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# DISEASES OF THE SPINAL CORD.

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# DISEASES OF THE SPINAL CORD.

## ANATOMY OF THE SPINAL CORD.

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AXEL KEY U. RETZIUS:—Studien in d. Anatomie des Nervensystems. Virchow-Hirsch, Jahreshb., 1870, I., S. 28. Arch. f. mikrosk. Anat., IX., S. 308, 1873. Stud. in d. Anat. des Nervensyst. und des Bindegewebes. Grossfolio. Stockholm, 1875 und 1876, I. u. II.—EICHHORST: Entwicklung des menschl. Rückenmarks. Virch. Arch., Bd. 64, S. 425. —KRAUSE, W.: Allgemeine und mikroskop. Anatomie. Hannover, 1876.—SCHIEFFERDECKER: Regeneration, Degeneration u. Architectur des R.-M. Virch. Arch., Bd. 67, S. 542.—FLECHSIG, P.: Die Leitungsbahnen im Gehirn u. R.-M. des Menschen. Leipzig, 1876.—Ueber Systemerkrankungen des R.-M. Arch. d. Heilkunde, Bd. XXIII. u. XIX., 1877.—LÖWE, S.: Ueber d. Bindesubstanz im Centralnervensystem der Säugethiere. Arch. f. Psych. u. Nerv., XII., S. 1, 1876.—MAYSER: Experim. Beitrag z. Kenntniss des Baues u. s. w. *Ibid.*, VII., S. 599, 1877.—HUGENIN, G.: Anatomie des centres nerveux. Paris, 1879.—UNGER, L.: Ueber den Bau der grauen Substanz des centralen Nervensystems. Allg. Wien. med. Ztg., 1879, S. 491.

*Axel Key* and *Retzius* describe the arachnoid as a membrane which lies pretty close to and in parts, especially in the cervical portion of the cord, is adherent to the dura mater. Anteriorly, as far round as the ligament. dentic., the space between it and the cord, the subarachnoidal space, is crossed only by a few fibres. Posteriorly, this space is divided up by a longitudinal septum, incomplete in many places, and by numerous parallel and oblique fibres and membranous septa. These fibres and septa are made up of fibrous connective tissues, and are covered with endothelium. The arachnoid itself is composed of layers of fibrillary network, and is covered with a layer of endothelium. The pia is covered with a subarachnoidal fibre-network, in which the larger vessels are located.

Attention is called to the asymmetry which so often exists between the different sides of healthy cords. According to *Boll*, *Key* and *Retzius*, the pia sends in funnel-shaped prolongations with the vessels, which are continued over the capillaries, and form a system of intercommunicating lymph-spaces, which open into the subarachnoidal space. The subarachnoidal, as well as the subdural space, is in free communication with the sheaths of the nerve-roots and the lymph-vessels of the peripheral nerves. The subarachnoidal space of the cord communicates freely with that of the brain.

*Unger* concludes that the fine, so-called nerve-fibre network discovered by *Gerlach* in the gray matter of the cord is really connective tissue; because, in the chick, it appears before any ganglion cells are visible.

Carrière has proved the existence of the *anastomoses between the multipolar ganglion cells.* (*Arch. f. mikrosk. Anat.*, XV., S. 125, 1877.)

Mayser has demonstrated that some fibres of the anterior roots pass through the anterior horns to the gray substance of the opposite side.

The most important recent contribution to the anatomy of the cord is that of *Flechsig*. By the study of the development of the central nervous system in numerous fetuses and in children, he discovered that certain tracts in the white substance of the cord develop at different periods. From this circumstance he was able to divide the white substance into certain tracts or *systems*, which in the fully developed cord are, for the most part, no longer anatomically separable. These, in the order of their development, are first, the anterior columns exclusive of their inner fourth (*principal mass of the anterior columns*), and the posterior columns exclusive of the columns of Goll (*wedge-tracts*); second, the anterior halves of the lateral columns (*anterior mixed region of the lateral columns*); third, the layer of fibres lying next to the lateral periphery of the gray matter (*the lateral boundary layer of the gray substance*); fourth, *the columns of Goll*; fifth, a thin layer of fibres at the periphery of the lateral columns, reaching from or near the posterior nerve-roots to about the middle of the lateral columns (*the direct cerebellar lateral column tracts*), and sixth, the inner fourth of the anterior columns (*pyramid tracts of the anterior columns*), and a mass of fibres, including the posterior half of the lateral columns, except the third and fifth (*the pyramid tracts of the lateral columns*).

The pyramid and cerebellar tracts, and probably the columns of Goll, unite the nerve-centres above the cord with the different centres, etc., scattered at different heights along the cord. The

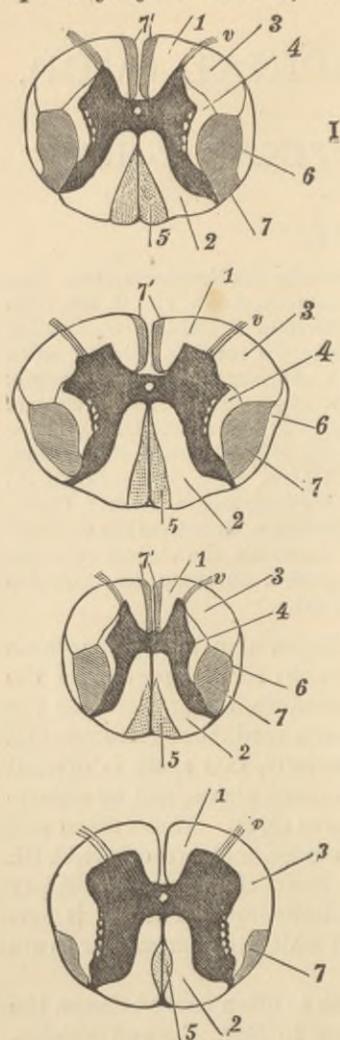


DIAGRAM OF THE DEVELOPMENTAL SYSTEMS OF THE SPINAL CORD AFTER FLECHSIG.

I, section at height of the 3d, II at height of 5th cervical nerves; III, at height of the 6th dorsal, and IV of the 4th lumbar pairs. 1, principal mass of the anterior columns; 2, wedge-tracts; 3, anterior mixed region of the lateral columns; 4, lateral boundary layer of the gray substance; 5, columns of Goll; 6, direct cerebellar lateral column-tracts; 7, pyramid tracts of the lateral columns; 7', pyramid tracts of the anterior columns; v, anterior roots.

principal mass of the anterior columns, the wedge-tracts, and the anterior mixed tracts of the lateral columns, for the most part unite the gray matter with peripheral organs, or unite different parts of the gray matter which lie at different heights on the cord.

The *anterior pyramid* tracts appear usually at the height of the lower dorsal vertebræ (variable), increase gradually in size, and finally pass upward into the pons without crossing over.

The *lateral pyramid tracts* appear in the lower half of the lumbar enlargement, and increase in size on going upwards. In the lumbar portion they are peripheral, but soon the cerebellar tracts appear, and separate them from the periphery, and they approach nearer to the gray substance. In the cervical portion they touch the periphery for a short distance again. In the pons each crosses over to the opposite side in the anterior pyramid.

The pyramid tracts are probably made up of fibres from the gray columns, which go either directly into the lateral columns of the same side, or through the anterior commissure, into the anterior pyramid columns of the other side, and are to be regarded as *indirect* continuations of the anterior roots—indirect, because interrupted by the ganglion cells.

The relative size of the pyramid columns varies. Usually the greater number of pyramid fibres are to be found in the lateral columns. Sometimes almost *all* the pyramid fibres are found in the lateral columns; sometimes almost all pass into the anterior columns. In the same individual most of the fibres of one side of the pyramid may cross over to the lateral column of the opposite side, while most of the fibres of the other side may not cross at all, *i. e.*, may pass into the anterior column. It is obvious how this irregular crossing may give rise to the asymmetry of the cord mentioned above. The exceptional non-decussation of the fibres is important in explaining the exceptional symptomatology of some lesions of the cord and brain (hemiplegia on the same side as the brain lesion).

The *direct cerebellar lateral tracts* appear in the upper part of the lumbar enlargement, partly as a compact bundle of fibres at the periphery of the posterior half of the lateral columns, partly as a number of isolated fibres scattered over its section. They increase in size in passing upward. They receive large bundles of fibres from the region of Clark's columns, and seem to be connected with these tracts and with the cells of Clark's columns (*Pick, Centralblt.*, 1878, No. 2). These tracts pass through the restiform bodies into the cerebellum.

The portion of the lateral columns not included in the above varies in size in different parts of the cord, corresponding to the size of the nerve-roots which enter at any given point. The connections and functions of the *lateral boundary layer of the gray matter* are little understood. The fibres of the *anterior mixed region of the lateral columns* come in part from the lateral parts of the anterior gray columns, in part they are to be regarded as the direct prolongations of the anterior roots. Their fibres pass in part back into the gray matter, in part into the medulla. Their function is wholly unknown.

The principal masses of the anterior columns do not increase in size from below upward. They come in part from the anterior roots, in part from the gray matter of the cord, and pass only in part directly into the medulla. Nothing more is known of them.

The columns of Goll are very small in the lumbar region; they increase steadily from below upward in size. Their fibres come partly from the posterior gray columns, partly from the posterior commissure. They terminate apparently in nuclei in the medulla.

The wedge-tracts vary greatly in size in different parts of the cord, increasing markedly at the enlargements. They are made up for the most part of direct continuations of the posterior roots, but there are also numerous longitudinal bundles of fibres in them. They terminate for the most part in nuclei in the medulla, but they enter also into the formatio reticularis, the olivary bodies, etc.

## PHYSIOLOGY OF THE SPINAL CORD.

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BALIGHIAN: Beiträge zur Lehre von der Kreuzung der motorischen Innervationswege im Cerebro-spinalsystem. Eckhardt's Beitrag zur Anat. u. Physiol., VIII., Bd., S. 193.—LUCHSINGER: Zur Kenntniss der Functionen des Rückenmarks. Arch. f. Physiol., Bd. XVI., S. 510.—SCHIFF: Ueber die Leitung der Gefühls-Eindrücke im Rückenmark. Allg. Wien. med. Ztg., 1879, S. 455.—KOCH, W.: Ein Beitrag zur Lehre der Hyperæsthesie. Virchow's Arch., Bd. 73, S. 273, etc.

The more recent investigations of Schiff confirm the observations of other writers, that when only a small portion of the gray matter of the cord remains intact, all the white matter and part of the gray having been severed, sensation to touch and pain remain in the parts posterior to the section. If, however, the intact portion is small, and in the extreme lateral periphery of the gray substance, sensation is retained on only one side of the body, together with a slight power of voluntary motion. The existence of different tracts for the different kinds of sensation has not been demonstrated, although Brown-Séguard, from certain facts in pathology, believes that they do exist. Vulpian denies their existence, believing that the different kinds of sensation only modify the sensitive tracts in different ways. Schiff confirms the observations of Woroschiloff, in that he proves that the white longitudinal fibres of the anterior lateral columns, although having chiefly to do with voluntary motion, "conduct a trace of sensation to the brain." Woroschiloff states that each lateral column contains sensitive fibres from both legs, the greater number crossing over to the opposite side of the cord. As the experiments were conducted only on the lumbar portions of the cords of rabbits,

*Erb* is not inclined to accept the results unconditionally. The complete decussation of sensitive fibres is not yet proved. *Koch* confirms the experiments of *Ludwig* and *Woroschiloff*, who found that section of the inner two-thirds of the middle portion of the lateral columns in the lumbar portion of the cord gave rise to hyperæsthesia of the parts lying below, and on the same side as the section. The fasciæ, periosteum, and joint-surfaces shared in this hyperæsthesia. In the cervical portion of the cord and in the medulla, section of the *outer* fibres of the lateral columns gave rise to this phenomenon. Section of the outer portion of this region gave rise to hyperæsthesia of the joints alone, cutting the inner, of the skin alone; but in neither case was the hyperæsthesia so marked as when the whole region was severed. Strong electrical currents caused a cessation of the hyperæsthesia.

From the recent investigations of *Flechsig*, it seems probable that the pyramid-tracts are the chief conductors of the voluntary motor impulses. *Luchsinger* has proved that under the influence of picrotoxin convulsions would occur in the extremities behind the section of the cord, proving that, for this poison at least, the *convulsion centre* is not exclusively in the medulla.

The following summary of the present theories concerning the *innervation of the blood-vessels* is taken from *Erb's* second edition, no modification of it being necessary from more recent publications. References and authorities must be omitted for want of space.

There are probably ganglion cells on the vessels, or in their neighborhood, which serve as local vaso-motor centres. These local centres are under the influence of the larger nerve-centres, and are connected with them through two varieties of fibres, which lie in the peripheral nerves, viz., the vaso-constrictor and the vaso-dilator fibres. These fibres connect with two varieties of vaso-motor centres, which are scattered throughout the cord, and perhaps the upper nerve-centres, possibly also throughout the cerebral convolutions; but the most important are in the medulla oblongata. The two varieties of centres are the vaso-constrictor and the vaso-dilator centres. Both kinds of centres may be directly irritated, the condition of the vessels depending on which is most so. The constrictor nerves seem to need a stronger irritation than the dilator, in order to react. After section of the nerves, their irritability sinks more rapidly than that of the vaso-dilator fibres. Reflex influences may cause dilatation or contraction of vessels. Vessel-reflexes from the spinal centres certainly occur, but they are more limited than those radiating from the medulla. Similar reflexes may be excited from the local centres, manifested by dilatation of the vessels when the irritation is slight, by contraction when it is powerful.

The location of these centres in the cord is unknown. The vaso-motor nerves, as they come from the spinal centres, lie, for the most part, in the lateral columns. How they make their exit from the cord is, for the most part, unknown. The vaso-motor nerves for the head come from the cervical portion of the cord, those for the upper extremi-

ties from the thoracic portion, those for the pelvis and legs from the lower dorsal and lumbar portions. The vaso-motor nerves of the sciatic nerve, however, do not pass into it through the sacral roots, but pass through the sympathetic. A similar condition of affairs seems to exist in the brachial plexus.

*Tschirjew* confirms by elaborate experiments the reflex nature of the *knee phenomenon* (*Berl. klin. Wochenschr.*, 1878, No. 17). *Gowers* proved that *ankle-clonus* resulted from direct stimulation of the muscle, the time between the tap on the muscle and the reaction being only .4 to .3 of a second, too short a time for a reflex.

*Nothnagel* (*Arch. f. Psych.*, VI., S. 832, 1876) and *Lewinski* (*Ibid.*, VII., S. 327, 1877) have proved that strong irritation of distal parts (skin nerves) may exert an *inhibitory action* on tendon reflexes. *Erb* has proved the same fact in a case of spastic paralysis. In *Stümpell's* experience, flexion of the great toe has no inhibitory influence on ankle-clonus, as stated by *Brown-Séguard*. Flexion of the whole foot, by relaxing the tendo Achillis, causes its cessation. *Langendorff* has proved that the brain has a crossed action in inhibiting reflex action, the right side of the body being under the influence of the left side of the brain, etc.

*Goltz*, *Luchsinger*, *Ostroumoff* and *Nawrocki* have proved the existence of nerves which pass through the peripheral nerve-stems, and which, on irritation, cause a secretion of sweat. The centres for these nerves lie chiefly (*Nawrocki*) in the medulla, but exist also (*Luchsinger*) throughout the whole length of the cord. The "sweat nerves" reach the nerve-trunks through the sympathetic system.

*Remak* (see *Poliomyelitis Anterior Subacuta*), by the analysis of certain facts in pathology, has sought to establish the supposition that there exist groups of ganglion cells in the cord, which control groups of muscles which act functionally together, but which receive their innervation from different nerve-trunks.

*Couty* (*Gaz. méd. de Paris*, 1876, No. 22) found that section of the posterior spinal roots in frogs had no noticeable effect on the *nutrition* of corresponding parts. He makes the spinal ganglia responsible for trophic changes.

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## GENERAL THERAPEUTICS OF DISEASES OF THE SPINAL CORD.

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In a case of traumatic paraplegia, in which there occurred strong tonic convulsions, *Nussbaum* stretched the crural and sciatic nerves with the effect of stopping the convulsions, but not influencing the paralysis. The seat of the lesion, whether in cord or in cauda equina, was questionable. *Vogt* comes to the conclusion that nerve-stretching has no effect on central lesions. It is indicated only when there is functional disturbance of a peripheral nerve, either from increased irritability or from disordered circulation. In traumatic tetanus the results are favorable. *Edlefsen* recommends for *catarrh of the bladder*, oil of turpentine (10–12 drops 4 or 5 times a day) and balsam of copaiba. He claims good results from the use of chloride of potassium (solution 1–20 of water, a tablespoonful to be taken every two or three hours).

## HYPERÆMIA OF THE SPINAL CORD AND ITS MEMBRANES.

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FABRE: Des phénomènes spinaux dans les affections cardiaques. *Gaz. des hôp.*, 1876, No. 147.

In the last stages of diseases of the heart, *Fabre* has observed pain, anæsthesia, paræsthesia, and slight paresis, rarely also convulsive phenomena, which are probably due to passive hyperæmia of the cord.

## APOPLEXY OF THE SPINAL MENINGES.

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WEBER: Spinal Meningeal Hemorrhage. *Boston Med. and Surg. J.*, July 8th., 1875.—DOWSE: Subarachnoid Hemorrhage of spinal Cord. *Trans. Path. Soc.*, XXVII., p. 1, 1876.—LUTKENMÜLLER, J.: Ein Fall von Hæmatorrhachis. *Wien. med. Bl.*, 1878, I., 964, 1879, II., 5.—DIXON, E. L.: Intermenigeal Spinal Hæmorrhage Simulating Strychnia Poisoning. *Lancet*, London, 1879, I., 333.—LANCEREAUX: Hématome de l'arachnoïde comprimant la moitié gauche de la moëlle épinière, etc. *Rev. méd. franç. et étrang.*, Paris, 1879, I., 22–25.

Of these records of cases, some of which the writer has not had access to, that of *Dixon* deserves mention. The patient was seized with violent tetanoid convulsions recurring at short intervals, and brought on by any movement. Consciousness was not affected. Death occurred in two hours. On autopsy, the spinal arachnoid cavity was found filled with

blood, all other organs being healthy. The resemblance of this case to one of *strychnia poisoning* is its chief point of interest. No chemical examination of the stomach and contents was made. There was no reason to suspect poisoning.

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## PACHYMENINGITIS AND PERIPACHYMENINGITIS SPINALIS.

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 BERGER, M.: Zur Kenntniss der Pachymeningitis spinalis hypertrophica. Deutsche medic. Wchnschr., Nos. 50, 51, 53, 1878. GIBNEY, V. P.: Cervical Pachymeningitis. Med. Rec., N. Y., 1879, XX., 20-22.—GLYNN: Internal Hypertrophic Pachymeningitis of the Cord. Brit. Med. J., 1878, II., 805.—SPENCER, W. H.: Case of Idiopathic Inflammation of the Spinal Dura Mater. Lancet, 1879, I., 836.—KOHLS: Gerhardts Handbuch f. Kinderheilk., Bd. V.

With reference to the etiology and pathology of these diseases, the following cases are of interest:

The possibility of the occurrence of peripachymeningitis from trauma is indicated by cases reported by *Lewitzky* and *Leyden*. The case reported by the former, *Erb* regards as inconclusive. That of *Leyden* resulted from a railroad accident. The autopsy disclosed caries of the vertebra as its probable point of departure.

*Spencer* reports a case of suppurative peripachymeningitis apparently primary, and resulting from exposure to cold. Pain in the back and lower extremities without paresis was the main symptom. *Kohl's* case was one of *tubercular tumors* on the outer surface of the dura mater in a child. The process seemed to originate in necrosis of a rib with (secondary?) involvement of the lung. The pressure of the tumor caused degenerative changes in the cord.

*Ollivier's* case was one of deposit of urates on the anterior surface of the dura mater, and on the sheaths of the nerves in a case of severe chronic *gout*. The symptoms during life, which were probably dependent on this lesion, were a sense of constriction about the neck, thorax, and abdomen, with fulgurant pains in the limbs.

*Glynn* reports a case of typical cervical pachymeningitis resulting in death in twenty months. Transverse myelitis existed at site of the lesion of the dura mater.

From a clinical point of view, the cases of *Berger* and *Gibney* are of

interest. Their cases recovered completely or almost completely after a tedious course of over a year. Cold was the apparent cause in *Berger's* case. Counter-irritation, galvanism, iodide of potassium, and frictions with alcoholic liniments were used. One of *Gibney's* cases, apparently traumatic in its origin (*Med. Rec.*, N. Y., 1880, Sept. 25th), recovered without systematic treatment. Ergot and the iodide of potassium seem to have had some influence over the disease in his experience.

*Joffroy* calls attention to this relatively favorable course of cervical pachymeningitis as contrasted with that of transverse myelitis.

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## LEPTOMENINGITIS SPINALIS.

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SCHWARZ: Méningite spin. Arch. méd. belge, Dec., 1874.—LASÈGUE: Méningite spinale, supposée de nature rhumatism. Arch. génér. de méd., Juin, 1874, p. 743.—SCHULTZE, F.: Das Verhalten des R.-M. und der Rückenmarksnervenzwurzeln bei acuter Basilar meningitis. Berl. klin. Woch., 1876, Nr. 1.—Beitr. z. Pathol. u. pathol. Anat. des centr. Nervensyst. Virch. Arch., Bd. 68, 1876.—VULPIAN: Leçons sur les malad. du système nerveux, 1877, p. 111.—Meningomyélite subaiguë. Chir. méd. de l'hôp. de la charité, Paris, 1879, 626-632.—DUNLAP: Meningitis Caused by Penetration of a Fishbone into the Spinal Canal. Brit. M. J., London, 1879, I., 289.

The predominance of the exudation in acute meningitis upon the posterior surface of the cord seems to be best accounted for by the numerous septa which exist in the subarachnoidal space and the consequent greater vascularity of the posterior meninges. This is more satisfactory than the previous supposition that it resulted solely from the position of the patient or (*Vulpian*) from the richer nerve-supply of the posterior meninges.

The case reported by *Dunlap* is of interest because of its unusual etiology. It was one of inflammation of the upper part of the spinal meninges and of those covering the under surface of the cerebrum and medulla, caused by a fishbone which penetrated from the pharynx partly through the second right inter-vertebral foramen and partly through the first inter-vertebral cartilage into the spinal dura mater.

*Vulpian* states that he has seen rapid improvement in a case of acute spinal meningitis, caused by cold, follow the administration of large doses of salicylate of soda.

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## TUMORS OF THE SPINAL MENINGES.

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DOWSE: Fibro-nucleated Tumor from the Dura Mater of the Cervical Portion of the Spinal Cord. Brit. Med. J., 1875, May 29th.—BIOT: Note sur un cas de sarcome intrarachid. Lyon méd., 1875, Nr. 31.—PEL: Myxom der Spinalmeningen. Berl. klin. Wehnschr., 1876, Nr. 32.—GOWERS: Myo-lipoma of the Spinal Cord. Transact. of the Pathol. Soc., XXXII., p. 19, 1876.—HÜNICKEN: Sarcomatöse Geschwulst im Wirbelcanal. Berl. klin. Wehnschr., 1878, 382.—BULTEAU: Tumeur sarcomateuse de la moëlle. Progrès méd., p. 181, 1878.—WOOD, H. S.: A Case of Spinal Hydatids. Austral. M. J., 1869, I., 222.

Of these newly reported cases little is to be said. *Gowers'* case was one of *lipoma* containing a large number of transversely striated muscular fibres situated at the conus medullaris; *Wood's*, one of *hydatid cysts* of the lower end of the dura mater, causing it to project out through some of the sacral foramina, hydatids being found also in the liver. In the case reported by *Bulteau*, a spindle-celled *sarcoma*, about the size of the first phalanx of the thumb, was found growing from the pia mater at the junction of the cord and the medulla. Nothing in the clinical histories of these cases calls for special mention.

## ANÆMIA OF THE SPINAL CORD.

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LAUENSTEIN, C.: Zwei Fälle von Embolie der Aorta. Deutsch. Arch. f. klin. Med., XVII., S. 242 u. 491, 1876.—MALBRANC, M.: Beob. über Aortenthrombose u. Aphasie.—*Ibid.*, XVIII., S. 462, 1876.—VULPIAN; Leçons sur les mal., etc., p. 98, 1877.—FRIEDERICH, A.: Recidivirende vorübergehende Rückenmarkslähmung. Virchow and Hirsch Jahresbericht, II., 1879, S. 113.

Under this subject the case related by *Friederich* alone deserves special mention.

The patient was a student, twenty years old, who, since he was eleven or twelve years old, had suffered from repeated attacks of paralysis, the first one following an exposure to cold. In the attack in which *Friederich* saw him, all four extremities became almost completely paralyzed in the course of a few hours without loss of sensation or of reflex action. In two days recovery was complete without treatment. The patient had an hypertrophied heart and a systolic murmur. *Friederich* suggests that a temporary anæmia of the cord was the cause of the attacks.

## SPINAL APOPLEXY.

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WEBBER: Fall von Spinalhämorrhagie. Schmidt's Jahrb., Bd. 170, S. 25, 1876.—FOX, E. L.: On Spinal Hemorrhages. Med. Times and Gaz., 1876, Aug. 23d.—REMAK, E.: Fall von atroph. Spinallähmung durch traumat. halbseit. Blutung in die Halsanschw. des R.-M. Berl. klin. Woch., 1877, Nr. 44.—VULPIAN: Leçons, p. 92, 1877.

Beyond the bibliography, the case reported by *Remak* alone will be alluded to. This was a case of atrophic paralysis on one side of the body, resulting from an injury which *Remak* supposed to be a hemorrhage confined to one-half of the cord in the cervical region.

## ACUTE TRAUMATIC LESIONS OF THE SPINAL CORD.

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FEINBERG: Wirbelfractur und Rückenmarksabscess. Berl. klin. Woch., 1876, Nr. 32.—HULKE: Three Cases of Broken Neck. Med. Times, 1876, July 29th.—HAYEM: Arthrite de l'articulation de l'axis avec l'atlas, etc. Gaz. des hôp., 1876, No. 147, Soc. d. Biol.—SEELIGMÜLLER: Fall von geheilt. Fractur d. Lendenwirbel. Deut. med. Wehnschr., 1877, Nr. 28.—HEYNOLD, H.: Fall von Luxation und Fractur des 6. u. 7. Halswirbels, verbunden mit ungewönl. nied. Temperaturabfall. Berl. klin. Woch., 1877, Nr. 39.—NIEDEN, A.: Ueber Temperaturveränderungen (Hyperpyrexie und Apyrexie), bedingt durch Verletzungen des Halsrückenmarkes. Berl. klin. Wehnschr., No. 50, 1878.—RIGLER, JOHS: Ueber die Folgen der Verletzungen auf Eisenbahnen, insbesondere der Verletzungen der Wirbelsäulen und des Rückenmarks, 1879.—BECK, B.: Ueber Verletzungen der Wirbelsäulen und des Rückenmarks. Arch. f. path. Anat., etc., Berl., 1879, 207-255.

Injuries to the spinal cord, without lesion of the spinal canal or of the soft parts, occur more readily when there already exists some anomaly of the spinal column, as in the case of *Hayem*, in which there existed hypertrophy of the odontoid process.

*Beck* records a case of softening of the cord in the dorsal region with a clot between the dura mater and the bone, which was traumatic in its origin, but without fracture of the vertebra.

## GRADUAL COMPRESSION OF THE SPINAL CORD.

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The secondary degeneration which, in the cases of *Westphal*, *Frommann*, and *Kadner*, was observed to extend upward for a short distance above the point at which the cord was compressed at the periphery of the cord, was evidently situated in the cerebellar lateral column tracts of *Flechsig*.

In regard to *vasomotor disturbances* in these cases, *Vulpian* speaks of various varieties as occurring from irritation and paralysis of vaso-dilator and vaso-constrictor fibres.

Although, as a rule, *motor disturbances* show themselves first in compression of the cord from Pott's disease, yet, as in *Ramskill's* case, disturbance of sensation may be first in order of time. On the other hand, compression from behind may cause predominant disturbance of motion, according to *Vulpian*, because the gray matter suffers less from pressure than the white.

Although, as a rule, there is increase of reflex action in these cases, *Kadner* has shown that in many this increase may not be present.

## CONCUSSION OF THE SPINAL CORD.

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In a case reported by *Leyden* as concussion of the spinal cord, peripachymeningitis with cheesy tubercular meningitis and secondary myelitis from compression were found to have developed. *Willigk*, in a case of concussion which died three months after the accident, claims to have found the capillaries and the small arteries and veins throughout both cord and brain widely dilated and showing slight fatty degeneration of their walls.

*Buzzard* has observed disturbance of speech and of the functions of the tongue with glycosuria as the result of concussion.

*Bernhardt* and *Leyden* have added to the list of cases in which the morbid symptoms appeared after a considerable time had elapsed since the injury. *Erb* has also seen several such cases.

## SPINAL IRRITATION.

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*Coghill* reports four cases of irritable spine as treated successfully by tonics and by the local application of *Corrigan's bouton*, heated to a blue heat, over the painful spots.

The cases reported by *Benedickt* (Neue Behandlungsmethoden der Spinal-Irritation. *Wien. Med. Presse*, 1879, 105, 173) are cases of pure hysteria, not of spinal irritation in *Erb's* sense.

## NEURASTHENIA SPINALIS.

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*Erb* protests against the classification of this disease as a manifestation of *hypochondria* (*Jolly*). Though often combined with the latter, it is to be regarded as of distinctly spinal origin. The recent literature given above calls for no comment.

## MYELITIS.

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Vallin reports three cases of more or less complete paralysis of the extremities, two being associated with disturbances of sensation, occurring in connection with *acute articular rheumatism*. In two, the symptoms rapidly passed away, a slight hemiplegia remaining in a third two months after the inception of the disease, at which time the patient passed from under observation. Erb considers it doubtful whether arsenic, phosphorus, mercury, bisulphide of carbon, alcohol, or lead can give rise to acute myelitis.

Leyden reports three cases of a peculiar form of myelitis resulting from *sudden diminution of barometric pressure*. One case was that of a laborer who, half an hour after coming out of a caisson, where he had been working under increased atmospheric pressure, became suddenly paraplegic; death occurred in the fifteenth day, the patient having the usual symptoms of acute myelitis with grave vesical symptoms. The cervical and lumbar parts of the cord were healthy. In the dorsal portion were seen, scattered through the cord, particularly in the posterior columns and in the posterior parts of the lateral columns, accumulations of large, round, nucleated cells which pushed apart the nerve-fibres, and contained among them only a few normal vessels—no neuroglia, no blood pigment. The nerve-fibres in their neighborhood showed in greater or less extent signs of parenchymatous myelitis. The gray

matter was normal. *Leyden* believes that the process consisted in a splitting of the masses of nerve-fibres by oxygen or carbonic acid set free from the capillaries without rupture of their coats or with rupture of only such small vessels that no noticeable hemorrhage took place, and in the subsequent filling up of these spaces with cells. The dorsal portion of the cord was probably affected because of its normally lesser consistency than the enlargements. The other two cases were similar, except that one patient was discharged completely recovered in nineteen days, the other recovered incompletely in thirty days, at which time he was discharged from the hospital.

*Hayem*, by tearing out the sciatic nerve of young rabbits, caused a cicatricial myelitis which became the point of departure of a progressive central myelitis with muscular atrophy. The process consisted in a degenerative atrophy of the ganglion cells. A similar, though slower result followed simple section of the sciatic nerve.

*Leyden's* latest experiments in the artificial excitation of myelitis have enabled him to demonstrate the passing over of acute myelitis into cyst-formation and sclerosis. *Vulpian* has observed similar processes after injecting nitrate of silver into the cord.

*v. d. Velden* described a case in which numerous small foci of acute myelitis were found in the upper thoracic and cervical portion of the cord predominantly in the anterior and lateral columns and in the anterior horns. The process was characterized by changes in the nerve-fibres, slight exudation around the vessels, and slight increase in the number of glyoma cells. *Lauinger, C.*, has published a case in which the process was even more purely parenchymatous. These two cases followed the clinical course of acute ascending paralysis. In that of *v. d. Velden*, the galvanic and faradic irritability was fully lost on the second day of the illness, while in that of *Lauinger* the faradic irritability was fully retained. The reflexes in the latter, which at first were fully lost, returned in a few days; an apparent exemplification of the *inhibitory effect* of the acute affections of the cord suggested by *Goltz*. In the case reported by *Leyden* (see above), faradic irritability of the muscles was good three days after the beginning of the disease.

A case reported by *Schuster* of acute myelitis is of interest on account of its probable syphilitic origin, and the good, although not complete, recovery after five months of treatment by warm baths, electricity and *mercurial inunctions*.

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## CHRONIC MYELITIS.

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Since the discovery by *Flechsig* of the various tracts or systems in the cord, of which an account is given above, several observers have endeavored to discover the relations which exist between these systems and cer-

tain previously classified diseases of the cord (lateral sclerosis, amyotrophic lateral sclerosis, etc.), and have also endeavored to differentiate a new class of diseases which is characterized by a primary affection of a number of these systems to the exclusion of others. This latter class, the *combined-system diseases* of the cord, will be here spoken of. The relation between *Flechsig's* discovery and the previously known diseases will be alluded to under the appropriate headings.

*Kahler* and *Pick* have described a case in which at least four systems were involved, and in which the degeneration confined itself approximately within their limits. Then there are cases in which the myelitic process is not confined strictly within the limits of any distinct systems, but is spread, more or less widely, over the section of the cord, as in cases described by *Westphal*, "in which there can be no question of a limitation of the affection to the systems of *Flechsig*. Such cases are very frequent and seem not unfrequently as if they were the result of a disease which began as a system disease, but which came to be examined at a later stage" (*Erb*).

The most important recent contributions to this subject have been from *Babesiu*, *Leyden*, and *Westphal*.

The case reported by *Babesiu* clinically presented all the symptoms of spastic paralysis, with the addition of atrophy of the optic discs, some loss of sensation in the feet, retention followed by incontinence of urine, and dragging pains in the feet and genitals. On autopsy, in addition to some lesions of the brain, etc., which had no probable connection with the symptoms observed in the limbs during life, degeneration of the lateral columns was found, most marked in the thoracic portion, where the degeneration reached from the posterior almost to the anterior horns. In the cervical expansion, the degeneration of the lateral columns dwindles down to a small triangle at their periphery, being separated from the posterior horns by healthy tissue. Downwards a similar diminution takes place. Only in the periphery of the cord does the degeneration approach the anterior columns. Wedge-shaped, sunken-in, sclerotic spots were found in the lateral cerebral tracts above the anterior pyramid. The posterior columns in their posterior periphery, and the columns of Goll were tolerably evenly affected throughout the cord. The central canal was filled throughout with growing cells. Ganglion cells of the anterior horns and Clark's columns normal. The pia mater of the cord was thickened, especially in its posterior and lateral parts, and was also congested and strongly pigmented. This short abstract of the case gives an idea of the class of cases which are grouped under the heading "combined-system diseases of the cord." It is unnecessary to state how manifold the symptoms may be in these cases, according to the particular system affected, and how still more confused may be the symptom-complex when the disease is of the irregular form.

*Leyden* believes that only two forms of primary "system diseases" of the cord have been demonstrated, *i. e.*, tabes dorsalis and atrophy of the motor parts of the cord (degeneration of the cells of the anterior horns,

motor tracts, spinal nerve-roots, and nerves of the muscles). A combination of these two may exist, giving rise to a "combined-system disease." *Kahler* and *Pick*, *Westphal* and *Babesiu*, he thinks, have systematized their cases more definitely than the clinical facts warrant. He cites several cases of his own, one of which resembled very closely that reported by *Kahler* and *Pick*, and states that he believes that all the cases of this class are cases of simple extension of disease by continuity of tissue with typical ascending and descending degeneration.

*Westphal* (Ueber combinirte (primäre) Erkrankung der Rückenmarksstränge, *Arch. f. Psych.*, Bd. IX., 691-737) analyzes his previously reported cases. He concludes that, although the paralysis in these cases bore no constant relation to the lesions of the lateral columns, yet this was the main element in its causation. The degeneration of the muscles was also an element. Implication of the lateral columns without paralysis, as in *Friedreich-Schultze's* case (*Virch. Arch.*, Bd. 70, S. 141), might have been due to non-destruction of the conducting fibres by the morbid process. Absence of muscular rigidity in his own cases, in which both lateral and posterior columns were involved, he accounts for by the extension of the affection of the posterior columns in the lumbar region to the posterior root-zones, by which extension reflexes were prevented. The supposition that the inflammatory process was transmitted by continuity of tissue or through the pia mater he rejects, because in all his cases healthy tissue intervened between the posterior and the affected portions of the lateral columns, and the pia mater was thickened only over the posterior surface of the cord. In one case, the affected portion of the lateral columns did not extend to the periphery. *Westphal* concludes that in certain cases of myelitis the process may extend in the long axis of the cord, at least in the lateral and anterior columns, and, in general, symmetrically; that there is no manifest single point of departure of the process; and that there are no *pure* system diseases, although here and there are indications that this or that system may be affected as such. Some undiscovered changes in the gray matter of the cord, which was apparently normal in *Westphal's* cases, may underlie these cases. The existence of some change is suggested by the *red* reaction of otherwise normal appearing ganglion cells, in diseased cords, with methyl-violet (*Jürgens*), whereas in normal cords the ganglion cells color *blue* with this reagent. *Westphal* rejects the idea that what in one of his cases looked like typical ascending and descending *secondary* degeneration (and is so regarded by *Leyden*), was so in fact, because the portion of the cord which would have to be assumed as that primarily affected was not involved in its whole diameter, and in cases of disseminated sclerosis the isolated foci of disease have never been observed to be the points of departure of secondary degeneration. He therefore concludes that the affection of the columns of Goll, the cerebellar-lateral tracts, and the lateral pyramid tracts observed in this case were *primary* degenerations of these systems.

*Atkins* found irregularly disseminated myelitis in three cases of *in-*

sanity of different types. In one case of general paresis extensive softening of the lumbar and sacral regions of the cord was found.

*Erb* quotes the following principles from *Renz*, of Wildbad, which should control the employment of *thermal baths* in myelitis: During the active stages of disease, harm is apt to be done unless the greatest care is exercised in their use. This rule holds good, whether the disease is acute or chronic. The baths must be cooler, less frequent, and of shorter duration the more obviously progressive a chronic disease is. Acute cases should not use them till they are convalescent. When a case is improving, the time and temperature of the baths may be increased cautiously. Only those cases in which the meninges are prominently affected endure baths of high temperature and long duration with benefit.

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## MULTIPLE CEREBRO-SPINAL SCLEROSIS.

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This abundant literature furnishes little new information.

The number of cases (all without autopsies) reported in children from five months to eight years of age is somewhat remarkable (*Dickenson*, *Pollak*).

Two of *Dreschfeld's* cases were brothers.

*Westphal's* case was one which presented the irregular complex of symptoms characteristic of multiple spinal sclerosis. The autopsy disclosed columnar degeneration of the posterior columns, with multiple sclerosis of the lateral columns.

*Leyden* reports a case in which all the symptoms of the disease appeared, but in which almost complete recovery (slight difficulty in

speaking, tremor and weakness of legs remaining) occurred after treatment by galvanism and a bath-cure at Rehme. This case he regards as one of acute, multiple cerebro-spinal inflammation which resolved and did not go on to sclerosis. He thinks that the case tends to prove that sclerosis is a late stage of an acute process, not a distinct process in itself.

## LOCOMOTOR ATAXIA.

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With reference to the *etiology* of tabes dorsalis it is to be noted that *Vulpian* affirms that *hysteria*, especially in its convulsive form, has a decided influence on the development of the disease. Among forty-four of the cases analyzed by *Erb* in a recent article, twenty-seven were found to have had *syphilis*, which usually had developed several years before the tabes, and which was usually light in its antecedent symptoms. *Fournier* found twenty-four out of thirty, and *Vulpian* fifteen out of twenty cases

of tabes which were syphilitic. *Erb* is therefore inclined to believe that syphilis has a more direct connection with tabes than he has formerly held.

*Kahler* and *Pick* advance the supposition that ataxia and other forms of spinal disease, occurring after acute infectious diseases, are the result of accumulations of the fungi which cause the acute diseases, in the central nervous system, and which give rise to nutritive disturbances of greater or less gravity and extent.

As examples of the previously known fact that the morbid process in tabes is in most, perhaps in all cases not confined to the posterior columns, cases reported by *Hayem*, *Prévost*, *Kahler* and *Pick*, and *Westphal* may be cited. In *Hayem's* case, sclerosis of part of the lateral columns, numerous changes in the gray substance, sclerosis of the ascending root of the trigeminus and of the so-called "respiratory bundle" existed in addition to a lesion of the posterior columns. The cases of *Westphal* and *Kahler* and *Pick* have been referred to under *myelitis* as cases of combined system-diseases of the cord. Also a case communicated by *Friedreich* (*Virch. Arch.*, Bd. 70) is to be mentioned, in which an extensive annular degeneration existed, resembling the lesion found in one of *Kahler* and *Pick's* cases. In all these cases, the *direct cerebellar lateral tracts* were involved in addition to the posterior columns. The supposition is advanced that the ataxia results from the implication of those columns and not from the disease of the posterior columns. This supposition is far from being proved.

In his recently published analysis of fifty-six clinical cases of locomotor ataxia occurring in his own practice, *Erb* gives a tabulated statement of the relative frequency and importance of the symptoms of the disease which cannot be transcribed here in full, on account of my limited space. In this paper he lays greater stress than formerly on the occurrence of *spinal myosis*, or, as he proposes to call it, *reflex immobility of the pupil*, to distinguish the condition from immobility of the pupil during accommodative effort and from complete immobility, and to include those cases in which the pupil fails to respond to light, but in which it is of normal size or is dilated. *Erb* found the symptom in fifty-four per cent of patients examined. *Vincent* found it in ninety-two per cent. It is probably not an early symptom. It is only found in progressive paralysis of the insane with equal frequency (nineteen out of twenty-one cases *Vincent*). *Atrophy of the optic nerve* occurred in only six out of forty-nine cases. *Tendon reflex* was absent in all but one of fifty cases. This is *probably* always an early symptom. Mechanical irritability of the quadriceps was always fully preserved (tested in thirty-two cases). *Erb* has found tendon reflex absent in only two other morbid conditions, *i. e.*, progressive cerebral paralysis and in cases of paresis of the quadriceps with atrophy and degeneration reaction. In *Erb's* experience, it is never absent in young or middle-aged people in health. Hence he thinks that its absence may be regarded as almost pathognomonic of tabes in its earlier diagnosis. *Cutaneous reflex* was absent in six out of forty-seven

cases. *Analgesia* to strong irritant (*Berger*) was present in thirteen out of twenty-nine cases. This symptom, which is not rare in other diseases, is often early present, but it must be remembered how widely sensibility varies in normal subjects.

*Oulmont*, by careful examination of the *disturbance of sensibility* in ataxic subjects, discovered that it was not only very frequent, but very widespread. He discovered certain spots of predilection (in the breasts, around the umbilicus, fingers and forearms, backs of the legs, the heels and toes, etc.).

Experiments to determine the farado-cutaneous sensibility in tabes conducted by *Drosdoff* (seven cases), and *Erb* (four cases), showed diminution all over the body. A broad, soft wire brush was used. It appears to be an early symptom and may prove of diagnostic value (*Erb*).

*Remak, E.*, in two cases discovered that sensibility could be exhausted by an abnormally small amount of irritation.

*Pierret* has found that all possible nervous *disturbances of hearing* may precede the ataxic symptoms. *Althause* has recorded a case of the same sort.

Among *unusual cases* may be mentioned the following: A case of "hereditary" tabes in which attacks, several hours long, of spasmodic coughing, great feeling of anxiety, rapid respiration, dyspnoea, and cyanosis with frequent and rapid pulse (bronchial crises) is recorded by *Kahler* and *Pick*. The same observers record a case of ataxia following *malarial fever*, with recovery after two months under quinine. Slight nystagmus and slight stiffness of the legs remained. The nystagmus, difficulty of speech, and absence of pains and of sensory disturbances seem to ally this case with that reported by *Leyden* as acute cerebro-spinal sclerosis with cure, rather than with locomotor ataxia. *Erb* records a case of not very typical but distinct ataxia following diphtheria (?) and exposure to cold which recovered rapidly, under galvanic treatment, in six months.

Ataxia associated with lesion of the left olivary body and a unilateral case with a tuberculous nodule in the middle of the left parietal lobe are recorded by *Kahler* and *Pick*. The same observers record a case of ataxia of apparently *cerebellar origin* in which the *tendon reflex was very powerful*. They record also a case of tumor of the third dorsal vertebra in which ataxia preceded all the symptoms of the transverse myelitis which subsequently developed.

*Robert* records a case of sclerosis of the posterior columns and of the anterior cornua of the brachial enlargement which, in life, was a case of typical tabes associated with marked atrophy of the forearms and hands. *Erb* and *Hardy* record two similar cases of this somewhat rare combination (both clinical).

*Kellogg* reports two cases very imperfectly, which seem to fall under *Friedreich's* form of tabes, and which developed in brothers at their sixth year. In other branches of the same family were other similar cases. *Kahler* and *Pick* have reported a case in which four systems

were involved, viz., the pyramid tracts, the cerebellar lateral tracts, with Clark's columns, the wedge-tracts, and the columns of Goll which, as the affection was associated with defective development of the cord, they have tried to rank as a case of hereditary tabes. As the ataxia was very slight, the paralytic symptoms prominent, and there were no disturbances of the bladder or of sensibility, the justice of this classification seems questionable (*Erb*).

The rules given by *Renz* (see myelitis) in regard to the use of *thermal baths* in myelitis hold good for tabes also. He begins with a bath of 31.0°–32.5° C., of 5–10 minutes' duration. Every two or three baths he decreases the temperature  $\frac{1}{2}$ ° until 29° or 28° is reached. When lancinating pains are present, baths from 33° to 34° of 8–15 minutes' duration are best. *Sée* obtained good results, when the pains were prominent, from salicylate of soda.

*Erb* recommends the energetic use of the combined antisyphilitic treatment, when syphilis coexists in a case of tabes, much more strongly than formerly. In his limited experience with it, he has seen encouraging results. In one case of atrophy of the optic discs, of two and a half years' standing, he saw material improvement of vision under galvanic treatment.

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## SPASTIC PARALYSIS.

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*Seeligmüller* calls attention to the possibility of the relationship of parents being an *etiological* factor in spastic paralysis in cases which he has observed among children.

No case has yet been published which proves the existence of a primary disease limited to the pyramid tracts satisfactorily. *Stoffella* has published a typical, uncomplicated case of spastic paralysis, in which, on autopsy, gray degeneration of both lateral columns, principally in their posterior portions and in the lower thoracic and lumbar portions of the cord, was found. In these portions of the cord, the degeneration reached to the meninges externally, and to the posterior horns internally. Unfortunately the absence of any record of the condition of the brain and medulla, and of any microscopical examination of the cord, renders the case incomplete, and therefore inconclusive.

*Erb* supposes that the lesions actually found in the lateral columns in *amyotrophic lateral sclerosis* represent the type which we may expect to find in an uncomplicated case of spastic paralysis. The degeneration in these cases is found predominantly in the lateral pyramid tracts, with a much less marked lesion in the principal mass of the anterior columns, and the anterior mixed region of the lateral columns. The anterior pyramid tracts are also sometimes degenerated. The degeneration of the pyramid tracts is marked, involving atrophy of the nerve-fibres, etc. In the other tracts of the white matter of the cord, which may be involved, an interstitial growth of the neuroglia is found without marked degeneration or atrophy of the nerve-fibres (*Flechsig* and *Pick*). The pyramid tracts are affected throughout the whole length of the cord, and the lesion has been traced through the pons into the crura cerebri.

The anterior gray columns are degenerated, there being nothing typical of this particular disease in the microscopic appearances. The cells of the tractus intermedio-lateralis, those of Clark's columns, and of the posterior gray columns are normal. The lesion of the gray matter is usually most prominent in the cervical enlargement.

The changes characteristic of bulbar paralysis are found in the medulla. The anterior roots of both cord and medulla are gray, degenerated, and atrophic. The muscles, especially of the upper extremities,

present about the same appearance as in typical progressive muscular atrophy.

*Flechsig* and *Pick* have concluded that, in *amyotrophic lateral sclerosis*, we have to do with a disease affecting the *whole system of nerve-fibres and ganglion-cells, which unite the motor centres in the cortex of the brain with the muscles*. Both these observers are inclined to regard the process as a primary disease of the nervous elements, a so-called parenchymatous degeneration or sclerosis. *Flechsig* seems inclined to regard the lesions of the anterior parts of the lateral columns, and of the principal masses of the anterior columns, as an accidental and immaterial complication. *Pick* regards them as being continued from the anterior roots and the primarily affected pyramid tracts.

With reference to the *symptomatology* of spastic paralysis, *Erb*, in the second edition of his work, calls attention to the great prominence of *contracture* in children affected with the disease. The increase of tendon reflex may usually be found in them if care is exercised in bringing the limbs into a proper position for its demonstration. In testing the ankle-clonus too strong or sudden flexion must be avoided.

Numerous cases of more or less typical spastic paralysis are reported clinically in recent literature. Many autopsies of cases, in which spastic symptoms have been prominent, have also been reported and shed some light on the affection. *Hallopeau* (*Des paralysies bulbaires*. Thèse, Paris, 1875, p. 121) and *Schulz* each reports a case of tumor of the medulla, with secondary degeneration of the pyramid tracts, in which spastic symptoms were prominent, though associated with other symptoms.

*Pitres* (*Revue mens.*, 1877, Dec., p. 902) reports a case which *Charcot* diagnosed as one of spastic paralysis, but which afterwards developed other symptoms. On autopsy multiple sclerosis was found. Both anterior pyramids and foci in the lateral columns were sclerotic. *Hydro-myelus of the cervical portion of the cord with symmetrical (secondary?) degeneration of the posterior portions of the lateral columns* in the lumbar portion of the cord, were found in a case of spastic paralysis, in which later disturbances of sensation occurred (*Sänger*).

*Shaw* reports a case giving a typical clinical history of bulbar paralysis with progressive muscular atrophy, *without contracture*, in which, in addition to the ordinary lesion of the medulla and anterior horns, sclerosis "of a very light character throughout the cord," was found in the lateral columns.

Two cases in which the symptoms of spastic paralysis were prominent, though complicated by others, in which *no lesions of the lateral columns existed*, are of special interest. *Schulz* reports a case of *internal hydrocephalus* of twenty-five years' duration, in which some disturbance of sensation was the only symptom complicating an otherwise typical history of spastic paralysis. The cord was normal.

*Mader* reports a case in which the *white substance of the cord was normal*. The prominent spastic symptoms which existed in life seemed to have been caused by a *lesion of the cauda equina*. The anterior gray

columns were sclerosed in part, and a peculiar, apparently inflammatory degeneration of the muscles existed. Inflammatory changes in the bones and knees were also found. Other symptoms besides those characteristic of spastic paralysis existed during life. A case reported by *Bramwell*, in which rigidity and paralysis of the legs, with numbness and greatly increased tendon-reflex, disappeared after the discharge of a lumbar abscess, may be ranked with this case as being possibly of peripheral origin.

The cases of *combined-system diseases of the cord*, above referred to, must be here alluded to, as in them spastic symptoms were associated with lesions of the lateral columns, *except* in *Westphal's* and *Leyden's* cases.

With reference to the *conclusions to be drawn* from clinical and pathological experience in respect to spastic paralysis, *Leyden*, writing in 1879, states that while the symptom-complex, known as *spastic paralysis*, is pretty frequent in spinal diseases of different kinds, it does not form a peculiar disease in itself. It occurs in some forms of meningitis and myelitis, which are susceptible of cure. He does not think that any decided relationship to sclerosis of the lateral columns is manifested in cases of chronic myelitis, in which the peculiar symptom-complex occurs, although in these cases the periphery of the anterior and lateral columns is, to a greater or less extent, affected. The spastic phenomena can be explained by the interruption of the conduction of motor impulses from the brain by the myelitic process, and the consequent increase of reflex irritability below the lesion. Other influences, however, may cause muscular contraction, such as associated movements from irradiation of voluntary impulses and descending neuritis and myelitis. *Erb* explains the absence of spastic paralysis in those complicated cases reported by *Leyden* and *Westphal*, by the coincident existence of degeneration of the gray substance and of the anterior roots, which must, of course, prevent contracture as well as increased tendon reflex. *Westphal* explains their absence, in his cases, by the implication of the white matter in the posterior root-zone, which prevented reflex action. *Erb* states that *complete* degeneration of the pyramid columns must cause only *paralysis*, since some power of conduction is necessary for the production of spastic symptoms. He concludes (1878) that a decided connection is manifested between sclerosis of the lateral columns and spastic paralysis. Cases recorded since 1878 seem to support his conclusion, although the peculiar symptom-complex of spinal paralysis has been exceptionally found *without* lesion of the lateral columns. These cases were not, however, perfectly typical.

*Erb* and *Charcot* lay great stress on the necessity of limiting the designation *spastic paralysis* to those cases which are *perfectly* typical. Any complicating symptom (disturbance of sensation, vesical weakness, etc.) throws doubt on the diagnosis. The history of the recently reported complicated cases illustrates fully the truth of the latter statement. The possibility that certain diseases of the brain may commence, especially in children, with the symptoms of spastic paralysis is to be kept in mind

(see case of *Schulz* and clinical observations of *Seguin* and of *Miles*, spastic infantile paralysis, *Med. Rec.*, N. Y., 1879, XVI., 217).

*Stümpell* calls attention to the *relaxation of the limbs*, which takes place in spastic paralysis, when the legs are deprived of the irritation of their own weight by the support afforded by water in a bath. The use of the bath is of value in determining the amount of paralysis, the spastic element being thus eliminated. The same observer saw *ankle-clonus* in typhoid fever and in phthisis without other nervous symptoms, except, in some cases, hyperæsthesia of the muscles. No change was found in the cords of the phthisis cases to explain the phenomenon. He found increased tendon reflex in a case of lead paralysis affecting only one arm, in a case of poisoning by strychnia and in three cases of poisoning by atropine.

#### TREATMENT AND PROGNOSIS.

*Mitchell* saw a case of amyotrophic lateral sclerosis improve in all respects under tonic treatment (elixir quiniæ, ferri et strychniæ, phosphates and cod-liver oil). He saw two cases resembling spastic paralysis recover in sixteen hours and three weeks respectively. *Henck, G.*, had a case, very nearly typical, which recovered in thirty-four days. Dry cups to the spine and purgatives were used. *Velden* gives one of recovery after thirteen months' illness. Case was not quite typical. Chloride of gold was used for two and a half months previous to recovery (chloride of gold, 0.3; distilled water, 15.00; fifteen to twenty drops three times a day). *Mitchell* obtained temporary relaxation of spastic contraction by the use of *massage*. In the absence of the spastic contraction, the patient could not stand up. No other cases are reported which shed light on the treatment or lighten the prognosis of this class of diseases.

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## HEMIPLEGIA AND HEMIPARAPLEGIA SPINALIS.

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A short mention of *Gowers'* case is all that calls for special mention here. In this case, a splinter of bone, knocked off by a pistol-bullet, injured the right half of the cord between the second and third vertebrae. Reflex action was diminished on the affected side. The sensibility to pain was abolished, that to touch was retained. The *posterior column was not directly injured*. The anæsthesia reached exactly to the median line.

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## POLIOMYELITIS ANTERIOR ACUTA.

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The important recent contribution to this subject is the case of acute poliomyelitis in an adult, with autopsy reported by *Schultze*. The patient was a woman, æt. 42. The disease began with a chill, followed by eight days of fever. The paralysis was typical in its way of occurrence and in the electrical reactions. Exposure to cold was the apparent cause. Death resulted from phthisis pulmonalis in thirty-four days after chill. On autopsy, degeneration was found in the anterior horns of those parts of the cord which corresponded to the affected limbs and degeneration of the corresponding anterior roots. At the junction of the lumbar and dorsal portion of the cord, a number of slightly and a few

greatly swollen axis-cylinders among a large number of normal ones were found, predominantly in the antero-lateral columns.

Clinical histories of this disease in the adult, quite typical in their course, are given by *Althaus*, *Sturge*, and *Sainton*.

*Seeligmüller* calls attention to the small number of exact observations of the *initial stage* of the disease which have been recorded. The initial symptoms are often so slight as not to give rise to careful examination of the cases. His last communication contains an analysis of seventy-five cases in children, which occurred in his own practice. The analysis led to no result which needs to be mentioned here. He records four cases, two of them doubtful, in which *progressive muscular atrophy* occurred in later life in patients who had infantile paralysis.

*Kirmisson* calls attention to the non-implication of the upper part of the trapezius in two cases of infantile paralysis, in which complete atrophy of the deltoid, biceps, supra- and infra-spinati, and lower two-thirds of the trapezius existed.

*Seeligmüller* saw complete recovery in one case of infantile paralysis of the right shoulder after four and a half months of electrical treatment.

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## POLIOMYELITIS ANTERIOR SUBACUTA ET CHRONICA.

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The etiological connection between *lead poisoning* and poliomyelitis anterior has been investigated by *J. J. Mason* (lead-poisoning in frogs, *N. Y. Med. Jour.*, 1877, July, p. 36). His results were negative. *Erb* alludes to them as needing confirmation. *Vulpian* found chronic myelitis with destruction of the ganglion-cells in a dog poisoned by lead. In a case of lead paralysis in the human subject, he found degeneration and atrophy of isolated ganglion-cells, with some increase of nuclei, and with sclerotic spots in the roots of the cervical enlargement. *Friedländer* (*Virch. Arch.*, 75, S. 24, 1879) records an autopsy of a case of lead paralysis in which degenerative changes were found in the muscles, nerves and spinal roots, but the *spinal cord was perfectly normal*. *Remak* criticises this case unfavorably on account of the imperfect clinical history.

As shedding some light on the *pathology* of the affection may be cited the cases of *Vulpian* and *Aufrecht*. These were complicated cases of irregularly distributed myelitis in adults, in which atrophy of the muscles and lesions of the anterior horns existed. The latter in his case found certain clear nucleated fibres, with here and there a spindle-formed mass of striated muscular substance, which he regarded as muscular fibres in the process of regeneration. Scattered through the nerves he found small nucleated fibres, in some places split into two parts, which he regarded as newly developing nerve-fibres.

*Kahler* and *Pick* report a case in which an excessive formation of vacuoles in the ganglion-cells of the anterior horns was the only spinal lesion found to explain paresis without evident atrophy, which existed during life in all the limbs. The only change in the electrical reactions was a diminished excitability to both currents in some muscles, principally in the extensors of the arms and hands. A similar lesion was found in a case reported by *Edes*, in which atrophy of forearms and legs began to develop less than four months before death. The abstract of this case given in the *Boston Med. and S. J.* is very imperfect. *Déjerine* publishes a case which, clinically, was not a typical one of poliomyelitis anterior, but in which degenerative atrophy of the muscles existed with degeneration of the motor nerves and anterior roots. *The ganglion-cells of the anterior horns in the lumbar enlargement had, for the most part, disappeared*. There was no indication of an irritative process in the connective tissue or on the blood-vessels. This same observer, from the examination of five cases of *diphtheritic paralysis*, concludes that in the anterior roots of the spinal nerves a lesion is *always* found, which corresponds to that which occurs in the distal end of a cut nerve, and is pro-

portionate in intensity to the duration of the paralysis. The posterior roots are never affected. A light alteration, to which the above is secondary, is always found in the gray substance, affecting the parenchyma and the interstitial substance equally, and not limited to any particular cell groups.

*Ricklin* speaks of a case (reported by *Debove* in the *Progrès Méd.*, 1879, p. 856) in which atrophy rapidly involved the legs, the forearms slightly, and the hands considerably, coming on with fever and pain in the limbs. These latter ceased after the second day in the arms, in the legs they diminished in violence, coming on in paroxysms three or four times an hour. Paralysis was nowhere complete. Sensibility and the sphincters were normal. Death occurred in three and a half months from intercurrent pneumonia. On autopsy, *the spinal cord was normal.*

Clinical histories of more or less typical cases are recorded by several observers, which confirm on the whole *Erb's* statements in regard to course and prognosis of poliomyelitis anterior chronica. *Renant* records two cases of *extensive lead paralysis*, which was preceded by *high fever*, with no other apparent origin than the process which resulted in the paralysis. *Bernhardt* records a case of *lead paralysis*, in which the electrical reactions were those of *Erb's middle form* of poliomyelitis anterior (see below).

*Erb* states that the *fibrillary contractions* of the muscles, which are usually present in the earlier stages of poliomyelitis anterior chronica, may be absent. The atrophying muscles are often painful on pressure, and may also be the site of spontaneous pains. In the more lightly affected muscles, reflex action may be only diminished, not, as is usually the case, entirely abolished.

Cases strongly resembling *lead paralysis* are reported by *Erb*, *Rosenthal*, and *Adamkiewicz*. This latter observer saw a formerly very free *secretion of sweat* disappear with the onset of the paralysis.

*Erb* has recently described what he calls a *middle form* of chronic poliomyelitis anterior, which differs from the ordinary type of the disease in that the *paralysis is never complete*, that the reflexes are not at all or only partly abolished, and that, while the *degeneration reaction in the muscles themselves is marked*, *the nerves are not at all or very little affected.*

*Erb* has seen four cases of this form of disease. Its course is very gradual. The evenly distributed muscular atrophy develops without fibrillary contractions. In other respects the symptoms are those of typical poliomyelitis anterior chronica. Three of *Erb's* cases improved, one recovering completely. The fourth developed slight symptoms of bulbar paralysis, but seemed to be stationary when it was recorded. The constant current applied over the spine was used in the case of cure.

In this place will be mentioned the results obtained by *Remak*, from a recent clinical study of twenty-one cases, including infantile, lead and chronic atrophic paralysis, the latter including four cases of progressive muscular atrophy. He finds that in these diseases there exists a marked

tendency toward the *isolated involvement of certain groups of muscles which act functionally together*, although the individual muscles of these groups are innervated by different nerve-trunks, and although other muscles innervated by these same nerve-trunks are not involved. The most important of these groups are the biceps, brachialis anticus, and supinator longus—the muscles on the dorsum of the forearm, exclusive of the supinator longus—the quadriceps extensor cruris and tibialis anticus, the sartorius and the perinei not being involved. These groupings have been previously observed in isolated cases. *Remak* regards this fact as pointing to the probability of the existence of *certain foci of ganglion-cells in the cord corresponding to these groups of muscles*, and therefore to the probability that any disease in which this grouping occurs is of spinal origin, although an affection of the spinal roots or of the plexuses near the cord may give rise to a similar grouping of affected muscles. He argues that this tendency in lead paralysis is an indication of its spinal origin. In a case reported by *Schultze*, in which all the muscles supplied by the sciatic nerves, except the tibiales antici were paralyzed, on autopsy it was found that the lower half of the lumbar enlargement of the cord was degenerated. The case thus demonstrated that the spinal centre for the tibialis anticus is in the upper half of the lumbar enlargement and disassociated with the centres of the other muscles supplied by the same peripheral nerve.

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## ACUTE ASCENDING PARALYSIS.

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In *Kétly's* case of acute ascending paralysis following poisoning by corrosive sublimate, the anatomical investigation was negative in results.

The case reported by *Jaffe* occurred in a syphilitic subject after excess in venery. The abolition of all the reflexes, and the almost complete abolition of faradic irritability of nerves and muscles with slight galvanic irritability, seems to point rather to acute poliomyelitis anterior. The reactions were tested by *Erb*. No autopsy.

*Fox's* case was almost typical in its clinical course. No changes were found in the cord except an apparent slight increase of connective tissue

in the cervical region of the cord. Sections of the medulla seemed to stain with abnormal readiness. Some increase of connective tissue seemed to exist about the cauda equina.

*Déjerine* claims to have found in two cases of acute ascending paralysis seen under *Vulpian*, an alteration in certain fibres of the anterior roots (parenchymatous neuritis). The myeline was broken up into fragments. Multiplication of nuclei in the white substance of Schwann, and disappearance of the axis cylinders were noted. The majority of the fibres were unaltered. The same lesions were found in the intra-muscular nerves of the affected members.

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## TUMORS OF THE SPINAL CORD.

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SCHULTZE, FR.: Beitr. z. Lehre von den Rückenmarkstumoren. Arch. f. Psych., VIII., S. 367, 1878.—ROTH, W.: Glyome diffuse de la moëlle, syringomyëlie, atrophie musculaire. Arch. de physiol. norm. et path., Par., 1878, 612-647.—KOHTS: Gerhardt's Handbuch der Kinderkrankheiten. Bd. V. (Tubercular Tumor of the Cord).—MADER: Einige Fälle seltener Spinalkrankheiten. Wien. med. Presse, 1879, 1058.

In the cases of *glioma* of the cord reported by *Schultze* and *Roth*, the process seemed to take its point of departure from the endyma of the central canal of the cord.

*Schultze's* case, a telangiectatic gliosarcoma myxomatodes, extended from the conus terminalis to the pons. For a while it presented the symptoms of a lesion of one-half of the cord. The peculiar combination of irritative and paralytic symptoms of striking inconstancy is brought forward by *Schultze* as a diagnostic point in tumors of the cord. The other cases reported call for no special comment.

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## SECONDARY DEGENERATIONS OF THE SPINAL CORD.

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DÉJERINE, J., et MAYER, A.: Recherches sur les altérations de la moëlle épinière et des nerfs du moignon chez des amputés d'ancienne date. Gaz. méd. de Paris, No. 39-40, 1878.—ISSARTIER: Des dégénérationes secondaires de la moëlle épinière consécut. aux lésions de la substance corticale du cerveau. Thèse, Paris,

1878.—TAYLOR, F.: A Case of Disease of the Brain with Descending Degenerations of the Spinal Cord. *Guy's Hosp. Rep.*, 1879, 169-184.

*Taylor's* case is the only one which calls for special mention. It was one of typical descending degeneration, originating in a peculiar gelatinous transformation of the corpus callosum, portions of the optic thalami, etc. The gelatinous substance of the cerebrum dissolved in alcohol, leaving only the blood-vessels, which showed nothing abnormal except a collection of small bright nuclei on their walls. The degeneration of the cord was darker and more sharply defined than usual. In the degenerated portion a structureless, almost homogeneous tissue took the place of the nerve tissue. The disease followed a fall on the back of the head. Paresis and spastic contraction of the limbs were the spinal symptoms of the case.

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## SPINA BIFIDA.

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RANKE, H.: Zur Aetiologie d. Spina bifida. *Amtl. Bericht der 50. Vers. d. Naturforscher u. Aerzte, München, 1877, S. 297.—Jahrb. f. Kinderheilk., XII., S. 116, '78.*

*Ranke* advances the theory that *spina bifida lumbalis* is caused by a growing together of the membranes of the cord and the external skin in embryonal life before the closing in of the spinal canal, the closure of the canal being thus prevented. His theory is based on three autopsies, in all of which the lower end of the cord was adherent to the sack.

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## SYRINGOMYELITIS.

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SCHÜLE: Beitrag z. Kenntniss der central. Höhlenbildung im R.-M. *Deutsch. Arch. f. klin. Med., XX., S. 271, 1877.—PICK, A.: Beitrag zur norm. u. pathol. Anatomie der Centralnervensyst. Arch. f. Psych., VIII., S. 283, 1878.—SCHULTZE, F.: Beitr. z. Lehre von der Rückenmarkstumoren. *Ibid., VIII., S. 367, 1878.—KAHLER und PICK: Beitrag z. Lehre von der Syring. und Hydromyélie. Vrtljsch. f. prakt. Heilk., 1879, II., 20.**

In *Schüle's* case, an enlargement of the central canal from softening of its walls occurred. The supposition that a congenital hydromyelitis may be the point of departure for a syringomyelitis later in life (*Leyden*) is supported by recent observations of *Pick*, who saw two

and even three central canals obviously formed by the growth of the ependyma and by the dividing off of diverticulum-like processes. *Schultze* considered that in his case the same thing might have happened.

*Kahler* and *Pick* saw in an adult cord a triangular dilatation of the lumbar portion, which was otherwise normal, obviously due to a defect of development. In a second case, the same condition existed, except that there was an increase of connective tissue around it, and the epithelium was in part destroyed. They found dilatation of the central canal in a case of amyotrophic lateral sclerosis and in one of progressive muscular atrophy with marked sclerosis of the posterior columns. They suggest that the condition noted in the first two cases may be regarded as an indication of deficient development, and therefore of predisposition to disease.

## TONIC CONTRACTIONS OF VOLUNTARY MUSCLES.

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SEELIGMÜLLER: Ueber spastische spinale Paralysen bei Kindern. Amtl. Ber. der 50. Vers. deutscher Naturforscher und Aerzte, München, 1877, S. 299.—Ueber spastische spinal Paralyse. Jahrb. f. Kinderheilk., XIII., S. 226.

*Seeligmüller* has observed two more cases of this rare affection. The first was a female public singer, whose mother had suffered from cramps in the calves. From her earliest childhood spontaneous tonic contractions of the kind described in *Seeligmüller's* first case (see *Erb*) would occur at times in the muscles of the calves, face, tongue, and hands. Otherwise she was perfectly healthy. The muscles of the legs, thighs, and upper arms were hypertrophied. Treatment effected very little, but some spontaneous improvement took place after its cessation.

The second case was a strongly developed healthy man, aged twenty-eight years, without hereditary tendency to disease. During four or five years he had stiffness all over the body. If he sat still for a while he would have to stretch and press his knees together before he could rise. If he wanted to dance he would have to try for some time before he could get going, and then there was no further difficulty. If any muscle was caused to contract by electricity it would not relax immediately. The right leg, right gluteal region, and the muscles of the right side of the back was more developed than those of the left side. Treatment with the induced current produced no effect. Massage caused some improvement.



