briefly we may say that the sections of this level present the usual appearance of an ascending degeneration after complete transverse lesion of the cord. But I want to draw attention to the often mentioned direct pyramidal tract. The examination of the serial sections in the oral direction, too, clearly shows that we have to deal with a really long

tract; as long as the anterior columns are not completely restored and we still note a degeneration of some short intraspinal tracts or fibers, we may recognize that small circumscribed tract near the ventral part of the antero-median fissure. In Fig. 9 it is still visible, and it is even more distinct in other sections of that level. When all evidence of degeneration of the surrounding parts has disappeared, it can of course no longer be traced.

Now, discussing the further ascending degeneration from this level onward, I can be brief about the spino-cerebellar and the spino-thalamic tracts. The upper thoracic part of the cord and the greater part of the cervical cord were not taken out, so I could only trace the degeneration again in the upper cervical segments; of the latter, the oblongata, the pons and the cerebellum, I made serial sections.

In the upper cervical segments, a slight degeneration of both tractus spino-cerebellares dorsalis (Flechsig) and ventralis (Gowers) can be observed, as a less darkly stained strand along the lateral border of the oblongata—this field of degeneration does not quite reach the outline of the bulb, an intact part of the dorsal spino-cerebellar tract is situated laterally of the degenerated part, as if the fibers, originating from the cells of Clarke orally of the lesion, had laid themselves outside the fibers of the lower part of the cord (in the same way as we have observed of the posterior columns). On the level of the decussation of the pyramids, a lighter spot is still visible in the lateral part of the bulb, but at this level its interpretation has become more difficult, for which reason it is indicated in Fig. 10 in the general way of "ascending degeneration of the lateral column."

Still more orally, it is no longer possible to trace the degeneration with any certainty, there is decidedly no distinct degeneration of any part of the corpus restiforme, nor can the degeneration of the ventral spino-cerebellar tract be traced higher up in the bulb. It is still more difficult to trace the degeneration of the tractus spino-thalamicus (Edinger). All certainty about it ceases to exist even in the lowest section through the medulla oblongata; in the upper cervical segments, its degeneration could be recognized medially of the spino-cerebellar tracts. It is an often discussed question whether there are in this tract, on its way to the thalamus, cells intercalated on the level of medulla oblongata. The problem, however, cannot be solved by tracing the degenerations in Weigert preparations; it is necessary to stain after Marchi. Weigert-Pal preparations only permit the tracing of well-defined compactly-degenerated tracts; therefore this

case is not suitable for a study of the degeneration of the spino-cerebellar and the spino-thalamic tracts to their termination.

Degeneration of the Posterior Columns and the Gracile Nuclei.—With the posterior columns, it is different. A sharply circumscribed degeneration of their medial part can be seen in the upper cervical segments. It is a striking fact, that the field of degeneration occupies only a very small part of the posterior columns, much smaller than is the case with a similar degeneration in an adult (see, f. i., Brouwer, 1915). It has the form of the upper piece of a bottle, the "neck" of which stretches along the postero-median septum and oc-

Degeneration of the columns of Goll.

FIG. 10. Degeneration of the columns of Goll at the caudal end of the nuclei gracilis. Weigert-Pal.

cupies about three quarters of its length. Only the lateral part of this field of degeneration contains some medullated fibers, as the fibers of adjacent segments are slightly mixed in the posterior columns (Schaffer, 1898). Just caudal to the beginning of the gracile nuclei, the field of degeneration is prolonged ventralwards to the central grey matter (Fig. 10).

The nucleus gracilis itself is nearly totally degenerated. It is also much smaller than normally, especially over its caudal half, just as the degenerated part of the posterior columns was found much smaller than is normally the case.

In our case these parts of the central nervous system had never functioned and therefore they failed to develop; in the same way,

the spinal cord between the sacro-lumbar and the cervical enlargements had remained much too small, as this part of the cord chiefly consists of long tracts, and as it is these long tracts that have not functioned for the greater part. The lumbo-sacral part of the cord, however, especially the sacral part, is remarkably well-developed; it is chiefly a reflex region, consisting of grey matter and short tracts.

I return once more to the gracile nuclei. When we have passed the caudal ends, we soon find a few groups of normal cells lateral of the degenerated part of the nuclei. The difference between the cells in the degenerated part and in the intact one is very striking in van Gieson preparations: in the degenerated part the cells are smaller and situated closer together, the number of glia cells is much greater; whereas the color of the degenerated part is brownish, the intact part is stained darker, with a redder tincture. Also in Weigert preparations, it is clear that only the most lateral parts of the gracile nuclei are stained normally. More orally, this aspect changes only little. The medial and, still higher, the dorso-medial part of the nucleus, remain degenerated, and it is by far the greater part (see Figs. 11 and 12). More orally, the nucleus of Goll takes a more dorsal position, bending round the dorso-medial surface of the bulb. Though the nucleus is less distinctly separable from the surrounding nuclei, it is clear, that also at the frontal top, the greater (medio-dorsal) part is degenerated.

We see, therefore, and this is a confirmation of Brouwer's results, that the lowest spinal segments are represented in the nucleus of Goll most medially, just as in the posterior columns; in this way they spread over the whole nucleus, though perhaps a little more in the caudal half than in the oral one.

It is also remarkable, that the remaining intact thoracic segments have such a small projection on the gracile nuclei; it is quite in keeping with the fact that they cover only a small portion of the columns of Goll (Winkler, 1918, l. c., p. 195);² evidently the posterior columns and their nuclei are chiefly provided with fibers by the spinal intumescentiae (extremities).

The further examination of the medulla oblongata, the pons-region and the cerebellum does not show any peculiarity. The lemniscus medialis is quite normal; the inferior olive, carefully examined in serial sections, has the usual size, form and histological features. Many hypotheses about the function of the olivary com-

² It is a well-known fact that the columns of Goll receive their fibers from the sacro-lumbar posterior roots and from the greater part of the dorsal roots (van Gehuchten, 1906; Edinger, 1911; Winkler, 1918).

FIG. 11. Degeneration of the nuclei of Goll (caudal half). Weigert-Pal.

FIG. 12. Degeneration of the medial part of the nucleus of Goll.
Weigert-Pal.

plex have been published, none of them, however, based on facts. It is often supposed, that the enormous development of the principal olive in man partly depends on the erect gait; as the child in question had never been able to walk or to stand, as the olive had always been deprived of the eventual sensible stimuli from the lower extremities, its form or size might possibly have been altered in some way—though I did not think it probable. This, however, is not the case. The cerebellum did not offer any abnormalities, neither macroscopically nor microscopically.

I shall now discuss some questions in connection with the clinical data and the descending degeneration.

Classification of Lesions of the Spinal Cord, Dependent on Dystocia.—I consider the present case an example of an almost complete rupture of the spinal cord at about the level of the tenth dorsal segment.

Lesions of the spinal cord dependent on dystocia may generally occur in three different ways:

1. By *venous engorgement*. In some children, born asphyctic, who died soon after birth, scattered hemorrhages were found in the central nervous system. These cases can be compared with hematomyelia in the adult in states of sudden venous congestion, such as vigorous movements, coitus, defecation, convulsions, etc. (Gowers, 1886, l. c., p. 282; Lewandowsky, 1911, l. c., p. 559; Oppenheim, 1913, l. c., p. 459; Lépine, 1900, l. c., p. 39).

Intracranial hemorrhages in new-born infants are not uncommon, intraspinal meningeal hemorrhages, though less universally known, have already been mentioned by Cruveilhier, Friedleben (1855). Jacquet (1873) was the first to describe hemorrhages in the cord itself, dependent on dystocia.³

The cases published, in which venous congestion is probably the only cause of these hemorrhages in the spinal cord of the newborn, are not many (Couvelaire, 1903; Jacquet, 1873; Schaffer, 1897; Pfeiffer, 1896; Schultze, 1895, 1896, probably also the case of d'Herbécourt, 1898);⁴ sometimes a single focus is found, but more often there are scattered hemorrhages, a fact which is easily understood, seeing that we often find minute extravasations in several organs after asphyxial diseases. It is therefore quite evident that at the same time all causes of asphyxia may bring about hematomyelia.

³ Already in 1870, Parrot communicated a case of rupture of the spinal cord caused by powerful extraction.

⁴ In this case, however, also vascular alterations were found.

Consequently it is impossible to state what sort of birth mechanism is apt to cause this form of medullary lesion intra or post partum. As regards prevention of therapeutics, nothing can be said. However, it appears that Schultze's swingings of the asphyctic child are particularly dangerous in this respect (Knapp, quoted after Oppenheim, 1913, l. c., p. 460).

2. The second way, in which the spinal cord may be affected in dystocia, is by a *fracture, rupture or luxation of the vertebral column* and a secondary meningeal and medullary hemorrhage or a more or less complete compression of the cord. Of this medullary lesion, the etiology, the pathological anatomy and the clinical symptoms are of course the same as those of fracture of the spine in the adult.

There are many casuistic communications about fracture of the vertebral column durante partu, for it is not an infrequent occurrence; Ruge found a vertebral rupture in 8 out of 64 autopsies of newborn children, Stoltzenberg (1911) in 9 out of 75 autopsies. According to Ruge and Küstner (quoted after Hofbauer), it is always the vertebral body⁵ itself that has been ruptured along the epiphyseal line, not the ligaments. According to Stoltzenberg, however, the mechanism of rupture in her own cases was the following: first of all, the lateral capsular ligaments were torn, then followed the adjacent part of the ligaments of the neural arch (which are very tender in the newborn), finally the cartilaginous part of the vertebræ themselves. The author could confirm this conception by experiments; by exercising on the bodies of newborn children a traction of a certain power. She obtained the same kind of rupture, the extent of which was proportional to the degree of deviation from the longitudinal axis (in a lateral direction), not to the power of traction. It is evident that especially cases of the aftercoming head with difficult development of the arms and shoulders, cause the spinal lesion in question, for in these cases traction at the column is combined with deviation from the longitudinal axis, and it is also easily understood that it is especially the cervical column that is most apt to undergo rupture, for the angle between the axis of traction and the child's longitudinal axis is situated there. Also, in all nine cases of Stoltzenberg, the lesion was found in the cervical column; the same was observed by Birnbaum (1906) and Hofbauer (1907). Moreover, the cervical column of the newborn seems, according to the experiments of Duncan (1874), the most fragile part of the whole vertebral column, also when the axis of traction coincides with the child's longitudinal axis.

Though the lesion of the vertebral column in dystocia has often been described, cases with a subsequent destruction of the cord are

⁵ According to Hofbauer (1907) it is the sixth cervical vertebra which is most frequently fractured.

rarely mentioned, most authors mention only intraspinal meningeal hemorrhages.

3. A third group is formed by the cases of a *partial or complete rupture of the cord*, while the vertebral column is quite intact. In the adult we also know those cases, in which a traumatic lesion of the cord comes about without any alteration of the vertebral column (see, f. i., Watts, 1897), but they are not analogous to our cases, in which there is no question of a direct trauma; moreover in those cases of traumatic lesion of the cord (in the adult) without affection of the spine, the cord is seldom ruptured, usually only a hemorrhage is found.

The present group can be compared best with those cases in which a traction at the nerve roots had taken place (f. i., by stretching the sciatic nerve in sufferers from *tabes dorsalis* or *ischias* (Rumpf, 1884; Petré, 1909), or by reposition of a hipjoint-luxation (Schlesinger, 1899), or with those cases in which the vertebral column is bent acutely and extremely (Thorburn, 1887; Oppenheim, 1913).⁶

It is only in order to make the several factors clearer, which play a part in the case of lesions of the spinal cord *durante partu*, that I have made this schematic classification. Of course these factors are often (probably as a rule) combined.⁷

It is also often impossible, afterwards, to decide by macro- or microscopical examination of the lesion, what the precise mechanism of its origin was. Most difficult of all is the differentiation of *hematomyelia* from rupture, as a rupture of the cord is of course always combined with hemorrhage and as a large hemorrhage may cause a complete destruction of the spinal cord. Especially in older cases, the difficulty is great; if the vertebral column had been only temporarily dislocated (a possibility which is also known in the adult), we may afterwards find no sign of its former lesion. The hematoma has disappeared and the differentiation between rupture and *hematomyelia* on the remaining cicatrice may have become quite impossible. In the present case, however, the diagnosis "rupture of the cord" can be made with certainty. Not only because the vertebral column was found totally intact, but also on account of the aspect of the cicatrice. The cord was found replaced by connective tissue over a considerable distance (except for small strands of nervous

⁶ It is true that also in those cases a rupture is not described, as far as I know; they only deal with *hematomyelia*, but then the manipulations to which the new-born are subject are much stronger.

⁷ Déjérine has pointed out that rupture of the spinal nerve roots can be combined with *hematomyelia* (quoted after Oppenheim, 1913, I, l. c., p. 460).

tissue); the central grey matter and the central canal were the first to be found normal again (in the examination of serial sections), whereas in the cases of primary hematomyelia it is just the grey substance, that is most injured. In this case the greatest destruction and the most striking development of connective tissue was found about the root fibers entering and leaving the cord, as if the traction had in the first place occurred at the nerve roots. I am therefore inclined to consider the present case as one of rupture.

It is a matter of course that also of this third group of medullary lesions the difficult delivery of the aftercoming head is the most frequent cause; also the cases of Parrot (1870), Handwerck (1901), Gött (1909), Lawatschek (1911), all more or less analogous to our case, concerned difficult extractions by the feet.

In our case, the application of the method of Müller (1898) may have been favorable for causing the lesion, as the vertebral column is bent extremely in a lateral direction by this manipulation.

Finally I intend to discuss three clinical questions, viz., that of the reflexes, of the bed sore and of the paralysis.

According to the law of Bastian (1890), in the case of complete transverse section of the spinal cord the tendon-reflexes are absolutely absent in the part of the body below the lesion. This law has been much discussed, it is accepted, f. i., by Dejerine (1911, '12, '15), while on the other hand Lewandowsky (1911), Oppenheim (1913, von Monakow (1905) do not believe it to be of general value.

Kausch's (1901) case has indeed convinced me that—though Bastian's rule may usually be confirmed, complete section of the cord need not include an abolition of the tendon reflexes with absolute certainty.⁸

Brouwer (1915) described a case of complete transverse section of the cord of traumatic origin, in which the knee and ankle-jerks were first absent, but reappeared and even became exaggerated a year later, to disappear once more some years afterwards. He attributes the latter fact to the bad conditions of the peripheral parts (muscles, tendons, etc.), a hypothesis already expressed before him by Gerhardt⁹ (1895) and Kausch (1901), and warns against the making of conclusions from cases with short survival of the trauma. Yet in other cases the reflexes may remain absent for life-time, even

⁸ The only objection to Kausch's case is that it concerns a man whose spinal cord had already been compressed for three years; the lower part of the cord, containing the reflex arc for knee- and ankle-jerks, might have become more or less independent before the complete transverse section took place.

⁹ For the rest, Gerhardt's case does not speak at all against Bastian's law. See the critique of Bruns (1895).

for a much longer time than in Brouwer's case (Lewandowsky (1910), l. c., p. 597). Of course the present case does not give absolute proof; for those fundamental questions a complete section of the cord is necessary. Still it is remarkable, that the knee-jerks, and still more the ankle-jerks, which have their medullary center in a segment (S_1-S_2) that had apparently not the slightest connection with the further central nervous system (total loss of all kinds of sensibility, total paralysis), were repeatedly found present and even exaggerated, whereas they gradually disappeared in the later period of life. Babinski's reflex, found present in 1912 and 1914, had in the same way disappeared in 1917 and 1918. As is well-known, this reflex usually coincides with the exaggeration of the tendon reflexes, whereas it is a rule (though not always, see f. i., the case of Brouwer) absent in the cases of complete transverse section (Déjérine, 1915). So our case is quite analogous to that of Brouwer, but for the reflex of Babinski.

That the reflexes disappeared once more, must be attributed to the bad condition of the peripheral parts (vide supra; see also the electric changes, mentioned on page 3).

Decubitus Acutus.—The direct cause of *acute bedsores* is still an open question. Samuel (1860) and Charcot (1884) considered it to be a trophic abnormality, later authors (Leyden-Goldscheider (1895), von Monakow (1905), are most inclined to see the cause of it in several other reasons: the permanent pressure of the integuments on account of the insensibility and paralysis, combined with defilement by the excrements, with infection and with vasomotor abnormalities.

Oppenheim (1913, l. c., p. 144), Cassirer (1912, l. c., p. 123), though believing that the above-mentioned factors can usually explain the acute decubitus, dare not exclude the possibility of trophic influences. Of course the problem cannot be solved from the present case. Yet it is worth while to dwell for a moment upon the fact, that in this new-born infant, decubitus appeared already in the first days after birth. The insensibility, the paralysis, the incontinentia cannot explain it in this case, for in this respect, the child did not differ from other babies, who also do not move themselves, but are turned about every three or four hours.

The child was carefully nursed from the beginning; in such a young baby, the pressure on the sacrum could only have been very slight—moreover, the child (it was especially noted in this case) was rather fat.

Are not these considerations in favor of the supposition of the

existence of trophic influences, since vasomotor abnormalities alone are known not to be sufficient to explain the decubitus?¹⁰

Function of the Ventral Pyramidal Tracts.—Finally I intend to discuss a certain point in connection with the intact ventral pyramidal tract. In the preceding pages (see also Figs. 1-9) I have described, that a small tract was left intact by the lesion of the spinal cord; by tracing it in serial sections in an oral and especially in a caudal direction, it could be ascertained, that it was for the greater part a long descending tract (perhaps also containing some short fibers). On account of its position along the ventral median fissure, this intact tract must be the ventral part of so-called direct pyramidal tract. Here I must remind the reader of the fact that the child had been able to flex both legs in the hip-joint, as was stated by the mother and several physicians. As the lesion of the cord occupied the ninth and tenth thoracic segment, the centers for the acting muscles were below the lesion, and so the tract in question is the only one, which could conduct motor impulses from the cerebrum to the moving legs. In this case the fibers of the right ventral pyramidal tracts must have reached the anterior horn of *both* sides—for the child had moved both legs during the first years of its life.

The way in which the ventral pyramidal tract ends is a well-known point of discussion.

According to van Gehuchten (l. c., p. 920) most authors believe that the fibers of this tract reach the anterior horn of the other side by the commissura anterior. Edinger (1911, l. c., p. 147) is of the same opinion. Lewandowsky (1905), on the contrary, believes the ventral pyramidal tract to be a really direct tract, of which the fibers end in the anterior horn of the same side.

Winkler (1918), also basing on observations made by Marie, supposed that the lateral and the ventral pyramidal tracts have different functions. There are various arguments in favor of this hypothesis. In cases of a hemiplegic contracture, there is often also a slight paresis of the other (not paralyzed) leg; not all the fibers of the ventral pyramidal tract reach the anterior horn of the other side, tracing its degeneration in slides, stained after Marche, the ventral pyramidal tract keeps the same size throughout the spinal cord, until the lumbo-sacral swelling, where it rapidly disappears. The thought arises, whether the ventral pyramidal tract might possibly serve as a path for bilateral innervations of the legs; this supposition would be in perfect accordance with the well-known fact that the lower ex-

¹⁰ See for a critique of the various vasomotor theories the excellent monograph of Cassirer, especially p. 56.

trémities show many common movements, whereas the upper extremities (with their unilateral innervation by the lateral pyramidal tract) are much more independent of each other.

It seems to me that the present case, in which both legs could move, and in which at the same time only the right ventral pyramidal tract comes into consideration for the conduction of the necessary stimuli from the cerebrum to the lumbar intumescencia, favors this hypothesis.

RÉSUMÉ

In this paper a case has been described of nearly complete rupture of the cord at the level of the ninth and tenth thoracic segment. The child was born by version and extraction, the shoulders were delivered by the method of Müller—from her birth until her death, about nine years afterwards—the little patient suffered from paraplegia (except for the flexion of the legs in the hip-joint), from total or almost total loss of sensibility of the legs, from incontinentia alvi et urinæ and from decubitus in the sacral region. The case demonstrates the danger which is always inherent to strong traction during delivery. (The method of Müller, by which a deviation of the traction axis from the longitudinal axis of the child is brought about, may be considered especially dangerous in this respect.)

From the microscopical examination it appears that the ventral pyramidal tract is the only long tract left intact by the lesion. It is supposed that this tract conducts motor stimuli to the anterior horns of the lumbar swelling on both sides, as the child could flex both legs. Though there was an almost complete destruction of the cord, the tendon reflexes were increased in the first period of life, whereas they disappeared in later years. Though the case cannot supply us with absolute proof, this condition is not in favor of Bastian's law. The acute decubitus in this case of rupture of the cord in a newborn infant is in accordance with the assumption of the existence of trophic influences of the central nervous system on the integuments.

From the examination of the ascending degeneration it appears that the lumbo-sacral segments occupy by far the greater part of the columns and the nuclei of Goll; the fibers of these lower segments end in the medial and medio-dorsal part of the gracile nuclei over their total length.

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