

A CASE OF ARRESTED DEVELOPMENT OF THE CEREBELLUM AND ITS PEDUNCLES WITH SPINA BIFIDA AND OTHER DEVELOPMENTAL PECULIARITIES IN THE CORD.

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WE have been induced to place this case on record by the fact of its exhibiting what appears to be an undescribed combination of deformities of the nervous system, all of which seem to us to be of much interest from the developmental standpoint. One of us (W. B. W.) has made himself responsible for the examination of the brain, the other (K. W. M.) for the clinical history and the examination of the cord.

I.—CLINICAL HISTORY.

E. M. H., a female infant, aged six weeks, was brought to the Infirmary for Children, Liverpool, on August 10, 1901. Family history as follows :—A grandmother and aunt died of phthisis ; the mother has one other child living ; after marriage she had two miscarriages, then the eldest living child was born, then followed another miscarriage and then this child was born.

Anamnesis :—At birth a swelling was noticed at the lower part of the back ; from this a creamy fluid was discharged for one day after birth ; it grew larger and then gradually smaller after

the discharge had recommenced. The mother noticed that the head was enlarging at the third week. The child was born with club feet and without any use in the lower limbs ; she also had *prolapsus ani*.

On August 12 it was noted—there is a linear ulcer with a few granulations over the posterior aspect of the fourth and fifth lumbar vertebræ ; the spine is bifid here but there is no bulging. There is well-marked hydrocephalus. The eyes are prominent ; measurement from external auditory meatus to external auditory meatus is $9\frac{1}{8}$ in. ; from root of nose to external occipital protuberance $10\frac{1}{8}$ in. The child shows no sign of being able to see but can hear. There is talipes equino-varus of the right foot ; the child moves the hands and arms freely but the legs are completely paralytic.

August 13.—Operation was undertaken to endeavour to drain the cerebro-spinal channels from the lumbar region. A flap was turned upwards exposing the second and third lumbar spines and laminae, the latter cut and a tightly distended tube of membranes exposed ; this was closed below at the level of the fourth lumbar by cicatrix. This tube was opened and a bunch of horse-hair strands passed into the opening ; the other ends were carried laterally into a bed dissected up for them between the muscular layers and the peritoneum. The wound was entirely closed.

For the first four days this drainage appeared to act satisfactorily, but on the fifth there was a considerable amount of tension of the structures around the wound, so much so that it was necessary to remove the stitches ; a slight amount of cerebro-spinal fluid came away into the dressings, but the drainage was much less satisfactory and the hydrocephalus persisted after showing at first some diminution. This condition of affairs continued until August 24, when the child showed signs of gastrointestinal disorder, with vomiting and diarrhoea ; she died on the 29th. The temperature chart shows nothing calling for comment, except for a rise of temperature on the 24th, at the commencement of the attack of gastro-enteritis ; there was apyrexia throughout.

II.—NAKED-EYE APPEARANCES OF BRAIN AND CORD.

Post-mortem Examination (August 29).

The cranio-vertebral cavity was opened throughout its extent, In the lumbar region an unhealed wound marks the position of the operative procedure ; the arches of L. 4 and L. 5 are represented by stunted projections from the lateral aspect of each

body; the arches of L. 3 and L. 2 have been removed. At the level of L. 4 the meninges and contents pass into a dense mass of cicatrix; above this the dura mater appears unusually tough and thick throughout. At the level of D. 4, the whole of the structures in the canal, membranes and contents, are divided into two apparently equal halves, an exostosis proceeding from the centre of the posterior aspect of the body of the vertebra, passing backwards between the divided cord and membranes, and then curving to the right and becoming attached to the right lamina near its junction with the spinous process. To the naked-eye the spinal roots appear normal. Above and below the exostosis the cord and membranes are undivided.

On slitting up the dura mater of the cord, this latter structure is found to be embedded in a casing of loose friable material, from the lumbar region to the cervical, where it becomes gradually less marked. The appearance of the cord after dissection from this bed is seen in the photograph (Plate I). It is a delicate attenuated structure; below there is no cauda equina distinguishable, but the cord becomes involved in the cicatricial mass at the level of the spina bifida.

The upper extremity of the cord is marked off from the medulla oblongata by a V-shaped depression, terminating in the well-marked anterior fissure. There are no prominences corresponding to the anterior pyramid and olive, and the medulla passes insensibly into the pons, no trace of the thickening caused by the transverse fibres of the pons being visible; the median raphé is well-marked, and on each side of this, opposite the rudimentary cerebellum, a slightly elevated ridge is present. The crura-cerebri emerge from beneath the thickened upper extremity of the pons, attached to each other mesially for some distance before dividing to enter the hemispheres. The optic chiasma and tracts are of normal appearance, and the corpora albicantia well formed. On the posterior surface, faint longitudinal lines indicate the fasciculi of the cord at its upper extremity. The inferior peduncle is very poorly represented. The cerebellum is represented by a very rudimentary structure with a few leaflets; it is impossible in this to distinguish the various parts; the worm is represented by a small oval prominence. The superior peduncle consists of a delicate flattened band of tissue on either side. The mesencephalon is represented by a single large structure; no trace of a division into anterior and posterior corpora quadrigemina being seen; the optic thalami are of moderate size, and the soft commissure is well formed.

The cerebrum exhibits marked flattening of the convolutions and extreme dilatation of the lateral and third ventricles. With these exceptions the hemispheres exhibit no peculiarities.

After removal the brain and cord were immersed in Müller's fluid.

III.—HISTOLOGY OF THE CEREBELLUM, MEDULLA, PONS, AND MESENCEPHALON.

The series of figures accompanying this article shows that we had to deal with a case of defective development of the cerebellum and of all those parts of the central nervous system which are connected with that organ. It is on account of the complete nature of this agenesis and the manner in which it is chiefly confined to the cerebellum and its annexes, that the interest of the anatomical findings in these regions arises.

Cerebellum.—The extremely rudimentary character of this organ was found to be accompanied by a similar absence of histological development.

The figures given represent accurately the outlines of its convolutions, which it will be at once seen were of an extremely simple nature. In many places the organ was entirely unconvoluted, and represented by an undifferentiated mass of embryonic tissue and small cells.

It was impossible to recognise the various named lobes. The lateral parts of the organ seemed to be slightly better developed than the remaining part of the hemispheres, and here were found almost the only myelinated fibres present. In these regions also, in a few parts, a differentiation into a cortex with its three layers of granular and molecular strata and Purkinje's cells could be seen.

No trace could be found of central gray nuclei. It is to be especially mentioned that there was no evidence of sclerosis, or of vascular degeneration.

The bulb.—The well-defined column of Burdach could be traced into the corresponding nucleus, the cells of which were large and numerous. It is doubtful whether a nucleus *gracilis* could be distinguished; this nucleus commences normally at a lower level than the cuneate nucleus, and

is situated medially. The first cell mass to appear in tracing the posterior columns upwards was situated laterally, and evidently in connection with the fasciculus cuneatus. The condition of the cord, especially in the lumbar region, is to be regarded as the explanation of this appearance. From this nuceus internal arcuate fibres were seen streaming inwards, fairly numerous and well coloured by the Weigert stain. Their decussation and the upward continuation of the fillet were well seen. The pyramids and their decussation were also fairly well coloured, though not so markedly as the fillet system.

No trace of the inferior olives or of accessory olives was found. Their usual position was represented by a well-defined area of embryonic tissue, entirely devoid of medullated fibres or of cells having any resemblance to the cells of the olives (figs. 5 and 6). External arcuate fibres, both anterior and posterior, were absent. Nuclei arcuati could not be recognised.

The cerebellar peduncles.—The three peduncles of the cerebellum were practically absent. The restiform body begins in normal brains to be visible on the outer side of the superior extremity of the nucleus cuneatus, but this region was almost devoid of medullated fibres. In the pons, where it is situated between the two roots of the eighth nerve, there was found a scanty number of irregularly-arranged myelinated fibres.

Both the corpus restiforme proper and its inner part appear almost unrepresented; no vestibulo-cerebellar fibres can be traced, although, as will be stated later, the end nuclei of the vestibular nerve appear well developed. The absence of the major part of the inferior peduncle is, of course, to be considered as connected with the absence of the olives and the scanty development in the cord of the direct cerebellar tract and posterior columns.

The figures show that the transverse fibres of the pons and middle peduncles were almost entirely absent; a few sections showed a band of medullated tubes situated laterally, which apparently represented all that existed of these structures.

The pyramids were seen to be lying in a mass of embryonic tissue. No trace of pontal nuclei could be discovered.

It would appear also that the superior peduncle was absent, a condition to be expected, considering that the nucleus dentatus and nucleus ruber could not be found.

Perhaps a few fibres in fig. 12 are to be considered as belonging to that system, and in the mesencephalon there is a decussation corresponding chiefly to the fibres of Meynert and Forel, but which may in part represent the superior peduncle.

The nuclei of the cranial nerves and tegmentum.—The deep origins of the cranial nerves were readily seen, and the various cell groups seemed normal.

Especial interest centres in the eighth pair. The large ventral cochlear end nucleus is well formed, and the lateral fillet system normal. The superior olives and corpora trapezoidea are well coloured by the Weigert stain. Fibræ acousticae are present, but not numerous.

With regard to the end nuclei of the vestibular nerve, so far as the method used will permit of observation, these cell groups were seen to be well represented, both by the central inner nucleus and the outer group of cells composing Deiters' nucleus. The various central and spinal fibres from these end nuclei could not however be recognised; in particular it was certain that no fibres passed into the cerebellum.

The posterior longitudinal bundle was well represented, but in the upper part of the pons, instead of occupying its usual position almost immediately beneath the fourth ventricle, it was seen to be separated from this by a mass of embryonic tissue (see fig. 11).

The nuclei tegmenti were fairly well seen.

Mesencephalon.—We have already mentioned the striking naked-eye appearance of this structure. On section the noticeable features were the entire absence of the red nuclei. The series of sections failed to reveal anything which resembled this important structure. The superior peduncles also were absent, with, perhaps, the exception of the rudimentary decussating fibres already alluded to. The feeble

development of the colliculi of corpora quadrigemina also arrested attention. In the pes the slight extent, laterally and medially, of the medullated tract, would appear to indicate that the *pars pyramidalis* only was represented—the fibres connecting the frontal and temporal lobes being absent. The third nucleus, fillet system, substantia nigra and fountain decussations were well seen.

IV.—HISTOLOGY OF THE CORD AND MEMBRANES.

Cord.—The cord after fixation and hardening in Müller's fluid, was divided into twenty-four portions, each of which was embedded in paraffin. Sections from each were stained (a) by the Pal-Weigert method, after impregnation with Marchi's fluid; (b) with hæmatoxylin and other chromatin stains.

The appearances found in sections from eight different levels will be described here, and any important points in intermediate sections also noted.

Series I. Upper Cervical (fig. 14).—The cord is reniform in shape. The central canal is represented by two channels; in section each shows as a slit, one transverse, the other antero-posterior. The transverse channel is lined by columnar epithelium, but only over part of its wall; the antero-posterior portion extends from within a few lines of the centre of the transverse towards the posterior margin of the cord; it is unlined by epithelium but bound by a membrane; between its anterior end and the transverse slit is neuroglial connective tissue. That these two portions each represent part of the canal is shown by the fact that they become continuous at a lower level. There is no trace of a posterior commissure. The gray matter shows numerous nerve-cells in the anterior horns, the medial, the anterior and the antero-lateral groups being well shown, the postero-lateral less so. The white commissure shows numerous medullated fibres. In the white matter medullated fibres are scanty throughout; there is a well-marked postero-external tract, however, and in the anterior ground substance medullated fibres are also fairly numerous; they are few and scattered in the direct cere-

bellar, antero-lateral ascending and descending tracts, and in the positions of the pyramidal tracts, and absent entirely in the position of the postero-internal column. In the latter locality and elsewhere, a fine neuroglial substance makes up that part of the cord outside the gray matter. The substantia gelatinosa is a very prominent structure.

Series II. Middle Cervical (fig. 15).—The canal is in the shape of a T, the vertical limb running towards the posterior aspect; the transverse slit is alone lined by columnar epithelium, and that in part only. In the gray matter the grouping of the cells is not distinct except that the mesial column is well marked; the cells of the lateral groups are numerous; the fibres of both anterior and posterior roots are distinctly medullated. Burdach's column is again well marked off, and contains numerous medullated fibres; the anterior ground substance also shows a considerable amount of medullation; but as before these fibres are few in the direct cerebellar, the pyramidal and the antero-lateral ascending and descending tracts, and absent entirely from the position of Goll's column. The medullated fibres in Burdach's column do not extend to the periphery of the cord. The substantia gelatinosa is as before a well-marked structure.

Series III. Lower Cervical. The Cord (fig. 16) is more flattened from before backwards than in the last series. This series is taken from the lowest cervical zone. The outline of the canal is in the form of a long transverse limb, and a short posterior extension from the centre of this. It is lined discontinuously with columnar epithelium; the apical part of the posterior limb is so lined, but not the remainder of this limb. The arrangement of the gray and white matter resembles that in the last series, that is to say, the postero-external column and the anterior ground substance are fairly well occupied, the remainder of the cord outside the gray matter being almost entirely without medullated fibres; in the gray matter the cells are numerous and well formed. By the lateral extension of the canal the anterior and posterior horns are cut off from one another. On the ventral aspect of the canal are a few medullated commissural fibres.

Series IV. Upper dorsal; just above the level of the division (fig. 17).—The cord is here a flattened ribbon-like structure, consisting of two lateral masses connected by a narrow strip. The canal extends from side to side across the middle line; on the one side it has a branched outline, on the other it is much smaller and spherical, the two lateral dilatations being joined by a slit with a narrow lumen; the epithelial lining is partial, the greater part being unlined. The gray matter is well represented on each side; in the anterior horns the mesial and lateral columns are large groups; by counting the cells of several sections it is found that the number is practically the same on each side. The only medullated fibres are a few in what appears to be the position of the postero-external column; the remainder of the cord structure is a fine gliosal tissue with numerous small cells.

Series V. At the level of D. 4; that is to say, at the division (fig. 18).—The halves of the divided cord are of about equal size, and irregularly circular in outline. The canal is represented on each side, but more extensively on one than on the other. On the left side the outline of the channel is oval, and it is completely lined by columnar epithelium; on the right side the outline is branched, and in some sections the branches appear as definite diverticula separated from the central channel by encroaching glial tissue. The cells in the anterior horns of each side are about equally divided in number; they are in two groups, one near the mesial, the other near the lateral margins of the horn. The amount of tissue outside the gray matter is now very scanty and staining with Pal-Weigert does not exhibit the presence of any medullation. The vascular spaces throughout the cord are large and numerous; that is to say, there appears to be unusual vascularity here.

Series VI. Lower dorsal; below the division (fig. 19).—The two halves have now reunited and form a cord of very diminished dimensions and reniform in shape. Here a remarkable change is met with, in that the central parts are disintegrated into a tissue of coarse fibrillar structure in which are numerous irregular cavities. This disintegration

has taken place posteriorly to the central canal, which is a transversely elongated slit. The outline of the canal is, however, largely obscured and the lumen invaded by the process of disorganisation. In addition to the coarse fibrillation of this disintegrated area, there are a large number of "colloid bodies" present throughout it, and also a few giant cells; fragments of columnar epithelium, evidently derived from the lining of the invaded central canal, are also found here and there; there is no round cell infiltration or any evidence that the process of change is of an inflammatory origin. There are a few well-formed ganglion cells in the anterior horn on each side. No trace of medullation in the fine gliosal tissue which constitutes the circumferential part of the cord.

Series VII. Mid-lumbar (figs. 20 and 21).—The cord is here reduced to very small dimensions; the kidney shape is noticeable. There are traces of the disintegrating process noted in the series above, but it is much less marked here; the sections show that this disorganisation, commencing at about D. 6 or D 7, reaches its maximum in the lower dorsal region, and disappears in the lumbar portion of the cord above the level of the spina bifida.

In this series there is no marked differentiation between gray matter and white matter; the whole structure is a fine gliosal tissue, the only exception being the presence of a few ganglion nerve-cells in the position of the anterior horns on each side. The outline of the central canal is slit-like from side to side, lined only in part by columnar epithelium.

Series VIII. Level of the spina bifida.—At this level the nervous tissue is represented by a central structure representing the cord, by nerves, nerve-roots and ganglia. All are embedded in a fine connective tissue containing numerous large vascular channels. The cord is more flattened than in the last series but is kidney shaped; there are a few nerve-cells in the position of the anterior horn on each side; no medullated fibres. The posterior ganglia exhibit numerous well-developed ganglion cells, but also show a marked hypertrophy of intercellular substance consisting of a highly cellular connective tissue.

Membranes.

Histologically the dura mater is of well-developed fibrous tissue. Within this the distinction of other coverings is entirely obscured by the material in which, as already described, the cord is embedded. This material consists of a highly cellular connective tissue containing numerous large thick-walled blood-vessels.

Ganglion Cells of the Cord.

Cervical region.—In this region there are three well-defined groups in the anterior horn. (a) Mesial column—large cells ten to fifteen in number. (b) Ventro-lateral column ten to twelve in number. (c) Dorso-lateral column, the largest of the three groups, seventeen to twenty cells. The numbers indicate the cells counted in each section and averaged.

Centrally to the dorso-lateral group are scattered cells of small size not marked off into groups but most numerous in the position of the middle cell column. No defined Clarke's column. No cells in posterior horn.

Upper dorsal region.—The cord here is elongated laterally. There are two well-defined groups of cells in the anterior horn. (a) Mesial group, large cells six to eight. (b) Lateral group not (subdivided) cells twenty-five to thirty in number. Between these groups but more centrally placed are a few scattered cells of small size five to seven in number. No cells in posterior horn.

Mid-dorsal region.—The total number of cells is smaller. The mesial and lateral groups are well defined and contain about an equal number of cells, twelve to eighteen in number, in different sections. There are also a few centrally situated, five to six in number. The cord is here divided. Immediately below the division—the mesial group is large—of large cells, fifteen to sixteen in number; the lateral group is composed of smaller cells, four to eight in number.

No cells outside these two groups.

Lower dorsal region.—The ganglion cells are, with the exception of a few scattered laterally, grouped into one mesial column, eight to ten in number.

Upper lumbar.—Cells very few in number; in some sections none are seen. The average is three in each anterior horn, placed antero-mesially.

Mid-lumbar.—The cells are here more numerous than in the upper lumbar region; they are in two groups, counted in eight sections at different levels:—

(1) Antero-mesial, 4, 3, 4, 2, 6, 1, 2, 4.

(2) Antero-lateral, 3, 1, 4, 3, 3, 5, 3, 2.

Sections of the posterior root ganglia show the cells to be numerous, large and well formed.

SUMMARY.

A child aged six weeks, presenting a lumbar spina bifida ruptured at or before birth, and now healed; hydrocephalus; paraplegia of the lower limbs. Death from intercurrent gastro-enteritis.

(1) In the brain. Arrested development of the cerebellum and of its efferent and afferent fibres, and of their nuclei of origin; of the restiform bodies, the olives, accessory olives, external arcuate fibres and nuclei arcuati; middle peduncles and transverse fibres of the pons, pontal nuclei, vestibulo-cerebellar tracts; superior peduncles, gray nuclei of cerebellum, red nuclei of the tegmentum; possibly the cerebro-pontal cortical tracts.

(2) In the cord. General attenuation of this structure throughout, division at the level of the fourth dorsal vertebra into two equal halves; marked enlargement of the central canal which divides with the division of the cord; the presence of various irregular cavities in the cord and disintegration of its central parts in the lower dorsal region; normal development of the gray matter with well-formed ganglion cells; scanty development of white matter, its place being taken by tissue of neuroglial structure.

(3) The coverings of the cord. The whole cord, as far as the upper cervical area, embedded in a fine connective tissue with large vascular channels; the laminæ and spines of the fourth and fifth lumbar vertebræ deficient; an exostosis dividing the canal at the level of the fourth dorsal vertebra.

V.—COMMENTARY.

The case exhibits a condition which may be generally described as arrested development of the central nervous system from the mesencephalon downwards, with a super-added local process of degeneration. In addition to this the abnormalities in the parts contiguous to the cord are to be considered, and on these we may first comment.

I.—The deficiency in the completion of the bony casing affects that section where the deformity is most commonly present. The exostosis which divides the canal and its contents is a rare developmental fault, but there are records of several cases presenting similar bony septa. Thus in the report of the Clinical Society on spina bifida four examples of this variation are recorded (1). One at the tenth dorsal vertebra, the whole of the laminæ and spines below being deficient; one in the lower dorsal region at the apex of a spina bifida involving the whole canal below, associated with an entirely bifid cord below; a third exhibiting a similar condition in the lumbar region, but with union of the cord below the exostosis; the fourth concerns a spina bifida of the last two dorsal and the upper two sacral vertebræ, the cord being divided into unequal parts and reuniting below.

M. Houël (2) has described a case in which a similar exostosis projected from the bodies of the third and fourth lumbar vertebræ, with its apex partially occluding the opening into the sac of a lumbo-sacral spina bifida; the cord at the point being divided into two parts and reuniting below. Sulzer (3) found in a lumbar spina bifida complete division with reunion below of the cord, apparently due to a similar exostosis.

It will be seen that these cases present one marked point of difference from that described here in that the exostosis and the division of the cord was in immediate relation with the deficiency in the vertebral arches constituting the spina bifida. M. Houël was of opinion that the exostosis in his case was in direct casual relationship with the deficient

laminar junction; on general grounds and in the light of this case it appears more reasonable to consider this variation in cases of spina bifida as merely concomitant, the deficiency in the laminæ and spines and the growth of such exostoses being all faults in the development of those groups of cells which form normally the bony casing of the cord. Another feature in which this case appears to differ from others similar is in the division of the meninges into two entirely separate tubes, each encasing one half of the cord. Apart from the presence of an exostosis such as was found in this case, the association of division of the cord and spina bifida has been recorded by several, *e.g.*, Recklinghausen (4), Koch (5), Ollivier (6). A division of the cord, a rachischisis, also occurs unassociated with spina bifida. A case of this kind is related by Recklinghausen (7), and another has been recorded by Foà (8). In each case, as in our own, the division was into two approximately symmetrical halves, with gray matter and white matter normally distributed, and with a central canal in each half. One of us (K. W. M.) has recently had the opportunity of dissecting a specimen which is also of interest in connection with this rachischisis. A fœtus born at full term exhibited a condition of anencephaly with spina bifida involving the cervical and dorsal regions. The nervous tissue representing the spinal cord was in the form of a tube with membranous wall, on opening which four prominent longitudinal ridges were seen to bulge into the lumen, arranged symmetrically, two on each side. These were found to consist of myelinated nerve-fibres; at the lower dorsal region the separation into two halves became distinct; on the right side the two ridges united to form a rounded cord, on the left the nervous tissue remained diffusely distributed on the inner surface of the membranous covering. The separation into right and left halves was complete but unsymmetrical.

This case and those of Recklinghausen and Foà, show that apart from the presence of a mechanical obstruction, a rachischisis occurs as a developmental fault, *sui generis*; in place of the union of the dorsal and ventral columns of nervous tissue on each side through the medium of the roof

and floor plates to enclose a single canal, each dorsal column unites with each ventral column and the floor and roof plates default. In the process of union of the dorsal with the ventral column, a portion of the lateral extension of the central canal is cut off, and in this way a channel is present in each half.

The presence of an exostosis, as in this case here recorded, affords an easy explanation of the etiology of the rachischisis; in the absence of such a condition the explanation of the division is not forthcoming; it is one of the rarest of deformities.

The tissue in which the cord was embedded is of much interest. At first sight it seemed probable that it was of the nature of an inflammatory effusion, but the histology entirely negatives this. The structure indicates an origin in mesoblast, in that portion of mesoblast which extends backwards in a double fold to enclose the neural tube, and which is spoken of by embryologists as the membranous primordial vertebral column, and from which the cutis, the vertebral arches and the cord coverings originate. As an erroneous developmental product it belongs to the same category as the maldevelopment of the vertebral arches. We are not aware of any quite similar observation recorded; Recklinghausen (9) has, however, placed on record a case of *spina bifida occulta*, in which a myofibrolipomatous tissue occupied the spinal canal in the lumbar region, and encircled the cord¹; the structure in our own case is also analogous to that described by Mr. Bland Sutton (10) in the variety of *spina bifida* to which he gave the name *myelocoele*. In this variety the medullary folds coalesce imperfectly, the central canal opens dorsally, and the tissue surrounding the furrow which is its continuation, shows microscopically nerve-cells, neuroglia and a few nerve-fibres embedded in a nœvoid mass of dilated capillaries and connective tissue.

II.—Probably in the great majority of cases of *spina bifida* there exists also some developmental peculiarity in the cord. Even in cases of the uncommon simple menin-

gocele, the cord not unusually shows some abnormality; *e.g.*, in a case of apparently simple meningocele lately examined by one of us (K. W. M.), the cord exhibited a marked degree of hydromyelia above the level of the spina bifida.

The case now under consideration shows this involvement of the cord in an extreme degree. The central canal is not only of very large dimensions throughout, but its outline is similar, generally speaking, to that which is found in the embryo up to about the eighth week, at any rate in the cervical region of this case. The way in which the posterior limb of the canal is closed by the approximation of its lateral walls gives strong support to the belief that the posterior median septum (the so-called fissure) is normally formed by the growing together walls of the dorsal part of the canal. It is not easy to explain the deficiency of the lining of cylindrical epithelium over a considerable proportion of the wall of the canal; it appears necessary to attribute it to the increased area of the wall and the consequent proportionate deficiency of those cells which normally are sufficient to give the canal a complete lining. A similar deficiency has been noted by Chiari in cases of hydromyelia recorded by him (11). The ependyma is, however, a formed structure very early; the cells are ciliated in the human foetus at the fifth week, and line the large canal, but this is, of course, of a much smaller surface area than the canal in the cord we are describing.

In the tissue of the cord itself the condition of first importance is that its substance is to a very large extent of a fine neuroglia structure, with marked defect in the development of the nervous elements proper. It must be remembered that the neuroglia is of epithelial origin, and derived from the cells of the neural tube; it is therefore the less specialised of the two elements which arise from these cells, and the development of the more specialised is at fault in this case. It is well known from the pathological anatomy of syringomyelia how frequently associated are abnormalities of the central canal and the presence of undeveloped embryonal nervous tissue of a fine neuroglial structure. The

presence of this glia tissue is differently explained; on the one hand it is widely held that in most cases the syringomyelia is secondary to an actual hyperplasia of glia tissue, a gliomatosis, an over-production; on the other hand a second view holds that the original starting point of the cavity formation is the presence of undeveloped glia tissue which should have been transformed, but which persists owing to the want of the normal guidance to transformation.

It seems clear that the preponderance of glia tissue in our own case is to be ascribed to checked development and arrested specialisation. v. Leyden (12), who holds that this glia tissue is a manifestation of developmental defect, recorded some years ago two cases of much interest for comparison with our own: one a child, aged two years, with syringomyelia enclosed by layers of embryonal glia tissue, and associated with encephalocele and a rudimentary cerebellum; the second case, a child aged two and a half years, with hydrocephalus, rudimentary cerebellum, and syringomyelia, accompanied by undeveloped embryonal glia tissue. Apart, however, from the distended central canal, we have in the lower dorsal region a localised area in which this embryonal tissue has undergone a definite degenerative process with resulting excavation of the central parts of the cord. Of the exact causation of this excavation we are unable to express a confident opinion. As before stated, there is no evidence which enables us to state that it occurred as a result of any actual inflammatory process strictly so called. The two most striking points in the histology are the presence of the "colloid bodies," and the occasional occurrence of a giant-cell system. The origin of these colloid bodies, which have been described in a variety of degenerative processes in the central nervous system, is variously interpreted. They are supposed by some to arise from myelin set free by the degeneration of the myelinated fibres, but this origin is out of the question here as there is no myelination in the region where they are found. The giant cell is, in our opinion, always a degeneration product, as a rule associated with the destruction wrought by a chronic inflammatory process, such as tuberculosis and

syphilis. On the whole we consider that the evidence is in favour of the incidence of an active disintegrating influence supervening on the congenital abnormality, and not of a simple degeneration from inherent or autonomous causes.

The case exhibits in a striking way the association of hydromyelus, gliosis, and true syringomyelia. It is known from the researches of His (13), Lenhossek (14), and others that the ependyma and neuroglia possess a common developmental origin; it is not surprising, therefore, to find abnormalities of the one associated with abnormalities of the other. Hoffman and Schlesinger have stated the dependence of the central gliosis, which is the basis of the majority of cases of syringomyelia, on a primary hydromyelia of the central canal; however this may be, the association is easily understood. As we have seen, in this case the central cells have provided but a partial lining for the canal, the remainder being lined by tissue of glia structure similar to that which constitutes the remainder of the cord outside. In relation to the genesis of syringomyelia the view is commonly held that a true hyperplasia of glial tissue is the first product, but authorities differ as to the question of referring this "gliosis" to a congenital abnormality. Hoffman says (15) "the basis and origin of the disease process is in most cases a congenital developmental anomaly, whereby nests of embryonal tissue remain behind the normal central canal; a plicated central canal possesses the same importance." Similarly Schlesinger: "I consider that the central gliosis originates in the central canal epithelium; with this are found for the most part other congenital anomalies, especially widening of the central canal; that the excavation of the new-formed tissue results from degeneration of the same, dependent on vascular abnormalities."

The case here before us does not conform to the most commonly observed type of syringomyelia in that there is no evidence of anything that can strictly be called gliosis or local proliferation of glia cells; but the whole cord shows a condition of gliosis, if this term means the presence of young glia tissue, and it is in this posterior to the central

canal that the degeneration and excavation is taking place. *It shows this cavity formation in progress, and its connection with an undoubtedly developmental anomaly.*

In Series IV. it is seen that on one side the canal has a branched outline, and that the branches tend to be cut off from one another by the encroachment of their walls; in this way also cavities partially or entirely lined by epithelium arise. It is doubtful whether persistent cavities in the cord result from such a process as this, as it appears probable that they will in the process of growth be encroached on to their obliteration.

The absence of myelinated fibres throughout the greater part of the cord is a matter which calls for remark. It is known that in cases of spina bifida where the cord is embedded in the sac wall, both it and the nerves originating from it exhibit an entire absence of medullated fibres. An example of this peculiarity is reported by the Clinical Society's Committee.

In our own case the number of myelinated fibres is very scanty throughout the whole cord, and they are chiefly absent from the system tracts throughout. These fibres are present only in (a) the anterior and posterior roots, and (b) the postero-external column, and the ground fibres of the antero-lateral region, that is to say in the ground tracts above the division of the cord. It is in these regions that the fibres normally first acquire their medullary sheaths, and the arrested development of this case confirms in this way the information which has been derived from the study of the natural process.

III.—A considerable number of cases of atrophy of cerebellum have been recorded since Combette's often quoted paper in 1831. Many of the earlier cases were not examined microscopically, or only by the older, obsolete methods, and hence do not possess much anatomical interest. In the last ten years however, several detailed and valuable accounts of this condition have appeared, notably the papers by Menzel, Max Arndt, Spiller, Thomas and Dejerine (*vide* Appendix). These writers also give a fairly complete account of the literature, especially is this so in the detailed analyses

given by Thomas in his "Thesis on Le Curvelet." Ferrier also in "Allbutt's System" tabulates the cases since 1879, correlating the clinical symptoms with the anatomical findings. He thus completed the list given by Nothnagel in his "Tophische Diagnostik."

Lastly, quite recently Dejerine and Thomas have given an account of a case with an unusual pathogenesis. The origin of these conditions, indeed, varies very greatly, and in an appendix we have endeavoured to classify the cases on a basis on what appears to be their mode of origin. The clinical interest of many of the cases is considerable, first because they show the symptoms resulting from simple defect of this organ, and secondly, because of the increased interest which these observations have thrown on the nature of the hereditary ataxia of Marie, Friedreich's disease, and certain allied conditions. The clinical aspect of the subject does not now concern us, but we may mention one fact which appears to be very clearly illustrated by a study of the cases, viz., the same symptom group may appear with very different anatomical lesions. Thus, the cases of Nonne and Muir showed a simple smallness of the cerebellum. Spiller's is one of sclerotic atrophy, Menzel's of sclerotic atrophy with lesions of the cord, and Meyer found that the cord alone was affected, yet in all these cases the symptoms were closely allied.

The interest in the present case is for the most part anatomical; in the brain at least the condition appears to be one simply of arrested development, and owing to its completeness the relationship which exists between the cerebellum and its annexes is demonstrated in its main features with remarkable clearness, and confirms if it does not add anything new to our knowledge on this subject.

As to the date at which the development became defective it is difficult to say; it must have been very early, for the olives can normally be made out at the end of the third month. The growth of the organ must, however, have been irregular, for Purkinje's cells, which were found to some extent, usually only appear about the sixth month, whilst the corpus dentatum, which also, according to Obersteiner, appears at that time, was absent.

The majority of anatomical findings which we hope will be obvious from a consideration of the figures and the text, do not require any comment, they contain what is established both by a study of similar cases and by various other methods of research.

A few points merely may be mentioned.

The absence of external arcuate fibres, combined with the integrity in part at least of the dorsal column nuclei, has been noticed by a good many observers, as in the cases of Menzel, Arndt, Cramer, Thomas, and Spiller. In these cases the nuclei arcuate were also absent, and the fact suggests, as Bechterew supposes, that these cells form relay nuclei. Menzel believes some of these fibres arise in the cerebellum.

So far as the method employed permitted, no atrophy was noted in the nuclei reticularis tegmenti; the cells appeared numerous and large. This is important in view of the finding of Cramer in his case of unilateral atrophy, that the nucleus on the contra-lateral side was atrophied. Cramer contends that his case shows the connection of this nucleus with the middle peduncle.

The fasciculus centralis tegmenti is described by Bechterew and Flechsig (*Neurolog. Centralb.*, 1885) as a descending tract terminating in the olive and forming by this body a connection between the lenticular nucleus and cerebellum. It is situated just external to the inferior olive, and higher up between the superior olive and fillet. Spiller believes it was atrophied in his case, and in ours also it cannot be seen. Short tegmental fibres appear numerous and well stained. It is also of interest to note that the fillet and posterior longitudinal bundle were both well developed, notwithstanding the absence of the olive and cerebellum. This is in accord with the experimental results of Ferrier and Turner.

Lastly, we have mentioned that there is some reason to regard the fronto- and occipito-temporal frontal tracts as being undeveloped. This is difficult of explanation, as it is generally regarded that both are corticofugal systems. It may be mentioned, however, that Obersteiner, in the last edition of his "Anatomy of the Nervous System," states that the latter tract is in part corticopetal.

In conclusion, we wish to direct attention to the history of the pregnancies of the mother of this child—two miscarriages after marriage, then a living child, then another miscarriage, followed by the child here described. Such a history suggests syphilis, but no further evidence could be obtained in support of this suggestion. If syphilis was concerned in this case, and we believe that the history of many cases of spina bifida is tainted by this disease, its activity produced a series of remarkable defects in the fœtus.

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- (2) *Bull. et Mem. de la Soc. de Chir.*, May 9, 1877.
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- (4) *Virch. Archiv.*, Bd. 105, p. 418.
- (5) *Beitr. z. Lehre v. d. Spina Bifida*, 1881.
- (6) *Traité d. Malad. d. l. Moëlle épiv.*
- (7) *Loc. cit.*, p. 409.
- (8) *Riv. sperim. d. Freniatria*, 1878.
- (9) *Loc. cit.*, p. 248.
- (10) *Lancet*, 1888, vol. i., p. 359.
- (11) *Denkschr. d. Wein. Akad.*, Bd. 63, 1895.
- (12) *Virch. Archiv.*, Bd. 68, p. 1.
- (13) *Arch. f. Anat. u. Phys.*, 1890.
- (14) *Der feinere Band. Nervensystems*, Auf. ii., p. 176.
- (15) *Deut. Zeitschr. f. Nervenheilk.*, Bd. iii., p. 82.
- (16) *Die Syringomyelie*, 1895, p. 188.

APPENDIX.

A Classification of Cerebellar Atrophies arranged on the Basis of their Probable Pathogenesis.

I.—Cerebellum almost completely absent on one or both sides. Condition primarily due to arrest of development.

1. COMBETTE.—*Bull. de la Soc. Anat.*, 1831.

An epileptic idiot died, aged 11. A gelatinous membrane which represented the cerebellum was connected with the medulla by two membranous peduncles. Pons absent. Cord and brain normal.

2. D'ANDRAL.—*Clinique Médicale*, vol. v. (abstract from Thomas, *Le Cervelet*).

A woman died, aged 45. Mentally feeble and liable to incoordinated movements in the limbs. Left cerebellar hemisphere practically absent.

3. LALLEMENT.—*Soc. Anatomique*, 1862.

Patient died from apoplexy, aged 53. No symptoms during life. The left cerebellar hemisphere was absent with the same sided superior peduncle and contra-lateral olive and nucleus ruber tegmenti.

4. LEYDEN.—*Virchow's Archiv.*, lxviii., 1876.

Two infants with meningocele, syringomyelia and absence of cerebellum.

5. HITZIG.—*Archiv. f. Psychiatrie*, xv., p. 267, 1884.

Patient died, aged 33. As a child was of slow intellect, gradually developed symptoms resembling general paralysis, with ataxia of both extremities and oscillation of the body.

Right cerebellar hemisphere reduced to a minimum, middle peduncle and pons with contra-lateral olive absent.

6. FERRIER.—“Functions of the Brain,” 1886.

Girl died, aged 15. Mentally feeble, with general muscular weakness and tremors of the hands.

The cerebellum was represented by a minute nodule. Pons and peduncles were absent.

7. NEUBURGER AND EDINGER.—*Berlin Klin. Wochen.*, 1898.

Man died, aged 46. He presented no nervous symptoms but suffered from bradycardia, death from cardiac failure.

Right cerebellar hemisphere practically absent.

Diminution of right superior and middle peduncles with the contra-lateral olive and red nucleus. Diminution in the central gray cerebellar masses and in the left nuclei pontis.

8. This case 1902.

II.—Cerebellum congenitally small, but of normal histological structure.

1. OTTO.—*Archiv. f. Psych.*, p. 730, 1874.

Man died, aged 39. Mentally feeble, but no physical inability.

Cerebellum was very small, pons rather narrow, but brain and cord normal in size.

2. VERDELLI.—*Rivista Clinica*, 1874 (abstracted from Thomas).

Man died, aged 19. Markedly rachitic. Epileptic convulsions from two years old.

Proportional reduction in size of cerebellum, medulla, and pons.

3. NONNE.—*Arch. f. Psych.*, 1891.

Patient died, aged 40. This is one of the cases in which Marie founded his “Hereditary Cerebellar Ataxia.” The whole central nervous system was small, but especially the cerebellum.

4. MIURA.—*Mittheil. der med. Fac. der Kaiserlich, Japan univ. La Tokio*, Bd. iv., Heft 1, 1898. "Abstract from Dejerine and Thomas" (*loc. cit.*).

One of two brothers exhibited symptoms similar to those in Nonne's case. Smallness of the whole central nervous system, especially of the cerebellum.

5. FRASER.—*Glasgow Med. Journal*, 1880.

Another case with symptoms resembling those associated together by Marie. Pathologically the examination was incomplete, but smallness of the cerebellum was the chief lesion with slight histological defects in its structure.

III.—Comprises the majority of cases. There is an atrophy with a general sclerotic condition. The cortex appears primarily affected, either the whole or in part. The disappearance of the white matter corresponds to the extent of cortical defect. There are often indications of chronic meningitis and vascular degeneration. The symptoms are often those of epilepsy with mental enfeeblement, and in a number of cases date from the occurrence of some acute infectious disease in early childhood. It is difficult to discern the exact pathogenesis of these conditions, probably an acute disease acts as an exciting agent in developing a previously existing morbid condition, and leading to retrograde changes in nervous tissue.

The vascular changes are in this division of cases usually secondary in great part.

1. DUGUET.—*Soc. Anatomique*, 1862 (abstracted from Thomas).

(a) Woman died, aged 39. No symptoms till 26, then epileptic fits and general weakness developed.

(β) Man, aged 72. No symptoms till three months before death, then convulsions occurred, followed by incoordination movements.

(γ) Patient died, aged 17. Epilepsy since 18 months old.

2. MAYNERT.—*Wien. Med. Jahr.*, 1864.

Died aged 62, from mitral disease. Symptoms began three years before death. They were incoordination, trembling, and affection of speech.

3. CLAPTON.—*Trans. Path. Soc.*, London, 1871.

Died at 33 from pleurisy. Measles at 4 years old, and then gradually unsteadiness in walking, enfeeblement of intellect, and inability to properly use his hands.

4. PIERRET.—*Archiv. de Phys.*, 1872.

Died aged 69. At 4 had a "fit," and never quite recovered. Staggering gait developed.

Cerebellum, middle peduncle and olives markedly atrophied.

5. MESCHÈVE.—*Virchow's Archiv.*, p. 559, 1880.
Died aged 40. Epilepsy with mental alteration, rotation in direction of an entering corkscrew. Gait oscillating, speech scanning. Sclerosis of right cerebellar hemisphere and nucleus dentatus.
6. CLAUS.—*Arch. f. Psych.*, vol. xii., p. 669, 1882.
An insane epileptic.
Partial sclerosis of cerebrum and cerebellum.
7. KIRCHOFF (Case II.).—*Arch. f. Psych.*, Bd. xii., 1882.
Woman of 20 died from phthisis. There was trembling and slow choreiform movements.
Cerebellum was very small and hard. K. believes a sclerotic process had become engrafted to an arrest of development occurring at the seventh month.
8. BORRELL.—*Neur. Centralb.*, 1883.
Died aged 83. Convulsions and mental defect since infancy, tottering gait, tonic spasm of the muscles of the neck. Pia mater thickened.
Left cerebral hemisphere practically absent, right much diminished. Olives invisible and pons very small.
9. MAYOR.—*Journal of Mental Science*, 1883.
Died aged 82. Epilepsy and idiocy since birth. No disorder of movement.
Right cerebellar hemisphere sclerosed, with some healthy patches in places.
10. SOMMER.—*Neur. Centralb.*, 1884.
Died aged 30. Severe cerebral illness at 3 years old; in the latter period of life was subject to psychological agitation, and a peculiar gait of propulsion and staggering.
11. INGELS.—*Bull. de la Soc. de Med. de Belgique*, 1884.
A dangerous epileptic. No motor disorder.
Pons as well as cerebellum atrophied.
12. BOURSONT.—*Annal. Medico-Psych.*, 1891 (quoted by Thomas).
Four cases of insane persons with atrophy of cerebellum.
13. MENZEL, 1891.—*Archiv. f. Psych.*, 1891.
Illness began at 34. Ataxia of all limbs, increased knee-jerks. Tetanic-like contraction of muscles of neck. Affection of speech.
Cerebellum very small, absent in parts altogether. Both cortex and white matter markedly affected. Atrophy of pons and pontal nuclei, olives, part of restiform body. In the cord the posterior columns, pyramids, and dorsal cerebellar tract were found diminished in size. Menzel regards the case as one of primary development error, and the spinal lesion as due to a chronic meningitis. No evidence of vascular disease or of increased connective tissue formation.

14. HAMMARBERG.—*Neur. Centralb.*, 1892.

Died aged 24. At 7 years old severe "cerebral inflammation," gradually followed by oscillatory movements and staggering gait.

Both hemispheres the seat of a sclerosis especially attacking the cortex, also patches of sclerosis in the frontal lobe and in left olive.

15. CRAMER.—*Beiträge z. Path. Anat.*, Bd. xi., p. 39, 1892.

Died aged 48. Epileptic since 14, with mental alienation. No particular motor disorders.

Left cerebellar hemisphere sclerosed and atrophied.

16. ROYET AND COLLET, 1893.—*Archiv. de Neurologie*, 1893.

Death at 56. Appears to have been well up to age of 48, then developed staggering gait, increased knee-jerks, slight tremors and nystagmus.

Cerebellum hard and sclerosed, with atrophy of the middle peduncle and pons. No microscopic examination.

17. MOELI.—*Neur. Centralb.*, p. 553, 1889.18. BOND.—*Journal of Mental Science*, p. 409, 1895.

Died aged 60. Mental deficiency since 17.

Ataxia and general tremor. Symmetrical atrophy and sclerosis of all parts of cerebellum.

19. SPILLER.—*Brain*, 1896.

At 5 years of age scarlet fever and diphtheria, and since then trouble in walking, gait staggering, mental deficiency. Died at 19 from tuberculosis. A sister affected in the same manner.

Post mortem.—Deficiency of corpus callosum, small size of cerebellum, atrophy of left cerebral hemisphere. Sclerosed areas in the cerebellum with thickened vessels. Atrophy of inferior olives, middle peduncle of pons, and pontal nuclei. External arcuate fibres and nuclei arcuati and diminution of nuclei reticularis tegmenti.

IV.—Primary vascular disease, attended by chronic interstitial inflammatory changes, chiefly affecting the white matter. Comparable to cirrhosis of the kidney.

1. SCHUTZE, 1887.—*Virchow's Archiv.*, 1887.

Death at 48. Diabetes insipidus since infancy. Developed a staggering gait, slow speech, traces of the intentional tremor, gradual paralysis.

Endarteritis obliterans of basilar and vertebral arteries. Cerebellum greatly atrophied from a patchy sclerosis, molecular and granular layers little affected, Purkinje's cells showed marked changes. Increase in neuroglia tissue and diseased vessels. White matter greatly destroyed. Dentate nucleus and superior peduncle, olives and middle peduncle were markedly atrophied. Cord normal.

2 MAX ARNDT, 1894.—*Archiv. de Psychiatrie*, 1894.

Death at 70. Symptoms for the last four years. Gait unsteady, ataxic movements with paresis in arms and legs.

Thickening and disease of arteries at base of brain, cerebellar sclerosis, evidence of chronic inflammatory changes in its substance, dilated capillaries, round-cell infiltration, thickened fibrous tissue. In the cortex Purkinje's cells mostly affected, the molecular and granular layers practically normal. The white matter markedly sclerosed, atrophy of middle peduncle and transverse fibres of pons, of olives. Integrity of the nuclei of posterior columns of cord, but some atrophy of fibræ arcuatæ, internæ and externæ.

V.—A primary atrophy affecting the cerebellar cortex, the nuclei of the pons and the inferior olives, accompanied by marked atrophy of the middle peduncle and partial atrophy of the restiform body. No increase of connective tissue or of altered vessels. This atrophy of cells is considered to be comparable to the Duchenne-Aran atrophy of the cells in the ventral cornua.

1. THOMAS, obs. iv.—*Le Cervelet*, Paris, 1897.

Patient died, aged 62. Symptoms began at 55. Staggering gait, oscillation of the body, scanning speech.

Cerebellar cortex greatly diminished in all its component parts. Gray nuclei intact. In the cerebellar medulla there was distinct diminution of myelinated fibræ. Atrophy of transverse fibres of pons. Middle peduncle, outer part of corpora restiforma, inferior olives, nuclei arciformi and superior external arcuate fibres.

2. THOMAS, obs. v.

Age 44 at death. Illness began at 25, with progressive enfeeblement of lower limbs, later, mental incapacity, paresis, tremors, universal loss of sensation of all form.

Cerebellar cortex markedly atrophied while white matter only slightly affected. Atrophy of olives, middle peduncle, transverse fibres of pons, restiform bodies (partially), cells of nuclei of Goll and Burdach, internal and external arcuate fibres. In the cord the posterior and dorsal and ventral cerebellar tracts affected.

3. DEJERINE AND THOMAS (two cases).—*Nouvelle Iconog de la Salpet.*, 1900.

Case (1).—Death aged 55. Illness began about 8 years earlier, gradual inability to walk owing to trembling and staggering, speech become scanning in character. No loss of power or of muscle sense, knee-jerks increased.

Atrophy of cerebellar cortex with degeneration and disappearance of afferent and efferent fibres, of olives, and pontal nuclei, while the central gray nuclei were intact with the superior peduncle and nucleus ruber tegmenti.

Case (2).—Clinical history only given.

DESCRIPTION OF FIGURES.

FIGS. 1 to 13.—*A series of transverse sections from the upper cervical region to the superior extremity of the mesencephalon. Drawn by means of Edinger's drawing apparatus.*

FIGS. 1 and 2. Show commencement of pyramidal decussation. Note the scanty peripheral fibres and the entire absence of fibres in the internal dorsal region of the cord. In fig. 2 the nucleus cuneatus is just becoming visible.

FIG. 3. Pyramid has almost crossed.

FIG. 4. Section at level of fillet decussation.

FIG. 5. Level of usual site of inferior olive, here represented by embryonic tissue. Note scanty peripheral fibres representing the spinal cerebellar tracts.

FIG. 6. Upper level of inferior olive, commencement of vestibular root and rudimentary corpus restiforme.

FIG. 7. Commencement of lateral fillet system, the anterior region of pons entirely devoid of nerve-fibres.

FIG. 8. Level of sixth and seventh nerves.

FIG. 9. Section of maximum development of the cerebellum.

FIG. 10. Level of fifth nerve. Note absence of transverse fibres in pons, possibly the medullated tract situated laterally is a representative of these.

FIG. 11. Upper level of pons.

FIG. 12. Lower part of mesencephalon. Note limited area occupied by medullated fibres in the pes. Well-marked fibres of Meynert and Forel, and perhaps also some representative of the superior cerebellar peduncle.

FIG. 13. Level of third nucleus. No nucleus ruber tegmenti seen.

FIGS. 14 to 21. A series of transverse sections from the upper cervical region to the upper lumbar, described in the text.

ABBREVIATIONS.

- pyr.* = pyramid.
s.g. = substantia gelatinosa.
n.c. = nucleus cuneatus.
dec. fl. = decussation of the fillet.
des. rt. v. = descending root of fifth cranial nerve.
cor. rest. = corpus restiforme.
cor. trap. = corpus trapezoideum.
s. ol. = sup. olive.
n. coch. = Cochlear nucleus.
n. d. = Deiters' nucleus.
n. c. v. = nucleus centralis vestibularis.
p. l. b. = posterior longitudinal bundle.
n. v. = fifth nucleus.
s. ng. = substantia nigra.
dec. M. & F. = decussation of Meynert and Forel.
a. r. f. = anterior root fibres.
ant. horn = anterior horn.
g. c. = Goll's column.
post. r. = posterior root.
c. c. = central canal.
a. g. s. = anterior ground substance.
a. f. = anterior fissure.
ar. deg. = area of degeneration.



FIG. 1.

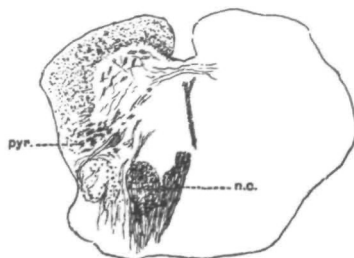


FIG. 2.

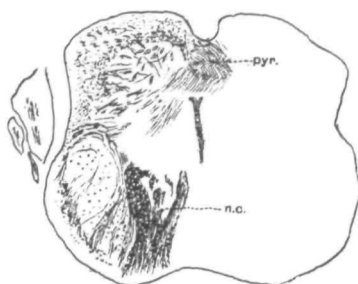


FIG. 3.

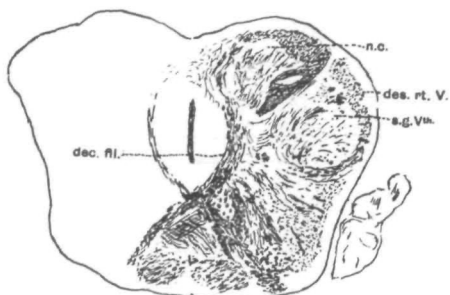


FIG. 4.

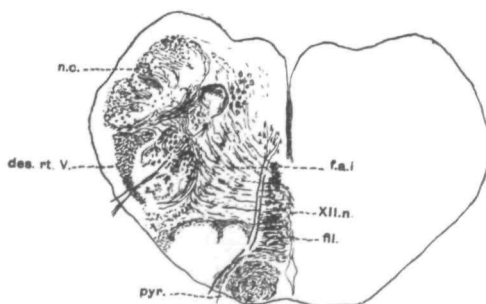


FIG. 5.

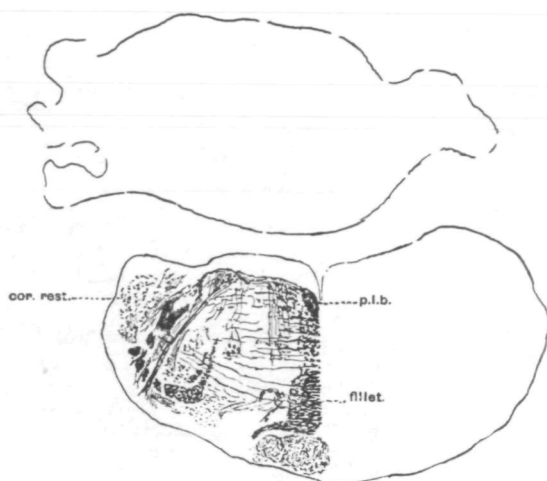


FIG. 6.

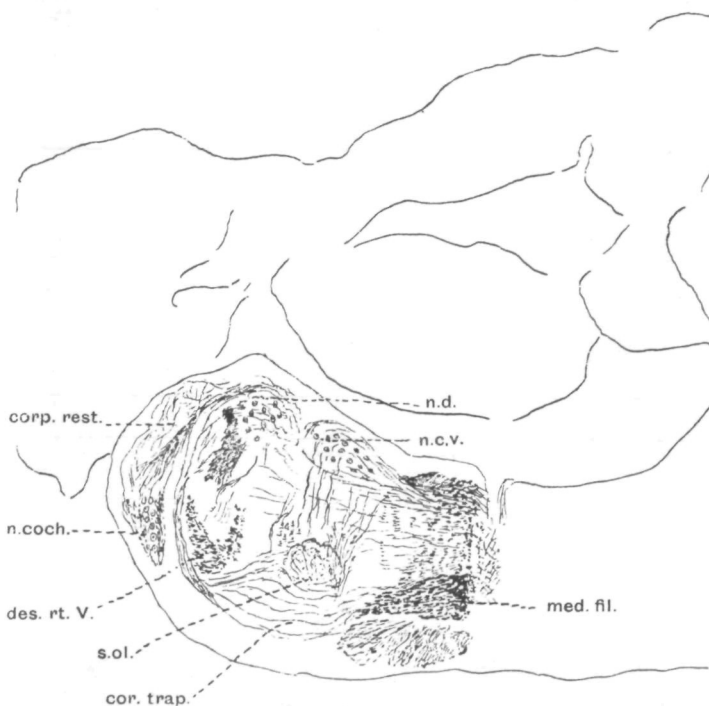


FIG. 7.

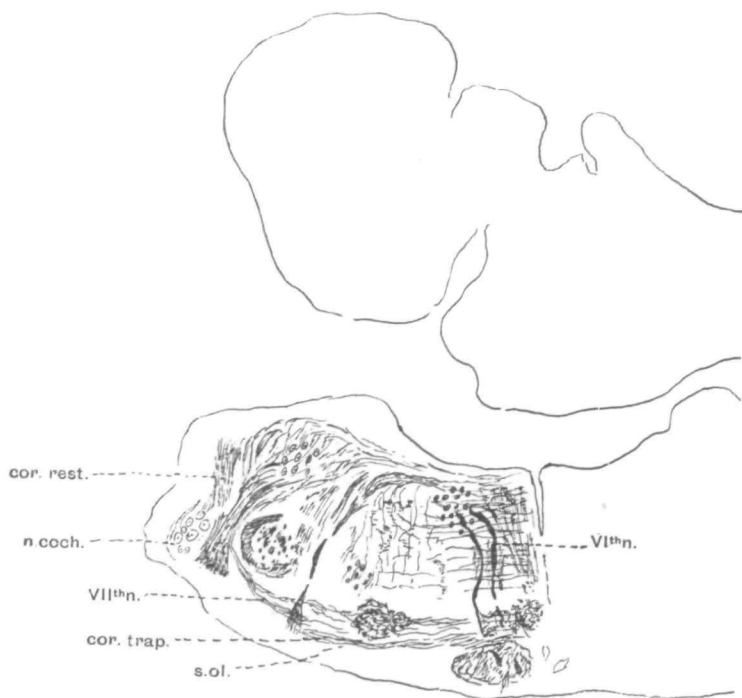


FIG. 8.

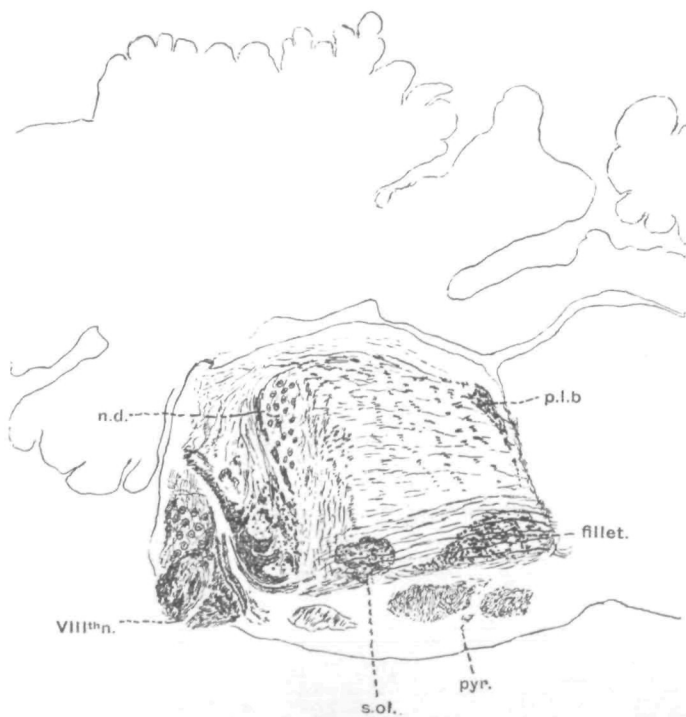


FIG. 9.



FIG. 10.

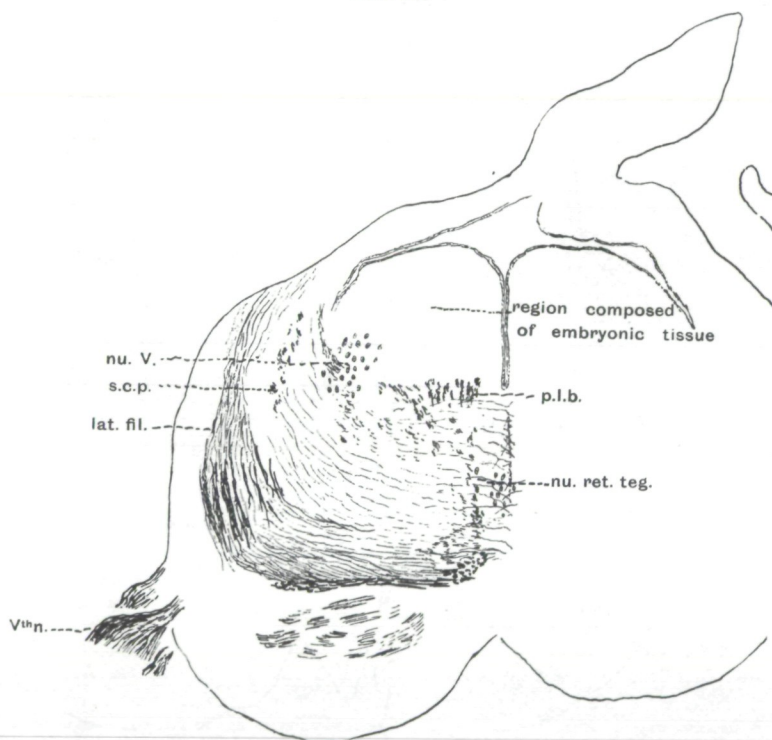


FIG. 11.

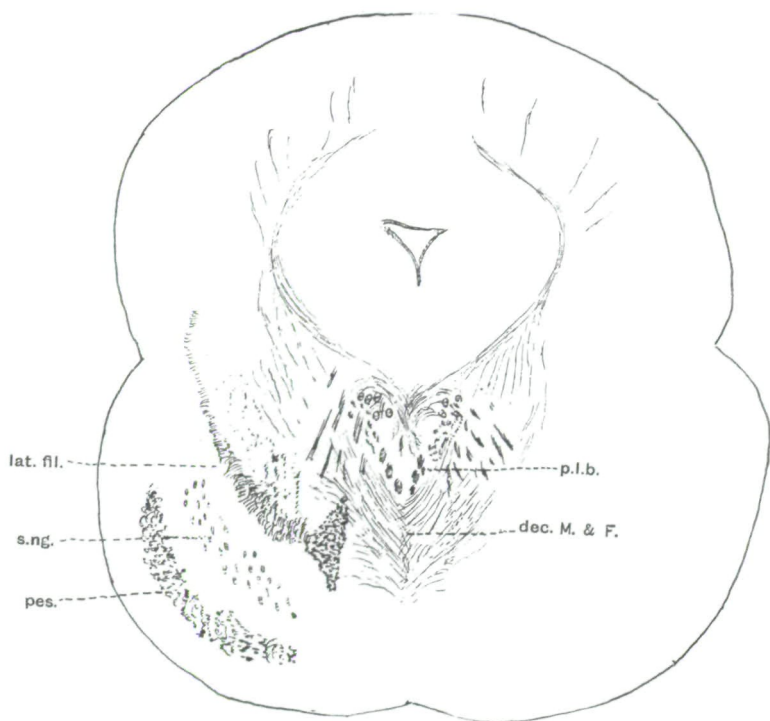


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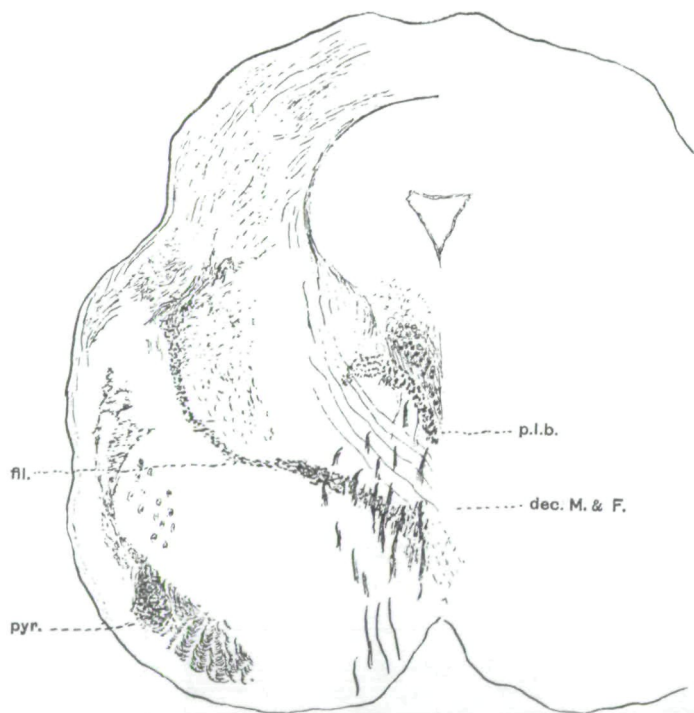


FIG. 13.

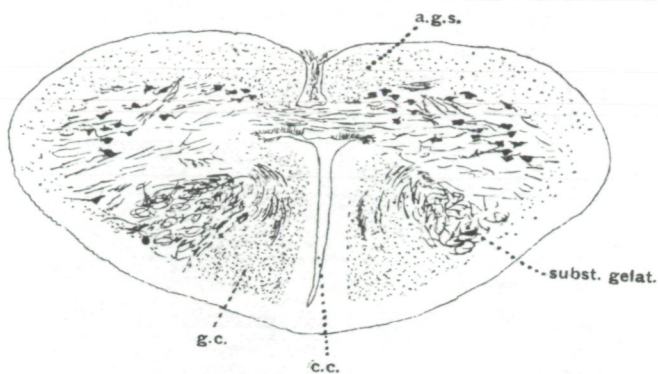


FIG. 14.

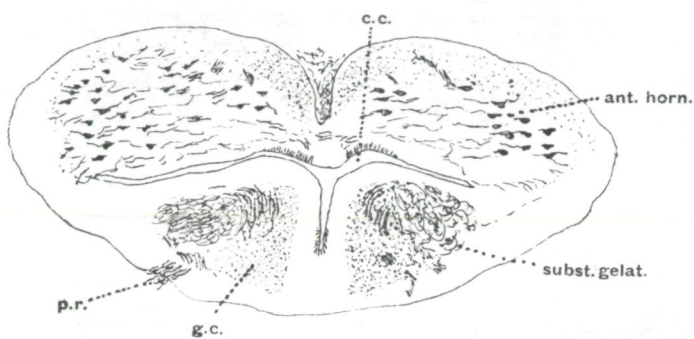


FIG. 15.

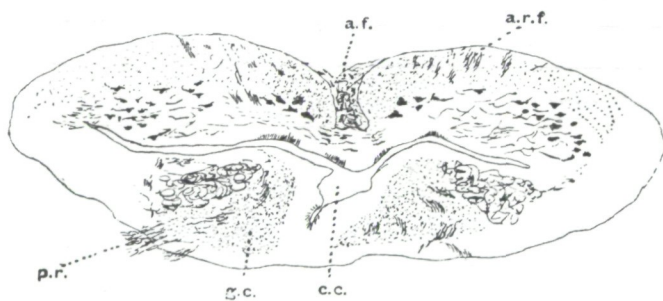


FIG. 16.

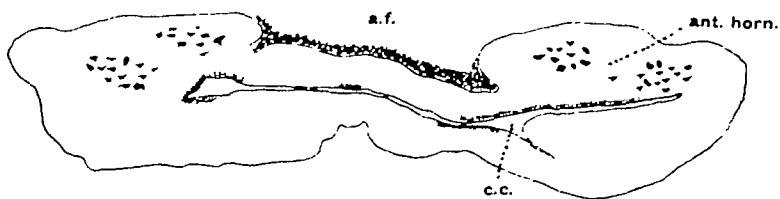


FIG. 17.

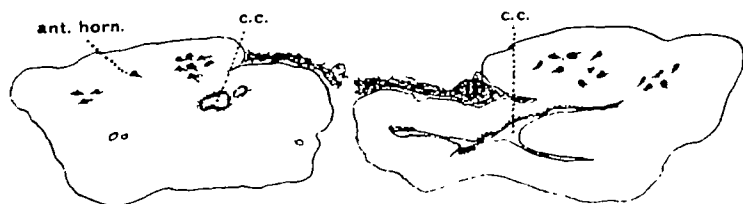


FIG. 18.

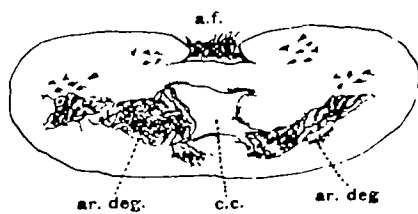


FIG. 19.

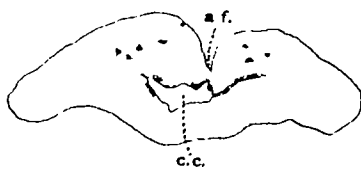


FIG. 20.

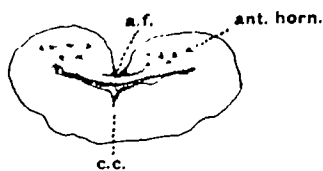
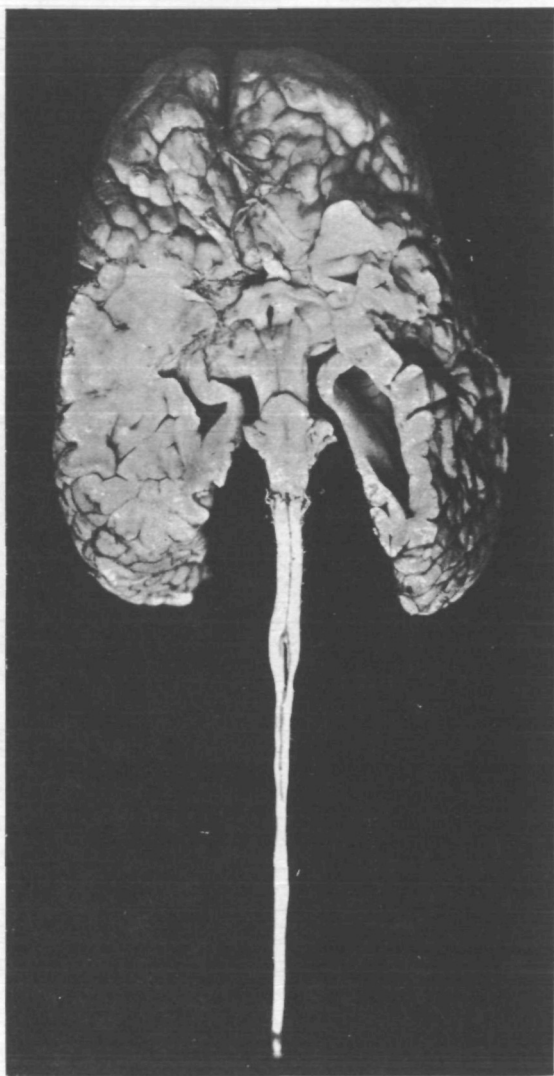


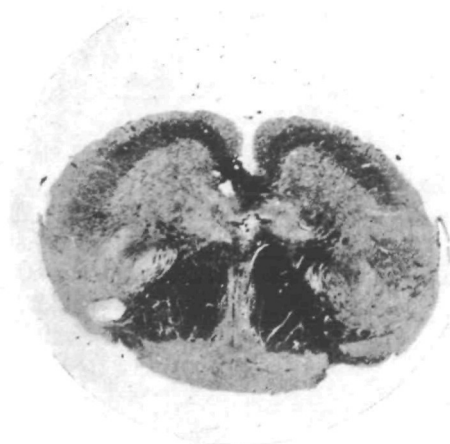
FIG. 21.

PLATE I.



Anterior view of brain and spinal cord.

PLATE II.



Photograph at the level of second cervical segment.