NEUROLOGICAL SURGERY

A. Spinal Cord
B. Peripheral Nerves

By JOHN B. MURPHY, A. M., M. D., LL. D., Chicago
Professor of Surgery, Rush Medical College, University of Chicago

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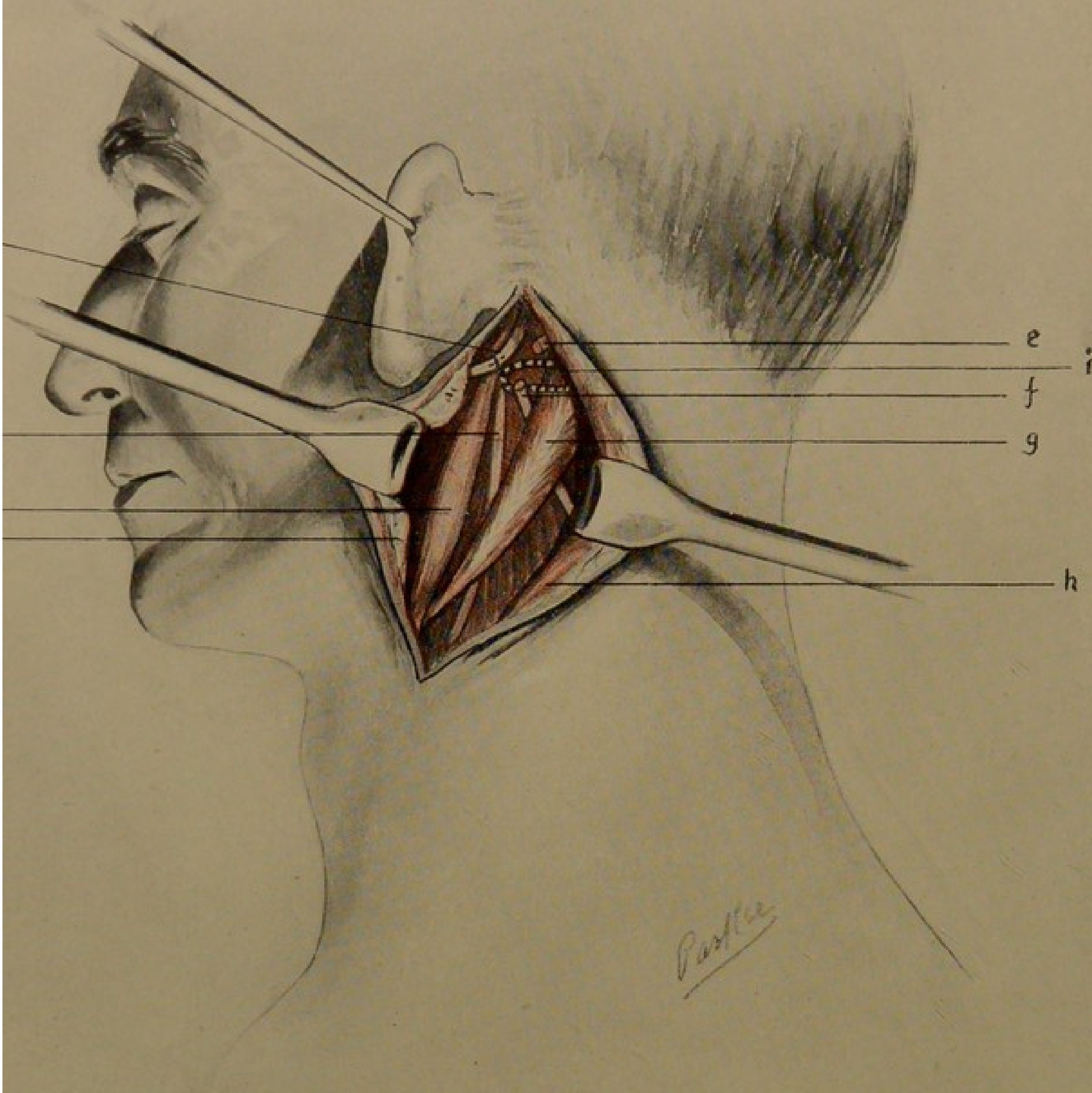

Fig. showing anastomosis between spinal accessory and facial nerve.

a. End-to-end anastomosis between facial and spinal accessory.
b. Hypoglossal nerve.
c. Stylohyoideus muscle.
d. Parotid gland.
e. Central end of facial nerve.
f. Periphral end of spinal accessory.
g. Posterior belly of digastric muscle.
h. Muscle.
i. Outline of transverse process.
(Original drawing from a dissection)

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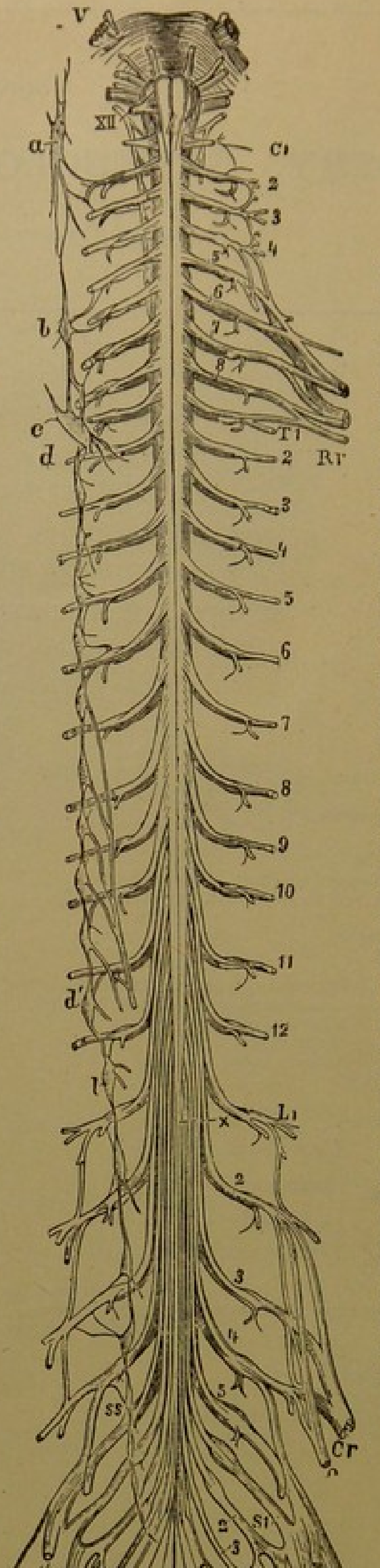
SYNOPSIS OF THE SURGERY OF SPINAL CORD

1. What lesions of the spinal cord are amenable to surgical treatment?
2. What lesions are not?
3. What is the histologic basis?

- Neurone; nervous unit.
  - Cell-body.
  - Dendrites.
  - Axone.
  - Neurilemmic axone.
    - Degeneration.
    - Regeneration.
  - Aneurilemmic axone.
    - Degeneration.
    - Regeneration.
  - Medullated. / Non-medullated. — Insulation effect.
  - Portion of neurones separated from cell-body.
  - Degeneration; regeneration; atrophy of cell-body.
- Anatomy (gross)
  - Spinal cord.
  - Medulla oblongata.
  - Cauda equina.
- Anatomy (minute.)
  - Columns. / Fasciculi. — Ararngement, origin, and termination of neurones: *a.* Motor; *b.* Sensory.
  - Gray matter. Conformation; structure.
  - Topography of
    - Motor nuclei.
    - Sensory nuclei.
  - Ependymal canal.
- Macroscopical and microscopical description of
  - Anterior (motor) roots.
    - Extraspinal. / Intraspinal. — Centrifugal impulses.
  - Posterior (sensory roots).
    - Extraspinal. / Intraspinal. — Centripetal impulses.
  - Spinal ganglia. / Cranial ganglia. — Location; structure.
- Location of Trophic Ganglion Cell-body.
  - Motor nerves.
  - Sensory nerves.
  - Sympathetic nerves.
- Degeneration of spinal cord after division.
  - Ascending (sensory).
  - Descending (motor).
  - Zonal.
    - Traumatic.
    - Ganglionic cells destroyed.
  - Description; histologic evolution of degenerative process.
  - Conclusion.
- Classification of nerves.
  - Cerebral.
  - Cranial.
  - Spinal.
- Degeneration of
  - Anterior (motor) roots. (Charles A. Ballance, Purves Stewart).
    - Extraspinal.
    - Intraspina
  - Cerebral nerves (four sensory).
  - Posterior (sensory) roots.
    - Extraspinal.
    - Intraspinal.
  - Cerebral nerves. (Four nerves of special sense.)
    - Optic.
    - Auditory.
    - Olfactory.
    - Gustatory.
- Regeneration of spinal cord after division.
  - Histology.
  - Experimental studies.
  - Is regeneration of spinal cord possible?
  - Partisans.
  - Opponents.
  - Conclusions.
- Surgical interventions and limitations in
  - *a.* Compressions.
  - *b.* Concussions.
  - *c.* Dislocations.
  - *d.* Contusions.
  - *e.* Fractures.
  - *f.* Bullet wounds.
  - *g.* Punctured wounds.
  - *h.* Partial or complete transverse sections.
  - *i.* Syringomyelia.
  - *j.* Spina bifida.
  - *k.* Motor paralysis of lower extremities.
    - Anterior pololiomyelitis, flaccid.
    - Hemiplegic.
      - *a.* Central, spastic.
      - *b.* Spinal, spastic below injury.
  - *l.* Hemorrhage.
    - Extraspinal.
    - Intraspinal.
    - Spinal puncture.
  - *m.* Tumors—Paralysis.
    - Degenerative.
    - Functional.
  - *n.* Tuberculosis.
    - Spinal column.
    - Spinal cord.
  - *o.* Meningitis.
    - Acute.
    - Infective.
- Laminectomy—Technique.
- FUTURE OF SPINAL CORD SURGERY.
- Conclusions.

WITHOUT a clear-cut, thorough knowledge of the anatomy of the cord — 1. As to its various columns and their locations; 2. As to the location of the ganglion or trophic cell-body of the axones which make up these columns, and which indicate the direction of the line of degeneration; 3. As to the origin of the axones, either in the spinal-cord ganglion-cells or in the spinal posterior root-ganglia and their directions; 4. As to the position of acquirement of the medullary sheath and neurilemma inside or outside of the cord; 5. As to the relation of the entrance of the nerve-roots into the anterior and posterior lateral fissures and the topographic relation of the spinal foramina to the point of entrance of fibers into the lateral fissures and thence to the various segments of the cord; 6. As to the ependyma in its anatomic and pathologic relations to the substance of the cord, and as to the essential genetic and histologic difference between the cauda equina and the remaining portion of the cord,—all effort of understanding or advancing the surgery of the spinal cord is futile. We must therefore devote a considerable space to these details, and solicit the indulgence of our readers for entering into such minute descriptions.

The neurone theory is herein appropriated for the interpretation of the histology, physiology, clinical manifestations, and pathology of the nervous system, and is particularly applicable to a knowledge of its surgery, notwithstanding the recent divergencies of theory as to "peripheral regeneration."



From Barker's *Nervous System*, p. 893, Fig. 572.

Fig. 1. Spinal cord in connection above with the medulla oblongata and pons. (After A. Rauber, *Lehrbuch der Anatomie des Menschen*, V. Aufl, Leipzig, 1898, bd. ii. s. 504, Fig. 485). V. Nervous trigeminus. XII. Nervus hypoglossus. C. First cervical nerve. C. 2-8. Second to eighth cervical nerve. T. 1-12. First to twelfth thoracic nerve. L. 1-15. First to fifth lumbar nerve. S. 1-5. First to fifth sacral nerve. 6. Nervus coccygeus. xx. Filum terminale of the spinal cord. From the root marked L to x, cauda equina. Rr. Plexus brachialis. Cr. Nervus femoralis. Sc. Nervus ischiadicus. O. Nervus obturatorius. The enlargements opposite L 3, 4, 5, represent the spinal ganglia on the dorsal roots. On the left side of the figure the sympathetic trunk is shown. *a* to *ss*, Ganglia. *a*. Ganglion cervicale superius. *b*, *c*. Ganglion cervicale medium et inferius. *d*. First thoracic ganglion. *d*. Last thoracic ganglion. l. First lumbar ganglion. *ss*. First sacral ganglion.

NEURONE

A neurone consists of a cell-body, its trophic ganglionic cell, of an axone or axones, and of dendrites and telodendrones. The intraspinal axone is composed of an axis-cylinder and medullary sheath; the extraspinal one has, in addition, a neurilemma. (See Fig. 4). The sympathetic axones, except a few abdominal fibers, have no medullary sheath, but have a neurilemma.

ANATOMY

Gross and Minute

From a surgical standpoint, the spinal cord commences above or on a line between the articular surfaces of the atlas and the condyles of the occipital bone. Anatomically, its lower limit is the apex of the conus medullaris, which is situated at the upper level of the second lumbar vertebra. Surgically, however, the lower limit is one to one and a half inches above this point, bringing it to the twelfth dorsal, as the sacral and coccygeal nerves originate from the side of the conus up to the level of the eleventh dorsal vertebra. The upper limit is an arbitrary one, corresponding to the decussation of the pyramidal tract. Anatomists differ in their description of the limitations of the conus. Fitzner considers its upper limit a line passing immediately below the point of origin of the coccygeal nerve. Poirier places this line above the origin of the first coccygeal nerve, just below the origin of the fifth sacral. This varies. In children it is as low as the second lumbar; in adults it is as high as the eleventh dorsal.

The cauda equina is a group of nerves which supplies the inferior extremities, buttocks, bladder, and genitalia. It is inclosed in a fibrous muff of the dura mater, which terminates in a cul-de-sac on a level with the second sacral vertebra. The exit of the lumbar and sacral nerves is through the side of this sac, while the filum terminale and coccygeal nerves pass through the apex of the cone. The openings in the dura for the exit of these nerves are very much lower than the exit of the nerves from the anterior and posterior lateral fissures of the cord. (See Fig. 2). The cord is held in a fixed relation with its bony canal by the ligamentum denticulatum and the spinal nerve-roots; above, it is suspended from the medulla, and below it is attached to the posterior surface of the coccyx by the coccygeal ligament, which is a continuation of the dura. The cord is composed of two halves, produced by the deep anterior and posterior fissures, which separate it into lateral halves for three-fifths of its diameter.

On both sides, external to the posterior fissure, there is another fissure — the sulcus lateralis posterior — in which are implanted the posterior sensory roots. This fissure subdivides the cord into a small posterior and a much larger anterolateral column. The two posterior columns thus formed are paths of sensory neurones. The anterolateral column is subdivided by a fissure — sulcus lateralis anterior — in which are implanted the motor roots. The fissure is about the same distance externally from the median line as the sulcus lateralis posterior, and subdivides the anterolateral white matter of the cord into sets of anterior and lateral columns. The posterior column is further subdivided by a groove into an internal fasciculus, gracilis or column of Goll, and an external fasciculus, cuneatus or column of Burdach. These columns, together with Gowers', and the small column of Lissauer,

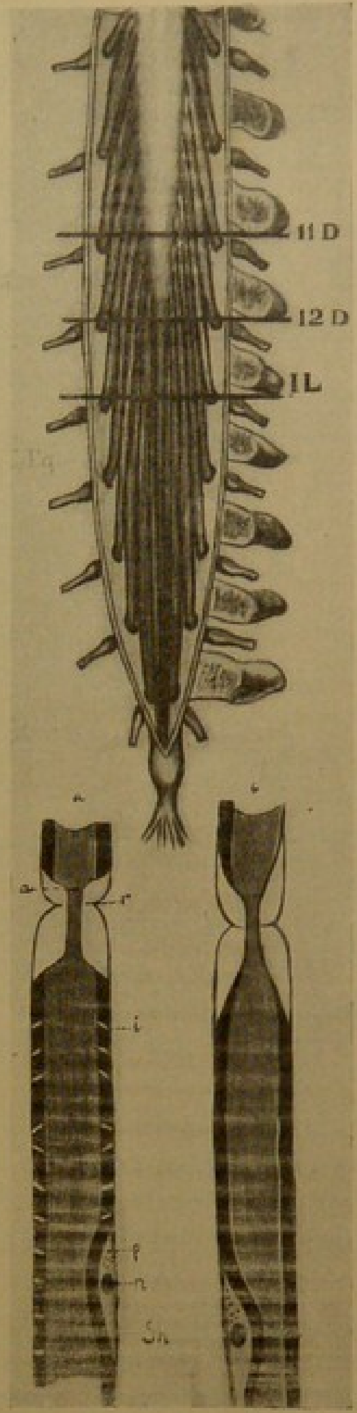


Fig. 2. Cauda equina. Showing the conus medullaris ending at the level of the second lumbar vertebra. *b, c.* Structure of a peripheral axone, showing axis-cylinder, medullary sheath, neurilemma-cell, and node of Ranvier. (Original).

which applies itself close to the outer margin of the posterior roots, are the only sensory tracts of the spinal cord.

The anterolateral column is divided into anterior and posterior sets of fasciculi. The anterior set is composed of: (*a*) Direct pyramidal tract; purely motor neurones which do not decussate in the bulb; and (*b*) The anterior ground bundle of short fibers connecting the spinal ganglion motor cells of different levels.

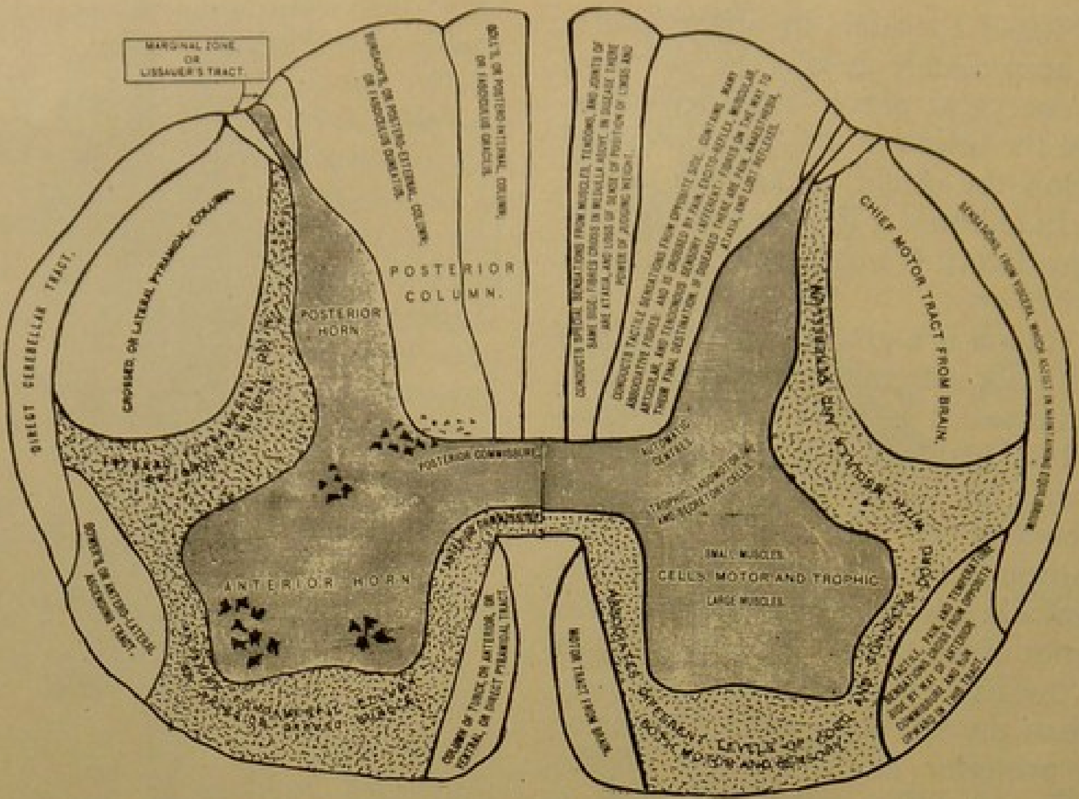


Fig. 3. Showing the tracts and functions of various tracts of spinal cord. (From Butler's *Medical Diagnosis*).

The backward set is composed of: (*a*) The anterolateral tract; (*b*) The direct cerebellar; (*c*) The crossed pyramidal; and (*d*) Gowers'. All of these surround the anterolateral ground bundle.

The gray matter has the aspect of a capital H, formed by two lateral cornua, connected by a transverse strand, the commissure. The gray matter is subdivided into anterior thick and posterior thin cornua. At the junction of these two there is a lateral expansion, frequently called the lateral horn. At the tip of the posterior horn, the point of entrance of sensory neurone, there is a translucent substance called the gelatinous structure of Rolando. In the center of the transverse commissure of gray matter there is a central canal which begins with an opening in the floor of the fourth ventricle, and ends blindly in the filum terminale. It is lined with epithelial cells, a continuation of the ventricular epithelium, both of which are ectodermic in origin. This canal has a great pathologic significance in syringomyelia, spina bifida centralis, and central hydrocephalus. It is centrally situated in the lumbar region; in the dorsal and cervical, it is more anterior; in the conus, it forms a fusiform dilatation one millimeter in diameter, called the ventriculus terminalis of Krause. The gray matter of the cord is made up of ganglion-cells, fibers, ground substance, and stroma. Here are situated the trophic cell-bodies of the lower motor neurones. The cells in the gray matter may be classified as follows:

1. Root-cells: (*a*) Anterior root-cells; (*b*) Posterior root-cells.

2. Column-cells: (*a*) Anterior column-cells; (*b*) Lateral column-cells; (*c*) Posterior column-cells.

The fibers originating from the column-cells may be divided into: (*a*) Those passing in the white substance of the same side; (*b*) Those passing in the white substance of the opposite side; (*c*) Those passing in the white substance of both sides.

3. Cells with short axis-cylinders, association neurones: (*a*) With fibers passing into the gray substance of the same side; (*b*) Into the gray substance of the opposite side.

Surrounding the ependyma there are isolated ganglionic cells, which send fibers to the same or opposite sides of the white substance. The ground substance or stroma, also called the neuroglia, is composed of delicate cells, which are distributed in both white and gray substances. There are two varieties, both of ecto-

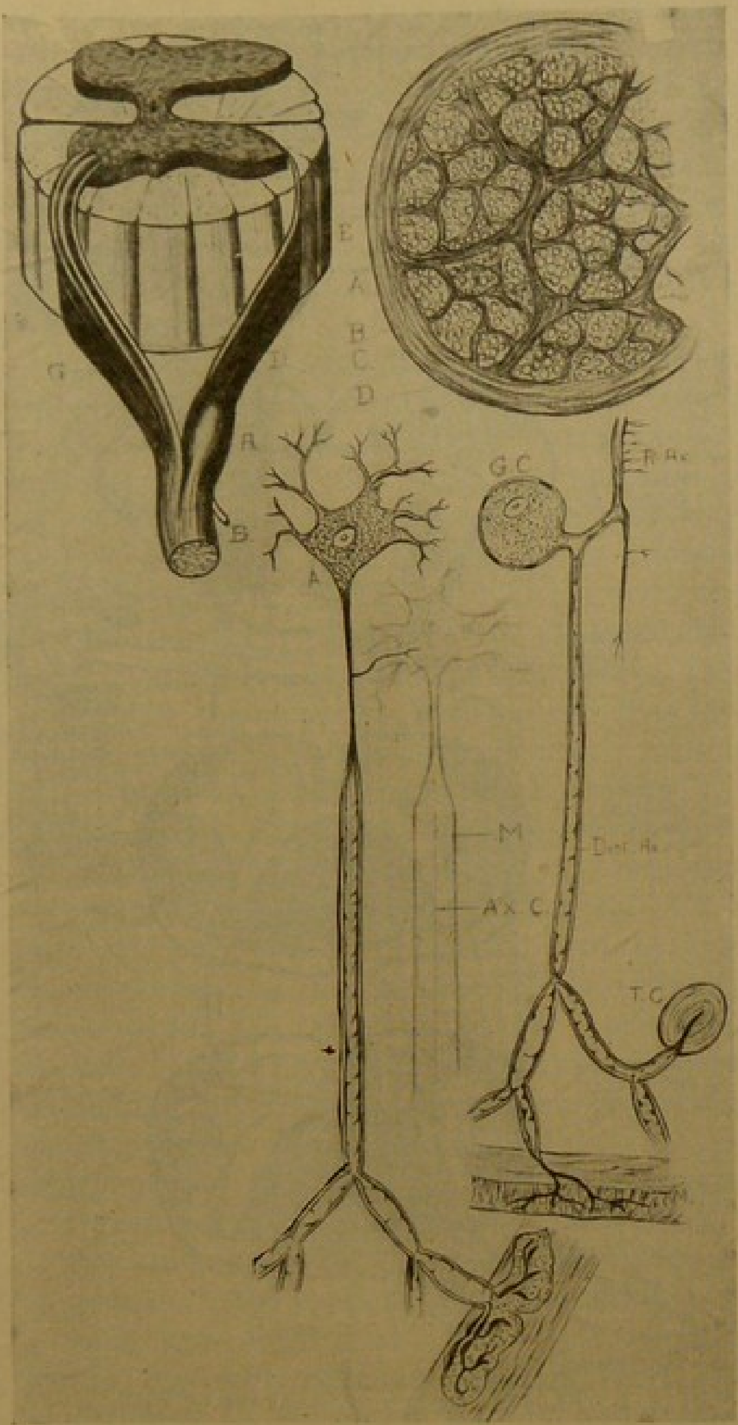


Fig. 4. Showing: *a*. Cross-section of spinal cord with anterior and posterior root and its ganglion. *b*. Cross-section of a nerve-trunk, showing the perineural and interfascicular connective tissue. *c*. Motor neurone. *d*. Sensory neurone. (Various authors).

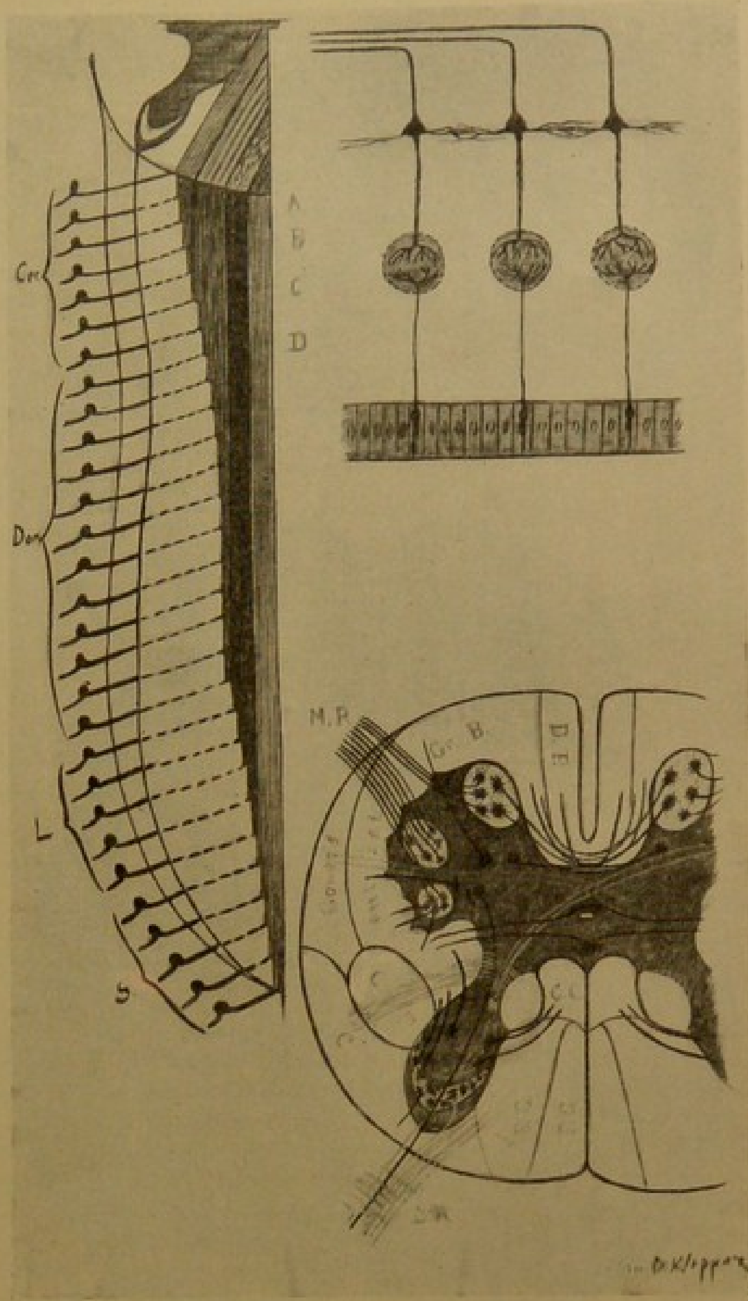


Fig. 5. *a*. Schematic drawing showing the formation of Goll's column. *b*. Schematic drawing showing the relations of the peripheral olfactory neurone with the central neurone. *c*. Distribution of cells in the gray matter. (Testut).

dermal origin; one the ependymal cells, already described, and the other, the neuroglia, Deiters' or spider cells. The latter have many projections, which anastomose in various directions, forming a framework which supports the delicate nerve tissue.

The fibers which make up the white substance or column may be briefly classified as:

A. Centripetal; long or projective axones: those which go from ganglia on the posterior roots and ganglion-cells in the posterior horns of gray matter: (*a*) Lower areas; (*b*) Upper areas in the cord, medulla oblongata, and cerebellum.

B. Centrifugal; those which descend from the pyramidal cortical cells to the spinal ganglionic cells.

C. Short axones; association neurones: which connect different levels of the cord with each other, and also the two halves of the cord

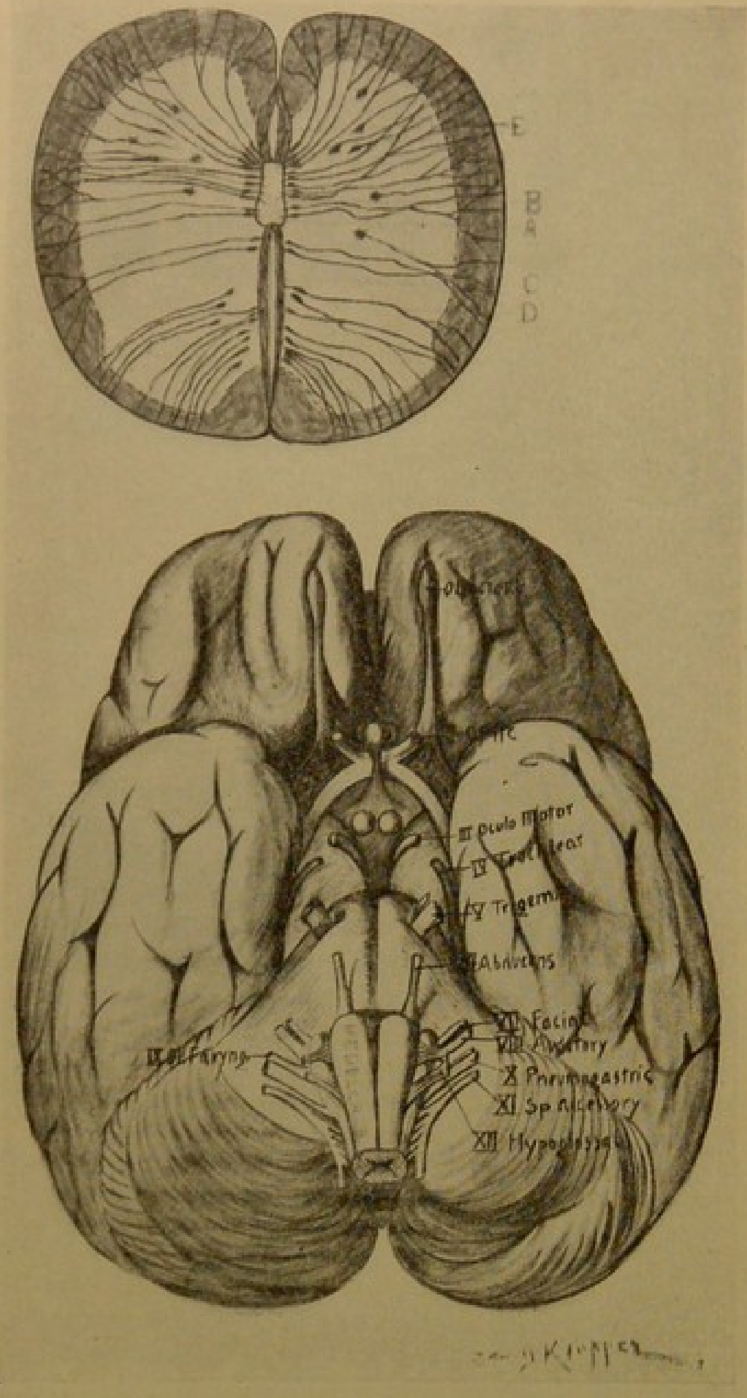


Fig. 6. A. Ependymal and neurogliar cells in the developing stage. B. Under surface of brain, showing origin of cranial nerves. (From Testut's *Anatomie Humaine*).

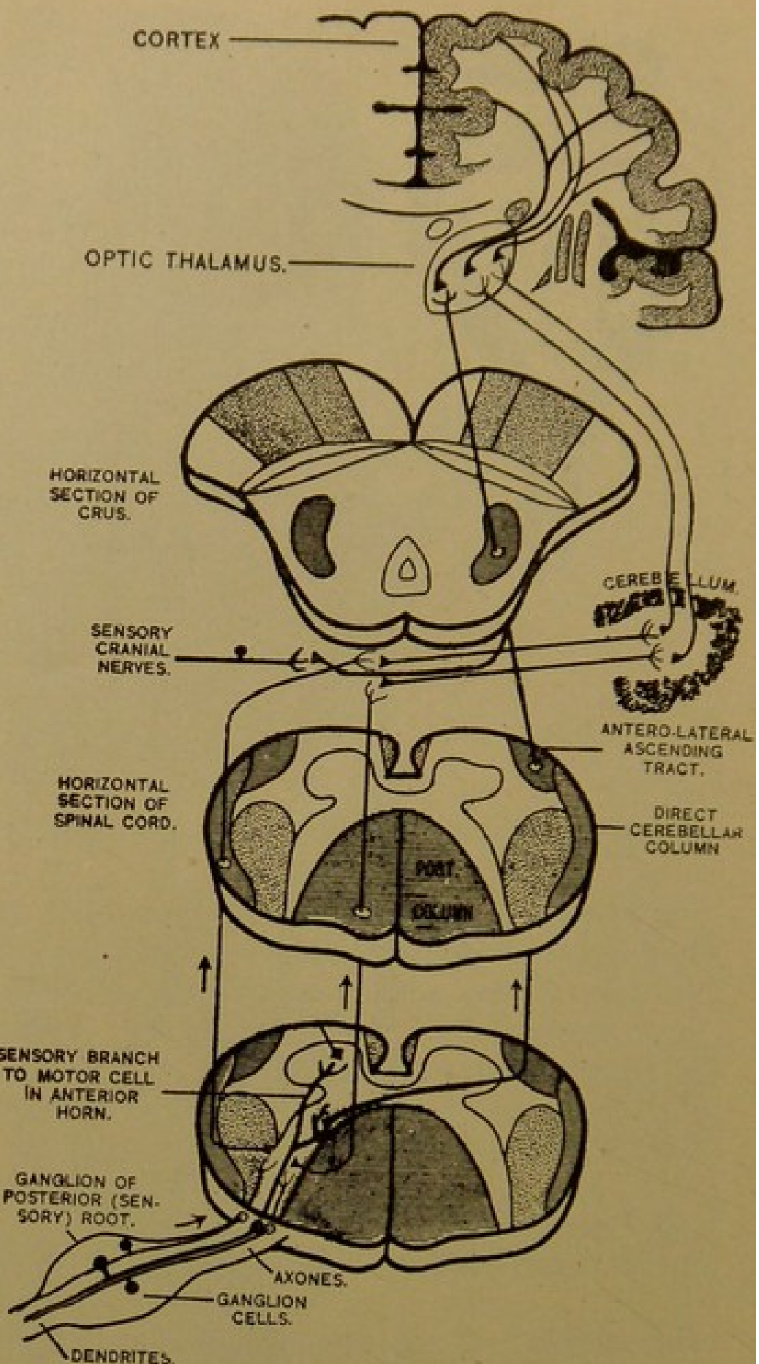


Fig. 7. Showing the sensory pathway. (Butler).

at the same level. All of the axones of the white columns are medullated and aneurilemmic (Böhm and Davidoff).

Anterior nerve-roots are the axones of the ganglionic cells of the anterior cornu. The cell-body and axone form the lower or peripheral motor neurone of first order (Barker). The cells of the anterior cornu are surrounded by two chief sets of "end-brushes"; one originating from the pyramidal tracts, the other from the posterior horn or roots. In this way, cells in the anterior horn are in relation to the brain and periphery.

The axones, from their motor ganglionic cells, pass out of the gray matter into the white, where they immediately receive their medullary sheaths, passing onward to the surface of the fissure, where they receive their neurilemma. From here the fibers are made up of an axone, a medullary sheath, and a neurilemma, which continue to the muscle-plate, where they lose their neurilemma and medullary sheath, the continuation being the axone subdivided into fibriles (telodendrones). All of the spinal nerves and all of the cranial nerves, except the nerves of special sense, have this anatomic structure. All axones supplied with neurilemma are, under favorable conditions, capable of complete regeneration. The trophic cell-bodies of the lower motor neurones are located in the anterior cornu of gray matter. When the ganglion-cells in the anterior column of gray matter are destroyed, as in anteropoliomyelitis, regeneration is impossible.

Posterior nerve-roots are made up of efferent axones, originating from ganglionic cell-bodies in the posterior root-ganglion. The latter are the trophic cells of the posterior root, as well as of the sensory neurones. The efferent axones enter the cord in the sulcus lateralis posterior, and divide in a T-like manner, one branch descending for a short distance, the other ascending for a longer distance; then they enter the posterior horn, where they terminate in end-brushes surrounding a nerve-cell, which is the terminal nucleus.

The sensory neurone is made up of the ganglion-cell (trophic cell) body in the posterior root-ganglia, an afferent axone leading to the cord and on upward through the cord, even to the cerebellum, as just described, and an efferent axone from the ganglion to the peripheral sensory corpuscles. Both of the axones have medullary sheaths and neurilemma, except the portion within the spinal cord, which has no neurilemma. The posterior root and peripheral axones are capable of regeneration.

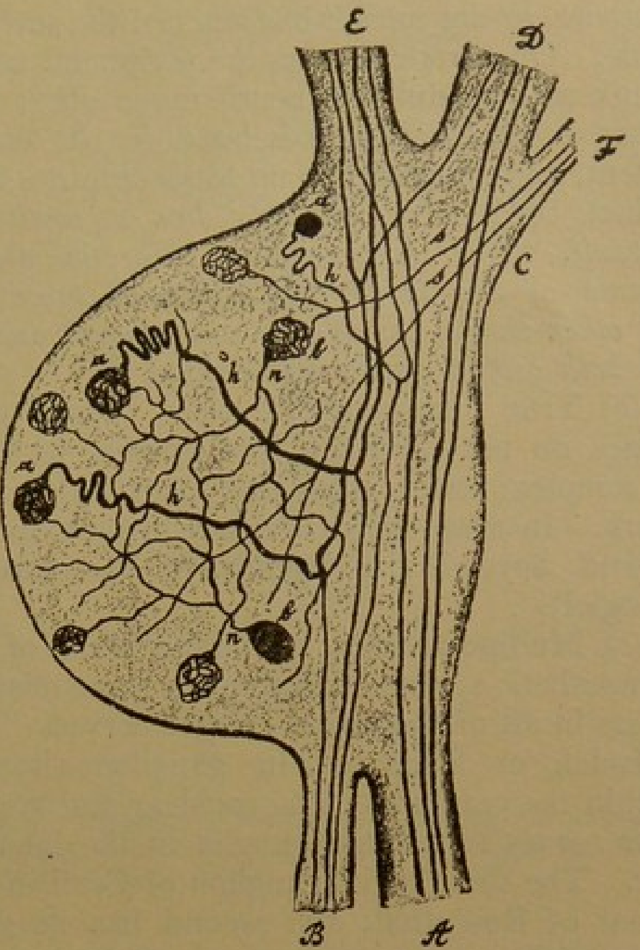


Fig. 8. Scheme of the reciprocal relations of the elements within the spinal ganglion, according to Dogiel. *A, B*. Ventral and dorsal roots. *C*. Spinal nerve. *D, E*. Ventral and dorsal divisions of spinal nerve. F. Ramus communicans (sympathetic connection). *a. b.* Spinal ganglion-cells of the first and second type. *h*. Trunk processes of cells of the first type which divide to form the axones of the peripheral and central fibers. *n*. Axones of cells of the second type which end as a pericellular feltwork about the cells of the first type. *s*. Sympathetic fibers which end as a circumcellular plexus about the cells of the second type. (Barker).

The spinal intervertebral or posterior root-ganglia are ovoid swellings of grayish color, with a long axis horizontal. They are situated within the intervertebral foramina, with the exception of the ganglia of the first and second cervical and all of the sacral and coccygeal nerves. The situation of the first cervical ganglion varies; it may be inside, within, or outside the dural opening; the latter most frequently. The second ganglion is situated between the anterior portion of the posterior arch of the atlas and the lamina of the axis, which represents the intervertebral foramen. The sacral ganglia are situated in the sacral canal between the lateral wall of the canal and the dural cul-de-sac. The latter ganglia are therefore extradural and intraspinal. The ganglion of the coccygeal nerve is not constant. Every spinal ganglion is surrounded by a connective-tissue capsule continuous with the perineural sheaths of the incoming and outgoing nerve-roots. The body of the ganglion is divided and subdivided by septæ, which originate from the capsule; these give support to the nervous elements. There are several types of cells in the spinal ganglion.

(*a*) Small and large unipolar, with Y-shaped or T-shaped division of their process. The cell-body is either round or oval; from it originates a single process, which becomes surrounded with medullary sheath soon after leaving the body. This makes several turns first, and then becomes straight. The branching of the process takes place, according to Dogiel, either at the second or third node of

Ranvier, or not until it has reached the seventh node. One branch enters the peripheral nerve trunk as a medullated neurilemmic fiber; the other becomes also medullated for its entire extent, and neurilemmic up to its entrance into spinal cord. *The cell-body has a nucleated capsule, which is continuous with the neurilemma of the single and bifurcated processes. In other words, the afferent and efferent axones are both provided with neurilemma.*

(*b*) True intraganglionic cells. Their processes do not bifurcate, but branch, forming a complex pericapsular and pericellular network. In other words, these processes do not extend beyond the bounds of the ganglia (Dogiel).

(*c*) Multipolar cells, resembling those of the sympathetic system. Exactly the same relation exists in all of the mixed cranial nerves. The trifacial, or fifth, has its ganglion situated within the cranium. The cochlear and vestibular nerves have their ganglia in the internal ear. The first has its ganglion of Corti in the canal of Rosenthal; the second has its ganglion of Scarpa at the entrance of the internal auditory meatus. The cells in these ganglia are bipolar, in contrast to other ganglion-cells.

The pneumogastric and glossopharyngeal have their ganglia extracranial, which is equivalent to extraspinal. They have, therefore, their efferent and afferent axones to and from the ganglia, and their trophic motor cell-bodies are situated in a continuation of gray matter of anterior cornu in medulla and pons. They are therefore all essentially spinal nerves, except those of special sense, which should be called cerebral, and in future will be called cerebral, nerves. The cranial nerves are, from a surgical standpoint, practically spinal nerves, governed by the same laws, having the same histology, anatomic relations, degenerative changes, and regenerative potency as spinal nerves.

The nerves of special sense, which we call cerebral, have an entirely different relation, as their ganglion or trophic cell-bodies are situated in the periphery or special-sense organs, and the axones extend from the periphery centripetally, passing inward to contact with the dendrites of special-sense ganglionic cells; that is, the axone trophic cell-bodies of the optic nerve are situated in the retina, the olfactory in the mucous membrane of the superior turbinated fossa, the gustatory in the taste-corpuscles situated principally at the circumvallate papillæ of the tongue, and the auditory in the cochlear membrane (organ of Corti). Their axones extend to the brain, and make up the nerve-trunks of special sense. With the exception of the optic and some fibers of the auditory, they are neurilemmic and capable of regeneration. The optic and the aneurilemmic fibers of the auditory are incapable of regeneration. All of the axones terminate in the opposite side of the cord or brain, from which they originate, except the axones of the direct cerebellar tract.

Origin and Termination of the Axones of the Columns (Fasciculi) of White Substance of the Cord

Ascending or descending degeneration means a degeneration upward or downward from the point of division only, and always in a direction opposite to the location of the trophic cell-body. From the point of division, proximally toward the cell-body, there is an atrophy, but not complete degeneration, of the axone. In neurilemmic axones there is a degeneration to the first node of Ranvier and an atrophy proximal to that point, even including the cell-body or ganglion-cell. The scheme below given shows distinctly the direction of degeneration in spinal-cord division, and of atrophy in contusion or compression. Still, we deem it necessary to give a detailed description of it, which for emphasis will be considered in the opposite order to that of the tabulation.

Posterior Columns: Fasciculus Gracilis (Goll) and Fasciculus Cuneatus (Burdach)

They contain fibers, some of them short, uniting the different levels of the cord, and others long, which go from both, uninterrupted, to the medulla oblongata. The most internal come from the lowest segments of the cord; the most external, from the highest. In the medulla oblongata, they terminate in brush-like endings in the nucleus gracilis (Goll) and nucleus cuneatus (Burdach), around the dendrites of cells situated in these nuclei. From these cells new fibers originate and go upward; some in the cerebellum, where they come in contact with central centripetal somesthetic axones; others in the cortex. The fibers originating from the cells of these nuclei cross soon after their origin, in a similar manner, to the motor fibers of the pyramidal tract. After crossing, they join the deep surface of the pyramidal tract, and, accompanying it, go to the cerebral hemispheres. It can, therefore, be seen that a hemisection of the left half of the cord, for example, would produce a paralysis of sensation in all the structures on the same side below the division.

The zone of Lissauer, or marginal zone of Lissauer, is situated in the sulcus lateralis posterior, between the external angle of the posterior column and the internal angle of the lateral column. The fibers of the posterior roots passing through it divide it into two halves, one external, the other internal. In the lumbar region, the zone of Lissauer has its greatest dimensions. According to Lissauer, the zone is composed of the fine fibers of the posterior roots, while other embryologists consider it as being formed by the collaterals given off by the fibers of the posterior roots when penetrating the network. Testut thinks that it is composed of both root-fibers, both ascending and descending, and their collaterals. The fibers in the zone of Lissauer enter, usually, after a short journey, some into the posterior horn, others into the fasciculus of Burdach.

The anterolateral ground bundles contain short fibers, which unite the different levels of the cord. They do not go to the brain. Thus, degeneration is always limited, both ascending and descending.

The Ascending Anterolateral Column or Tract of Gowers

The trophic cell-bodies of this column are situated, according to Sherrington and Edinger, in the posterior horn, close to the commissure. The axones originating from these cells mostly pass to the opposite side in a transverse manner until they reach the periphery of the cord; then they change direction and become vertical, in order to make up the column. In other words, the fibers of this column are crossed within the gray substance of the cord. The ascending fibers of the column reach the lateral surface of the medulla oblongata in the region of the

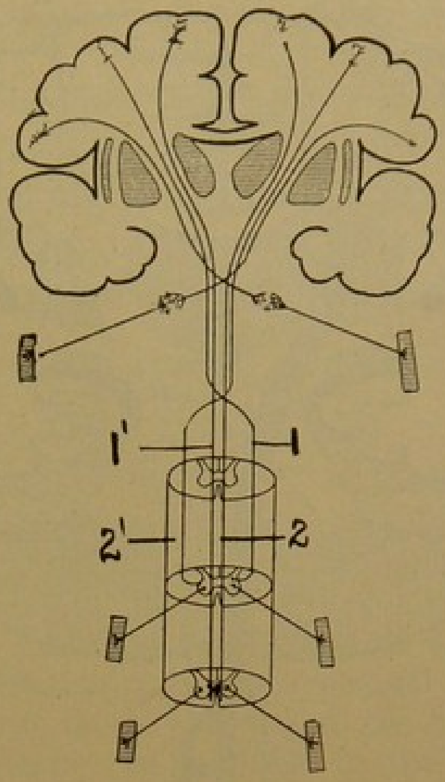


Fig. 9. Diagram of motor path, showing the crossing of the path, which takes place in the upper segment. (Osler, after Van Gehuchten).

inferior olivary body, where they partially or totally terminate in the nucleus of Bechterew, whence they approach the peduncular island of Reil, and from there go to the cortex cerebri.

The Direct Cerebellar Tract

The axones of this tract originate from a group of cells situated at the posterointernal region of the posterior horn and close to the commissure, forming there a distinct column of Clarke. In the lumbar and cervical region, where the column is not distinctly formed, they originate from cells which are homologous to the column of Clarke. The uncrossed fibers of this tract terminate in the dorsal portion of the vermis superior of the cerebellum. Degeneration is therefore ascending, and on the same side with division. A hemisection of this column would involve the loss of automatic equilibrium on the same side of the lesion.

The Crossed Pyramidal Tract

The crossed pyramidal tract contains axones originating from the pyramidal motor-cells of the cortex; they descend down the bulb, and, after the crossing of all of its axones (decussa-

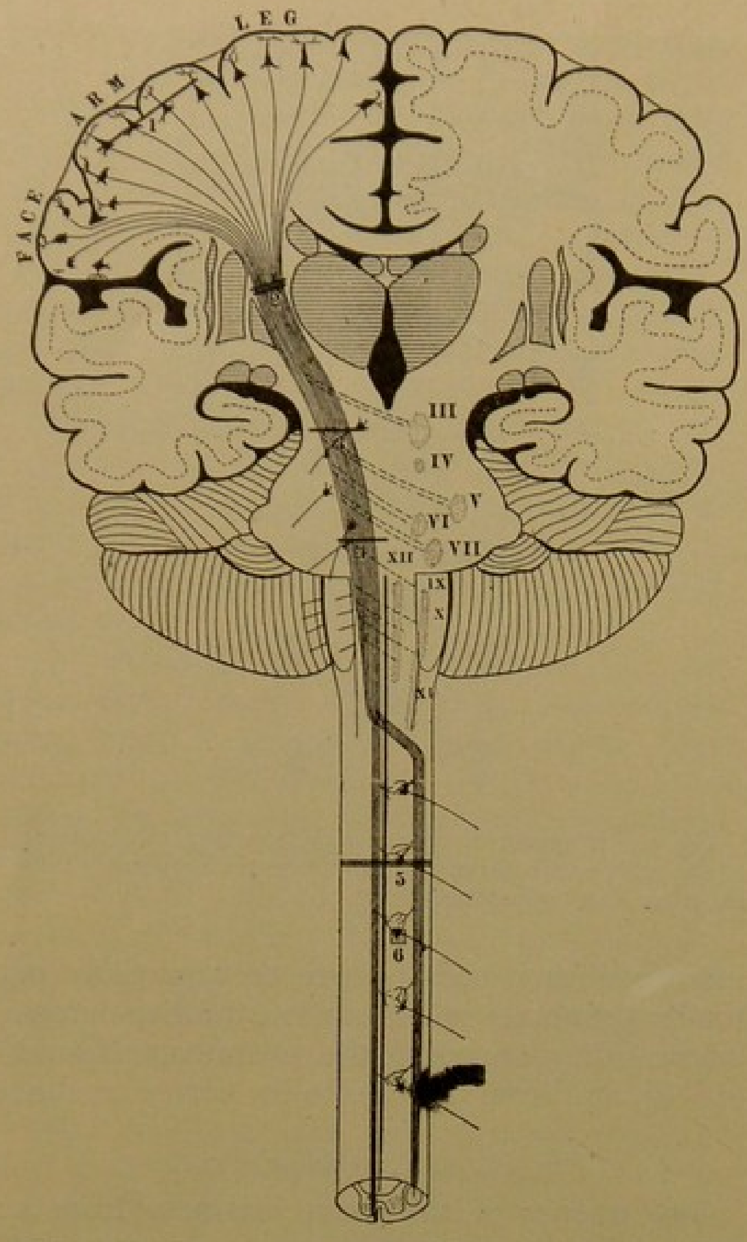


Fig. 10. Diagram of motor path from right brain. The nuclei of the motor cranial nerves are shown on the left side; on the right side the cranial nerves of that side are indicated. A lesion at 1 would cause upper-segment paralysis in the arm of the opposite side (cerebral monoplegia); at 2, upper-segment paralysis of the whole opposite side of the body (hemiplegia); at 3, upper-segment paralysis of the opposite face, arm and leg, and lower-segment paralysis of the eye-muscles on the same side (crossed paralysis); at 4, upper-segment paralysis of opposite arm and leg, and lower segment paralysis of the face and the external rectus on the same side (crossed paralysis); at 5, upper-segment paralysis of all muscles below lesion, and lower-segment paralysis of muscles represented at level of lesion (spinal paraplegia); at 6, lower segment paralysis of muscles localized at seat of lesion (anteropoliomyelitis). (Van Gehuchten, modified. Osler, p. 818).

tion of pyramids), descend in the cord of the opposite side, so that fibers originating from the right half of the cortex are situated in the left half of the cord, and *vice versa.* Degeneration is therefore descending. In a hemisection in the cord, involving this tract, there is almost complete paralysis of muscles of the same side, the only remaining motion being supplied from the direct pyramidal tract from the opposite side. This remaining motion explains some of the errors in the reports in cases of supposed regeneration of the cord after hemisection.

Direct Pyramidal Tract

Its axones originate from the pyramidal cells of the cortex. They descend in the bulb and cord on the same side to a point in the white substance, where they cross and pass to contact with lower motor neurones, whose axones pass directly out of the cord through the anterior root. It ultimately must be considered a crossed tract, because its fibers cross at the level of entrance into the gray substance. Degeneration is therefore descending, and a division of this tract produces a partial paralysis or paresis of the opposite side.

TRACTS OF DEGENERATION OF SPINAL CORD AFTER DIVISION

Recapitulation

Six Sensory Fasciculi

Tracts which undergo ascending degeneration:

1. Posterointernal fasciculus gracilis (Goll).
2. Posteroexternal fasciculus cuneatus (Burdach).
3. Cerebellar tract.
4. Zone of Lissauer.
5. Anterolateral tract, or ascending tract of Gowers.
6. Column of Clarke.

Two Motor Fasciculi

Tracts which undergo descending degeneration:

1. Crossed pyramidal tract. (It is the chief motor tract, and contains ninety per cent of the motor fibers.)
2. Direct pyramidal tract, fasciculus of Turck. (Contains only ten per cent of motor fibers. Division of the direct pyramidal tract produces a diminution of the muscular tonus).

Two Atypical Fasciculi

Tracts which do not undergo either ascending or descending degeneration, or, at least, for no great distance:

1. Anterolateral ground bundle.

Columns	Fasciculus	Trophic Cells of Origin	Termination	Degeneration
1. Anterolateral columns.	1. Direct pyramidal (motor), same side.	Cerebral cortex.	Brush-ends of motor cells of anterior cornu.	Descending.
	2. Crossed pyramidal (motor), same side.	Cerebral cortex.	Brush-ends of motor cells of anterior cornu of same side.	Descending.
	3. Direct cerebellar (fasciculus of equilibrium), same side.	Clarke's column.	Cerebellar cortex of same side.	Ascending.
	4. Anterolateral (Gowers).	Graysubstance of cord of both, same and opposite sides.	Partly in cord, partly in cerebral substance.	Ascending.
	5. Anterolateral ground bundle association, same side.	Gray substance of cord.	Spinal cord.	Limited degeneration, ascending and descending.
2. Posterior columns.	1. Cuneatus (Burdach); sensory, same side.	(*a*) Spinal sensory ganglion (exogene fibers). (*b*) Gray substance (endogene fibers).	Either gray substance of cord or in nucleus cuneatus, whence new fibers originate, cross, and terminate, partly in cerebellum, partly in cerebrum.	Ascending.
	2. Gracilis (Goll); sensory, same side.	Spinal ganglion (exogene fibers).	Same.	Ascending.
	3. Lissauer.	Gray substance of cord (spinal ganglion).	Either in posterior horn or in fasciculus cuneatus.	Ascending degeneration.

2. Short commissural tracts or association neurones.

MEDULLA OBLONGATA

The medulla oblongata is nothing more than the intracranial portion of the spinal cord, or the spreading out of the cord. Its lower limit is a plane passing through the decussation of the pyramids, which corresponds to the foramen magnum; above, it is continued by the pons varolii, and is outlined from the same by the abrupt margin of the pons.

In its lower portion it resembles perfectly the spinal cord. We find the anterior, posterior, and lateral fissures, and the various columns delineated by them. A cross-section in the lower part of the medulla shows all the elements of the spinal cord, with their white and gray substance arranged in a similar manner as in the latter.

On the posterior surface of the bulb, the posterior columns, instead of ascending, diverge so that they form a sharp angle. These are the lower angular limits of the fourth ventricle, its upper limits being formed by the superior cerebral peduncles.

A cross-section of the upper third of the medulla presents an entirely new aspect. The organ is still divisible into two symmetric halves, but they are not separated any longer by the anterior median fissure. The posterior median fissure has also disappeared. The ependymal canal has become enlarged, in order to form the fourth ventricle. The gray substance of the bulb at this level has lost its H-shaped arrangement. Instead of this, we find nuclei, which are small masses of gray substance formed by the decapitation of the anterolateral and posterior horns in the crossing of the motor and sensory fibers. In addition to the neuronic elements transmitted from the spinal cord and cortex, we find the bulb elements proper, which originate here.

Of the seven fasciculi of the spinal cord, three cross in the bulb — the crossed pyramidal tract, fasciculus cuneatus, and fasciculus gracilis. All the others are situated in the bulb, on the same side as in the spinal cord.

A slight restriction must be made as to the direct pyramidal tract, which occupies the same side in the bulb and in the spinal cord; this, however, only to the level at which its axones enter the gray substance of the opposite side of the cord to contact with the lower motor neurones. The axones of the posterior columns terminate in the bulb, in their respective nuclei (nucleus of Goll and Burdach), in end-like brushes, around the cells of these nuclei. From the latter new fibers originate, which shortly after cross those of the opposite side, and they ascend in order to form the fasciculus of Reil. It will be seen, therefore, that the medulla is a continuation of the cord and its tracts. Most of the cranial nerves originate here, and, from a surgical standpoint at least, are practically spinal nerves. We will speak of all these nerves, except those of special sense, as cranial nerves, while those of special sense will be classified as cerebral nerves, for the more positive anatomic and histologic reasons given above.

ZONAL DEGENERATION AND REGENERATION OF SPINAL CORD

(a) Degeneration

In addition to the ascending and descending degeneration, there is traumatic or zonal degeneration. Almost immediately after division, degenerative processes begin, and are completed in a few weeks. At first the myelin sheaths swell, gradually break down, and disintegrate; the axis-cylinder is next involved; the glial tissue proliferates, and replaces both cells and axones with connective tissue until the entire degenerative tissue is finally replaced by connective tissue. This zonal degeneration gives all the evidence of a complete destruction of the ganglionic cells of motion over that particular zone, and in its restricted peripheral zone there is an absence of reflex manifestation; while reflex phenomena remain intact above and below that point.

In cortical lesions — hemiplegia, for example — the degeneration extends through the entire axone of the upper motor neurone down to the motor ganglionic cells in the gray matter of the anterior horn of that particular segment, but there is no degeneration of motor cells in the anterior horn, or of their axones in the peripheral motor nerve.

The following is a detailed description of degenerative processes, as given by Schiefferdecker: Shortly after section of the cord in dogs, there is "a traumatic degeneration," extending upwards and downwards for a distance of six millimeters. In the first few days after division, there is an extravasation of blood, which, later, is absorbed. There are clefts in both white and gray substances, containing shining plates, possibly derived from the swelling of the axis-cylinders; these are absorbed later. After fourteen days, the ends of the cord are firmly united by connective tissue; later, the line of division is barely discernible from the outside. There may be a slight retraction of the dura at the point of division. Schiefferdecker was unable to find, after 238 days, the slightest attempt at regeneration of axones at the point of division. We have, therefore, a perfect, afunctional mechanic reunion.

Enderlen, studying the changes after punctured wounds of the cord of rabbits, mice, and dogs, found, after two hours, swelling of the axis-cylinder, followed by the appearance of fat-droplets, and a progressive degeneration of ganglionic cells, leading on to fatty degeneration. On the second day, there was a beginning proliferative process, in which the glial connective tissue and vessels took part. In the ganglionic cells there was an increase of chromatine, but never a karyokinesis and division. On the fourth day, vessels from the pia penetrated the injured region. The fat-droplets diminished, and newly formed glia-cells appeared in great numbers between the eleventh and twelfth days. From the thirteenth day star-like and spider-like cells increased, forming a dense glial network in which there were still some degenerated nerve-fibers and ganglionic cells.

Stroebe observed about the same changes at the beginning of the injury. After two days he saw a marked proliferation of connective tissue in the pia mater, leading to a formation of connective tissue, rich in cells and vessels, which contained many phagocytes. In the stumps of the divided cord there was a great proliferation of connective tissue.

(b) Regeneration

In the lower forms of life, regeneration of cord was supposed to be possible, as demonstrated by the fact that, after amputation of the tail of a lizard, not only the tail is reproduced, but also the spinal cord. This observation was made by Spallanzani. His findings on the reproduction of the cord in the lizard have been confirmed by many other experimenters, such as Müller, Colucci, Caproso, and Magini. This should not be construed to mean a regeneration of true cord. The caudal zone of the spinal cord of man, also, regenerates, but it is not true cord, as the cauda is composed of axones with a neurilemma which originate from the spinal ganglionic cells in the anterior cornu of the true cord. Even in lizards there is no record of regeneration higher up than the conus of the cord, which supports the position we take, *that regeneration of cord proper, that is, above the cauda, has never been observed and will not take place.*

Regeneration of the cord in the tail region of *Lacerta viridis* is less complete than in that of *Triton*. Some doubt the regeneration of nerve-cells in the former.

Sgobbo Francesco has observed, in the lizard, regeneration of the cord in the dorsal region after division. The degeneration which apparently originated in the epithelium of the ependyma was not so marked in the cicatrix of the dorsal region of the tail. These remarks evidently refer to histologic appearance, and not to function, which is the point that concerns us as surgeons.

Massius and Vanlair removed one to two centimeters of cord in frogs, and, after a few months, found that the absent portion was replaced by a yellow gelatinous mass, which they considered to be composed of ganglion-cells and Remak's fibers. It is stated that there was also restoration of function. This is inserted as a direct contradiction to the results of so many other experimentors, but it is explained by the fact that the portion of the cord removed from the frog must have been the "caudal zone," and this zone in man has also the power of regeneration, as it is made up of neurilemmic axones. From our personal examination of the spinal cord of frogs we can say that the caudal zone extends very high up, almost to the lower angle of the scapula; therefore a division of the spinal cord a little below the middle of the body will involve only the caudal zone, and not the true cord.

In spite of these observations on the frog, Sgobbo Francesco was unable to notice any karyokinesis or other evidence of nerve regeneration at the point of transverse section of the cord.

Voit, Duval, and others support the theory of regeneration of neurones. They observed, in cases of removal of the brain in pigeons, that a mass similar to brain substance appeared in the skull after a certain length of time. This, however, must not be interpreted as a physiological genetic material.

Brown-Séquard found that, after complete tranverse division of the cord in pigeons, there was apparent restoration of function. The ends of the cord were physically united, and in the cicatrix, ganglion-cells and nerve-fibers developed. This, we believe, was only an apparent restoration, as the movements proved to be only reflex, exactly what they should be, and are, after division of cord in man, and is evidence against rather than in favor of regeneration. His experiments were contradicted by Picolo, Santa, Selina, and Francesco.

Regeneration of nerve-cells in dogs has been described by Danten, Massius, Baer, Dawson, and Marshall. Eichhorst and Naunyn have described penetration of the cicatrix by nerve-fibers "without regeneration of nerve-cells." Westfall and Schiefferdecker deny these findings.

Experiments on dogs by Danten, Marinesco, Sgobbo Francesco, Eichhorst, and Schiefferdecker may be summarized as follows:

(*a*) Transverse hemisection of the cord was sometimes followed by restoration of function without the appearance of new fibers in the cicatrix. *The function was due to the impulse traveling along the fibers in the uninjured half of the cord.* This would explain, we believe, the erroneous statements in the reports of clinical cases, as the direct pyramidal tract is not interfered with in hemisection, and it supplies about ten per cent of motion below the line of division on the divided side.

(*b*) Complete transverse section of the cord was followed by apparent limited restoration of function. The cicatrix showed nerve-fibers, although there was no evidence to prove that the cut fibers were united end to end (Eichhorst). These experiments, however, show that apparent restoration was none other than reflex action, and if Massius and Vanlair had demonstrated that reflex action existed after exsection of the frog's cord, and also between that and the time of supposed restoration, then we should have proof positive that the excised portion was not the caudal cord, but true cord, as the cauda has no ganglion-cells for reflex action. This, however, was not done.

Contrary to these findings, Schiefferdecker, Francesco, Piccolo, Santa, Selina did not find, after transverse section of the cord, either restoration of function or regeneration of new fibers in the cicatrix. These and many other experiments prove that the cord of the superior vertebrates, which includes man, is not capable of regeneration after its division or destruction.

Stroebe, however, observed regeneration in the posterior root, the axones advancing from the spinal ganglia to the cord, and even penetrating the connective-tissue scar of the cord. Furthermore, Stroebe saw, at the seat of the degeneration of the cord, new fine axones endeavoring to penetrate the scar. He mentions the fact, without saying definitely that these fibers were newly formed neuronal substance. Even if this is the case, he thinks the connective tissue interferes with the transmission of impulses, so that his results may have been histologically positive, but, functionally, they were negative.

Marinesco, who, once, was among those who denied the possibility of histologic regeneration of axones at the seat of degeneration after division of cord, in a recent article in the *Semaine médicale* for April, 1906, confesses that his previous statements were not quite correct. He has made some very beautiful and instructive observations on the regeneration of nerve-roots and spinal cord. He showed that the reason why he and the others did not find any regenerated axones at the seat of division was because of the improper staining methods. He states that the stains previously used had no affinity whatsoever for the axis-cylinder. By using the special stain of Ramón y Cajal for axis-cylinder, he found that there was an effort at regeneration of the spinal cord axones from the ganglionic side, and that these embryonic axones penetrated for a short distance into the new connective tissue, and then ceased. He concludes that it is this new cicatricial tissue which prevents the advancement of the axones. This, we believe, is erroneous. Practically all cells make an effort at reproduction from the nuclear or trophic remnants, but they span only very limited distances. In spinal axone regeneration the new axone from the proximal side must penetrate not only the new glial connective tissue, but the entire degenerated axones as well. There are no new embryonic axones in the spinal tracts, as the spinal axones have no neurilemma to produce them, differing from the peripheral nerves in this vital particular.

According to Ludwig, Tirelli, and others, karyokinesis is not possible in the spinal nerve-cells of higher vertebrates after the stage of development at which the dorsal white columns are sufficiently formed to produce a distinct median fissure in the cord.

In the human subject, regeneration of the degenerated fibers of the cord has not as yet been demonstrated, so far as function is concerned. The nerve-roots outside of the cord, however, regenerate in the same way as the peripheric nerves. Weissman's *dictum* can be properly inserted here: "Ganglionic nerve-cells, once destroyed in the gray matter of the cord, are never reproduced or replaced." We may add that ganglionic cell-bodies and aneurilemmic axones are never functionally reproduced. This includes all ganglionic cells, the axones of nerves of special sense, and the axones of the white column of the cord.

Lewellys F. Barker says: "Regeneration of severed nerve-fibers within the spinal cord and brain is, unfortunately, very much less complete than in the peripheral nerves." (*The Nervous System*, p. 246).

DEGENERATION AND REGENERATION OF MOTOR AND SENSORY ROOTS

Anterior Root

The division of an anterior root is followed by degeneration of the entire peripheral segment and a small portion of the central segment, generally not extending beyond the first node of Ranvier. The trophic ganglionic cells of the respective fibers are situated in the gray matter of the anterior horn. They suffer, more or less, great changes, according to some authorities (van Gehuchten and Nelis). Others, however, believe that "chromolysis" takes place after the division of a cranial nerve only, while after division of a spinal nerve there is no chromolysis of the ganglionic cells of the spinal cord.

Whether partisans of "peripheral or central school," all authorities agree that if the ends of a divided motor root are united, restoration of function takes place. This is in strict accordance with the histology and minute anatomy; the axones of the motor nerves are neurilemmic from the time they leave the cord, and should therefore regenerate completely if accurate aseptic apposition is made.

All these statements refer to fibers of the motor roots outside the cord; that is, after they leave the lateral fissure. If, however, the motor fibers are divided within the spinal cord, then *restoration never takes place.*

Posterior Root

If a posterior root is divided between a spinal ganglion and spinal cord, the fibers degenerate proximally or cordward from the line of section. In the cord there is a degeneration at the level of division. Below this level there are few degenerated fibers in the so-called cornua tract. Above the level there is a degeneration in the external portion of the Burdach column, while higher up the degeneration takes place in the internal portion of Burdach's column and the external portion of Goll's. Still higher, the degenerated fibers are confined exclusively to the fasciculus gracilis, whereas the cuneatus is free from degeneration. These upper axones which do not degenerate are association axones and axones from higher ganglionic cells, as seen from our analysis of the anatomy of the posterior columns, and should not degenerate, as they are not separated from their respective trophic cell-bodies.

William G. Spiller and Charles H. Frazier performed some experiments on the regeneration of spinal roots. Their results point to the conclusion that after section of the cord the intramedullary roots were permanently degenerated, as no newly formed fibers could be found. No regeneration took place within ten months, and the authors say, "We may assume that it would not have occurred had this dog lived much longer."

Stroebe, after injuring the spinal cord and some of the roots, observed a few fibers penetrating the scar tissue of the spinal cord. Biekeles noted similar phenomena in man, but remarked that the penetration was very imperfect. His observations corresponded with those of Kahler, in that the extramedullary roots are capable of regeneration, while the intramedullary ones, regardless of favorable conditions, are not capable of restoration. The maximum regenerative potency of the axone is limited to a very short distance, probably the length of a dendrite. This has never been experimentally demonstrated, but, from the anatomy and from the functional restoration of peripheral nerves, as well as from the observations made by Biekeles on the divided end of spinal cord, seems plausible. The potency of regeneration is controlled or governed by the regenerative energy which the axone receives from the ganglionic cell-body, and is analogous to the limited distances spanned in regeneration of bone, muscle, tendon, etc. There is no more striking illustration of the relation between the size of the ganglion and nerve regenerative potency than that furnished us in both the clinical and experimental operations on the trifacial. The axones of the Gasserian ganglion — the largest ganglion in the body — will penetrate an enormous amount of connective tissue, and even circumscribe foreign bodies, in their efforts to reunite with the peripheral sensory bulbs. These observations explain the frequency of recurrences of trifacial neuralgias after nerve division, root resection, and chemical necrosis. The period of time for reapproximation or re-establishment of the axone is expressed in the period of relief obtained after the operation.

HISTORICAL DATA CONCERNING SURGERY OF THE SPINAL CORD

Paulus Ægineta, in the seventh century, was the first to advise operation for fractures of the spine, but it was not until the eighteenth century, when Paré and Heister recommended operative treatment, that the subject received serious attention.

The latter recommended extraction of the fragments in cases of fracture, where the depressed fragments compressed the cord.

The first operation for fracture of the spine was performed by Louis in 1762, but the first attempt worthy of consideration was made by Cline in 1814.

In 1820, the subject was extensively discussed before the Royal Society of Surgery of London. In 1823, Sir Astley Cooper wrote: "If you could save one life in ten, one in a hundred, by such an operation, it is your duty to attempt; and he who says it should not be attempted is a blockhead."

In the beginning of the nineteenth century, a very earnest debate was carried on between Sir Astley Cooper and Charles Bell, the former a great advocate of surgical intervention, and the latter opposed to it. Brown-Séquard was at first opposed to operation, but later advocated it.

Leyden, in his clinic on spinal cord diseases, advised operation in case of fracture of the arch of the vertebra, and since the successful operation of Macewen, laminectomy has been performed many times. In 1887, Horsley and Gowers first successfully removed a subdural tumor. In 1899, Chipault reported 167 operations for spinal fracture from his personal experience.

SURGERY OF THE SPINAL CORD

Surgical intervention in spinal cord affections has been at all times a matter of hesitation on the part of surgeons. Results were so varied and indefinite that for a long time no conclusions could be drawn as to the advisability and probable outcome of operative treatment. Statistics were meager, and their reliability doubted where favorable results were recorded.

In this paper, we do not intend to consider in detail the entire field of surgery of the spinal cord, or to offer anything particularly new. We will deal mainly with the different types of injuries to the spine and spinal cord, and endeavor to bring out the important points in diagnosis and definite indications for surgical treatment in cases amenable to it. We also hope to be able to show that certain cases should not be operated on, as they are absolutely hopeless, from our histologic knowledge of spinal cord degeneration. The chapters to be covered are:

(*a*) Contusion.

(*b*) Concussion.

(*c*) Punctured wounds of the cord.

(*d*) Hemorrhage or clot in spinal canal and cord.

(*e*) Dislocations and fractures: 1. Contusion of the cord; 2. Compression without division; 3. Partial division of the cord; 4. Total division of the cord.

(*f*) Gunshot wounds.

(*g*) Tuberculosis of the spine and spinal cord.

(*h*) Tumors.

(*i*) Spina bifida.

Contusions

Contusions of the spine may be of various degrees. They may affect the superficial tissues, as the muscles and ligaments, cause an effusion of blood into the spinal canal or cord, or cause traumatic zonal inflammation, *with paralysis without division* of the axones. The symptoms following a contusion to the spine may be widely at variance with the apparent degree of trauma, and a classic clinical picture would be impossible. In many of the cases of contusion to the spine, even in those of a severe character, we may be surprised to find no symptoms of cord injury at the beginning. I have had several cases under my personal observation in which no initial symptoms were present, but in which, in a short time, varying from a few days to several weeks, a traumatic reaction set in, and the patient developed complete paraplegia of both motion and sensation.

The following cases will illustrate this statement:

On January 20, 1891, a patient, Mr. G. M., aged 23 years, while carrying a carboy of acid across a bridge fell a distance of twelve feet, and struck on his buttocks. He felt some pain after the fall, but was able to resume his work in a few hours. For three days following the injury he was able to walk and work. On the fourth day, pain in his back obliged him to discontinue work. From that time on, weakness and incoordination of the limbs gradually developed, although he was able to walk with the assistance of crutches until February 10th. At this time, upon examination, I found that the spine was sensitive to pressure at the eleventh lumbar junction, but no fracture could be detected. There was no numbness of the limbs, but incoordination of motion. February 27th, his paralysis was complete below the level of the dorso-lumbar junction, with incontinence of fæces and retention of urine. This continued for three months. At the end of this time, sensation began to return. There was partial restoration of motion at the end of four months. In June of the same year, patient was again able to walk with the assistance of crutches, and finally, at the end of fourteen months, motion and sensation were completely restored.

This case illustrates two points: 1. The absence of initial evidence of injury to the cord and development of paralysis five weeks later; 2. Complete restoration of function, without surgical intervention, after fourteen months.

Cicero B., aged 59, farmer. Admitted to Mercy Hospital, August 20, 1906.

History of Present Trouble. October 7, 1900, patient was working in the field, plowing potatoes. As the plow ran over a little hill, it suddenly turned over to the right. Patient fell on his right side with the plow, but was not hurt by it. He got up without assistance, and was able to resume his work a few minutes after the accident. This happened in the morning. At 3 P. M. of the same day, patient met with a similar accident. This time, however, his feet became tangled up with the reins, while his body fell on the right side. The horses stopped immediately after the plow turned over. With the assistance of a man, patient was able to get up, and resumed his work immediately. After this second fall, he was free from pain and from any other symptom in his lower extremities. He had, however, considerable pain in his back, along the lumbar portion of the spinal column. He continued to handle the plow until 6 P. M., notwithstanding the intense pain in his back. After supper-time of the same day, patient walked to town. There was no difficulty or abnormality of gait; the backache persisted, however, with the same intensity. At 2 A. M. the morning following the accident, patient woke up on account of the pain in his back. He got up, and as soon as he stood on his feet the pain ceased entirely. He walked around the room and the yard for some time, and when he returned and sat down in the rocking-chair, he experienced the same pain as before. Concluding that sitting down brought on the pain, he decided to either walk or stand up.

The day following the accident, he walked to town to consult a physician. The latter diagnosed it as "sprained back," and applied a plaster to the lumbar region of the spine. Returning from town, he was practically free from pain when sitting down. At noon of the same day, patient experienced numbness in his feet. At 3 P. M. he lay down, and at 5 P. M., while endeavoring to get up, he noticed a considerable heaviness and numbness in his lower extremities. With some difficulty, he got up and stood on his feet for a short time. The limbs, however, were unable to support the weight of his body. With some assistance he stood up for a short time. At 8 P. M., 29 hours after the last accident, the limbs were completely paralyzed and unable to support any weight at all. At 11 P. M. of the second night, patient recollected that he had not urinated for 15 hours. At midnight he was catheterized, and a considerable quantity of urine extracted.

October 9th, patient was in bed with an almost complete paralysis of the lower extremities. This, however, of motion only. He was able to move his toes very little for a few days. October 11th, there was complete paralysis of motion of both lower extremities. The first three days after the accident, patient was constipated, and purgatives were ineffective. At the end of the third day, he had a bowel movement. Ever since, he uses an enema every morning. Whenever the fæces are liquid, patient is unable to retain them. For a year and a half, patient made use of the catheter, not because he was unable to pass the urine, but simply because he wanted to avoid the annoyance caused by its incontinence. In the past four and a half years, patient has not used the catheter, and is able to urinate voluntarily. Involuntary micturition was rather an exception during this period of time.

After two months of complete paralysis of motion, he commenced to move the toe of his right foot. There was slow but gradual return of motion in the muscles of the right foot, leg, and thigh, and at the end of one year he was able to flex, extend, etc., his right lower limb. Yet he was unable to support the weight of his body. There was no improvement whatsoever in the left lower limb. In September of 1905, patient used braces, and with the assistance of crutches he was able to move about, but states that his legs were unable to support him. In December, one of the braces broke, and patient fell on his left hip. Ever since then he has had considerable pain in that hip, especially when he is moved from one side to the other.

The backache, which was experienced soon after the accident, covered for a few days a large area in the lumbar region. The same localized on one point on the middle line of the back, which has remained painful all of these six years. The pain was neither exaggerated nor relieved by change of position. Of late, has, in addition to the pain mentioned, pain on the side of the lumbar column and about one inch from it. At various times he used faradic currents without avail. Patient's general health was good.

Previous Illnesses. For years, patient had backache when working. "The weakness of the back," as patient chooses to call it, was especially marked before the accident. In infancy he had measles, whooping cough, and a few other similar diseases, which he cannot definitely remember. As a young man, he was always very healthy. Denies gonorrhœa and syphilis.

His habits have been good. Drank alcohol occasionally, but never to excess. He was married for years. Has four children living; one died in infancy. His wife had no abortions. She died two years ago.

Family History. Negative.

Examination of Patient. Patient is tall, stout, and very well nourished. His estimated weight is about two hundred pounds. Pupils react to light and distance. Mentally, normal. Thoracic and abdominal organs, negative.

Examination of Lower Half of Abdomen and Lower Extremities. 1. *Sensibility* — Right Side: Beginning with a line situated four fingers above the umbilicus and circular around the body to the spinal column, and up to the tip of the toes, the condition of the sensation is sensibility to light touch, tested by light wool, is absent in about ninety-five per cent of the cutaneous area. In a few scattered, irregular areas, the light touch of wool is barely noticed. Sensibility to prick is present, yet considerably diminished as compared with the left side. Above the circular line described, there is hyperæsthesia. Sensibility to pain is present, but it has the same character as sensibility to prick. Caloric sensibility. Patient cannot discriminate between heat and cold. The application of hot water for five minutes does not give any other sensation than that of touch. Left Side: Sensibility to prick, light touch, pain, heat and cold, are all present, and exaggerated to a certain degree. This is especially true about sensibility to prick and that of extremes of heat and cold. 2. *Motion* — Right Limb: There is no visible muscular atrophy. The tonus of the muscles is decidedly diminished. He is able to flex, extend, abduct, adduct, and rotate the foot, leg, and thigh. The movements are, however, slow and hesitating. The limb is unable to support the weight of the body. Left Limb: There is a complete paralysis of motion in this limb, except a very insignificant excursion of the toes. The paralysis is of the flaccid type; there is a visible muscular atrophy.

Superficial and Deep Reflexes. Tickling or pricking the right plantar region produced a sudden reflection of the limb, also a slight flexion of four toes, and an extension of the great toe. The latter, however, is not marked. The same action in the left plantar region produces the same sudden retraction, flexion of four toes, and a very pronounced extension of the great toe (positive Babinski). Upon pricking the plantar region three or four times, the leg suddenly retracts and enters into a spasmodic condition which lasts a fraction of a minute.

The patellar reflexes are greatly exaggerated on both sides.

Muscular Sense. Only very slightly impaired. It can be said that it is practically normal.

No ankle clonus can be produced in the right leg. If the patient touches the ground with his left toes, then the ankle clonus is marked in this leg. Babinski's sign is very pronounced in the left leg.

Response to Electricity. Right Limb: All the muscles of the anterior and posterior region of the thigh, all of the muscles of the leg, respond to faradic currents. While the extensors of the foot proper do not seem to be powerful enough, yet they respond fairly well on faradic stimulation. As a whole, however, the response to electric stimuli is not so quick as in another group of healthy muscles. Left Limb: The muscles of the thigh do not respond to faradic stimulation. There is a very slight fibrillary contraction on the anterior aspect of the quadriceps. The muscles have absolutely no potency of contraction. Muscles of the posterior region of the thigh are in the same condition. The extensors of the leg, the peroneal group of muscles, the muscles of the posterior region of the leg, show a slight response to faradic stimulation. This is similar to what would happen if the crossed pyramidal tract were divided and the muscles were under control of the direct pyramidal tract only. Occasionally, application of the electrode fails to produce contraction in the same muscles which previously responded to stimulation. Again, the application of electrodes may produce three or four successive reflex-like contractions. In one instance, the leg entered into a spasmodic contraction after the application of the electrode, and the spasm continued even after the removal of the electrode. Once, the application of a strong current elevated the leg above the plane of the table; a movement which patient had not been able to make for the past six years.

Diagnosis. Traumatic zonal paralysis of extreme severity, with possible destruction of the left half of the cord, but, more likely, one of extreme inflammatory deposit in the left half and a lesion in the right.

In such cases, while there is no gross anatomic change in the spinal cord and its membranes, we are convinced that some changes must have taken place later in its vessels and neuroglia, which interrupted the transmission of impulses. Cases of this type have been clinically interpreted as destruction of spinal cord with regeneration. They do not deserve such interpretation. They are simply zonal paralyses which are produced without division of the cord or axones, and are completely restored after the inflammatory condition has subsided and the products of the inflammation have been removed. There is no regeneration of spinal axones. The observation of Schmauss — namely, œdema and swelling of the ganglionic cells and of axones in the traumatized cord of rabbits — does not necessarily mean degeneration; we interpret it as an atrophy, as shown by the frequent recoveries in similar cases in man. These have proven the truth of this assumption. Several observers have noted cellular changes in the gray matter and neuroglia of the cord, after applying great force to the spinal column, insufficient, however, to produce fracture or dislocation. Following laminectomies, we frequently have complete paraplegia with full restoration of function, illustrating operative zonal inflammatory reaction.

An insignificant trauma to the spine may cause marked paralysis.

August 18, 1886, I had under my care a patient, Mr. K., who, while sitting in a hammock, tipped backward a distance of two feet to the ground. Immediately after the accident, he developed a paralysis of motion and a numbness of sensation in both the upper and lower extremities. There was no special deformity or displacement, but great sensitiveness at the fifth cervical spinous process. In November of the same year, the patient was able to walk, though hesitatingly, and had still a numbness of sensation in the previously paralyzed members. In May, 1887, he was still coming to my office. At the same time he was practically well, except for some unsteadiness of gait and uncertainty of equilibrium when he turned suddenly. The index and middle fingers of the right hand were the last to recover.

This case is a good illustration of the traumatic transient type of zonal paralysis.

Concussions

By spinal concussion is meant an impairment or loss of function of spinal cord due to injury which is not sufficient to produce gross anatomic changes. The term "spinal concussion" is accepted by some surgeons and rejected by others. While we admit that spinal concussion is a temporary reaction on the part of the cord, yet we do not consider it as a definite morbid entity; but we do not accept as spinal concussion cases other than those with symptoms of pronounced zonal hyperæsthesia or anæsthesia and with a zone of diminished reflex, or paralysis partial or complete. Those who deny spinal concussion base their views on anatomic grounds; namely, the small size of the cord, as compared with the spinal canal, and its suspension in the canal by the denticulate ligaments and nerve-roots which protect it from injury in traumas short of fracture or dislocation. In this connection we must mention that hemorrhage in the spinal canal may produce an exact clinical picture of spinal concussion. Therefore, differential diagnosis between these two conditions is quite impossible. There are several cases on record, however, where the disappearance of paralytic symptoms in a short time point to the probability of spinal concussion. On the other hand, many post-mortem examinations have not shown any anatomic changes in cases where marked paralysis following injury was noticed, and continued up to the time of death. The following classical case is recorded in one of the Guy's Hospital Reports:

A woman, 59 years of age, fell on her back and immediately developed marked paralysis of the upper and lower extremities. On the tenth day, she died of pneumonia. A careful post-mortem examination was made, and no anatomical changes were found, either in the brain or cord. There was no evidence of effusion of blood in the spinal cord or canal.

We have had, in our experience, several cases of injury to the spine in which physical examination did not reveal evidence of dislocation or fractures, although the patients developed marked paralytic symptoms. In all of these cases perfect recovery took place in from six to twenty months.

Punctured Wounds of the Cord

The knife is usually responsible for such wounds. The cord may be injured without fracture to the vertebræ. Wagner-Stolper has collected 86 cases of punctured wounds of the cord. Of these, more than half occupy the cervical portion and the remainder the upper region of the dorsal. There was no case of puncture of the lumbar cord. The cause of the cord being punctured more often in the upper part is very simple. The object of an assailant is generally the head, and anatomically there is a considerable interspace between the vertebræ in this region for the admission of the knife-blade. As a rule, such wounds involve one half the cord, though sometimes the knife may pass beyond the median line and injure the other side. The clinical picture of punctured wounds of the cord is that of a hemisection lesion. Out of Wagner-Stolper's 86 cases, 44 presented the picture of hemisection.

The symptoms appeared immediately after the injury, and the course was generally an aseptic one. Twenty per cent of the cases mentioned by the above author died. The rest of the cases improved; but few recovered. So far as treatment is concerned, very little can be done beyond rendering the external wound aseptic, and keeping it so. Operation is never indicated except where infection of the spinal canal has occurred, or when there is compression from hemorrhage. If this is manifested early, it can be relieved by a lumbar puncture with a large needle. The divided axones of the cord do not regenerate, as they have no neurilemma, and suture of the severed segments is not only useless, but adds to the danger. Such paralysis as results from effusion or traumatic zonal inflammation will subside without operation. Therefore these cases all contra-indicate laminectomy.

Hemorrhage — Effusion of Blood in the Dura and Spinal Cord

The blood may occupy the spinal canal, extradural, may be beneath the meninges, or may be in the substance of the cord itself. In the second case, it is called hæmatorachis; in the third, hæmatomyelia. The most important ætiologic factor of hæmatorachis and hæmatomyelia is trauma from a blow, fall, gunshot, or stab wound of the spinal canal. The symptomatology of hæmatorachis is not definite. Patients usually complain of pain in the spine, paroxysmal and burning in character, along the nerves, and occasionally muscular spasms, opisthotonos, convulsions, and finally paraplegia. While the paraplegia is sudden and complete in fractures and lesser injuries to the spine, in hæmatorachis it is gradual and progressive, often developing two to four days after the injury. The above-mentioned symptoms of hæmatorachis usually develop in the first twenty-four hours, increase in severity up to the fourth day, and disappear in from four to six weeks. There are, however, cases in which the paraplegia is sudden and complete, depending upon the size of the vessel, as in a case of mine of bullet wound of the cervical region, and one of Edward Janeway.

The best test for hemorrhage (in fractures, bullet wounds, etc.) is spinal puncture. Twenty to thirty cubic centimeters of fluid may be drawn off. If the cord is injured above the spinal puncture, then the spinal fluid may be bloody.

The hemorrhage may be extradural or intradural, or combined. When the dura is lacerated, hemorrhage is usually due to rupture of the venous plexus, and rarely to rupture of arteries. The blood has a tendency to extend downward, and therefore the hemorrhage is more extensive if the lesion is situated high up. As the lumen of the spinal canal is large, the quantity of blood is considerable. Diagnosis of intradural hemorrhage can readily be made by lumbar puncture, and the pressure of the cord relieved by this procedure if the puncture is made early.

While hemorrhage is generally due to injury, occasionally it may be non-traumatic in origin. Edward Janeway, for example, reports a case of spontaneous subarachnoid hemorrhage in a bleeder. The young man, after having recovered from a slight attack of influenza, suddenly lost sensation and motion in the lower extremities, and fell to the floor. The autopsy revealed extensive hemorrhage in the subarachnoid space up to the middle cervical region; there was also some blood between the dura and the arachnoid. Patient's brother was also a bleeder.

Hemorrhage into the spinal canal may produce marked paralytic symptoms, even to complete paraplegia. In such cases, diagnosis cannot easily be established, as illustrated by the following case from my practice in Cook County Hospital in 1887:

A man was shot in the neck. The bullet passed externally to the jugular vein just above the clavicle. He said he did not fall immediately after this wound. A second bullet passed through the shoulder and hand, when he fell to the floor. He was taken to the hospital shortly after the accident. There was complete paralysis of the upper and lower extremities. He was able to move the muscles of the neck only. The diaphragm acted on both sides. It was concluded that the patient's statement was wrong, and in all probability the first bullet entered the shoulder and hand, while the second one struck and divided the cervical region of the cord. He died on the third day. A post-mortem showed that the cord was not injured by the bullet. The meninges were divided and the intravertebral artery was severed, which permitted an extensive hemorrhage within the dura; this was filled with blood from the occiput to the cauda. In all probability, the man's life could have been saved if the tension had been relieved by immediate tapping of the spinal canal in the lumbar region. From the post-mortem findings it was evident that the first bullet entered the neck, as was stated by the patient, and the second the shoulder and hand, and that his fall was due to the blood-pressure on the spinal cord from the hemorrhage in the canal.

Hæmatomyelia. Hemorrhage into the substance of the cord usually occurs in the region of the fourth, fifth, and sixth cervical vertebræ. According to Thorburn, the hemorrhage in that region is due to overflexion of the spine. If the hemorrhage is confined to the gray substance only, there is wasting of muscle and anæsthesia of the upper limbs; also zonal absence of reflex. If the hemorrhage involves the white substance, we have paraplegia below the level of the lesion. Prognosis must be guarded in cases of hæmatomyelia. Even in cases of recovery, there is always a weakness left, due to the destruction of the ganglion-cells of the motor neurone at the point of hemorrhage. Hæmatomyelia may be due to gliosis spinalis. A patient of von Bergmann, a girl 19 years of age, died from this condition, as shown by the post-mortem examination.

Dislocations and Fractures

Dislocations. A dislocation without fracture practically never occurs. Rarely, we find luxation alone of the upper cervical segment, but, as a rule, dislocation is associated with fractures, and the symptoms are then those of compression. Recovery is possible, if the cord is not divided and the compression is relieved early. The subject will therefore be treated under the head of fractures, and included with them will be associated luxations.

Fractures. Fractures may be due to direct or indirect causes, as, for instance, forced flexion. The direct force may fracture a vertebra, but, as a rule, the fragments are not displaced. The laminæ and spinous processes may be driven into the spinal canal and cut off and compress the cord. This, however, is a rare type of accident, and readily diagnosed. The bony fragments do not compress or lacerate the cord, at least primarily. The following history is a striking illustration of the exception to this rule:

Tony Y., aged 38, Italian, laborer. Admitted to Presbyterian Hospital, May 3, 1906; died May 7, 1906.

History of Present Trouble. April 27th, while attempting to get off a moving train, he fell or was thrown off, and was unconscious for fifteen minutes. On regaining consciousness, he was unable to move the lower extremities, and complained of severe pain in the back. Has had retention of urine since that time, and it has been necessary to catheterize him. Patient does not know how he fell at the time of the accident.

Physician's Examination. Man a laborer of fair muscular development. Head, neck, and chest negative, except congestion of the lower lobes. Abdomen negative; back, marked hæmatoma over lower lumbar region. Vertebral column shows a pronounced angle at level of third lumbar; the lower fragment is displaced forward for about an inch and a half. A superficial abrasion is present at this point. The displacement cannot be reduced by manipulation or extension.

Extremities: Left foot shows a hæmatoma on internal aspect of heel. There is a complete paralysis of sensation and motion below the second lumbar zone.

Reflexes: Abdominal, present; cremasteric, abolished; patellar, absent; Babinski, absent.

Diagnosis. Fracture of spine, with anterior displacement of the lower segment; division of the caudal fasciculi.

Operation. May 3, 1906. Exposure of fractured zone by single incision. It was found that the arch of the second lumbar vertebra was driven in and compressed the cauda. The third and remaining vertebræ below were displaced forward; the arches of the third and fourth and the body of the third were fractured, and the spinous processes driven down on the cord. Almost all of the caudal filaments were divided by the great displacement forward of the lower segment. The second, third, and fourth spinous processes and laminæ were entirely removed, the dura opened for a considerable distance by a longitudinal incision and the fasciculi picked up, and the divided filaments united by end-to-end suture. The right and left halves of the proximal ends were easily determined by their position. The distal ends were considerably entangled, but the right and left could be differentiated by applying the faradic excitor to the filaments. This brought a muscular contraction in the respective muscles, notwithstanding that four complete days had elapsed since the division. When applied proximal to the contusion, there was no muscular response. This muscular response, after division, corresponds to that obtained in our examinations of divided peripheral nerves the same length of time after their separation. After completing the approximations, the dura was sutured in position, and

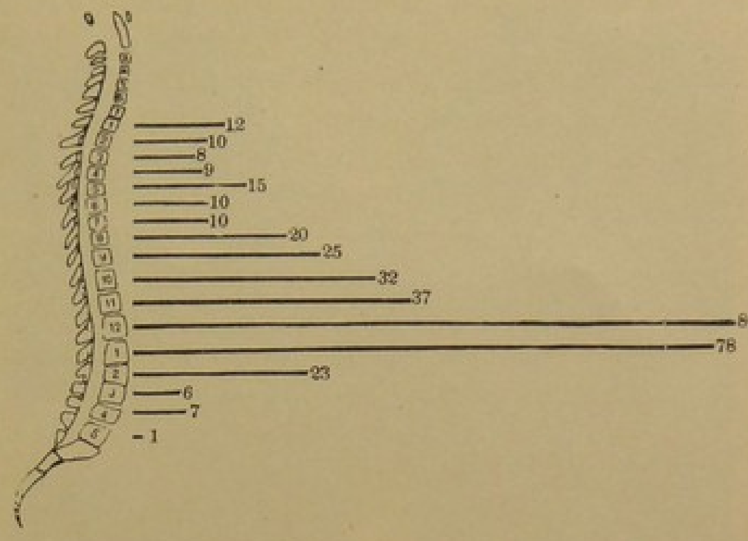


Fig. 11. Frequency of fracture in regions of spine. (Mènard).

a muscle-flap drawn from each side, filling the gap to the skin level. The patient withstood the operation fairly well, although his breathing was noticeably more rapid than at the beginning. From these physical findings we conclude that the man was struck in the back with great force, which first fractured the spinous process and laminæ of three of the vertebræ, and then carried the lower segment forward for a full inch.

May 4th, the day following the operation, the patient was restless throughout the night, and his respiration increased to 56. His pulse ranged from 118 to 132; temperature 101.6°.

May 5th, patient delirious; had rather a restless night; respiration 64; some hypostatic consolidation of both lower lobes with râles extending up to the spines of the scapulæ behind. Temperature 102.4°; pulse 124 to 140; urinary incontinence; bladder quite distended. From this time on, patient's pulmonary manifestations were more and more pronounced. Temperature rose to 104.6°. He died on the morning of the 7th, apparently from a double pneumonia. No post-mortem.

Indirect force may produce fracture and displace the fragments of the vertebræ, so as either to compress or divide the cord; as, falls on the buttocks or shoulders, or extreme flexions or extensions of the body. In these cases the site of fracture is usually at the dorso-lumbar junction. Where the paralysis is immediate, uniformly transverse and complete, of motion and sensation, there is no hope of recovery, with or without operation, as the spinal cord is divided. Surgical intervention is of value only in cases where the fragments press upon the cord without having caused complete division. None of our cases (and there were many operated on, in our twenty years' service in the Cook County and Alexian Brothers hospitals), in

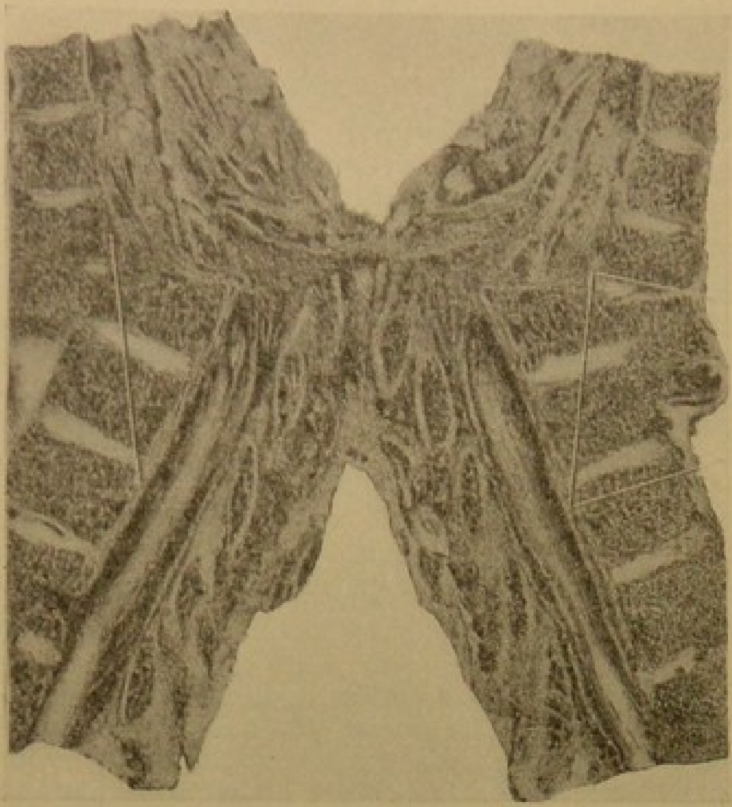


Fig. 12. Severe fracture of the upper dorsal spine in a woman 25 years old. (*Handbuch der Chirurgie.* Von Bergmann, Bruns, Mikulicz).

which the cord was divided by a fragment, recovered. The degree of pressure on the spinal cord cannot easily be determined, and it is difficult to tell at the beginning how much pressure is being exerted on the intact cord. One can fairly accurately estimate when the cord is completely divided, as then there is a *complete immediate annular paralysis of both motion and sensation* below the line of fracture, which cases are beyond repair.

Fracture with contusion of the cord may give a motor or sensory paralysis below the line of union. The paralysis is rarely immediate, and is never complete and annular of both motion and sensation. The irregularities indicate that some columns of the cord are still intact; usually this paralysis comes on hours, days, or weeks after the trauma. It is preceded by a zone of pronounced hyperæsthesia, and is accompanied by abolished reflexes in this zone. The motor or sensory manifestations will predominate, depending upon whether the anterior or posterior columns of the cord have suffered most. Compression may be associated with contusion. There is no known means of making differential diagnosis of continued compression in addition to contusion, the degree of displacement of spinous processes being the only physical manifestation which we have for suspecting compression in addition to contusion. Both may have the same degree of primary paralysis; both may have the same speedy or slow increase in the paralysis; both may have a stationary degree of paralysis for a considerable period of time; both may have a late manifestation of paralysis, as shown in the cases cited under "Contusions." Therefore differential diagnosis is impossible without laminectomy, which, however, is a procedure to be avoided, as a contused cord suffers additional insult by every operation, no matter how delicately performed. However, where compression is suspected on account of the great displacement, one must not hesitate, but should operate immediately.

Partial divisions of the cord give a symptom-complex resembling contusion, in that there is an irregularity in the transverse line of

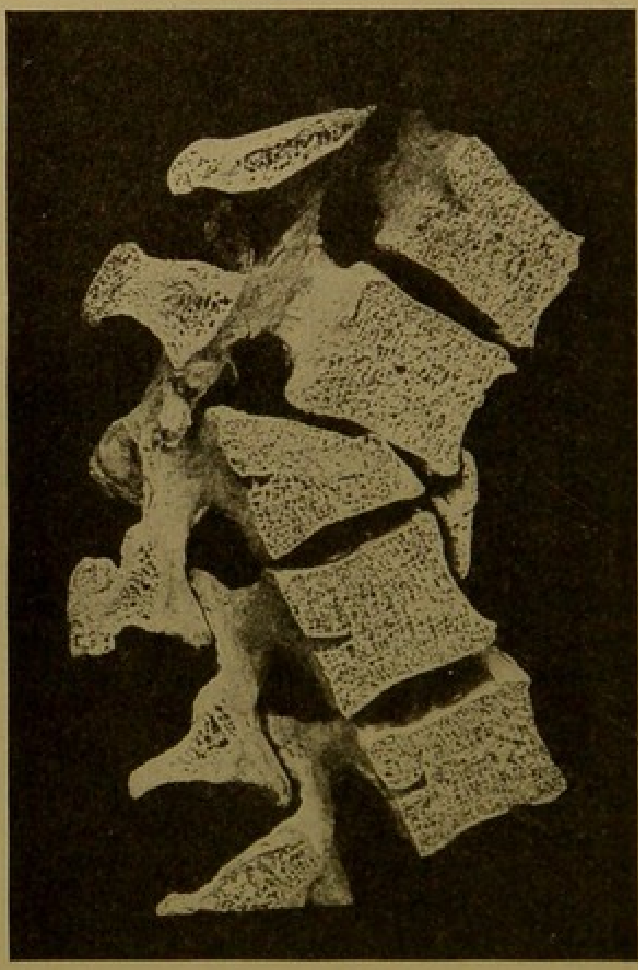


Fig. 13. Showing fracture of the first lumbar vertebra, with displacement forward of the upper segment of the spine (Guy's Hospital Museum). (From *International Text-book of Surgery*).

paralysis, and either sensation or motion may be present to a limited degree. Operation is contraindicated, except where there is suspected continuous compression. Dr. Walton says: "We have no symptoms from which we can assert from the outset that the cord is crushed beyond at least a certain degree of repair. It has been said that where there is complete loss of sensation, motion, and patellar reflexes, the cord is completely crushed, and, consequently, recovery cannot be expected." It is stated in the same article, that the patellar reflexes may be present in a case of complete transverse destruction of a segment of the cord. This is possible, as the ganglionic cells of the motor neurone below the point of division may be in perfect condition, though their contact axones of the central neurones are divided.

In fractures with division of the true cord, operation with suture of the cord is absolutely worthless, as we have shown above that functional regeneration of the columns or gray matter never takes place. The improvements recorded by many authors were in cases in which only contusion or hemorrhagic compression of the cord existed, or where the type of traumatic zonal paralysis was present, which would have improved as well, if not better, without operation. Laminectomy in the dorsal and cervical regions is, therefore, never indicated for division of the cord.

When the fracture occurs in or below the twelfth dorsal, the treatment and prognosis are entirely altered. The cauda equina, which begins here, is made up of essentially peripheral nerve fasciculi, and not of spinal cord fasciculi, as the axones of the motor root in this portion have their ganglion trophic cells above this level in the conus, and the motor axones in the cauda are covered with the sheath of Schwann, or neurilemma. They therefore degenerate after division, and have the power of regenerating, the same as peripheral motor axones. The sensory neurones of the posterior roots of the cauda have their ganglion-cells just inside the sacral and lumbar foraminæ. Their proximal axones, which run through the cauda to the spinal cord, are medullated, and have a sheath of Schwann. They are capable of regeneration, at least up to the posterior commissures, and, from clinical observation, we believe, can again functionally contact with the posterior horn of gray matter. In other words, both the motor and sensory neurones in the cauda outside of the cord are histologically capable of regeneration under favorable conditions; that is, after accurate suture and exact approximation of the ends of the divided caudal fasciculi under aseptic conditions. It therefore becomes imperative for us to establish this approximation in every case of fracture of the lumbar spine with division of the caudal fasciculi and paraplegia. It is easy to determine which are the right and left fasciculi by a mild faradic current up to the seventh day after the injury. If the faradic current be applied to the individual nerve bundles *distal* to the trauma up to seven days after division, there is a muscular response in the zone supplied by that bundle, as shown in the case of Tony Y., *ante*. If it be applied, as will be seen in the above reported case of caudal suture, *proximal* to line of division, or even severe contusion, there is no electrical response. This corresponds exactly with the electrical response after division of peripheral spinal nerves external to the bony canal. Laminectomy with suture in this position is not extremely difficult. It has in it, however, the danger of sepsis, as the spinal canal and dura are open.

In every case of fracture, stab, or bullet wound, where the cauda has been supposedly divided, exposure and suture of the fasciculi are positively indicated, the individual points of suture to be surrounded by egg membrane to lessen connective-tissue interposition.

The transposition of caudal fibers in the treatment of unilateral paralysis of the entire lower extremity will be described in the article on peripheral nerve surgery, in its subdivision, "Nerve Anastomosis in Anterior Poliomyelitis."

Gunshot Wounds

Theodore Prewett collected 49 cases of gunshot injuries treated since the antiseptic era. Operated: Recovery, 11; dead, 13. Non-operated: Recovery, 8; dead, 17. Mortality: Cervical region, 55 per cent; dorsal region, 66 per cent; lumbar region, *nil*. Three cases of the latter operated on; all recovered.

Conclusions of Prewett. (*a*) Immediate operation if wound is in accessible region, and if it involves posterior or lateral portions of the spine. (*b*) It is imprudent sometimes to wait for repair to be effected by nature.

In bullet wounds of the spine involving the spinal cord itself, operation is contraindicated, as from our knowledge of the absence of regeneration of the spinal cord proper, operation would be useless. If the cord is not severed, such fibers as are not divided will recover from the zonal paralysis resulting from the contusion and concussion without operation, while the fibers which are divided cannot derive any benefit from surgical procedure. If the bullet remains within the spinal canal and compresses the cord, as the skiagrams will readily show, it should be removed. If the cord is compressed by fragments of bone, which may also be determined by the radiograph, operation is indicated. If the cord be compressed by accumulated blood within the meninges, a lumbar puncture for its relief is indicated. The blood is kept fluid by its intermixture with the cerebrospinal fluid.

Mortality. The causes of mortality from fractures of the cervical region are due to interference with the essential function of the centers of the spinal cord. Death may occur on the third or fourth day as the result of the degree of fracture. In fractures in the neighborhood of the fourth and fifth cervical, the temperature reaches as high as 106°, 107°, 108°, and even higher (as in one of my cases, as high as 111°). This is a central temperature from the destruction of the automatic center of temperature control. Respiration may be compromised from the injury of the phrenic segment of the cord, which corresponds to the fourth and fifth cervical. With fracture in the upper dorsal region, the functions of the abdominal organs are greatly disturbed, and there is a paralysis of peristalsis, and enormous tympany may result; respiration may be impaired by the intra-abdominal pressure upon the diaphragm. In fractures of the spine above the second lumbar, sphincteric control is destroyed. Of the secondary causes of death, the most prominent are vesical infection and ascending sepsis to the kidney. Next in frequency is the decubitus infection. The former can be avoided in a large percentage of the cases by permitting the bladder to overflow from the beginning, and refraining absolutely from using the catheter. If the catheter has once been used and decomposition takes place, it must be continued. Relaxation of the vesical sphincter is favored by transproctеal massage. The decubitus can be avoided or retarded by the water-bed air-cushion, frequent changes of position, and by dermal cleanliness.

The following interesting case is reported by Richard H. Harte and Francis T. Stewart in the *Philadelphia Medical Journal*, June 7, 1902:

Woman, aged 26, was shot twice with a 32-caliber revolver. Operated on by Stewart. One ball was lodged just beneath the skin; the other entered about an inch to the right of the seventh dorsal spine and traversed the spinal canal. Sequence, paralysis of both motion and sensation below the level of a line drawn from the tenth spinous process to a point three and one fourth inches above the umbilicus. The zone of hyperæsthesia manifest just above the line of anæsthesia. Temperature 97.6°; pulse 120; mind clear. Operation three hours after accident. Seventh and eighth dorsal spinous processes and laminæ were removed, and bullet extracted from body of vertebra. Removal of bone fragments and contused cord *débris;* this left a gap between the segments of cord of three fourths of an inch. Wound flushed with salt solution and ends of cord approximated with three chromicized catgut sutures inserted by a small staphylorrhaphy-needle. One was inserted anteroposteriorly and the others transversely. It was impossible to approximate the edges of the lacerated dura. A gauze drain was inserted and retained twenty-four hours. Patient withstood the operation very well; she was in better condition immediately after than preceding the operation.

On the fifth day there was evidence of sensation in the lower extremities. Superficial reflexes returned on the fifth day, and the patellar reflex was manifest on the twenty-first day. There was, at the end of sixteen months, voluntary flexure of toes, flexion and extension of legs and thighs, and also rotation of the legs. While sitting, patient could extend the leg so as to raise it from the floor. Patient able to slide in chair. Bowels moved every other day; perfect control. One pint of urine was evacuated at each of three urinations in twenty-four hours. There was occasional incontinence of urine during sleep. Temperature remained below 100°, except on the fifth day, when it reached 101°.

From the rapid return of sensation it is evident that some fasciculi of the cord were not divided, and from the return of motion, even at the end of sixteen months, there is still more forceful support of the same idea. The primary complete paraplegia was typical of traumatic zonal paralysis without complete division of the cord, with the possible exception of the speedy return of sensation. What portion of the cord was cut it is difficult to say from the report given, but it is evident that it was not completely divided.

These authors advise myelorrhaphy in transverse division of the cord, and state that it is especially indicated in cases in which the cord has been cut by a sharp instrument or severed by a projectile.

Dr. W. L. Estes of Bethlehem, in 1902, reported a similar case in which there were positive signs of some regeneration.

From our knowledge of the histology and from our clinical experience, we feel certain that the restoration of function was misinterpreted as a restoration of regeneration. The former is a uniform result from impaired functions in contused, inflamed spinal axones, while the latter — regeneration of the spinal axones — never takes place.

In the case of Dr. W. Braun (*Berliner Verh. der Deut. Gesellschaft f. Chir.*, 1906, p. 56) we have a striking illustration. A bullet five millimeters in diameter was imbedded in the center of the spinal cord, at the sixth dorsal body, without dividing many of the spinal cord fasciculi, as is shown by the sequence of events. Three weeks after the removal of the bullet, which operation was performed thirty-seven days after the accident, there was return of function in the bladder and rectal control. Paralysis of motion and sensation was complete shortly after the accident, also the reflex action below the seventh dorsal segment. Twenty-three months after the operation, when the case was demonstrated, the patient was able to walk quite a distance with the assistance of a supporting apparatus, and was able to ascend and descend low steps. These restorations must not be construed, however, as regeneration of spinal axones. They are merely the return of function in axones, where conductivity was impaired by the trauma and the traumatic inflammation as shown by the speed with which function returned, which would have been impossible in regeneration.

Statistics. John Chadwick Oliver reports 57 cases of fracture and dislocations of the vertebræ. Fifty of these developed nerve symptoms after the injury; seven showed no evidence of special involvement.

Areas involved: Cervical region, 23; dorsal, 18; lumbar, 10; dorsolumbar, 2; in 4 cases, location not specified. Deaths, 39 (68 per cent). Of the 23 cases in cervical area, 22 died (95 per cent). Of the 18 dorsal cases, 10 died (55 per cent); 5 were discharged as improved and 3 as cured; recoveries, 16.6 per cent. Of 10 lumbar cases, 6 died, 1 improved, 3 remained unchanged, and no complete recoveries. Of 2 dorso-lumbar cases, 1 death (50 per cent). Of the 4 where location was not given, 2 died (50 per cent); considering only the 50 cases which showed nerve symptoms, 41 died (82 per cent). Of the 50 cases, 8 were operated on; 6 of these died (75 per cent); 2 unchanged. Of the 8 operated on: Cervical, 4 — 4 deaths (100 per cent); dorsal, 2 — 1 death, 1 unchanged (50 per cent); lumbar, 2 — 1 death, 1 unchanged (50 per cent). Of the cases operated on, therefore there is a *nil* percentage of improvement or recovery. Non-operated cases: Mortality — Cervical, 94.8 per cent; dorsal, 72.8 per cent; dorso-lumbar, 50 per cent; lumbar, 55.5 per cent.

Thorburn collected 56 cases operated upon. Result: 38 deaths, 67.8 per cent; 18 recovered from the injury; 2 recovered from cord symptoms; 16 showed little or slight improvement.

Dr. Samuel Lloyd collected the following statistics on operated cases:

	Immediate Operation	Later Operation
Cervical region —		
Deaths	21	2
Recovery	....	2
Improved	2	1
Not improved	....	4
Subsequent death	4	3
	27	12
Dorsal region —		
Deaths	23	5
Recovery	4	10
Improved	9	18
Not improved	6	16
Subsequent death	7	16
	49	65
Lumbar region —		
Deaths	4	4
Recovery	1	6
Improved	1	6
Not improved	....	4
Subsequent death	....	2
	6	22
Sacral region —		
Deaths	....	....
Recovery	....	1
Improved	....	3
Not improved	....	....
Subsequent death	....	....
	....	4

The results of these operations are anything but gratifying. Out of 185 operations, there were only 24 recoveries and 40 improvements. There were only 12 improvements by immediate operation; that is, there were only 12 that improved from the symptoms at time of operation. From various statistics of cases not operated on, and from our experience, the immediate mortality is no small percentage. The number of recoveries in 82 immediate operations was 5. The recoveries without operative intervention far exceed this number. The number of recoveries with late operation is a little more gratifying. There were 19 recoveries in 113 operations, which, when compared with the number of late recoveries without operation, may be considered as giving us some justification for the late operation. Lloyd's results are about fifty per cent better than Chipault's. The latter published, in 1894, 167 cases operated on for fractures of the spine; 12 recovered and 24 improved.

Indications for and Time of Operation. In the cases that have come under my observation it has been difficult, and many times impossible, to ascertain whether I was in the presence of a case of division of the cord or reaction of the cord due to trauma. Therefore I always hesitate to operate until I am satisfied that the cord is not completely divided. The immediate complete bilateral circular paralysis of both motion and sensation is the only positive evidence of division of the cord. Where there is definite evidence of fracture or dislocation with division of the cord by fragments, operation is contraindicated, and should not be performed. Where there is evidence of compression by blood or small bone fragments with fracture or dislocation, as shown by the fact that the paralysis is not immediate, not circular, and not complete, of both motion and sensation, an immediate operation may be indicated. Where they are persistent, the operation should be performed.

John Chadwick Oliver divides the surgeons into three groups: 1. Those who advocate operation in every case of injury to the spine in which cord symptoms are present. 2. Those who abstain completely from operation. 3. Those who advise operation in selected cases. We belong to the last class of surgeons.

Eugene Hahn[1] advises operation as early as possible, and particularly in cases of compression due to fractures of the arch, which always result from direct violence. However, he says the question of early operation is not settled as yet.

Andrew McCosh condemns the waiting and watching policy. He advises early operation as being of the utmost importance, unless severe shock is present.

Kocher is of the opinion that operation can be delayed, and advantageously performed after long-continued pressure has been present. There are several cases on record where patients were *apparently* benefited by late operation. This is in consonance with a mild and gradual paralysis from neoplasms within the canal, but one cannot see how that type of pressure would be exerted by a displaced fragment, but it might be by the callus of bone repair.

Carl Lauenstein records a case in which an excellent result was obtained by intervention five weeks after injury. Paraplegia, rectal and urinary symptoms, and trophic changes were present in this case. This is no proof at all that there was compression, or that operation had anything whatever to do with the result, as the train of symptoms, and the result obtained, occur frequently from contusion and with fracture without compression.

McCosh records a case in which paralysis had been present eight months, and yet complete recovery occurred after operation.

L. A. Weeks reports a case by Huss, in which recovery followed surgical intervention one month after the injury.

M. Allen Starr reports a case of compression of the cauda equina of one year's standing. Operation was followed by partial recovery. Some anæsthesia persisted.

Samuel Lloyd says: "In my opinion, we should wait until the period of shock is passed, and until there is evidence that there will be no spontaneous recovery." If, after this period, the patient still continues to improve, no operation should be considered, but as soon as the symptoms begin to show retrograde phenomena, or seem to have reached the end of improvement, operation should be undertaken. This rule is not applicable with any

[1] Deut. Zeitsch. f. Chir., 1902, vol. lxiii, p. 995.

degree of force or regularity, as far as our observations tend. *Recovery after operation before a period of two and a half years has elapsed cannot positively be interpreted as playing any part in the production of the result, except in the cases where fragments or foreign bodies were compressing the cord.*

Syringomyelia. Syringomyelia is a cavity situated in the posterior horn or horns of the gray matter, either connected with or independent of the central canal, and extending upward and downward, for a limited distance or throughout the entire spinal cord. The disease has been known since the sixteenth century. It has occasionally been found in post-mortems of premature fœtuses, or in those of individuals who did not exhibit symptoms intra vitam. Morgagni and Santorini found in a post-mortem of a Venetian fisherman a tubular cavity in the spinal cord independent of the central canal. The present name of the disease was given to it by Nonat[1] in 1830. In 1869, Hallopeau advanced the theory that syringomyelia is not merely a dilatation of the central canal, but an independent cavity resulting from the softening or degeneration of the periependymal glial tissue, which was previously inflamed. Several of the early writers on this subject emphasized the relationship between the formation of the tubular canal within the cord substance and a hydromyelia of the ependymal canal. In 1875, Th. Simon and Westphal considered syringomyelia as being absolutely independent of the central canal. Hoffman of Heidelberg admits that syringomyelia results from degeneration of periependymal glial tissue, but he does not deny the fact that congenital ependymal epithelial displacements of the central canal may have something to do with the proliferation. Wald. Gerlach (1894) was of the opinion that congenital anomalies of the central canal were predisposing factors of syringomyelia. More recently, Schlessinger, Hoffman, Minor, and others favored this relationship. It is therefore certain, say Leyden and Goldscheider, that "in the anomalies of development of the central canal, there is a hyperplasia of the periependymal glial tissue, the relation and importance of which, however, is not yet fully established." The disease has been carefully studied during the last quarter of the nineteenth century, and about three hundred cases have been reported in the literature. Schultze and Kahler deserve credit for their clinical descriptions. Schlessinger's classical monograph has elucidated many points on this subject.

[1] Traité de la moelle epinière et de ses maladies. Paris, 1827.

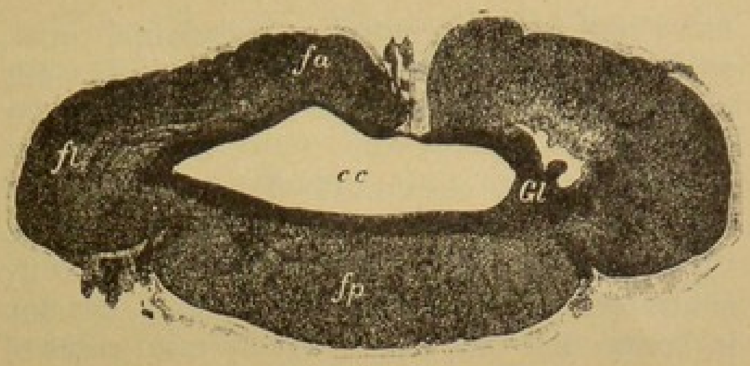


Fig. 1.

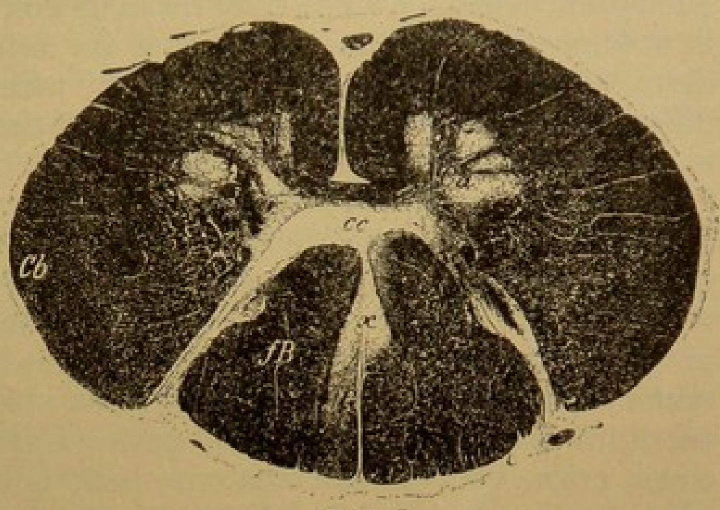


Fig. 2.

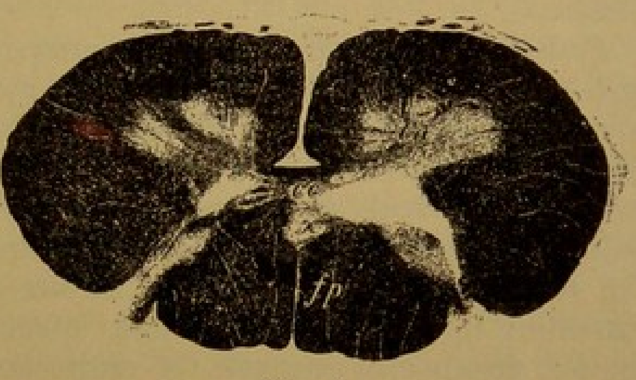


Fig. 3

Fig. 14. Cross-section of cord, showing syringo-myelic cavities. (Jakob's *Atlas of the Nervous System*).

Embryologic Considerations. About the sixth week of intrauterine life, the central canal is very wide, and occupies almost the entire cord. In the course of time, it gradually contracts, and subdivides itself into two halves. The anterior half forms a central or ependymal canal, while the posterior half contracts until its walls come together and finally form the posterior septum.

A failure of closure of the posterior half leaves a congenital cavity within the cord. "The central canal may remain imperfectly contracted and a syringomyelia may develop in connection with it. Probably most cases of syringomyelia are developed on the basis of embryonic defect." [1] The central canal, which is nothing else than an infolding of the ectoderm, is lined with epithelial cells, which differentiate themselves in the course of time and become cylindrical. It should not be forgotton that they are of epiblastic origin. The periependymal glial cells, spread throughout the substance of the cord, and are differentiated epithelial cells. A proliferation of glial cells and fibers (dendrites) forms a tumor mass which has frequently been mistaken for a sarcoma, which is a gross embryologic error, because sarcomatous tissue is of mesoblastic origin, while glial cells are epiblastic.

Pathologic Anatomy. While neuropathologists disagree as to the origin of syringomyelia, their findings are uniformly the same. Examination of a cord, even before its section, reveals a bulging on the posterior half of the cord, which fluctuates on palpation. On section, one or several cavities are found extending upward and downward, either for a limited distance or through the entire cord up to the bulb, and occasionally involving the latter. Primarily, the cavity is more commonly found in the cervical swelling of the cord. The cavity occupies, almost invariably, one or both posterior horns, just behind the commissure and close to the central canal. The latter is not necessarily dilated, and in some cases is so contracted that it is almost impermeable. Again, the syringomyelic cavity may communicate with the central canal and form one large cavity, impressing one that the condition might be a primary hydromyelia. The tube may be patent or may be subdivided by septæ, either lengthwise or laterally, making single or multiple cavities. It may communicate with the fourth ventricle, or end near it in the subarachnoid space. The shape of the cavity is tubular or triangular, and it is lined with cylindrical epithelium similar to that of the central canal. From this histologic fact we are led to believe that it is not likely that columnar epithelial cells can develop from glial cells, notwithstanding they were both originally ectodermic cells before differentiation took place. This epithelial membrane is surrounded by partly proliferated, partly degenerated glial cells. The consistency of this cellular element is sometimes so sclerotic, so hard, that it can be extracted like a foreign body — a rod or a pencil — from the substance of the cord. Here and there the sclerotic tissue softens and forms new smaller cavities. There are scattered areas of hemorrhage and of myelitis resulting from it. The arteries are sclerotic and the veins dilated. The cells in the anterior horn exhibit degenerative changes. The lateral columns of white substance may be either displaced without being injured, or they may be atrophied by compression. The posterior columns, with the exception of the comma tract of Schultze, are free from degeneration. The anterior roots are atrophied only where there are degenerative changes in the anterior horn. The posterior roots suffer changes from compression. The spinal gangla are usually normal. The pia mater is thick in many cases. In the peripheral nerve there is a thickening of the perineurim or endoneurim (Steubeuer, Langhans). If the tube reaches the medulla, the nuclei of the trifacial, hypoglossal, vagus, and glossopharyngeal may be involved.

Ætiology. There is no definite ætiologic factor in this condition. The disease is dependent on some embryologic abnormalities, and there are ætiologic factors, not well understood, which produce changes in embryonal defects. Trauma seems to be one of the most important factors in developing the congenital tendency. Infectious diseases have been deemed the exciting factors, but it seems that they are mere coincidences. Syphilis and alcoholism, which are the chief exciting factors of so many diseases, do not seem to play any rôle in syringomyelia.

Age and Sex. Sclessinger's statistics of 190 cases show:

[1] Dana. Treatise on Diseases of Nervous System, p. 337.

Ages	Males	Females
1–10 years	4	1
11–23 years	36	18
21–30 years	53	25
31–40 years	30	12
41–50 years	4	7
51–60 years	3	3
Above 60 years	3	1
	133	57

From these statistics one must conclude that trauma has little if any ætiologic influence, as the male spine in middle life suffers enormously more trauma than the female.

Symptoms. One cannot understand the symptoms of this disease, and in fact of any of the spinal diseases, without a definite knowledge of the anatomy and physiology of columns and ganglionic cells. The syringomyelic process involves one or both posterior horns, immediately behind the commissure. It occasionally compresses the posterior columns; it is associated with changes in the anterior horns; it displaces or compresses the lateral columns of the white substance. It will therefore have sensory and motor disturbances, changes in the reflex and trophic conditions of the extremities, and derangements of gait.

Disturbances of sensibility are typically characteristic of this disease. Patients have an almost perfect tactile sensibility, but have lost that of heat and pain. Occasionally heat is mistaken for cold, and *vice versa*, a phenomenon which is called syringomyelic dissociation. In very rare instances an anæsthesia of the skin and mucous membrane has been noticed (Déjerine). Subjectively, patients complain of pains, usually in the upper extremities. They are lancinating in character, and are often mistaken by the patient for rheumatic pains, and by the physician for tabetic pains. The preservation of tactile sensibility is explained by the fact that in the majority of cases the posterior columns of the fasciculus cuneatus and fasciculus gracilis are not involved. They carry the tactile impulses; the perversion of loss of sensibility to heat or pain is due to the destruction of a zone of ganglionic cells from which originate the axones that go to make up Gowers' column, which conduct the impulses of heat and pain.

Motor Changes. There is muscular twitching and muscular atrophy, either unilateral or bilateral. The upper extremities are usually involved, and begin with the small muscles of the hand, resembling, in this respect, the clinical picture of progressive muscular atrophy, Duchenne-Aran type. Occasionally the muscles of the shoulders only may be involved. The muscles of the trunk may next be involved, those of the abdomen, and finally, rarely, however, the lower extremities. The extent of the muscular involvement and the group of muscles injured depends upon the extent and topography of the destruction of the anterior horn.

The cutaneous reflexes are generally increased. The reflexes in the upper extremities are absent, while in the lower they are exaggerated. This is the case if the lesion is located in the cervical region. If the disease involves the lumbar region, destroying the center of the patellar reflex, then, of course, the latter disappears.

There are trophic changes, viz., glossy skin, hypertrophic nails, herpes, and ulcers; accidental cuts heal either slowly or never; there are trophic changes in the bones and joints, producing a syringomyelic arthropathy. Dr. Hugh T. Patrick, in the *Journal of Nervous and Mental Diseases*, December, 1898, draws attention to some striking manifestations in his cases: the great sensory disturbance and the spontaneous loss of toes; and in 1897, before the American Neurological Society, he reports a case of syringomyelia in which anæsthesia and analgesia more closely corresponded to the tegumentary sensory representation of the segments of the spinal cord than any reported up to that time. A second case had a broad area of analgesia and smaller scattered areas of anæsthesia. The reverse is obtained in trunk anæsthesia of tabes, as shown by Laehr, Bonnar, and Patrick. Scoliosis or kyphosis are often present. As in tabes dorsalis, there is here a tendency to spontaneous pathologic fractures. There may be changes in taste, in vision, bulbar symptoms, disturbances of the vasomotor centers, etc. If the bulb is involved and there is compression upon the brain, there is a complex of cerebral symptoms, rendering the clinical picture more obscure.

Schlessinger, in his monograph published in 1895, describes five distinct types:

1. One in which motor trophic sensory symptoms, and few other irregular symptoms, are present.

2. A motor type similar to amiotrophic or spastic paralysis, in which the chief disturbance is motor, while the sensibility remains normal for years.

3. One in which the sensory disturbances predominate.

4. One in which the trophic disturbances are more conspicuous. If there is mutilation of the extremities, it affects the type of Morvan's disease. In this type there are intense neuralgic pains, anæsthesia of the skin, and destructive whitlows.

5. One in which the posterior columns are invaded with gliosis (Oppenheim).

From the pathologic changes and the clinical course, it is fairly safe to assume that the great element of destruction is the pressure from accumulating products of degeneration, or the excess and retention of secretion of the columnar cells lining the cavities. This pressure can be relieved when circumscribed or even fusiformed by a subdural drainage, the same as indicated in cerebral hydrocephalus. For this purpose, however, a horsehair seton passed through the cord into the cavity, and knotted on its surface beneath the dura, should suffice for permanent relief of pressure, on the same principle that metallic and glass tubes were used by Charles Ballance for ventricular hydrocephalus. Where the disease is pathologically represented as a solid ligneous mass, it might be extracted through the posterior columns or commissure of the cord by careful blunt dissection, as one would by a foreign body, and as Braun removed the intramedullary bullet.

Diagnosis. The disease can be, and has been, confounded with progressive muscular atrophy, leprosy, multiple neuritis, hæmatomyelia, tabes dorsalis, scleroderma, acromegalia, and others. In this the surgeon should co-operate with the neurologist. It is with the hope that surgical intervention may avail for the relief of this otherwise incurable condition that we have devoted so much space to its detailed consideration. We have not yet had an opportunity to operate on a case.

Prognosis. The prognosis is "finally fatal," but the course of the disease is very long, ranging from five to twenty years. Occasionally, patients may not develop marked symptoms during life, as was shown by post-mortems of individuals with syringomyelic cavities who had had no pathognomonic or recognized symptoms.

Prognosis without Surgical Intervention. It seems to us, from a theoretical standpoint, that this disease, at least in its circumscribed form, offers a field for surgical intervention. As soon as opportunity presents itself, I will perform a subdural drainage with an inabsorbable seton which will keep a permanent communication between the syringomyelic canal and subdural space, and insure an equalization of pressure, which should stop the advancement of the disease, even if it does not permit restoration of function in some of the compressed ganglion-cells and axones.

Tuberculous Osteitis of the Vertebræ — Vertebral Caries — Tuberculous Granulomata

In this chapter we do not intend to describe in detail vertebral caries. We will consider only that phase of the pathologic process which directly or indirectly may produce pressure symptoms upon the cord. In other words, we will deal principally with the tuberculoma, which should be regarded as an extradural tumor.

Spinal caries was first described by Percival Pott in 1779. Since that period, the pressure symptoms of the cord have been differently interpreted by various authorities. Ollivier thought that the cord was compressed by the bony deformity pressing upon it with its sharp angle directed posteriorly. This has been proved, in the course of time, to be untrue. Repeated post-mortems have demonstrated beyond a doubt that the cord may be free of compression, even with very sharp angular deformities of the bony elements. The spinal cord follows the angulation of the bony canal, and unless there is displacement or fracture of the respective vertebræ, the cord does not suffer from pressure. Kahler and Schmauss ascribe the compression symptoms to a lymphatic œdema, the result of compression. Ziegler believes that the compression produced by the bone leads to an anæmia of the cord, which is responsible for the symptoms. The partisans of the lymph stasis theory state later that it is followed by a swelling and softening of the nerve substance in the cord, which is due to an active inflammation or reaction myelitis. The inflammatory process may reach the cord by a propagation from the meninges, the tuberculous process invading the cord either by direct continuity of destruction through the septæ or blood-vessels. The latter may be obstructed, the arteria centralis principally, producing a softening of the medullary substance. This, however, is of no great pathologic significance. Strümpell is the first who conceived the idea that compression of the cord was mechanical in origin, and due essentially to the pressure of extradural tuberculomata acting as extradural tumors. Tuberculous caries of the spinal column is of importance when we consider the enormous number of cases, and also the amount of damage which it causes to the spinal cord. Vulpius, studying the statistics of Billroth, Mohr, Lorenz, Hoffa, and Beuthner, found that, among ten thousand surgical cases, forty-four were vertebral caries. He also estimated that the latter represent one fifth of all bone diseases.

In order to understand thoroughly the cord symptoms in vertebral caries, it is essential to have a clear knowledge of the evolution of the pathologic process which takes place in the bone. The tuberculous process originates practically always in the body of the vertebra, principally in the cancellous substance. Occasionally — very seldom, however — it invades the small articulations and the arches. If this be the case, the first two cervical vertebræ are concerned.

The bone changes may be subdivided into two stages:

(*a*) Granulation focus (fungous osteomyelitis). One or several small cavities form in the cancellous substance of the bone, containing tuberculous granulation tissue. If coagulation necrosis take place, the cavities are filled either with cheesy material or tuberculous pus.

(*b*) Tuberculous necrosis. If there be considerable destruction of the bone, absorption is not possible. The destroyed segment forms a sequestrum. The cheesy mass is separated from the normal bone by a zone of tuberculous granulation tissue; at first it is reddish in color, and at this period it is very difficult to distinguish from the healthy tissue; later, however, on account of circulatory disturbances, this mass becomes grayish, and finally yellow. If absorption of the fluid take place, it becomes an encapsulated caseous focus. As the substance of the body of the vertebra is being destroyed, it gradually compresses, the result being a deformity. When the tuberculous process ruptures through the posterior wall of the body of the vertebra, it elevates the periosteum, and gradually invades the spinal canal, compressing the cord with the accumulation products of the tuberculous disease in its circumscribed connective-tissue walls. This is always situated anterior to the cord and nerve-roots. The spinal cord molds itself to the bony deformity, but the granuloma encroaches upon and compresses it, producing paraplegia. The destruction may have been going on for a considerable time without compression symptoms being present. Trauma is generally responsible for the rapid collapse of the body. For a short period of time, the cord may be compressed by a large abscess under tension, as well as by a tuberculoma. The segment of the spinal column usually involved is from the middle dorsal to the middle lumbar. The cervical region may be the seat of attack, although rarely. Generally speaking, one vertebra is affected. There are cases, however, where two or three, and even more are invaded.

In sudden collapse with displacement of the fragments, the cord is damaged exactly as in a fracture and dislocation of the vertebræ.

The tuberculous process invades the meninges, and some authorities think that compression is due to a localized meningitis; furthermore, that the inflammation is transmitted to the cord substance itself. These changes, however, are pathologic curiosities.

In compression due to a tuberculoma, the cord is usually bent and flattened. Its nerve elements are crowded and contracted. For a long time, there may not be trophic or degenerative changes in the cord, because it adjusts itself. Long-standing compression, however, produces degeneration of the axis-cylinders and ganglion-cells. There is a sclerosis of the walls of the blood-vessels, an invasion of granulation-cells at the seat of the trouble, and the glial and connective-tissue elements undergo fungoid changes. Finally, the cord elements may be replaced by connective tissue, with ascending degeneration of the posterior columns and descending degeneration of the anterior columns.

Symptoms and Diagnosis. The cases which come to the surgeon have long passed the lymphatic stage, or anæmia of the cord. They are, generally, cases in which the compression is affected by a tuberculoma. It is true, however, that it is impossible to differentiate from the symptoms, whether we have to deal with a simple reaction myelitis produced by the tuberculous inflammatory process, or whether it is a compression myelitis due to a tuberculous tumor; the former condition, however, is very rare. The only criterion in this differentiation is the long-standing symptoms of myelitis without the slightest attempt to amelioration.

As indicated in the beginning, we will consider the tuberculoma principally. It is practically an extradural tumor situated on the posterior portion of the vertebral body and in front of the cord; if it is of considerable size, it may spread to the sides of the cord. It is never, however, posterior to it. From this we can understand that the primary symptoms will be purely motor, as the pressure concerns the anterior or the anterolateral columns. Sensory disturbances may take place, not by compression of the posterior columns, but by pressure against the posterior roots at their exit, and also on Gowers' column. From the symptoms, one can accurately estimate the location of the tuberculoma; that is, which cord segment and which column is compressed. One cannot, however, judge from the symptoms whether the cord is merely compressed without having undergone degeneration, or whether there is degeneration of the cord substance. In localizing the tuberculous tumor, it should be borne in mind that one is likely to make the error of estimating the tumor too low. A given cord segment is situated above the respective vertebra, as indicated in the charts (see cord tumors). The topography of cord segments, in their relation to the vertebral bodies, must be clearly pictured in the mind of the diagnostician. The splendid work of Victor Horsley, who has done so much to elucidate this condition, should be carefully studied by every one contemplating work in this line.

Prognosis. The prognosis in paraplegia of spinal caries is always grave. Statistics from Billroth's clinic, quoted by Oppenheim, show that out of 97 patients, 48 died, 22 were cured, and 11 remained unchanged. Gowers states the paralysis due to vertebral caries is the most favorable of all types of paralyses. Generally speaking, the prognosis does not depend so much upon the age of the individual, nor upon the intensity and extent of the symptoms, as upon the amount of damage done to the cord substance which does not allow of repair, if the pressure has been long continued. Operative procedure must therefore be timely.

Operation. The operation for tuberculoma is a simple laminectomy. (See "Laminectomy.") Displace the cord to side, expose the granuloma, remove it with a bone curette, fill the cavity with a Moorhof plug to lessen the danger of suppuration and hasten the powers of repair; replace the laminæ and close without drainage. It is usually not necessary to open the meninges. One should not hesitate to curette extensively the vertebral bodies. If the wound heals without suppuration, and the cord has not long been compressed, the paraplegia rapidly disappears. If a mixed infection follows, the cord may be destroyed and a permanent paraplegia result, as occurred in one of my early cases.

Technique of Laminectomy. Before proceeding with a primary incision, it should be definitely determined by the operator whether his plan is to extirpate the spinous processes and laminæ entirely, or to preserve them. If the former is decided upon, longitudinal incision should be made parallel to the spinous processes on the operator's side of the middle line. The incision should extend along down the side of the processes close to the bone. After thoroughly exposing the muscle, a blunt dissection may be made on the lateral surface of the processes to the laminæ. The attachment of the flap to the tip of the processes is then divided, and a similar blunt dissection made on the opposite side of the processes down to the laminæ. The hemorrhage, which has been considerable, can now be checked by gauze packing. The interlaminar spaces of one, two, or three vertebræ should be exposed,

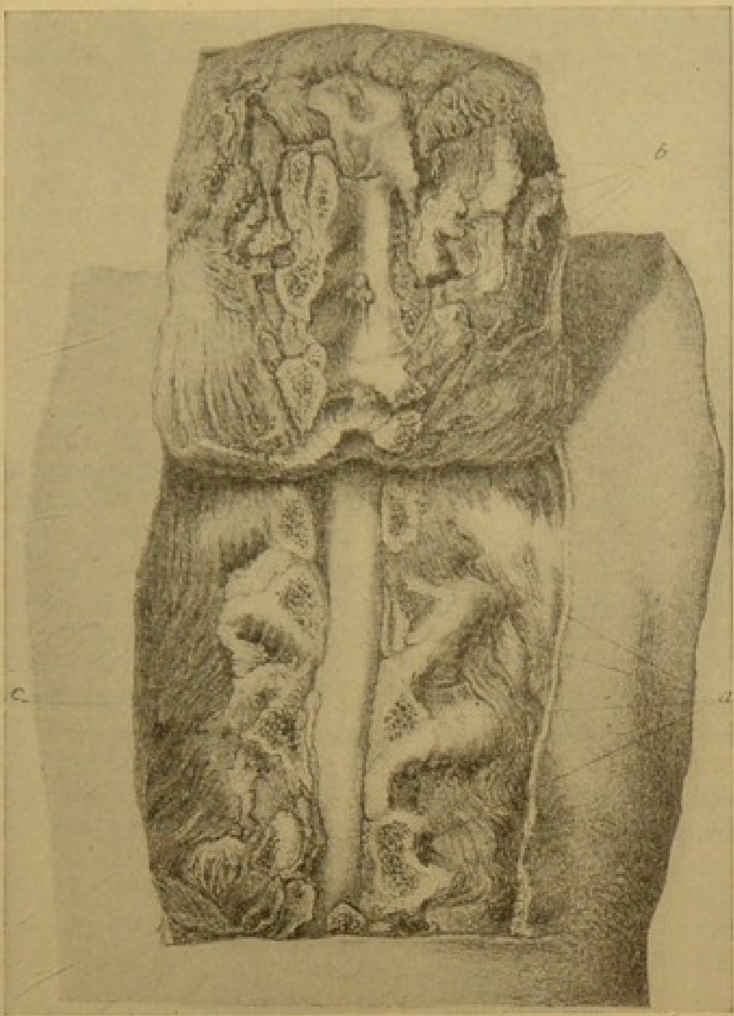


Fig. 15. Urban's osteoplastic resection. (*Handbuch der Chirurgie.* Von Bergmann, Bruns, Mikulicz).

and the conical drill rongeur which I have adapted for this purpose should be used to puncture the laminæ at the base of each transverse process. The tips of our rectangular guarded bone-cutting forceps can be inserted in each of these punctures, and the laminæ divided. The interspinous ligament is next divided. The spinous processes and laminæ can be readily lifted up with a sequestrum-forceps. This plan avoids all malleting and concussion of the cord, and guards against its possible puncture; it hastens the procedure very materially.

The dura may now be opened, if desired, and the cord inspected, or if searching for a tuberculoma, the cord can be pushed to the side, and the tumor recognized on one or the other side, anterior to the cord in the body of vertebræ where it always forms. The dura should be carefully sutured after the operation by catgut. If it is the purpose of the surgeon to conserve the processes and laminæ, then the H-shaped incision is preferable, leaving a septum of tissue, muscle, and skin one inch in diameter between the two parallel incisions, making the muscular blunt dissection one half inch from the bone to the side. The laminæ should be punctured in the same way, and divided with rectangular bone-forceps. Then the interspinous ligament is divided and the flap turned half up and half down. In replacing this, care should be exercised that the bone-flap does not compress the cord. The flap should be closed by deep catgut sutures and superficial silkworm-gut. Horsehair should not be relied upon, as tension is so great in these cases that this suture may give way. I had a case of compression by bone-flap after operation, resulting in permanent paralysis. Since

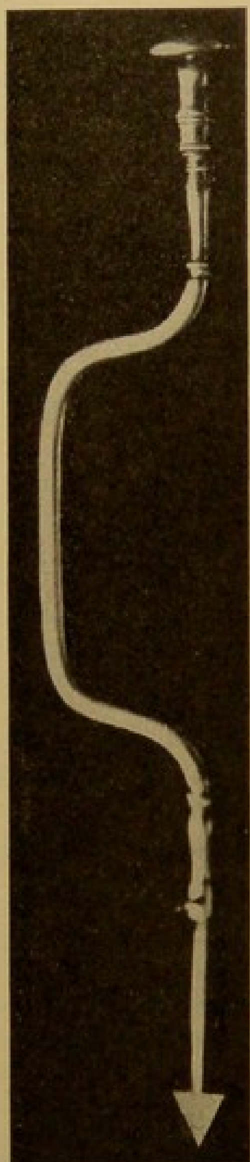


Fig. 16*a*.

Figs. 16*a*, 16*b*. Instruments used in laminectomy. (Original).

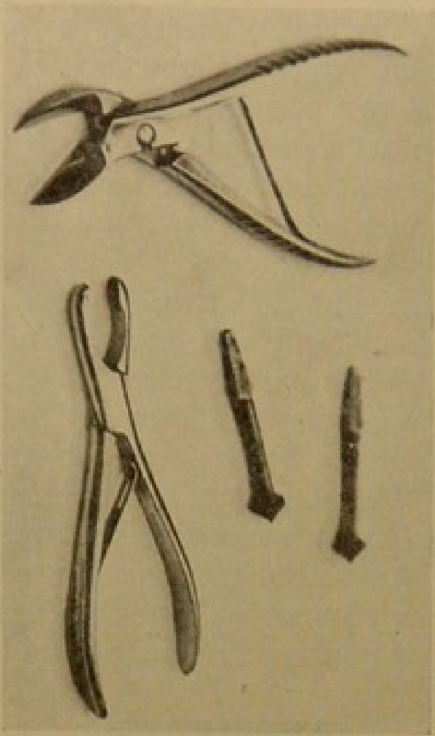


Fig. 16b.

that time I have removed laminæ and spinous processes completely, and found no permanent weakness of spinal column following this procedure. In fracture and dislocation it is always desirable to remove the laminæ, as the luxation frequently cannot be reduced at the operation, and there is danger of compression of cord after the parts are replaced. In pronounced deformities following fractures, one should not hesitate to remove the protruding edge of the fractured body of vertebra. This may necessitate a division of one of the spinal nerve-roots. Traction should be made on the nerve to draw it well into the canal; it may then be divided and sutured at the close of the procedure. The greatest care must be exercised in the manipulation of the cord, as it requires but a slight degree of trauma to produce a zonal paralysis. In most of my cases a complete temporary paralysis immediately followed the operation.

Mr. S., printer, aged 32. Admitted to Mercy Hospital, August 22, 1905.

History of Present Illness. In 1898, patient noticed for the first time some difficulty in walking. While endeavoring to catch a car, he noticed that he was not as agile as formerly. In the following weeks the difficulty became more manifest. It increased slowly and gradually, until in 1901 the gait was distinctly impaired. From 1901 to 1904, the patient was compelled to make use of a cane. In the last three months he was forced to make use of crutches. Three years ago, patient noticed pain in both lumbar regions, along the posterior region of the thighs, and in both knees. The pains were not continual, were sharp at times, but not sudden, so that they were entirely different from the classical shooting pains usually experienced in tabes dorsalis. At times the pain took the form of cramps; again they were neuralgic in character. From time to time, patient noticed muscular spasms, which he chose to qualify as "twitchings." Since September, 1902, patient did not experience any pain. Had no gastric symptoms at any time; no gastric crises; was usually constipated. In last four years, had great difficulty in making his bowels move. Lately, he has noticed a weakness of the rectal sphincter, especially when taking purgatives. He describes the phenomenon by saying, "I can't control my bowels." The same derangement has been noted on the part of the vesical sphincter. Patient has frequent desire to urinate, but sometimes fails to urinate when he has the desire. If that difficulty appears, he usually overcomes it by pressing upon the bladder, which is followed by the evacuation of the urine. He has several involuntary emissions of urine at night. These sphincter troubles appeared about one and a half years ago.

Patient is not nervous; has not changed in character and temperament of late. His memory has not been as good during the last five years as it was previously.

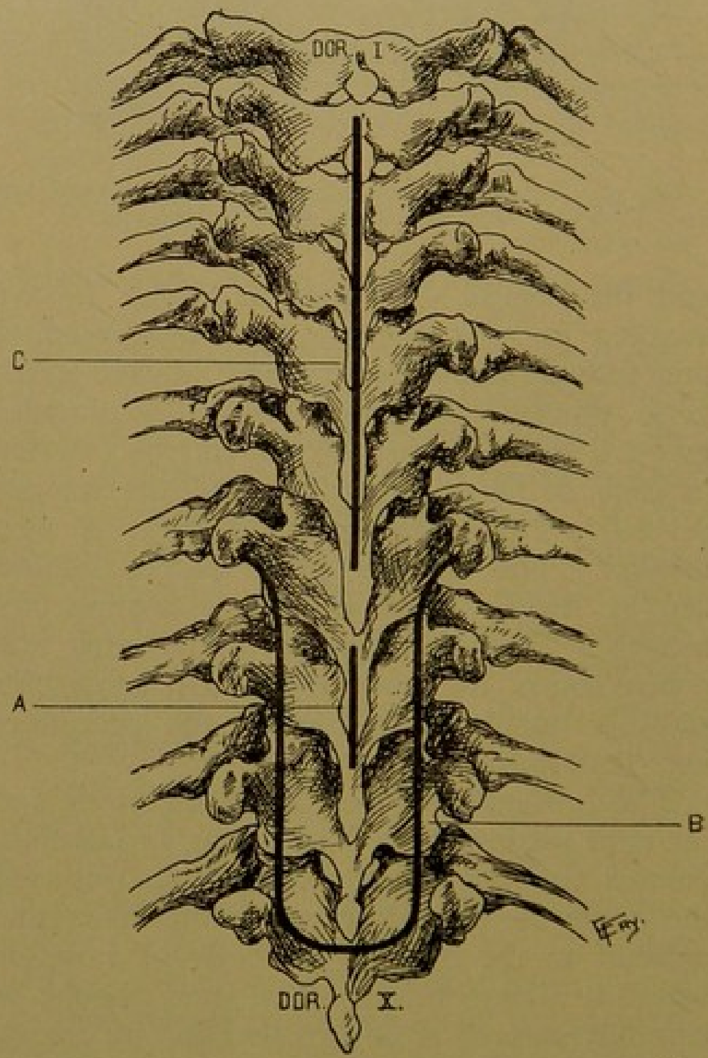


Fig. 17. Bickham's incision for laminectomy. (*Annals of Surgery*, 1905, p. 380).

No change in vision, speech, or deglutition. Lately, however, he noticed a slight hesitancy when beginning a new sentence. Sleeps well; has not lost in weight. For the past four years, patient has been deaf. The condition was not induced by any accident; he cannot account for its appearance. In the last six months, his sexual appetite has considerably diminished. He seldom noticed an erection; has no night emissions.

Previous Illnesses. Patient had mumps, measles, and chicken-pox in childhood, without sequelæ. Had a specific venereal ulcer at the age of 16, when he was treated for one year. Four or five years later, he took antisyphilitic treatment for eight months. The disease was diagnosed by a physician, and was fully confirmed by post-syphilitic symptoms. At various times, he had mucous patches in the mouth and the throat. In 1896, he had what he calls sores on the hands and feet, accompanied by severe nocturnal headaches. The administration of iodide of potassium greatly improved his condition and finally cured him. He used, and probably abused, the use of alcohol up to three years ago. Smokes, but not immoderately. Has been married four years; no children. His wife has had no abortions.

Family History. Father died with general paresis at the age of 56. Mother died with tumor of the kidney at the age of 55. Wasting symptoms preceded her death. Patient says: "We had a good many cancers in the family. Cousins, aunts, and collaterals of the family died with cancers of the throat, breast, stomach, etc."

Examination of Patient. Medium stature; general nourishment fair; teguments have a normal appearance.

Eyes. Pupils are equal in size, regular in their contour, and respond very well to light and accommodation. Wears glasses; is able to read without difficulty.

Speech. By paying special attention, and for some time, we notice a little hesitancy in speech, particularly when beginning a sentence. The tongue shows a fine fibrillary tremor.

Examination. Lungs, negative. Heart, negative. Abdominal organs, negative.

Reflexes. The reflexes of the face are increased. The cutaneous reflexes, in general, are slightly increased. *Patellar reflexes, however, are absent.*

Sensation. Patient has a perfect sensibility, both tactile and caloric. We were unable to find even a small area in which the sensation was impaired or absent. By running a needle along the surface of the skin, patient feels equally the touch of the point of the needle.

Motion. In recumbent position, patient is able to flex, extend, rotate, abduct, and adduct his lower extremities. Flexion of the knee is accomplished almost normally on the left side; flexion of the right knee with some difficulty. He is able to extend his legs, but toward the end of this action the heels fall down on the bed involuntarily. He flexes and extends his toes almost normally on the left side; flexion and extension of *right* toes is difficult, limited, and imperfect. He is able to lift his legs. The left heel can be elevated eighteen inches above the level of the bed, while the

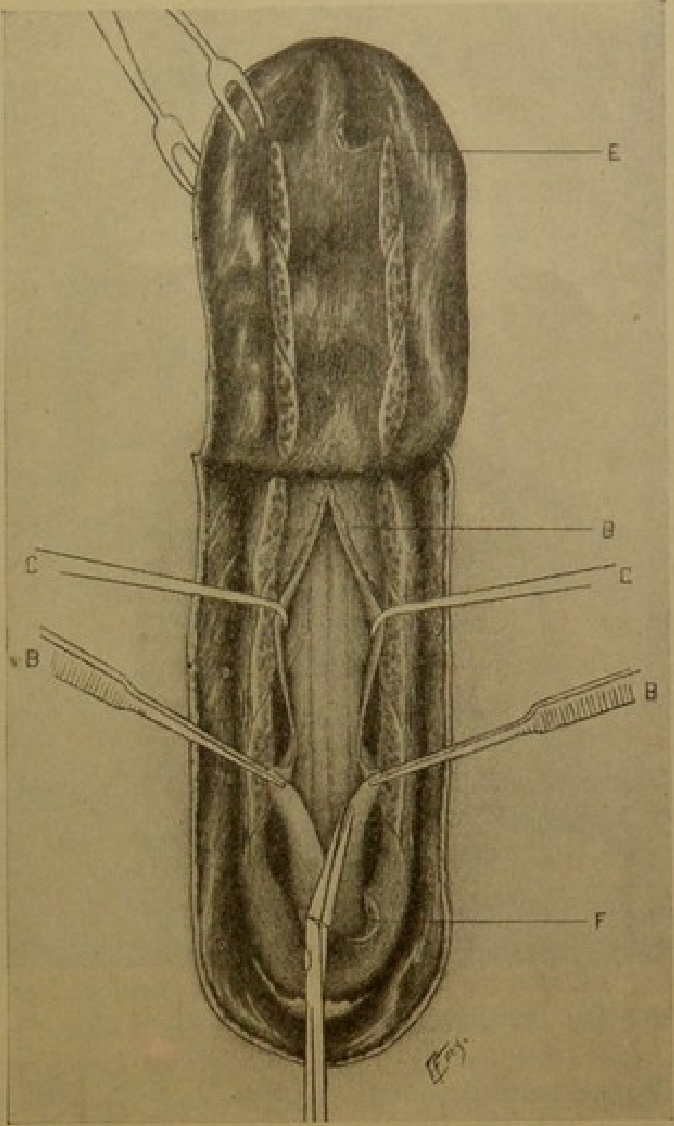


Fig. 18. Bickham's osteoplastic flap reflected upward, dura incised and retracted, and cord exposed. (*Annals of Surgery*, 1905, p. 388.)

right one can be elevated only twelve to fourteen inches above that level. The distances above mentioned are considerably reduced if he elevates the legs four or five times in succession. Generally, we may say that after repeated muscular contraction a fatigue is distinctly noticeable. Patient calls it exhaustion. Muscular sense is perfect. No muscular atrophy. Tension of the musculature of the thigh and leg is slightly diminished. Patient is able to walk eight blocks with the support of crutches. Without crutches, he walks with difficulty one half block, maximum. Gait decidedly not ataxic. The first step is very difficult, but once started he is able to make the following steps. Walking, patient rotates his trochanters around an anteroposterior axis of the pelvis. His gait can best be compared to the gait of children with congenital double dislocation of the hips. He is unable to walk in the dark. Standing up with his heels together and closing his eyes, he loses his equilibrium.

Examination of the Spinal Column. The spinal column shows no deformity; no deviations. By pressing along the spine and spinous processes, we find the

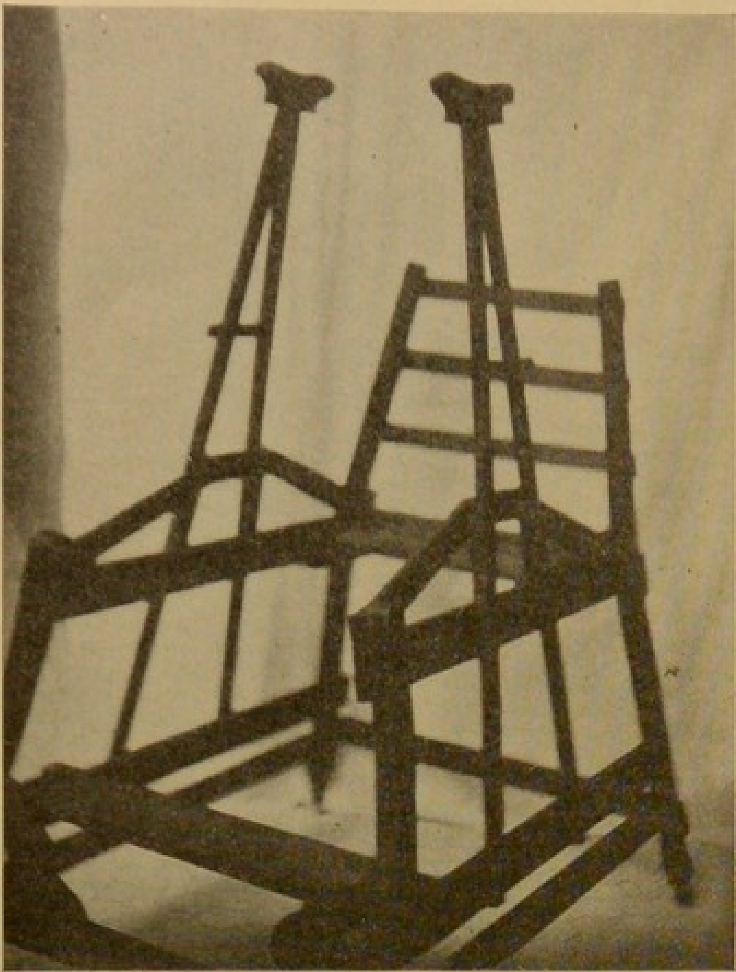


Fig. 19. Chair for the re-education of gait.

following painful points: (*a*) Fourth, fifth, sixth, and seventh cervical spinous processes; (*b*) The middle dorsal spine; (*c*) The lumbosacral junction.

Babinski's sign present, and more marked on the right foot. August 24, 1905, pain spasmodic in character; that is, short, sudden pains located on the outer surface of thigh only. Is able to flex toes, but very little. August 25, 1905, same condition. Babinski's sign not noticeable. Slight muscular twitchings in the extensors of the thigh. August 26, 1905, no improvement in flexion and extension of toes. Patient is able to flex his left knee, but very little.

Diagnosis. Dr. Hugh Patrick: Compression of the spinal cord on its right side with a tumor at the eleventh dorsal vertebra.

Operation. August 26, 1905. Compression of spinal cord from the right side behind, pushing the cord forward and to the left, making a groove or depression in the cord. Enlargement of lamina and base of transverse process at their junction, involving only the eleventh dorsal process. Meninges appeared normal; no elevation on the vertebral body anteriorly. Cord was *not* compressed anteriorly for the distance of four inches examined. Operation: Laminectomy and removal of exostosis.

Paraplegia complete after operation. This occurs even in cases where paralysis was only partial preceding the operation. It is, however, only a zonal or traumatic paralysis, and entirely disappears as long as the centripetal and centrifugal axones are not divided or ganglionic cells severely traumatized; complete restoration always follows. One of the unpleasant features of this work is the length of time necessary to obtain results, six months to two years being required for a restoration of function. However, the time is not too long nor the labor expended too great, as otherwise the patient has before him no hope except a permanent paraplegia. In handling these patients after operation, care should be exercised that too great pressure is not exerted on the vertebral bodies while moving the patients from one cart to another and to their beds.

January 22, 1906, five months after operation, patient can move all muscles and stand in his training-machine without support. Since that the improvement has been slow, but progressive.

It is unnecessary to say that all these cases should be temporarily drained and all aseptic

Fig. 20. Patient in the chair.

precautions taken, as infections are more likely to occur here than in the peritoneum. If the meninges become infected, the case is likely to terminate fatally. Every precaution against bed-sores (decubitus) must be taken. The bladder should not be catheterized; the patient should have a vaginal or procteal massage until overflowing is produced. The urine, when started, will continue to flow. If the catheter is once used, it will have to be used continually, and decomposition of urine will result, which will necessitate frequent vesical irrigation, greatly increase the labor in caring for the patient, and very materially hazard his life, as vesical and ascending renal infection is the most common cause of death after operation for injuries to the spinal cord.

Suppurative Meningitis. The operative treatment of suppurative meningitis has been ably handled by Dr. Hermann Kümmell (*Arch. für klin. Chirurgie*, vol. lxxvii, h. 4).

The writer advocates the operative procedure in suppurative meningitis, which he believes at times may prove successful, as it has in the drainage of suppurative peritonitis. The attempts have been unsuccessful in tuberculous meningitis, where the condition of the patient was, clinically, beyond aid. Yet even in such cases there was a temporary improvement, or, better, relief, inasmuch as the intellect of the patient revived temporarily and the pain diminished considerably. The successful lumbar punctures in cerebrospinal meningitis, as practised by Lenhartz, speak in favor of an active treatment in this affection. The writer proposes to drain the spinal canal in cerebrospinal meningitis whenever the simple puncture proves to be a failure. Localized meningitis of otic origin has often been treated surgically, and a great number of cases recovered. Hinsberg and Brieger saw an otic meningitis subside by simple incision of tympanum. In a similar case, Cohn saw subsidence of symptoms after a lumbar puncture. Lucæ and Jansen had a recovery from a leptomeningitis of temporal region by simple incision of dura and drainage. Witzel lost two cases of suppurative meningitis in spite of proper and timely drainage. Macewen refers to a number of cases of meningitis treated surgically; some of them with success. Hinsberg collected twelve cases of otic meningitis; of these, eight recovered and four improved.

The problem is a little different in diffuse suppurative meningitis.

Poirier obtained a cure in a case of diffuse meningitis subsequent to fracture of the skull, by double trephining. A case of otic meningitis reported by Bertelsmann in which the cerebro-spinal fluid was turbid and contained diplococci, and which recovered by operation, could be well mentioned as diffuse meningitis.

Macewen refers, in his work, to six cases of cerebrospinal leptomeningitis; five of these were operated on, with one recovery. He is convinced that by accurate diagnosis and timely treatment, results could be better. In cases of leptomeningitis involving the entire cerebrospinal tract up to the cauda equina, he considers operation valueless.

Kümmell reports a case, a man 33 years old, who suffered a severe fracture of the base, the sequence of which was a suppurative meningitis. Lumbar puncture revealed pus in cerebrospinal fluid. The patient recovered after bilateral trephining. The smear from the turbid fluid, however, showed no cocci; blood was sterile. The clinical picture was that of a very severe type of diffuse, generalized suppurative meningitis.

In 1902, Gussenbauer, at the meeting of the Vienna physicians, expressed himself as follows: "The treatment of meningitis belongs to surgical therapy."

Von Bergmann ascribes the success in the operated cases of diffuse meningitis to the fact that the ætiologic factor was staphylococcus albus and not aureus. This is the same deduction formerly made in respect to the peritoneal infections now known to be erroneous.

In a case of meningitis in a girl 27 years of age, in which there was clinically no hope for recovery, the drainage of subdural spaces gave a remarkable temporary relief of all of the chief symptoms; temperature, cephalalgia, rigidity of neck. She finally died.

Kümmell believes that in diffuse suppurative meningitis in which proper and timely drainage is instituted, a certain group of cases will recover as they do in peritonitis.

We have in general suppurative meningitis the same essential problems of pus infections as in the peritoneum:

1. The virulence of the infection.
2. The source of supply of infective material.
3. The pressure under which the pus is retained.
4. The duration of the infection before the pressure is relieved.
5. The relief of the pressure for a sufficient length of time for tissue infiltration (local immunity) to develop.
6. The development of constitutional immunity — hyperleucocytosis.

In the epidemic meningitis, the infections are fairly uniform, each individual case closely resembling in virulence that of its neighbor. While the sporadic cases of otic, nasal, or cryptic origin may vary greatly, they are rarely streptococcic. To the absorption of all these, the tissues offer a considerable resistance, and the element of time increases this resistance rapidly under favorable circumstances. The source of supply is usually limited and self-circumscribing, whether it is of otic, nasal, or hæmatogenous origin. If we could, therefore, tide the patient over the immediate effect by reduction of tension and drainage of infection products, we should put him in a good way to recover. The pressure can be relieved and the drainage established: (*a*) Temporarily, by repeated spinal punctures, with the withdrawal of large quantities of cerebrospinal fluid; indeed, the spinal tract can be flushed with its own fluid; (*b*) Permanently, by lateral ventricular punctures with a canula and needle (as subsequently described), and combined with spinal puncture so that saline solution may be washed through the lateral ventricles and out through the lumbar puncture; the subarachnoidal space may be handled in the same way. By using the blunt trocar needle with an inner tube, the presence of fluid can be detected without the admission of brain substance into the needle at any given depth.

In an operation by me in the lumbar portion of the spine, in which infection took place, a drainage-tube was inserted and kept in for five days; the patient recovered. This, as well as the clinical fact that in spina bifida and in intranasal and intraotic fractures, large quantities of cerebrospinal fluid can escape for weeks or months without any untoward effect on the patient, unless the tract becomes infected, shows that a unilateral laminectomy with opening of the dura and the insertion of a drain may be accomplished without untoward effect on the patient. All of this can be done under spinal analgesia. It is the rational treatment and operation in cerebrospinal suppurations, as it not only relieves the pus tension in the spinal canal, but it also relieves the pus tension in the ventricles and ependymal tract, when the foramen of Magendie is patent. If the tension can be kept off, I believe that here, as has been so incontrovertibly demonstrated in the peritoneum, the cases will go on to recovery without the aid of saline or other antiseptic fluid. The cone-shaped trephine can be used to advantage in perforating the laminæ.

The pus or infection products (*tension* in the cranium and spinal canal) are most important factors in producing the fatal termination by the ischæmia which the pressure causes in the vessels and brain; here, as no place else, except in the shafts of long bones, we have an unyielding, inelastic bony wall, which does not give in the least to the accumulating products. The only concessions made to the increase in the infective products are made by the diminution in the size of the blood-vessels with its resultant ischæmia. The ischæmia in the brain, as in all other tissues, favors the destructive influence (biotic and toxic) of micro-organisms. It is this increase in the pressure from inflammatory products that causes the speedy deaths from acute traumatic meningitis. The absorption of infection products by the meninges is feeble compared to peritoneal absorption, so that the patients do not die from a general toxæmia in these diseases. Drainage, therefore, and relief of infective tension is the most important factor. This can be made through the ventricles in the parietal region or through a submuscular occipital trephining near the median line, where the fourth ventricle can be drained and the velum partially excised if the foramen is closed, as I have successfully done in central hydrocephalus.

It will avail little here, as in other positions of the body, to drain after ischæmia necrosis or fatal absorption has taken place. The drainage must be instituted timely and at the earliest indications of brain pressure. By an early relief of the pressure the local infiltration is permitted to take place, its local immunity zone. Here, as in all tissues of the body, the infiltration, induration, and œdema of tissues around infective foci, which the doctors have for centuries been endeavoring to "scatter, absorb, and dissipate," are nature's coffer-dams, nature's barriers against absorption, nature's defense-works against the invasion of micro-organisms. In cases of sepsis in which these defenses are meager or absent, the life of the patient is in the greatest danger from general constitutional infection. The relief of pressure favors the circulation in the zone, diminishes the degree of absorption, and permits tissue reaction against the infection.

During the time that the local lymphocytosis and infiltration are retarding the advancement of the infection, the hyperleucocytosis is developing in the blood to defend the general organism. The fear of diminution or low tension of the cerebrospinal fluid as result of drainage is not justified by either the clinical or experimental facts, provided infection is avoided. It must be borne in mind that the serous membranes of the brain and spinal cord have much less resistance to microbic infection than the peritoneum. The indications for operative intervention in acute meningitis may be formulated as follows:

1. Lumbar spinal puncture for the diagnosis as well as for the relief of cerebrospinal tension. Large quantities of fluid may be withdrawn at individual sittings in this way, and the sittings may be as frequent as the symptoms of cerebral pressure occur. A canula with a double opening at its lower end may be substituted for the needle in cases of rapidly recurring cases of hyperpressure. This must be very cautiously protected against the entrance of air and micro-organisms.

2. Continuous drainage of the spinal canal with secondary drainage of the ventricles may be established by a single laminectomy with the insertion of a very small tube. This, however, will be rarely indicated if the canula is used judiciously and to its best purpose.

3. Ventricular drainage, transoccipital, can be made through a musculocutaneous flap with a trephine opening in the occipital bone to the right or left of the median line, an inch posterior to the foramen magnum. The dura may then be excised, the bony opening enlarged, if necessary, with bone-forceps, the velum exposed, opened if indicated, or the tube may be inserted in the subarachnoid space without opening the velum. This can be utilized in connection with spinal puncture or laminectomy.

4. Transparietal ventricular drainage can be established by following the description given in the average work on surgery for the insertion of the tube into the lateral ventricles. A small trephine opening is all that is necessary for this procedure. It is an operation that will be rarely undertaken, as lateral ventricular is the least common of the meningeal infections. The duration of the drainage is governed by the general surgical principles of drainage. I prefer rubber to the metallic type of drain.

Technique for Irrigating and Draining the Subdural Space in Cases of Acute Leptomeningitis

The body must be so placed as to give the maximum convexity to the lumbosacral region. A U-shaped incision three inches long is made over the sacral region, the most convex points of the curve touching the last sacral tubercles, while the branches of the U run upward along the line of the postero-external tubercles of the sacrum.

The skin, subcutaneous tissue, fat, and muscles are divided down to the bone; the flap is dissected until the sacral foramena are exposed. With the bone-cutting forceps, one blade in the sacral canal, the laminæ are divided on either side until the sacral dura is exposed at the third body. This is easily accomplished. The sacral dura bulges very conspicuously in the field, and corresponds to the middle of the third sacral body, one and a half inches from the coccygeal tubercles (or lower postero-internal tubercles of sacrum). It forms quite a large sac or pouch, which is called the sacral cerebrospinal cistern (Fig. 21). Before opening the cistern it is advisable to aspirate the blood by a siphon, so as to have a clear view. In fact, the siphon should always be in action for the clearing of the operative field when deeply situated. This accomplished, the dural sac is split parallel to its long axis, sufficient cerebrospinal fluid allowed to escape to relieve the tension, and artery-forceps put on the opening so as to close it temporarily. At this step the region should be temporarily abandoned, the field well protected with a pad of sterile gauze, and the attention directed to the occipital region.

An incision is made parallel to and below the occipital ridge on one side down to the bone. The horizontal incision is joined by a vertical one along the middle of occipital bone, which is prolonged down to within one half inch of the foramen magnum. After dissecting and reflecting the musculocutaneous flap downward, the occipital bone is trephined either over the left or right cerebellar fossa, close to the middle line. The bone is comparatively thin at this point, and the openings easily made and enlarged to the desired size; bone removed. The dura at this point bulges conspicuously over what is commonly known as the cerebellar cistern. It is incised with a small scalpel, and the blunt point of a common intravenous transfusion outfit, which is connected with a fountain-can of normal salt solution, is introduced into the opening; the fluid is allowed to flow through the spinal canal with the desired pressure, readily regulated by the height at which the can is held. In order to demonstrate the certainty of the flow from the subdural cerebellar cistern into the spinal canal and down to the sacral cistern, it may be colored with carmine. The forceps temporarily applied on the sacral dura is removed and the irrigation instituted. Tubes can be inserted into both cerebellar and sacral openings and kept in place. The cerebrospinal fluid tension can be regulated by clamps on the tubes. The irrigation may not be needed, as a simple drainage with relief of pressure or pus tension is often all that is needed to conduct to a cure. It is the tension that favors absorption and tissue necrosis, and tiding over the primary acute pressure of the products of infection is life-saving.

If the foramen of Magendie is closed, a V-shaped piece of the velum can readily be removed with the scissors, establishing a direct communication from the fourth ventricle to the subcerebellar cistern.

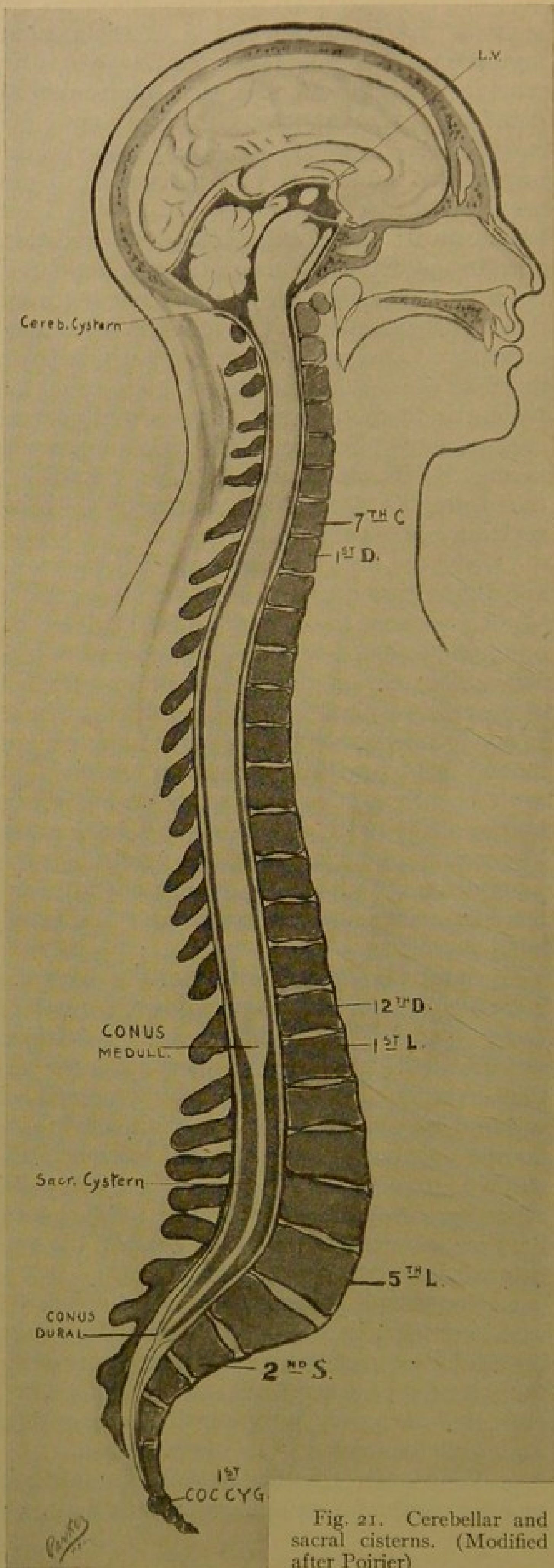


Fig. 21. Cerebellar and sacral cisterns. (Modified after Poirier)

This operation should be performed in every case of central hydrocephalus, whether of tuberculous or traumatic origin. Eleven years ago I performed this operation for the first time, and the patient, who was almost blind from a rapidly increasing optic atrophy, retained the then remaining fragmentary vision, was relieved of his periodical, maniacal headaches, and enjoyed perfect freedom from all of the symptoms during the succeeding years. This shows that when optic atrophy is threatened from a ventricular fluid retention, excision of the velum should be resorted to; it is neither difficult of execution internally, nor externally dangerous to the patient. Asepsis is a requirement to success. Central ventricular drainage cannot be maintained with the foramen of Magendie occluded. The velum should be divided freely, when this closure is suspected, in all cases of leptomeninigitis.

Spina Bifida

Spina bifida is a congenital malformation of the spinal column, meninges, cord proper, or ependyma, consisting of a protrusion either of the meninges or of meninges and spinal cord through a cleft, resulting from failure of coalescence of the borders of neural groove, or absence of the vertebral arches.

Embryology. The nerve elements of the spinal cord are of ectodermic origin exclusively. The cord is an invagination of the ectoderm, forming a groove which is widely open at first, gradually closing to form a tube—the neural tube. The mesoderm supplies the serous, bony, and muscular elements which arrange themselves around the neural tube. A malunion or absence of the involuting mesodermic elements which form the vertebral arches and spinous processes will leave a cleft through which the meninges alone, or together with the spinal cord, will protrude posteriorly, because of lack of support.

The defective coalescence of the spinal processes may be either partial or complete. In the first instance, the opening involves two or three vertebræ, either in the lumbar or cervical region, as these are the last to close. The lumbar is the more frequent. Complete absence of union is called "rachischisis totalis", and is an uninterrupted opening, extending from the cervical to the sacral regions. This variety, however, is extremely rare.

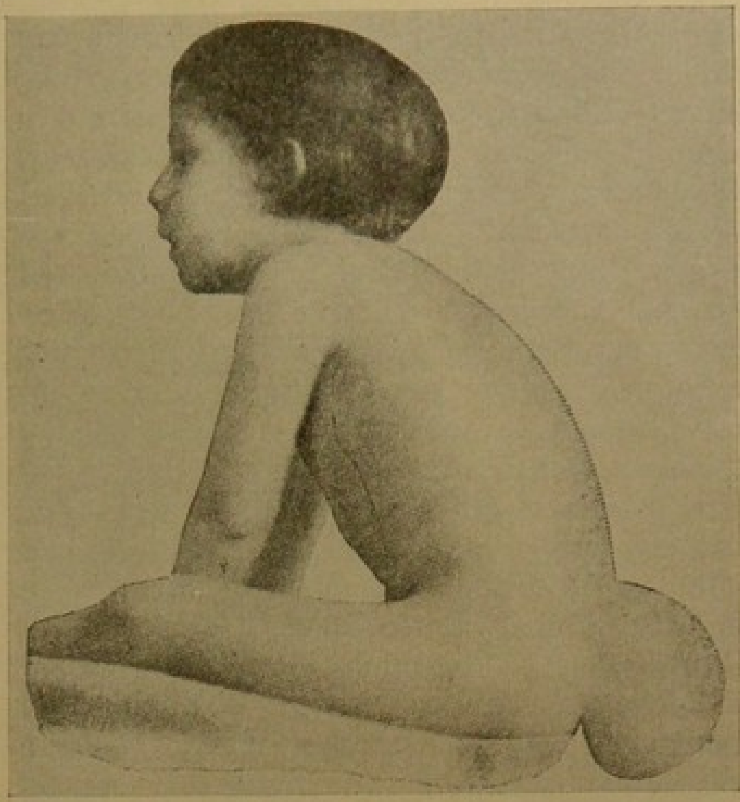


Fig. 22. Case of spina bifida (paraplegia.) (*System of Practical Surgery.* Von Bergmann, Bruns, Mikulicz).

In the circumscribed type of spina bifida, we find a protruding tumor, the size of which may vary from three to fifteen centimeters in diameter, having either a broad or pedunculated base. The sac usually protrudes posteriorly. Occasionally — very seldom, however — the sac bulges anteriorly; it escapes through the defective body or process, or through the spinal foramina, as shown in the following cases, and is discovered by accident. Several cases of this type are recorded in the literature. De Forest Willard reports a case, a child two months old, with a large cyst palpable through the right iliac fossa. The cystic tumor, which communicated directly with the spinal canal, bulged anteriorly when coughing. Its walls were the meninges, and the contents cerebrospinal fluid. Emmet reported a case, a woman 36 years of age, with a very large cyst extending down into the pelvis. It was aspirated through the rectum, and three quarts of fluid extracted. The patient died on the seventh day. Postmortem examination revealed a cyst formed of meningeal membranes, which protruded into the pelvis through the sacral foraminæ. Robinson operated on a case, a child 11 months old, who had a large cystic tumor occupying the entire right side of the abdomen. The patient had club-feet; there was no tumor to be found posterior to the sacrum. A diagnosis of parovarian or broad ligament cyst of fœtal origin was made. During the operation the surgeon found that the cyst communicated directly with the spinal canal, and that it was an anterior spina bifida. Patient died after ten days. Post-mortem showed extensive defects on the right side of the last dorsal and first lumbar vertebræ. The pedicles of the transverse process of this vertebræ were absent. The first two lumbar vertebræ had an undeveloped point of ossification on the right half, while the third lumbar vertebra had no point of ossification at all. The spinal column was curved laterally, the concavity being directed toward the left. There was a marked dilatation of the ependymal canal. Bryant reports a case of anterior spina bifida in a woman 25 years of age, who died from a trauma. The tumor was discovered accidentally.

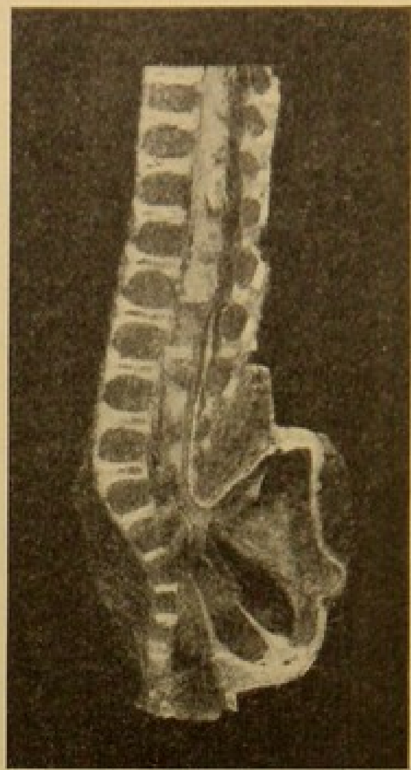


Fig. 24. Spina bifida (*meningomyelocele,*) showing cord and nerves crossing sac (Guy's Hospital Museum). (*International Text-book of Surgery*, p. 836).

Varieties of Spina Bifida. Dana distinguishes three varieties:

(*a*) Meningocele; a protruding sac composed of meningeal membranes and cerebrospinal fluid only. This may be either: 1. Anterior, abdominal, or pelvic; or 2. Posterior or dorsal tumor.

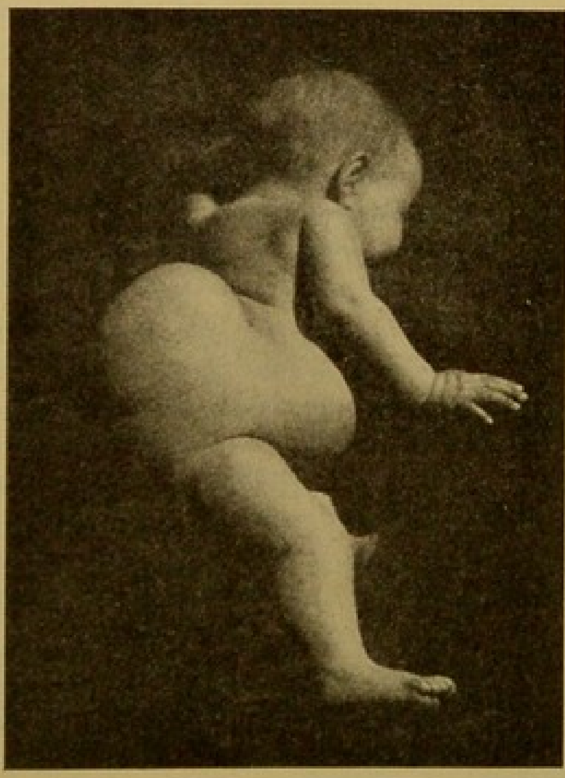


Fig. 23. Anterior and posterior spina bifida. (*Annals of Surgery*, 1904, p. 613).

(*b*) Meningomyelocele; hydromyelia; a tumor composed of meningeal membranes, cerebro-spinal fluid, and spinal cord, including cauda.

(*c*) Syringomyelocele; composed of meninges, cerebrospinal fluid, and spinal cord, with an

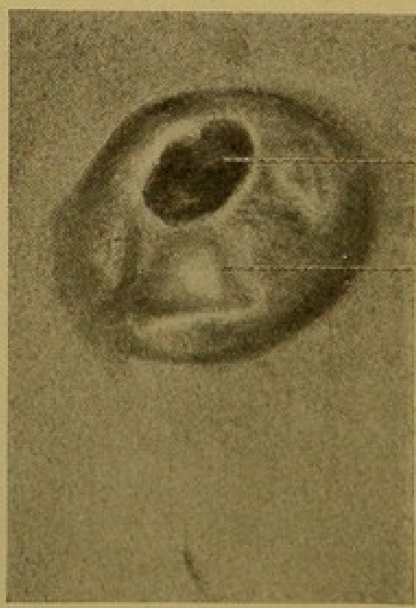


Fig. 25. Myelominingocele. (*System of Practical Surgery*. Von Bergmann, Bruns, Mikulicz).

enormous dilatation of the central canal or ependyma.

To these we may add spina bifida occulta, which is a cleft of the spinal column without any visible protusion of the contents of the spinal canal. In great defects, the cord is a velvet-like band containing blood-vessels and flattened nerve elements spread out under the meningeal covering. In circumscribed varieties, the sac may be covered with skin and hair, or simply with a thin epithelial layer which, together with the pia, form an epithelio-serous covering.

In the first variety of Dana, the cord elements may be intact. In the second, the cerebrospinal fluid lies in the subarachnoid sac. The nerve elements of the cord protrude into the sac in two thirds of the cases. The fibers are attached on the posterior and median surface of the sac, *and form a part of the cyst-wall.*

In the third variety there is a considerable dilatation of the ependymal canal with complete, or almost complete, pressure atrophy of the spinal cord at the point of dilatation, with a paraplegia below that zone.

De Y., aged 3 years.

Family and Previous History. Absolutely negative.

Present Trouble. Mass the size of a silver quarter was noted at birth, in median line of back, at about the level of the dorsolumbar junction. This at first was flattened and of a fluctuating consistency. A serosanguineous fluid discharged from the most dependent portion of tumor. This continued during the first year, but finally yielded to local treatment. The tumor mass gradually increased in size, but the growth during the past six months, i. e., before coming to the hospital, was very rapid. On entering the hospital, the mass was the size of a small orange, globular in shape, rather tense, fluctuating in consistency. There was no tenderness, nor evidence of inflammation in the tumor mass or in the adjacent skin. The head of the child from birth has been large, and tended to approach the hydrocephalic type. From birth the child presented a complete paraplegia with absolute motor paralysis, anæsthesia, and analgesia; also loss of sphincter control. Mother says the teething and talking began at a normal age; that is, as compared with her other children.

Diagnosis. Syringomyelic spina bifida. Three injections with Morton's fluid were made. Some reaction, but no appreciable benefit.

Decision. Cure of spina bifida would not materially benefit without curing paraplegia.

Operation Freeing of sac. Dilatation was central; that is, it was a dilatation from an accumulation of material within the lower canal portion and filum terminale portion of the ependymal canal, producing a

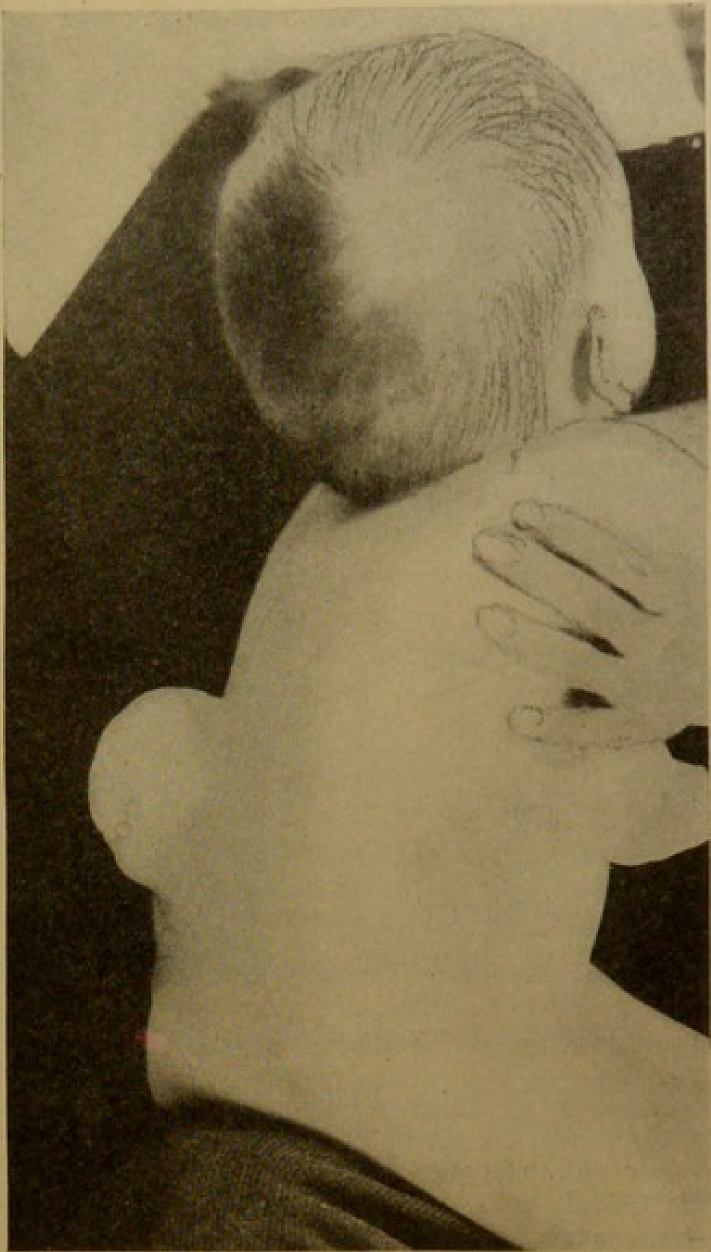


Fig. 26. The sac protruding posteriorly.

pressure destruction of the cadaul nerves just below the exit from the conus, and not roducing a destructions of the conus ganglionic cells or paxones. Careful disection was made, separating the dura and sac from other tissues down to the bone border. Cord was found dilated from center, with complete pressure destruction for an inch or more. Central cicatricial mass dissected out. Ends of caudal fasciculi, upper and lower, united with catgut suture, and the divided lateral nerves (lumbar plexus) attached to line of spinal suture. Electric test was then made, one pole at upper and one at lower end of cord, and with both poles on lower segment there was pronounced muscular response in both lower extremities. Dura then closed with single row of catgut sutures. Closure of opening in bony spine. Both spinous processes were fractured at the attachment to transverse processes and drawn to median line and united with heavy catgut sutures. Drain was placed (one eighth inch perforated rubber) beneath the line of suture. Aponeuroses of

Fig. 27. The sac protruding posteriorly.

quadratus drawn together over seam so as to retain lumbar muscle in center line. Skin-flaps supported by four silkworm-gut sutures through lead plates. Skin united with horsehair. Silkworm-gut drain. Cerebrospinal fluid was discharged for two weeks. Temperature 102° F. It, however, subsided, and case recovered. The feet changed in their temperature and color, and "life expression" was very materially noticed before the patient left the hospital December 20, 1905. Letter dated January 14th: "Patient progressing nicely; no motion in extremities; the legs and feet have an expression of life about them." Another letter, January 16th, informs me that the patient is in splendid physical condition. The tumor in the back has not reappeared, and the spine is apparently normal.

If the dilatation had been two inches higher operation for the cure of the paraplegia would have been worse than useless, and dangers of operation greatly increased, for in that position resection of sac and restoration of conformation of spine is all that could have been under taken or justifiable. It must be borne in mind that the sacral and lumbar nerves pass upward a considerable distance in the spinal canal before they enter the cord or conus and have their neurilemma.

Ætiology. The ætiology of spina bifida is very obscure. Occasionally, it is one of a series of congenital malformations. A plausible explanation for the ill-closure of the medullary groove is the traction by amniotic bands, preventing the union of its borders. Still, it appears to us that there is also an element of increased pressure from within, as in the anterior varieties failure of cleavage does not occur, but pressure seems to force the meninges through the foraminæ and bodies.

Symptoms. The general nourishment of the individual is usually below par. The mentality is defective. In cases of meningocele there are no motor or sensory disturbances. In meningomyelocele and syringomyelocele there are usually sensory disturbances, sphincteric derangement, and, not uncommonly, motor disturbances. In the latter variety, paraplegia is usually present. Associated with the motor and sensory disturbances, we find club-feet, spinal curvature, and other deformities.

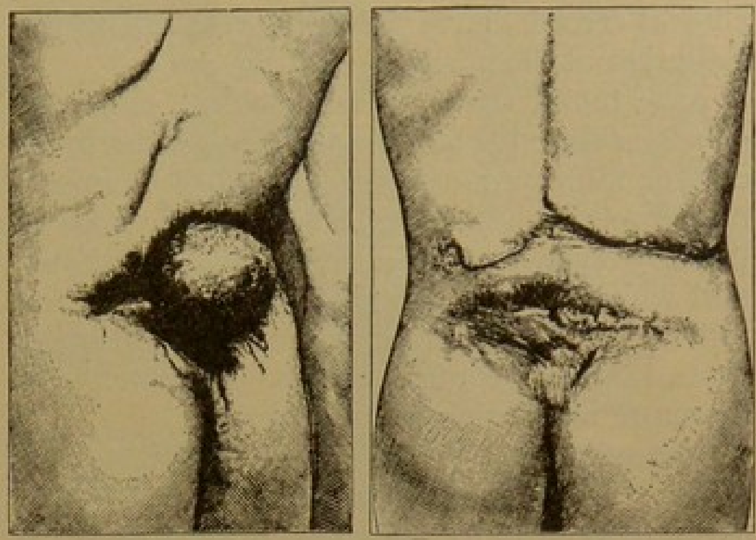
Diagnosis. If the spina bifida is posterior, the diagnosis is generally not difficult, except in spina bifida occulta. In this type the vertebral cleft is covered with skin and hair, and there is no protrusion of the cord or membranes. Patients affected with this variety, however, are not free from symptoms; we may find in them anæsthesia, club-feet, and perforating trophic ulcers. The cases of anterior spina bifida are impossible of diagnosis before abdominal section.

Treatment. Treatment consists either in injection or excision of the sac. Injection has long been and still is in vogue. Morton's fluid, which has the following composition, is the one generally used: Iodine, 10 grains; iodide of potassium, 30 grains; glycerine, 1 ounce. Injection is not free from danger, and its efficacy is very questionable. We used it three times in the case reported above, without effect, except a temporary disturbance of the central nervous system.

Excision of the sac seems to be the radical and more desirable procedure. The sac is best attacked from the side, because the fibers of the cord are more frequently spread and attached to the median portion of the posterior wall, within the line of embryonal cleavage. When the sac is pedunculated, it should be first freed from its attachment to the skin and neighboring structures, except its highest point, which is adherent to the skin; there, an elliptical cutaneous flap should be permitted to remain attached to the sac. When the latter is freed to a level with the cord, a provisional ligature should be thrown about its neck without tying. The sac should be opened, carefully examined, and if the cord is not protruding, the ligatures should be firmly tied; an overstitch of catgut should give it additional support. If the base is broad, the same procedure should be followed, except that after opening the sac and inspecting it, the ligature should be removed. It should then be amputated at its base and carefully sutured if it be a case of syringomyelia, and if the cord protrudes into the sac, the fibers of the cord should be freed from their attachment to the meninges and replaced in their normal position, and the opening in the sac closed with a continuous chromoform-catgut, or, preferably, kangaroo-tendon suture. If a complete syringomyelia exist, the cord should be amputated in both directions from the center of elevation to a normal anatomic zone; then a careful end-to-end suture of fibers should be made if the spina bifida be situated in the caudal zone. If above the caudal zone, the cord should be freed and replaced within the dural sac without suture, the opening closed, with no hope of motor improvement offered the patient. In localizing the muscles supplied by the distal fibers, the faradic current should be used. Our results have been most satisfactory after this technique, both immediate and remote.

Mayo-Robson uses a median incision. He also leaves a segment of the skin in place if separation of the dura from it is difficult. He advises that the meninges and skin should not be united on the same longitudinal line. In the clinic at Breslau, the meninges are united longitudinally, and the skin transversely. After removal of the sac, in order to prevent subsequent hernia of the contents of the spinal canal, the defect is repaired either by a muscular flap (Bayer) or by osteoplastic flaps (Selenko Boboroff). Mayo-Robson once transplanted periosteum and bones from animals for this purpose.

Prognosis. Of 647 cases of spina bifida which died in England in 1882, 615 were under one year of age. Of the 90 not operated upon, the great majority died within the first few weeks. Only 20 reached more than 5 years of age. The mortality in the treatment of spina bifida by injection of Morton's solution is twenty per cent. Statistics of the Clinical Society in London, which includes 71 cases treated by injection, showed 49 per cent cured, 5.4 per cent improved, 7 per cent unchanged, and 38 per cent mortality.



a. *b.*

Fig. 29. Meningocele. *a.* Before operation. *b.* Twelve weeks after operation. (*System of Practical Surgery.* Von Bergmann, Bruns, Mikulicz).

TUMORS OF SPINAL COLUMN, MENINGES, SPINAL CORD, AND CAUDA EQUINA

The progress in our anatomic knowledge of the spinal cord, both gross and minute, has materially advanced the surgery of tumors of the cord. The clear understanding of the process of degeneration and regeneration of nerve-tissues is the only scientific guide as to the feasibility of surgical intervention in cord tumors. The topographical diagnosis of the cord segments in relation to cutaneous sensation segments is to-day definitely established, and by consulting one of the well-recognized tables or charts of Sherrington, Head, Starr, Edinger, Kocher, Wichmann, or Seiffer, the clinician will come to definite, sometimes even mathematical, conclusions as to the anatomic location of a certain tumor.

One must acknowledge that the repeated post-mortems, compared with the ante-mortem clinical records, have played a great rôle in the evolution of diagnosis of cord tumors.

As a tumor damages nerve-tissue, either by compression, displacement, encroachment, or destruction, we must view the word "tumor" more broadly, and consider as such any mass of tissue which, anatomically, is an alien tissue, and which, by its presence, interferes with the function of a certain portion of the cord or its roots. Any swelling, abscess, cyst, etc., will therefore be considered as a tumor.

Schlessinger, who has thoroughly studied spinal-cord tumors, and who perhaps examined the greatest number of post-mortem records, found 151 cases of tumors of the cord, meninges, and vertebral column among 35,000 autopsies at the Vienna General Hospital. In 104, or two thirds, the spinal cord was disturbed either directly or indirectly. There are no definite ætiologic factors in tumors of the meninges or cord. Trauma, however, seems to be of some ætiologic importance, and has frequently been noted in reported cases. Children are prone to tumors of the cord and brain.

Classification of Spinal Tumors

Tumors of —

A. Spinal column: 1. Originating in its own structures; 2. Involving the spinal column by direct extension.

B. Meninges: (*a*) Extradural; (*b*) Intradural.

C. Cord *per se:*

D. Nerve-roots and cauda equina, including conus medullaris.

All of the tumors must be subdivided into: (*a*) Benign; (*b*) Malign: 1. Primary; 2. Secondary.

A. Tumors of the Spinal Column

We will not consider the pathology of tumors of the spinal column, because it is similar to those of the bony system in general, and as such does not belong to this section. Furthermore, because most of the tumors of the vertebræ exert about the same influence upon the cord as vertebral tuberculosis and osteitis, which have been considered in another place. They constitute extradural tumors, and will be discussed below.

We will make an exception in one, however, which demands special attention, and that is myeloma. It is a multiple tumor, involving the ribs, skull, pelvis, spinal column, etc., and has its origin from the cells of the bone-marrow. The multiplicity in this tumor is by no means an evidence of malignancy, as it has the pathologic peculiarity of appearing simultaneously in various places. Myelomic tumors of the spinal column act as extramedullary tumors; they have no special symptomatology, as the type of compression does not differ from the pressure exerted by other tumors, except for the albumosuria, or Bence-Jones's reaction, which is of great diagnostic value in myeloma, and must be considered pathognomonic. It might be advisable to mention here another class of tumors, which may encroach upon the nerve-tissues by their size, and that is the congenital sacrococcygeal tumors, parasitic tumors, or teratomata. These are irregular, pendulous tumors attached to the coccygeal region. They are composed of undifferentiated tissues, and very frequently contain rudiments of skeleton or other organs. To these we might add post-rectal dermoids and pilonidal cysts, which are remnants of the post-anal gut and neuro-enteric canal.

B. Tumors of the Meninges

(*a*) Extradural; (*b*) Intradural

(*a*) Extradural tumors are of many varieties, and one can gain great information on this subject by consulting Victor Horsley's work. He found, among 20 extradural tumors, 4 lipomata, 5 sarcomata, 4 tuberculomata, 3 echinococci, 1 myxoma, 1 fibrochondrolipoma, 1 fibrosarcoma, and 1 carcinoma. Echinococcus cysts are usually extradural. According to Horsley and Schultze, extradural tumors are less frequent than intradural. The symptomatology of the former will be treated later, in connection with that of intradural tumors. We can state beforehand, however, that they have this special characteristic, that the compression exerted upon the cord is slow, gradual, and continuous. In a case of Schultze, for example, the compression symptoms appeared about eight years after the diagnosis of an extradural tumor was made. Death ensues sooner in extradural than in intradural tumors, because of their greater malignancy. Muscular cramps, marked spasticity, tonic reflex cramps, are considered pathognomonic symptoms. This has been denied by Schultze, who found these symptoms to be present in many other affections of the spinal cord, and particularly in the multiple sclerosis.

(*b*) Intradural tumors are mostly benign. They originate from the inner surface of the pia or dura, seldom from the arachnoid, occasionally from a nerve-root; they are usually single. Sarcomata, neuromata, and parasitic tumors may be multiple. Intradural tumors are of moderate size, egg-shaped, and they are mostly seated laterally. In the caudal zone the intradural tumors attain considerable size. If a tumor, for example, originates from the segment of meninges, which forms the sacral cistern which has a capacity of six to eight drams, it may attain the size of a walnut or even larger. The tumors are usually primary. Among 38 intradural tumors, Horsley found 12 myxomata, 7 sarcomata, 7 fibromata, 4 psammoma, 4 tubercles, 2 parasitic and 2 syphilitic tumors. Occasionally we find a neuroma, a congenital lipoma in connection with spinal bifida, and also a myeloma. Echinococcus cysts are very seldom intradural. Cysticerci are occasionally found, but no special diagnostic symptoms. Once, Taube* found a lymphangioma of the pia growing intradurally. The chief symptom in intradural tumors is compression of the cord and its roots, and in this respect they differ very little from extradural tumors. The compression symptoms do not depend so much upon the size of the tumors, at least at the beginning, as upon their consistency and location. Some writers speak about stagnation of cerebrospinal fluid in the arachnoidal space, and also in the central canal, but it is very doubtful, to my mind, if this has special influence in intradural tumors. The result of continued compression upon the cord leads to œdema, softening, degeneration at the point of compression, of centrifugal axones below and centripetal above that area, and thickening of the meninges. It should be borne in mind, however, that the conductivity of the cord is still present to some extent, even after long-continued pressure. This is of great importance in determining whether or not a case in which there have been long-continued compression symptoms is still amenable to surgical treatment.

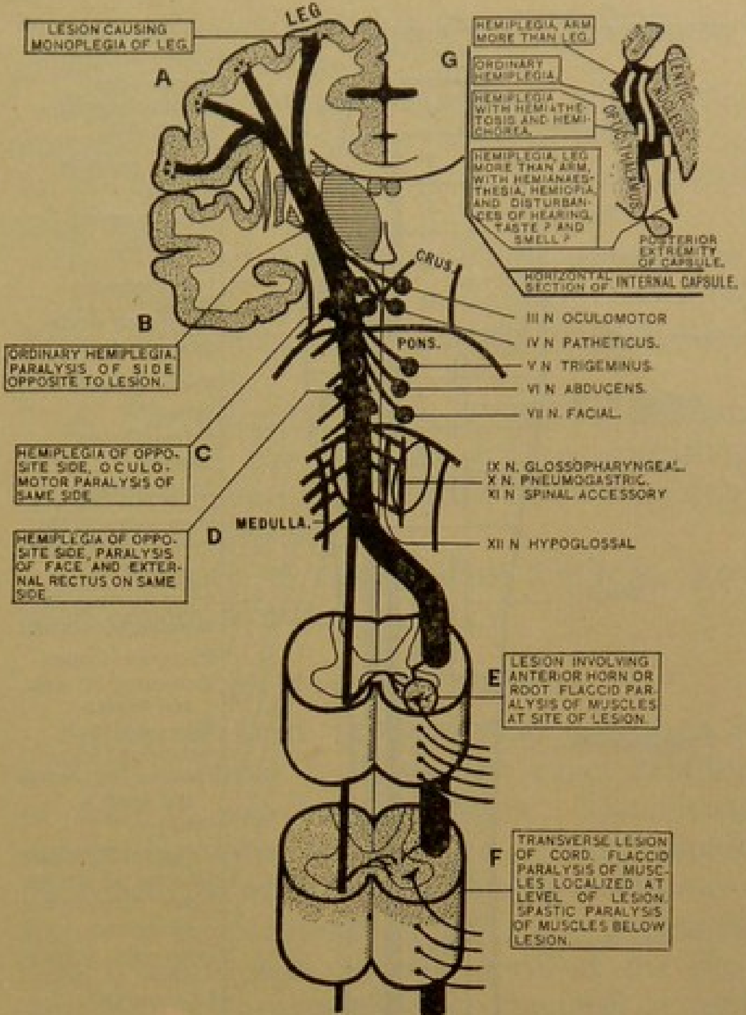


Fig. 30. Showing the effects of various lesions of the motor path in the brain and spinal cord. (Butler's *Medical Diagnosis*).

General Symptomatology of Tumors of the Meninges

Sensation. The chief sensory disturbances are neuralgic pains, hyperæsthesia, paræsthesia, and anæsthesia. The pain can be excited by pressing upon the cutaneous segment corresponding to the cord segment involved; it is increased on percussion and also on active and passive motion. The pain radiates along the nerve-trunks. It is of a severe character, and is either intermittent or remittent. It is usually the initial symptom, and always precedes motor disturbance. Unilateral pain means unilateral involvement, while bilateral pain indicates the growth of the tumor on both sides. The neuralgic pains, or so-called root symptoms, are the most conspicuous in the clinical picture of tumors of the meninges, as they precede the anæsthesia and motor disturbances. This is the reason why so many unfortunate errors in diagnosis have been made. We know how often patients suffering from tabes dorsalis, in

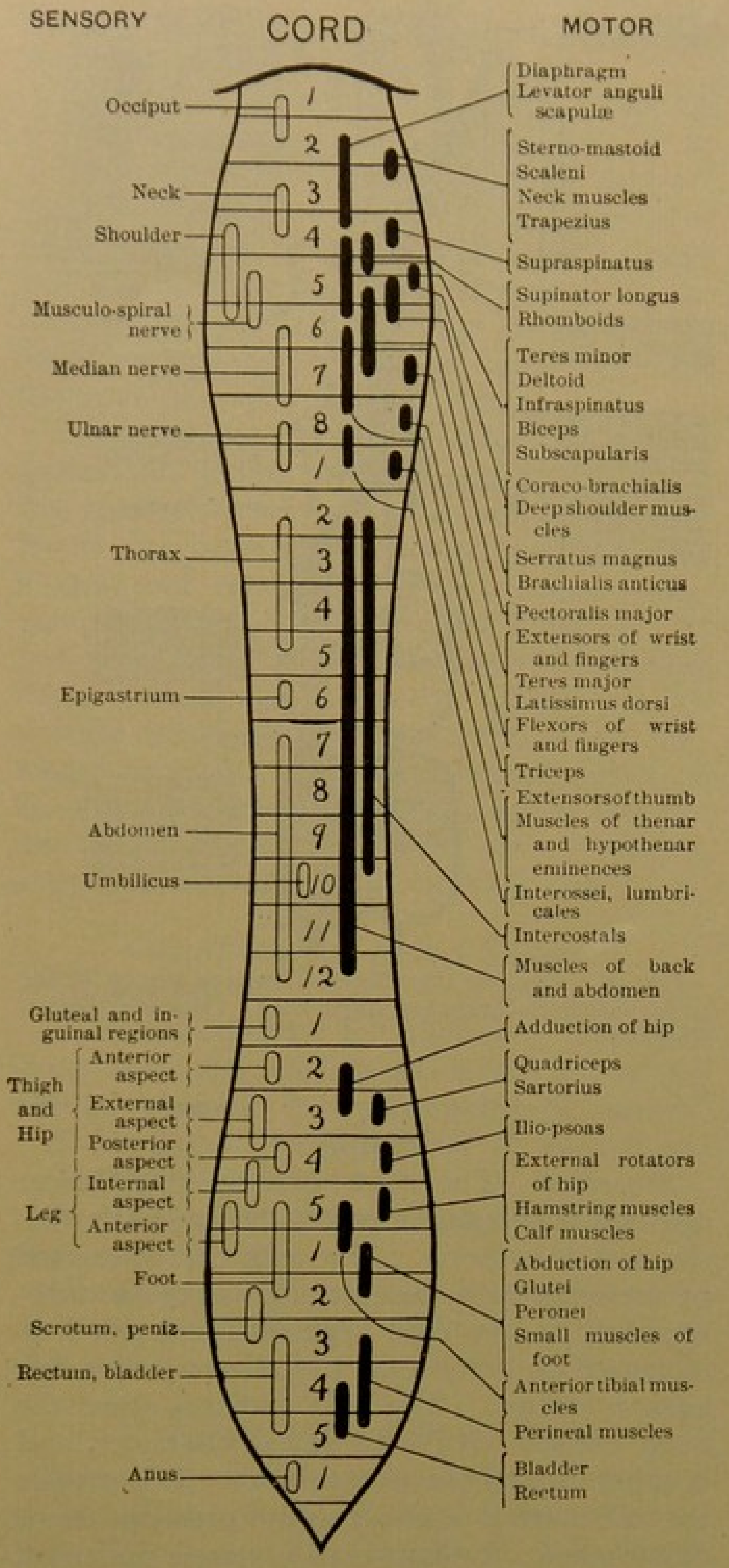


Fig. 31. Showing the location of the spinal segments for sensibility and motion. Based on Jakobi (sensory), and Starr, Mills, Sachs and Dana (motor). (Butler's *Medical Diagnosis*, p. 537)

which gastric crises are the only conspicuous symptom, are operated on for gastric ulcer or gall-stones. The long-standing root symptoms, in the absence of any other gross sensory or motor disturbances, frequently lead to wrong diagnoses. In both European and American neurologic clinics, it is not unusual to see patients, in which the neurologist endeavors to demonstrate a meningeal tumor, presenting a scar, either in the epigastrium, over the right hypochondrium or in McBurney's zone, indicating an unnecessary removal of the appendix, opening of the gall-bladder, or inspection of the stomach and duodenum.

Motor Disturbances. The usual motor derangements are muscular spasms, twitchings, atrophy, reaction of degeneration, and finally complete paralysis. At the beginning of the illness, the paralysis may be limited to one extremity, or it may be of the Brown-Séquard type. If the tumor involves both sides of the cord, it leads to complete paraplegia, which, together with the pains, gives the clinical picture of the paraplegia dolorosa of Cruveilhier.

The reflexes are at first exaggerated and finally abolished.

The trophic changes consist in herpes zoster, glossy skin, and decubitus. There are vasomotor disturbances, as congestion, pallor, cold skin, etc. The bladder and rectum are paralyzed. The spinal column, as a whole, is movable, except for a small section corresponding to the tumor. It is necessary, therefore, to exercise great care in the inspection of the spinal column during active and passive motion, and not to overlook limitation of motion of a definite segment, especially if there are other symptoms suggesting compression of the cord.

The local pain, the long-standing root symptoms preceding a slow and progressive paralysis of motion, constitutes the clinical picture of extramedullary tumor. The peculiarity of sensory and motor paralyses is, that they do not extend upward in spite of the gradual increase of the degree of the paralysis, or if they do so, they do not extend beyond one cord segment. This is perhaps the only clinical feature which may be considered pathognomonic of extramedullary tumors. It should not be forgotten, however, that sometimes the local sensitiveness and the preparalytic neuralgia may be absent in extramedullary tumors. The slow and gradual paralysis of motion,

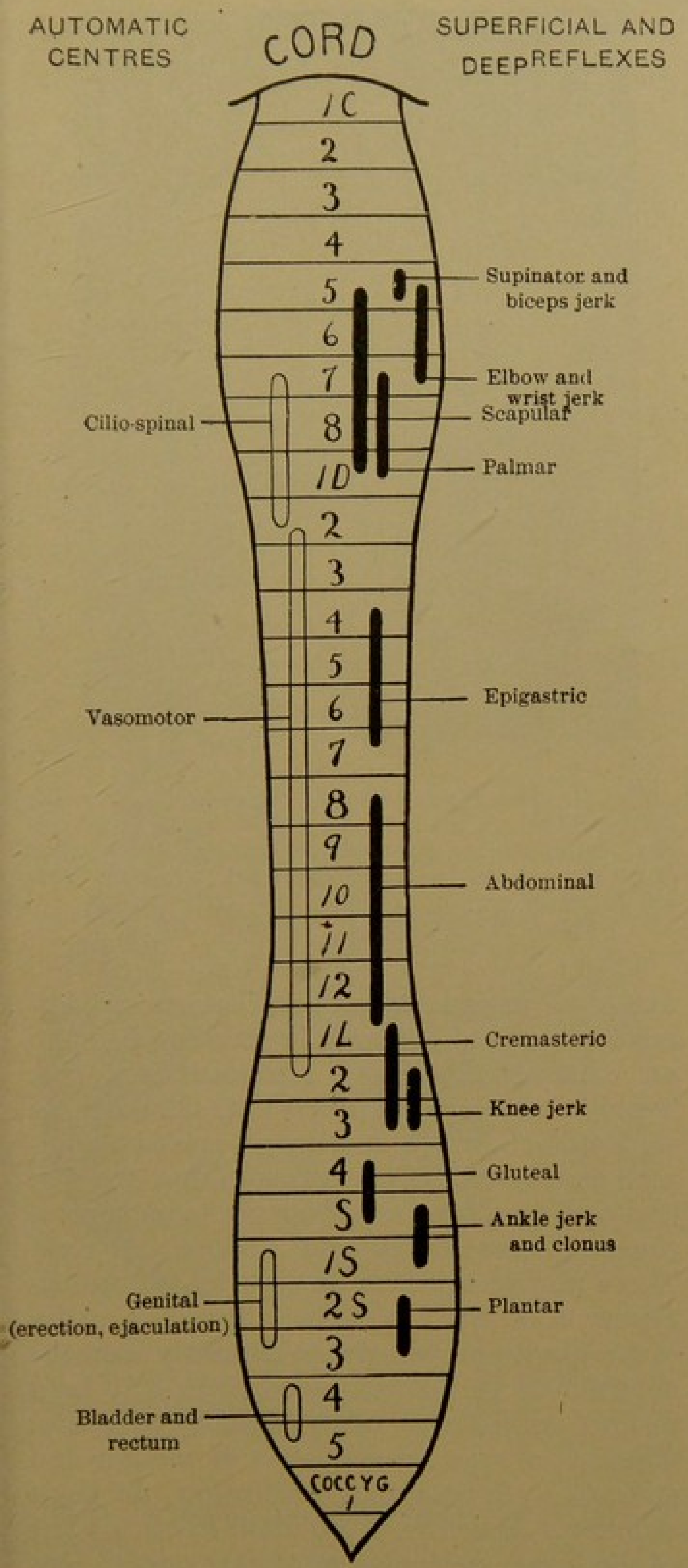


Fig. 32. Showing topography of anatomic centers and of superficial and deep reflexes. (Butler).

however, is highly suggestive of the presence of such tumors.

Oppenheim gives the following clinical picture as characteristic of extramedullary and endovertebral tumors:

(*a*) Long-standing neuralgia, which disappears as soon as the paralysis of motion sets in, but which is still noticeable on sneezing and coughing.

(*b*) Gradual loss of conductivity of impulses.

(*c*) Marked spasticity and exaggerated reflexes with persistence of complete paraplegia after the onset.

(*d*) The exaggeration of pressure symptoms due to local compression without spreading the pathologic process above or below the initial point of compression.

(*e*) Absence of tuberculous diathesis, and good general health.

Topographical Diagnosis

For an accurate topographical diagnosis, it is first essential to know the exact relation of cord to cutaneous segments. The charts referred to later on in this section should be consulted whenever attempting to locate a tumor of the meninges or cord. As a general rule, it can be said that the level of the sensory paralysis corresponds to the level of cord segment involved; yet, in spite of all this mathematical *data* on the exact sensory distribution, it has been clinically demonstrated that there is always danger of estimating the tumor too low. According to Leyden and Goldscheider, the tumor should be looked for, not at the upper level of anæsthesia, but two intercostal spaces above it. Sir Victor Horsley, Oppenheim, and Schlessinger, also, warn the diagnostician of this danger. Starr states, "It is safe to say that in the dorsal region the tumor is situated four inches higher than the level of anæsthesia on the back." A more conservative statement would be to say, two to four inches, instead of four. While the tables to which we refer are sufficient in the diagnosis of cord tumors, it might be of value to know the topographical *data* given by Sir Victor Horsley: "The second cervical nerve arises opposite the neural arch of the atlas; the third, opposite the interval between the second and third spinous processes. The lower four cervical nerves arise each opposite the spinous process of the second vertebra, a little above the point of exit of the nerve from the spinal foramen; the origin of the upper six dorsal nerves is on or about a level with the spinous processes of the third, while that of the lower six, with the spinous process of the fourth vertebra, are a little above their respective points of exit; the lumbar nerves arise in the neighborhood of the tenth and eleventh dorsal spinous processes, while the sacral arise between the eleventh dorsal and first lumbar spinous processes."

Differential Diagnosis

As tuberculosis of the spine is by far a more common cause of compression of the cord than tumor, and as it occurs so frequently, one must, first of all, exclude tuberculosis before considering a tumor and before differentiating between an extramedullary and intramedullary one. Syphilis must also be ruled out, although, as we stated elsewhere, it is by no means a common occurrence. Generally speaking, the root and cord symptoms are about the same in compression from caries and tumors. A clear X-ray picture made by an experienced radiographer is of great value in tuberculous caries of the spine, as it will always distinctly show the deformity. In marked cases, there may be a distinct kyphosis, or other palpable deformity. There is always absolute loss of motion and stiffness at, or both above and below, the point of lesion. Heredity, tuberculosis in other parts of the body, vesperal fever, facial aspect, peptonuria, and antecedent trauma are evidences of tuberculosis as a cause of the compression. The long-standing root symptoms preceding a progressive paraplegia are rather an evidence of tumor. A rapid, progressive paraplegia following root symptoms of short duration

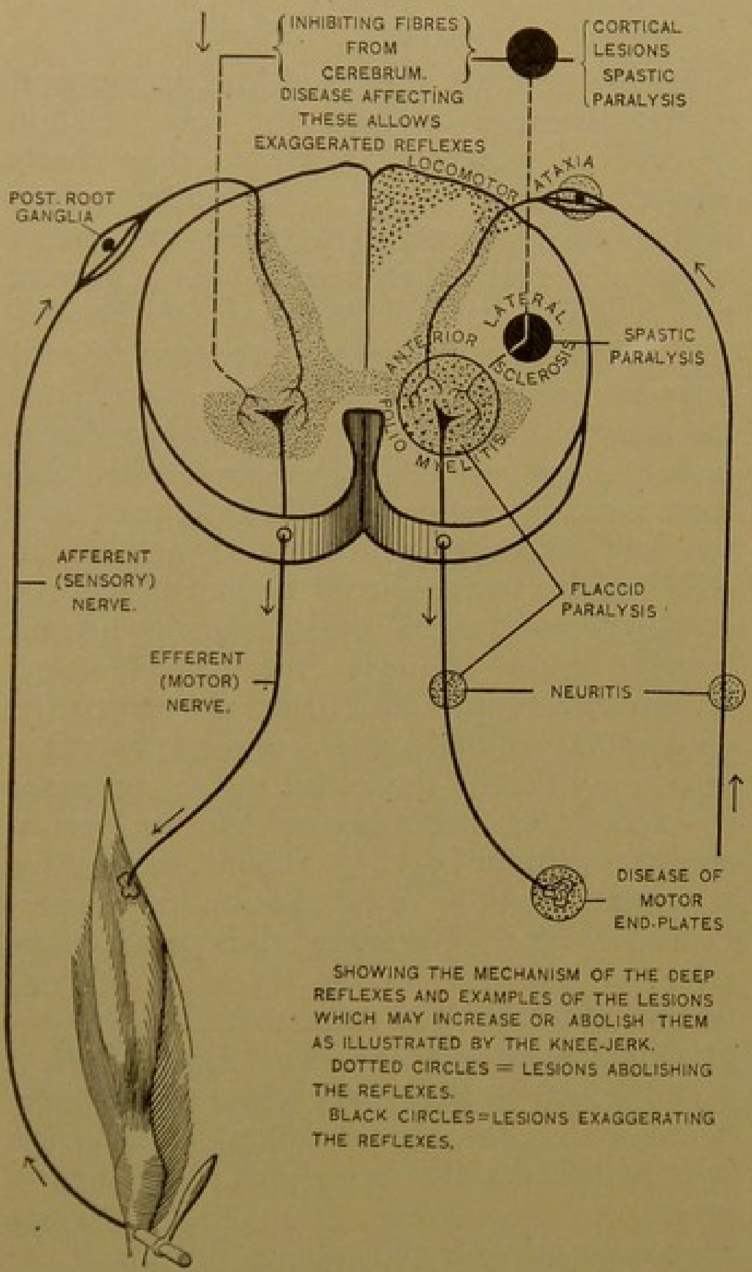


Fig. 33. (From Butler's *Medical Diagnosis*, p. 540.)

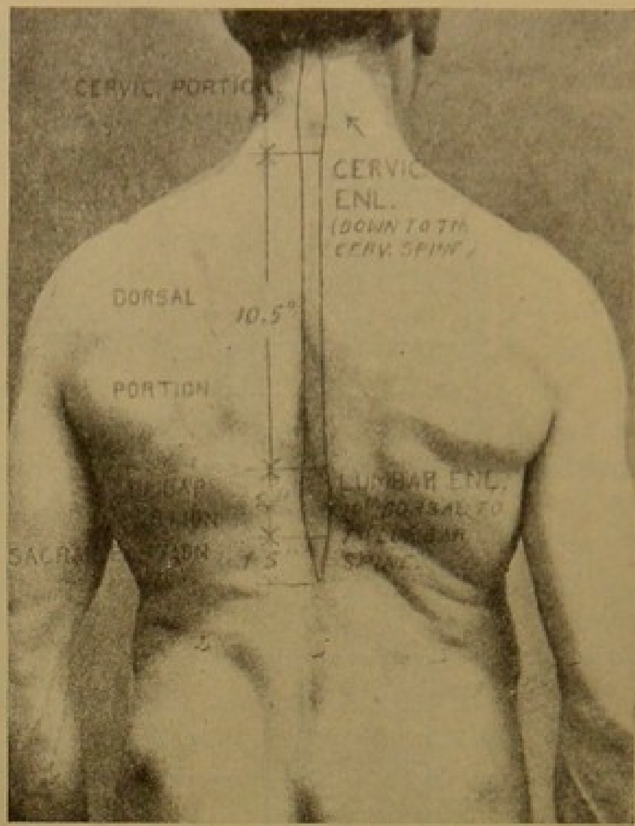


Fig. 34. Showing the relation of the spinal cord to the dorsal surface of the trunk; the relative length of the cervical, dorsal, lumbar, and sacral portions; and the position of the cervical and lumbar enlargements. (Butler's *Medical Diagnosis*.)

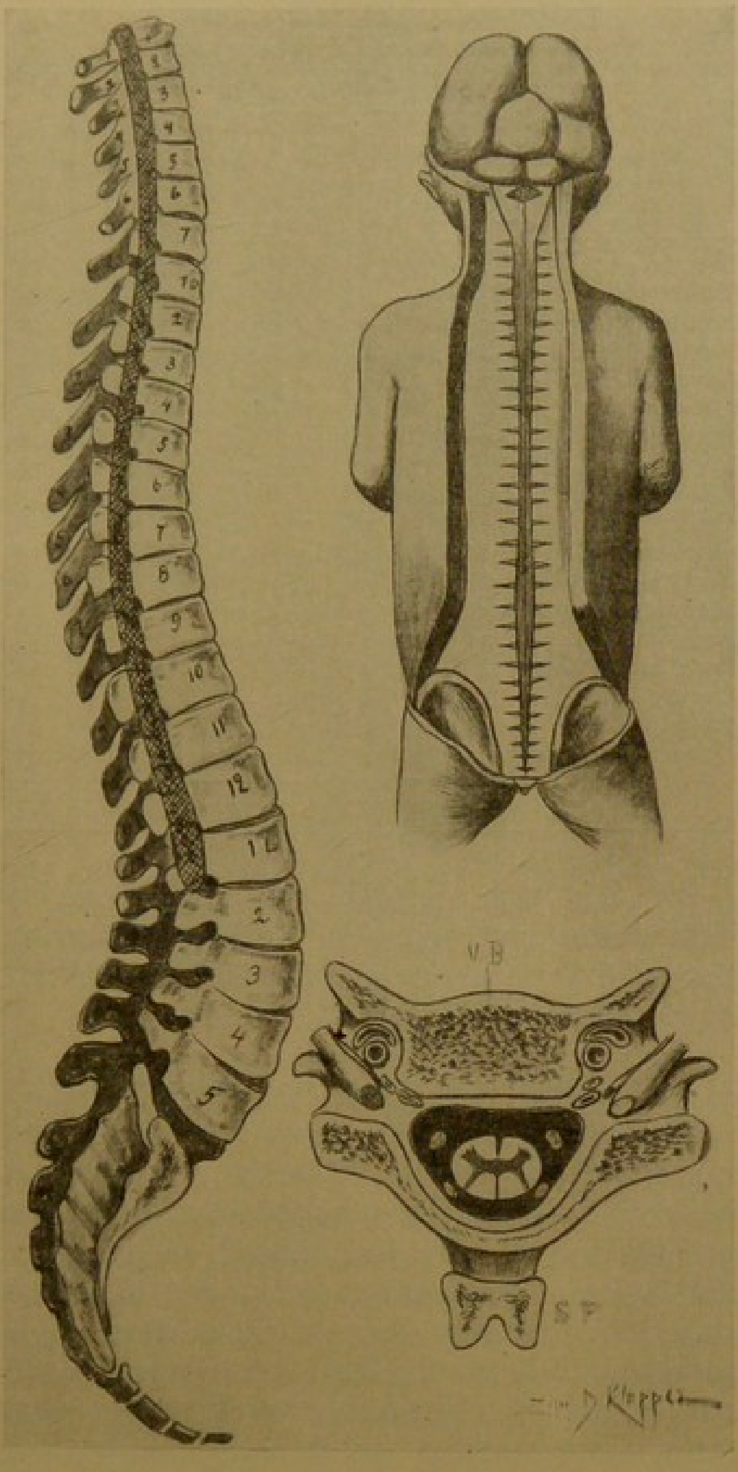


Fig. 35. *a.* Showing relation of cord segments to vertebral bodies (Church and Peterson). *b.* Showing cord of embryo reaching the coccygeal region (Testut). *c.* Cross-section of spinal column and cord (Testut).

speaks in favor of tuberculous caries of the vertebræ.

In a differential diagnosis between extradural and intradural tumors and other conditions, one must consider chronic pachymeningitis, meningomyelitis, and myelitis. In 1869, Charcot first described in detail the clinical picture of pachymeningitis hypertrophica cervicalis. The affection closely resembles localized tumors of the cord. The progress of the disease is slow, sometimes extending over a period of twenty years, and is usually syphilitic in origin. It involves the dorsal region, which is known also to be invaded by tumors. Osler described a case of pachymeningitis limited to the lumbar and caudal zone.

Meningomyelitis has usually a very rapid progress, except when syphilitic in origin. It is almost always associated with involvement of one or several cranial nerves, which is of great value in a differential diagnosis. In pure myelitic affections there is, as a rule, no pain, but it should be remembered that this may occasionally be the case in tumors.

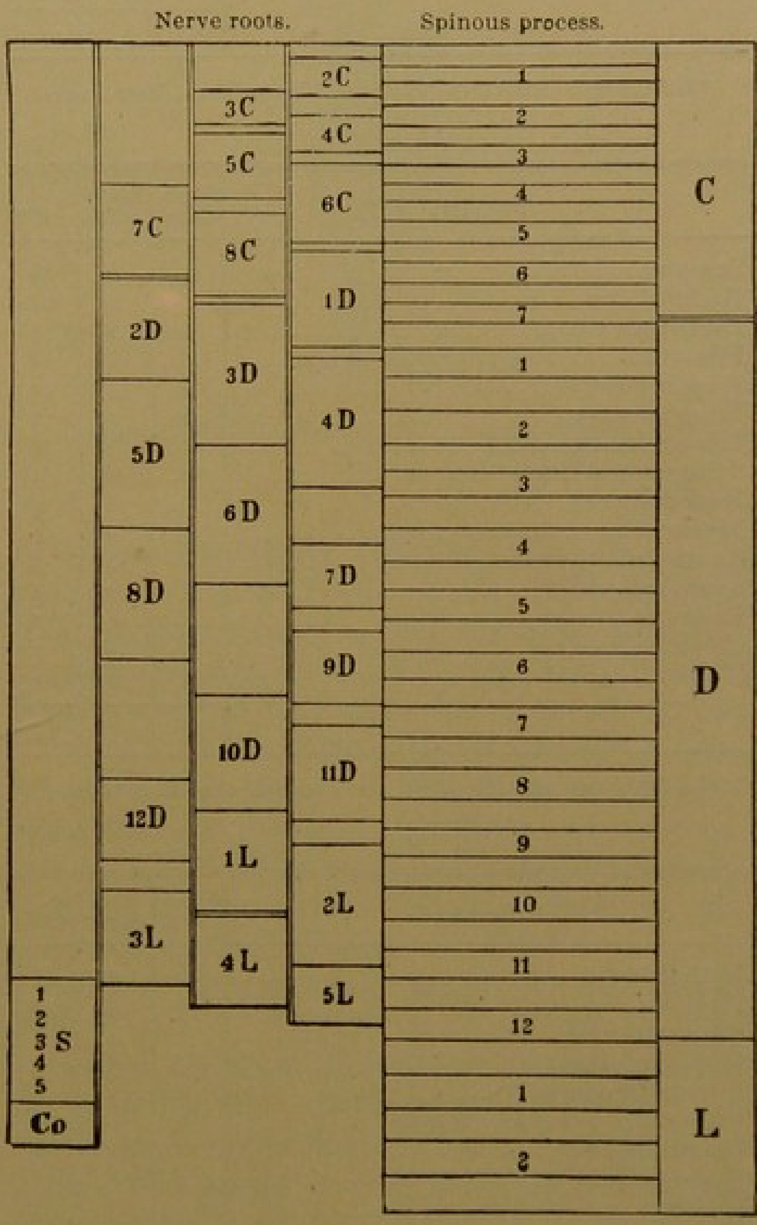


Fig. 36. Regions within which the point of exit of the nerve-roots from the cord may be found in relation to the spinous processes. (Reid.)

Tumors*	Extra-dural	Intra-dural	Total
Sarcoma	18	10	28
Hydatid cyst	15	1	16
Myxoma	1	12	13
Tubercle	4	4	8
Fibroma	1	11	12
Fibromyxoma	0	1	1
Fibrosarcoma	3	1	4
Fibromyxosarcoma	0	1	1
"Connective-tissue" tumor	2	0	2
Psammoma	0	8	8
Lipoma	5	0	5
Fibrochondrolipoma	1	0	1
Endothelioma	2	0	2
Myeloma	1	0	1
Exostosis	1	0	1
Lymphangioma	1	0	1
Chondrosarcoma	1	0	1
Carcinoma	2	0	2
Syphilitic gumma	0	2	2
Cysticercus	0	1	1
	58	52	110

*Williamson's tabulation of tumors. Med. Chron., Sept., 1902.

Tumors*	Extra-dural	Intra-dural	Total
Sarcoma	13	3	16
Hydatid cyst	12	0	12
Psammoma	0	4	4
Fibroma	1	4	5
Fibromyxoma	0	1	1
Fibrosarcoma	2	2	4
Fibromyxosarcoma	0	1	1
"Connective-tissue" tumor	2	0	2
Endothelioma	2	0	2
Myeloma	1	0	1
Exostosis	1	0	1
Lymphangioma	1	0	1
Lipoma	1	0	1
Chondrosarcoma	1	0	1
Cancer	1	0	1
	38	15	53

*Williamson's collection of 53 cases operated upon since 1887.

Tumors*	Extra-dural	Intra-dural	Total
Hydatid cyst	5	0	5
Fibroma	0	3	3
Fibromyxoma	0	1	1
Fibrosarcoma	2	1	3
Fibromyxosarcoma	0	1	1
"Connective-tissue" tumor	2	0	2
Psammoma	0	2	2
Exostosis	1	0	1
Sarcoma	4	1	5
Lympangioma	1	0	1
	15	9	24

*Williamson's tabulation of 24 tumors successfully removed.

TABLE OF SPINAL CORD TUMORS
(Allen Starr, Organic Nervous Diseases)

Variety	Intradural			Extradural				
	Medullary	Meningeal	Both	Meningeal	Not Meningeal	Single	Multiple	Total
Sarcoma	14	53	9	17	11	80	27	107
Tubercule	62	0	0	2	0	55	9	64
Echinococcus	0	5	0	39	0	8	36	44
Fibroma	0	20	2	5	0	15	18	33
Gumma	7	4	15	2	0	19	9	28
Glioma	20	0	0	0	0	20	0	20
Psammoma	0	18	0	0	0	18	0	18
Myxoma	0	7	0	4	0	11	0	11
Lipoma	1	8	0	0	1	8	3	11
Cysticercus	2	5	0	0	1	4	4	8
Gliosarcoma	0	3	4	0	0	0	7	7
Endothelioma	0	5	0	1	0	4	2	6
Melanosarcoma	1	0	3	0	0	1	3	4
Neuroma	4	0	0	0	0	3	1	4
Lymphangioma	0	1	0	1	0	1	1	2
Cysts	0	1	0	1	0	1	1	2
Cholesteatoma	1	0	0	0	0	1	0	1
Uncertain	13	12	2	3	0	24	6	30
	125	142	35	75	13	273	127	400

On this page we give two tables, one by Williamson[1] and the other by Starr,[2] showing the relative frequency, nature, and multiplicity of extradural and intradural tumors.

Course of Tumors of Meninges

The course depends upon the nature and rapidity of the growth. Generally speaking, it is slow; in time, the symptoms become more numerous and more pronounced. Tumors in the region of the cauda equina are of longer duration, probably because they have ample room for extension. For some unexplained reason, tumors in the lumbar and dorsal region grow more rapidly. This is especially true with sarcoma, which, according to Westphal produces infiltration more rapidly in the lower dorsal and lumbar region s than in the cervical region.

INTRAMEDULLARY TUMORS

The variety of intramedullary is not so great as extramedullary tumors. Glioma is the most common. Next in frequency comes tuberculoma. The order is reversed in children; in them, tuberculoma is the more frequent. Myxomata, sarcomata, etc., are usually secondary,

[1] Med. Chron., Sept., 1902.
[2] Organic Nervous Diseases, p. 390.

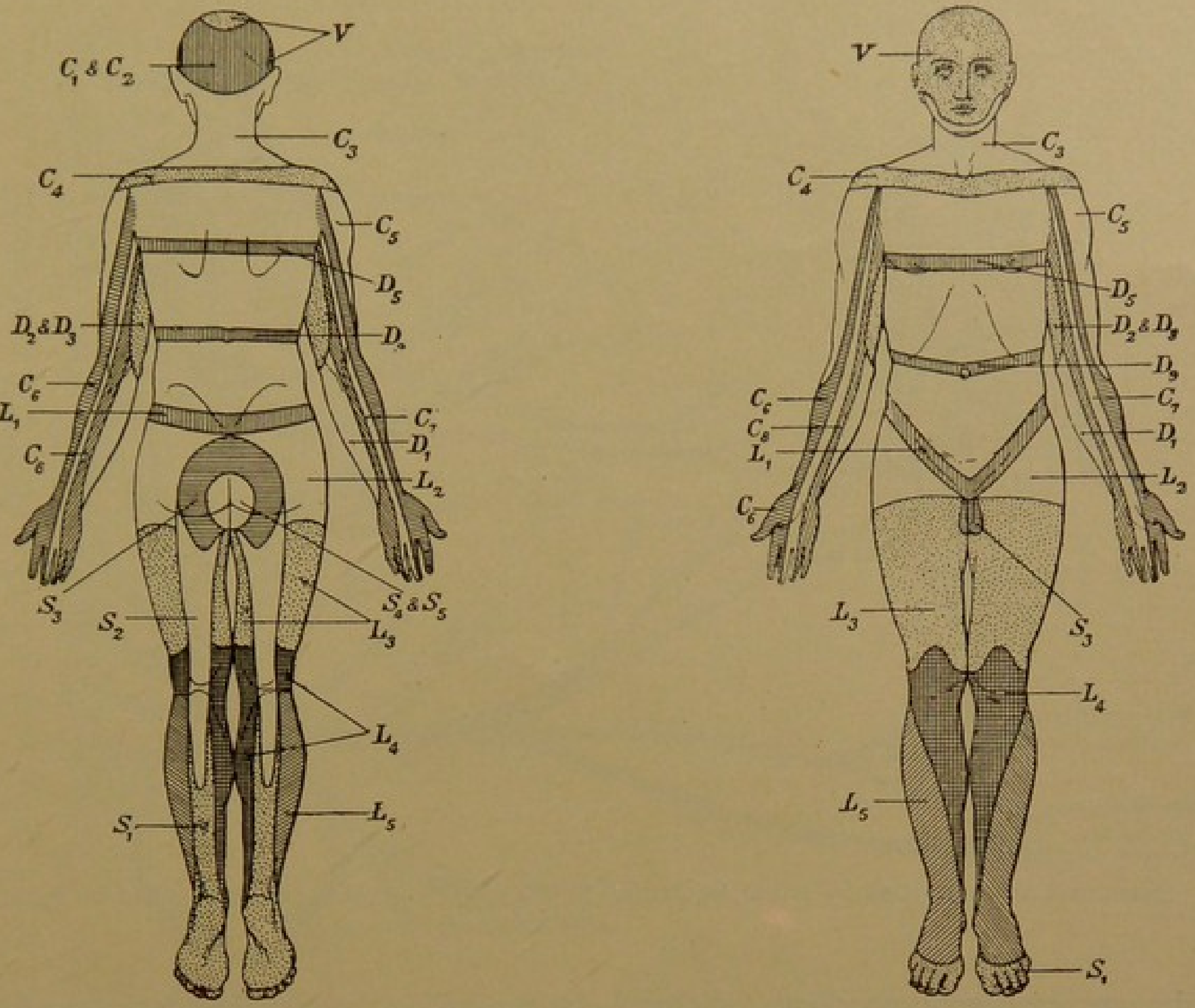


Fig. 37. Diagram of cutaneous areas of posterior roots (after Collier and Purves Stewart). (Purves Stewart, *Diagnosis of Nervous Diseases*).

extending from the pia into the cord. With the exception of glioma, most of the intramedullary tumors are fairly circumscribed; they are of comparatively small size, and usually oblong or oval in shape. Gliomata originate mostly in the vicinity of the central canal, and tend to spread along the entire length of the cord. Some, however, are relatively circumscribed. Tuberculomata are usually solitary tumors, varying in size from a pea to a hazelnut. They occur both in children and adults, but they are very rare after the age of 35. According to Schultze,[1] they have a predilection for the anatomic swellings of the cord. Gliosarcomata are occasionally found in the cord. They may be circumscribed, but more often diffuse, and invade the entire length of the cord and medulla oblongata. One might well classify them as mixed tumors, because sarcomatous tissue is of mesodermic origin, while glial is a modified ectodermic product. Glioma has often been mistaken for sarcoma, or classified as such. This, in my estimation, is an error in pathology, as glial tissue is a modified epithelium derived from the spinal-cord infolding of the ectoderm. It is strange that primary carcinoma of the cord has never been observed. When we stop to consider that the greatest percentage of cord tissue is epiblastic in origin, we must naturally wonder at the absence of carcinoma in the tissue of the cord proper.

Gumma is not so common as one might believe.

Among the unusual varieties of tumors of the cord we mention cavernoma, or tumor cavernosus and angioglioma. The tumor is composed of irregular cavities communicating with one another and also with the surrounding capillaries. It is interesting, not so much because of its unusual occurrence, but simply because cavernomata are common occurrences

[1] Deutsche Klinik, 1906, p. 949.

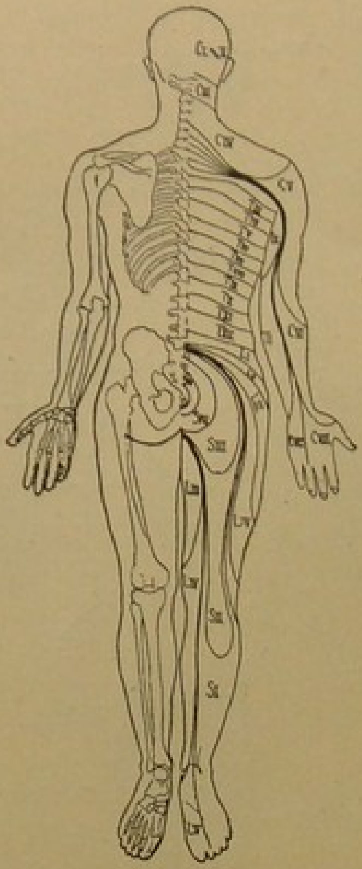


Fig. 38. Cutaneous areas supplied by spinal segments. (Osler.)

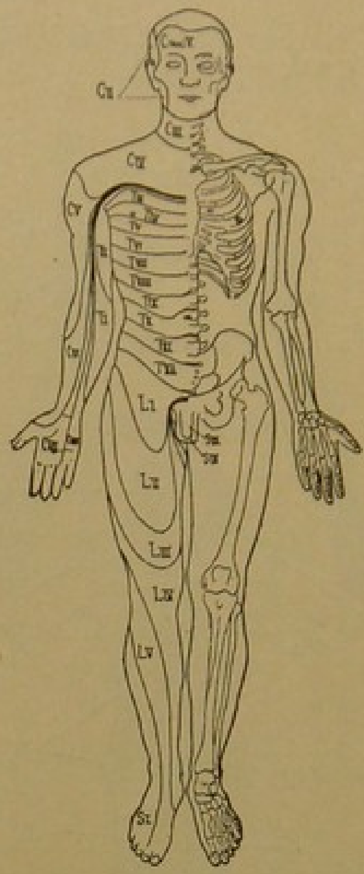


Fig. 39. Cutaneous areas supplied by spinal segments. (Osler)

in the skin, an ectodermic product. Richard Hadlich [1] reported a case.

The ætiology of cord tumors is only imperfectly known. Trauma, a blow upon the spine, commotion, and particularly pregnancy, have been considered ætiologic factors. The table below shows the comparative frequency of extramedullary and intramedullary tumors at varying ages (Schlessinger).

Age	Intramedullary	Extramedullary
Under 10 years.	Tubercle.	Lipoma or sarcoma.
10–20	Tubercle and glioma.	Multiple and metastatic sarcoma and hydatid cysts.
20–40	Tubercle and glioma.	Sarcoma and hydatid cysts.
40–60	Gumma and tubercle.	Solitary sarcoma, psammoma.

Symptoms. Clinically, it is practically impossible to differentiate between extramedullary and intramedullary tumors. Perhaps the only difference is the absence of local stiffness, and possibly the fact that flexion and extension of the spine do not increase the pain. The neuralgia is less severe in intramedullary tumors. The pain is not in the spine, but in the part of the body corresponding to the cord segment involved. As the result of compression, we have atrophy and paralysis of muscles with the reaction of degeneration. The reflexes are exaggerated at first, and finally abolished. Anæsthesia is present if the tumor involves the posterior columns. Trophic changes, as, for instance, herpes zoster, ulcers, and eschars, are usually present. Dissociated sensation is more commonly found in intramedullary tumors; if present in extramedullary ones, it is usually temporary (Starr). The lines of thermal tactile and pain sensation vary in the elliptical or polypoid spinal tumors, while in the compact, clean-cut neoplasms they are on the same level.

Differential Diagnosis. The long-standing pain preceding the paraplegia for months, and even years, favors the diagnosis of extramedullary tumors. In the absence of pain, and in the presence of the clinical picture in which there is only motor disturbance for a long time,

[1] Virchow's Arch., iv, 1903, bd. clxxii, p. 429.

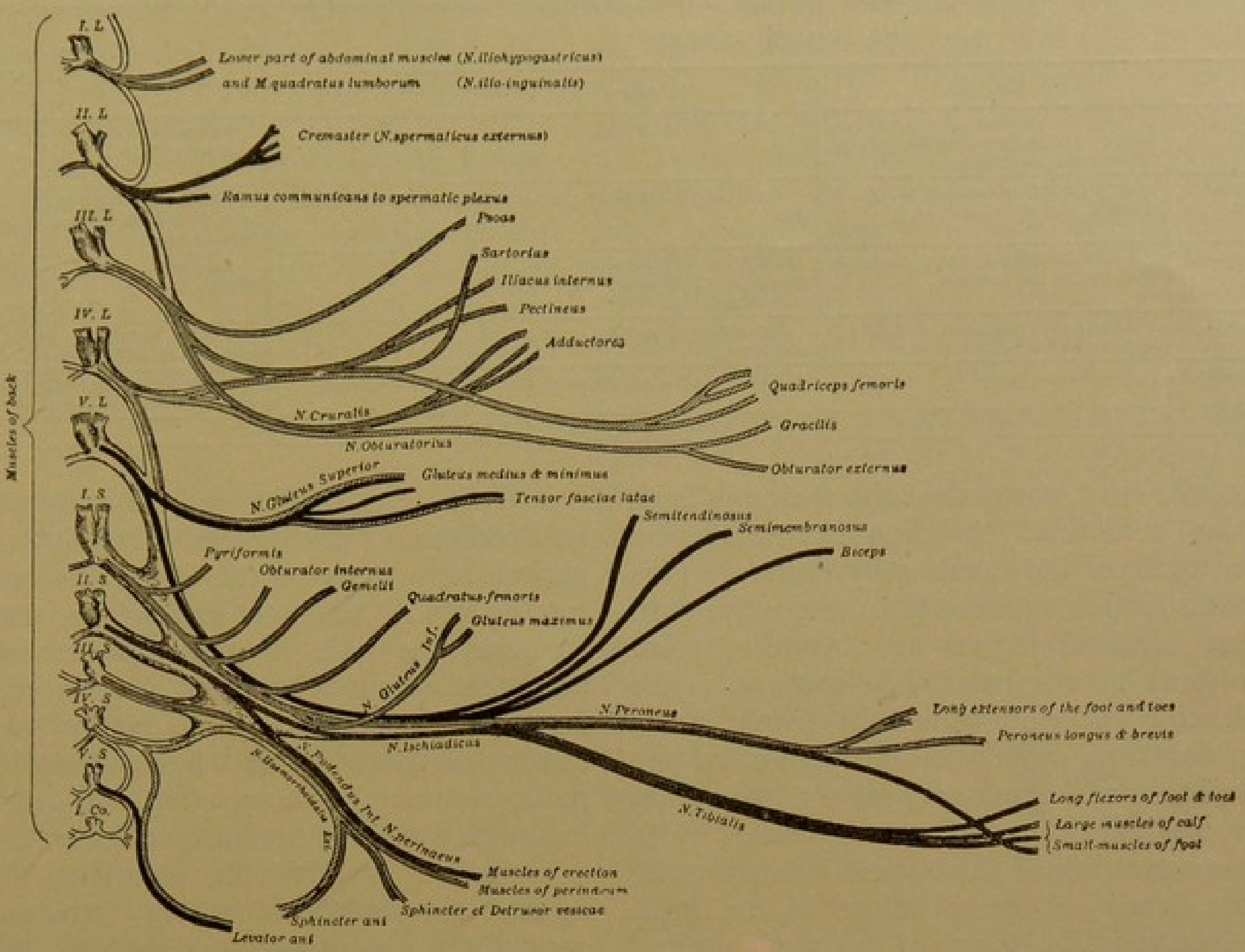


Fig. 40. Lumbosacral plexus and its branches (Kocher). (Purves Stewart, *Diagnosis of Nervous Diseases.*)

the tumor is, in all probability, intramedullary. The Brown-Séquard paralysis can be present in both extramedullary and intramedullary tumors.

TUMORS OF THE CONUS MEDULLARIS AND CAUDA EQUINA

Conus Medullaris

The tumors involving the conus medullaris *per se* or secondarily offer an almost similar clinical picture to that of caudal tumors, yet, in spite of all difficulties, an attempt at differentiation must be made, because the affections of the cord are always more or less amenable to treatment, while those of the conus are not, as a rule. The affections of the conus have been analyzed by B. W. Sippy. He states that in involvement of the conus there is a symmetrical impairment or loss of sensation. The integuments of the penis, scrotum, perineum, anus, inner aspect of the buttocks, and posterior surface of the thighs are anæsthetic. The sensibility of the penis and rectum may also be dulled. In advanced lesions, the bladder and rectal control may be seriously impaired, sexual power may be lost, and decubitus manifest.

Cauda Equina

We have emphasized in our section on anatomy that the cauda equina is a bundle of nerve-trunks inclosed in a meningeal tube, and, as such, have nothing in common with the spinal cord. A lesion of the cauda, from a surgical standpoint, must be regarded as a peripheral nerve lesion, and it would perhaps be more appropriate, from an anatomic standpoint, to treat this subject with the peripheral nerves instead of with the spinal cord. Before entering into consideration of lesions of the cauda, the reader must be reminded that the axones which make up the trunks of the cauda are neurilemmic from the time they leave the sulci of the cord, and, as such, they are potent of regeneration, after division and proper reapproximation of healthy axones. The trunks egress from the commissures at a very high

RAYMOND'S TABLE				SENSORY SUPPLY (According to Seiffer)
	MOTOR	SENSORY	REFLEXES	
Fifth lumbar. (Abd.)	Gluteus medius and minimus, tensor fasciæ, semimembranosus, semitendinosus, biceps.	Outer side of thigh.		External surface of leg, dorsum and sole of foot.
First sacral. (Ext. rot.)	Pyriformis, obturator internus, gemelli, gluteus maximus.	Posterior surface of thigh and leg.	Plantar.	External surface of leg, dorsum and sole of foot, outer part of posterior surface of leg.
Second sacral.	Tibialis anticus, peroneal muscles, center for erection.	Exterior surface of leg and foot, sensation of bladder and upper part of rectum.		Median part of posterior surface thigh.
Third sacral.	Center of ejaculation, ischiocavernosi and bulbocavernosi.	Sensation of urethra, penis, and scrotum.		Penis, scrotum, vulva, perineum, and region of buttocks.
Fourth sacral.	Muscles of bladder.	Perineum, sacral region.	Ankle-jerk.	An oval area including the region of sacrum as far as the anus.
Fifth sacral.	Levator ani, sphincter ani.	Coccygeal region, anus.	Anal.	

point of the cord, compared with their exit from the spinal foramina. A lesion or a tumor of a certain trunk of the cauda may therefore give symptoms similar to those of its respective cord segment situated higher up. Another point which must be emphasized beforehand is the considerable space in which the trunks practically float. A tumor in the region of the sacral cistern will grow very slowly, and attain quite a size before producing symptoms. With the understanding of these facts we may enter into the details of the affections of the cord. Those interested in lesions of the cauda equina are referred to the monograph of Dufour.[1]

Ætiology. Trauma is the most significant ætiologic factor in tumors of the cauda, and is of greater ætiologic moment than in tumors of the meninges and cord. According to Stanley Barnes,[2] the cerebrospinal fluid may carry sarcomatous products from a primary sarcomatous involvement of the meninges of the brain and cord, and disseminate them throughout, also involving the caudal. Neurofibromata originating from nerve-trunks are not uncommon in the region of the cauda. It is essential, before going into the symptomatology of tumors of the cauda, to refresh our memory concerning the motor and sensory distribution of the lumbar and sacral roots. To this end the tables of Raymond and Seiffer,[3] the latter referring to sensory disturbance only, are of distinct value.

The symptoms as given by Leyden and Goldscheider are:

[1] Contribution à l'étude des lésions des nerfs de la queue de cheval et du cône terminal. 1896.

[2] Brain, 1905, vol. xxviii, p. 30–51.

[3] Nouvelle Iconographie de la Salpêtrière, 1902, vol. xv, p. 471; also, W. B. Warrington, Lancet, 1905, p. 749.

1. Pain situated deeply in the sacral region and radiating into the legs. It is more widespread here than in tumors of the cord, and movements of the lower extremities increase it. In time, the pain becomes spontaneous and persistent, and is subject to exacerbations. Lassègue's sign in sciatica is always present in tumors of the cord, and is considered almost pathognomonic, in connection with other findings which suggest caudal involvement.

2. Sensory disturbance involving the perineum, anus, gluteal regions, scrotum, and genitalia. The sensory paralysis is slow and progressive.

3. Paralysis of the bladder and rectum. They are usually involved very early and precede the anæsthesia. In this connection, it is interesting to know that the bladder and rectum have a double innervation, as shown by the work of Langley and Anderson and others. One set of nerves comes from the lower dorsal and upper lumbar roots, through their white rami, to the sympathetic, and the other set from the sacral roots to the pelvic splanchnic, or nervi erigentes. The exact physiology of these two different sets of fibers is not known. Clinically, however, it has been observed that in affections of the conus medullaris, or its roots, there is motor and sensory paralysis of the bladder and the rectum. Erection is usually absent. The paralysis of sensation and motion are not always associated, as pointed out by Raymond. In a case of his there was incontinence of urine without loss of sensation. The reverse happened in a case of Van Gechuchten,[1] where the mucosa of the bladder was anæsthetic, but where there was no incontinence of urine. This

[1] Le Névraxe, 1902, vol. iv, fasc. 1, p. 93.

was explained by him on a possibility of the sympathetic acting independently of the central nervous system. In paralysis of the lower sacral segment of a cord or injury of its corresponding roots, the bulbocavernosi and ischiocavernosi muscles are also paralyzed, and we know that ejaculation and the expulsion of the last few drops of urine depend upon the integrity of these muscles. Van Gechuchten states that the diagnosis of uninjured conus and its corresponding roots can be made only with good function of these muscles and in the presence of the anal reflex.

4. Muscular weakness following long-standing pain. The motor paralysis is similar to the sensory paralysis; that is, it is slow, but progressive, and involves one extremity more than the other. Finally, the paralysis is complete, and it affects the type of paraplegia dolorosa.

5. Impairment of erection and ejaculation.

6. Reflexes, exaggerated at first, gradually diminished, and finally lost.

In the differential diagnosis of involvement of the cauda, one must, first of all, exclude sacral tuberculosis and fracture with pinching of the roots. But, far more important is differentiation between involvement of the root, a possible lesion of its respective cord segment, and affections of the conus medullaris. According to Sippy and Davis,[1] in diseases of the conus "the symptoms develop more rapidly; the various sensory disturbances may not be alike; pain and temperature sensation are more frequently seriously affected than tactile sense." Severe pain is absent, provided the conus lesion does not involve the cauda fibers. Disease of the conus is more likely to cause decubitus than caudal disease. The most important symptom of caudal disease is pain. A conus lesion may be associated with pain, provided the caudal meninges are also involved. The absence of pain is indicative of conus lesion.

Therapeutics of Tumors of the Meninges, Cord, Conus Medullaris, and Cauda Equina

In all cases of tumors it is advisable to give antisyphilitic treatment for a period of three or four weeks. If no amelioration is obtained, no further attempt should be made. Gummata are not common, as can be seen from Schlesinger's table of four hundred cases of tumors, of which twenty-one were syphilitic. These statistics show that only one case in twenty should be relieved by this treatment. The policy of "benefit-of-the-doubt" treatment is very frequently fatal, because much precious time is lost. It should also be remembered that a mass resulting form tuberculous decay may form a circumscribed granuloma acting as a tumor and pressing upon the anterior portion of the cord. If such a tumor does not improve by medical and hygienic treatment, immobilization of the spine and relief of local pressure, then the granuloma must practically be regarded as a tumor, and, as such, removed by laminectomy. The next step in the consideration of surgical intervention is to ascertain whether a tumor is single or multiple, benign or malign. It can readily be understood that multiple tumors situated in distant segments are not amenable to treatment. Malign tumors should be removed only if circumscribed. The final and most important of all is to know which tissue is involved and to what extent.

[1] Jour. Am. Med. Assoc., March 19, 1904.

Division of Posterior Roots and Crushing of Spinal Ganglia in a Case of Severe Neuralgia Following Amputation

Dr. G. W. F., German, physician, aged 61.

Family History. Negative.

Personal History. Born and always lived in Pennsylvania. Habits regular. Married; no children.

Previous Illnesses. Diseases of childhood. Had an acute illness 25 years ago, diagnosed as typhoid fever; was sick 5–6 weeks at this time; recovery without complications. At the age of 14 was struck by a steer just above and to outer side of right knee. Patient was able to walk after injury. Complained of considerable tenderness on this area; had considerable pain when walking. The acute pain and tenderness soon subsided, but never entirely left. About one year later, pain and tenderness began to be more marked, and the knee began to swell. Patient does not remember whether he had chills and temperature at this time or not; thinks he had a chill. The skin over one knee retained its normal color; it did not become red, or feel hot to touch. The pain, tenderness, and swelling gradually increased, and he consulted a doctor, who amputated the limb, July 9, 1865. Amputation took place about four years after swelling began.

Present Trouble. For 25 to 26 years following amputation, patient was free from trouble. Then commenced to have pain, at times crushing and again shooting in character. The pain seemed to be located in the heel and dorsum of the toes of amputated foot. Did not include upper surface of the foot or the limb. The attacks would last 5 to 10 minutes, and at first occurred every 6 or 7 months. They gradually increased in frequency and severity for next 18 months. Since then they have been nearly constant. Has some tenderness over stump. For the past 18 months used morphine constantly; takes 6 to 8 grains daily; never less than 6 grains since November, 1906.

In September, 1902, was re-operated on, and some of the nerve in the stump removed. Was free from pain for three months. Did not use morphine in this period. Then pain returned.

In January, 1905, was again operated on, and more of the nerve removed. Was free from pain for three months again; he did not use morphine; then pain returned. January 25th, had sciatic nerve injected with 75-per-cent alcohol. Pain was very severe after last injection, and continued ever since.

His general health is good; appetite good; bowels regular; states they have always been regular (in spite of morphine); no gastric or urinary symptoms; no loss in weight.

Operation. A U-shaped incision was made over the sacrum, the vertical branches of the U being parallel to the external tubercles of the sacrum, while the curved portion united the last two tubercles. [For more accurate details as to the cutaneous incision, see section on Meningitis.] The skin, subcutaneous tissue, and fat were divided to the bone; then with a heavy bone-cutting forceps the sacral foramina were opened, and the osteoplastic flap reflected upward. There was considerable bleeding, which was partly checked by pressure made by an assistant, and partly by siphoning the blood with an ordinary siphon, such as is used in cystoscopic work. The respective posterior ganglia and roots were exposed, the ganglia were crushed, and the roots severed from their respective ganglia. After controlling the hemorrhage, a small drainage-tube was introduced, the osteoplastic flap placed back in position, and cutaneous incision closed. This was the only logical operation for the relief of the severe neuralgic pains in the amputated toes, which made the patient's life so miserable. Unfortunately, the patient developed signs of pulmonary embolism, and died the next day.

SURGERY OF THE PERIPHERAL NERVES

SYNOPSIS

Introduction.

- Classification of nerves.
  - Cerebral.
  - Cranial.
  - Spinal.
  - Sympathetic.
- Anatomy of nerves (myelinated and non-myelinated fibers).
  - Macroscopic.
  - Microscopic
    - With sheath of Schwann (neuroblastic cells). — Neurilemmic
      - Cranial (except olfactory and optic).
      - Spinal.
      - Cauda equina.
      - Sympathetic.
    - Without sheath of Schwann — Aneurilemmic.
      - Nerves of special sense.
- Trophic centers of nerve-fibers.
  - Special sense.
    - Optic.
    - Olfactory.
    - Auditory. } Mixed nerves.
    - Gustatory. } Mixed nerves.
  - Sensory.
    - Tactile.
    - Heat.
    - Pain.
  - Motor.
    - Upper cerebral neurones.
    - Lower, spinomedullary neurones.
  - Sympathetic.
  - Vasomotor, caloric, and trophic centers

NEURONE DOCTRINE.

- "Central School."
  - Historical.
  - Conception and definition.
  - Anatomic and pathologic value.
  - Arguments sustaining it
    - Embryologic.
    - Histologic.
    - Clinical.
    - Pathologic
- "Peripheral School."
  - Neurofibrils. Partisans: Apathy, Bethe, Nissl, Kennedy, Ballance and Stewart et al.
  - Arguments sustaining it. Incontrovertible evidence of the importance of the neurilemmic cell nucleus.
- Division of peripheral nerves, followed by reunion.
  - Immediate
  - Remote.
- Division of cauda equina, followed by reunion.
  - Experiments.
  - Clinical cases.
  - Future of surgery of the cauda
- Essential conditions for reunion of peripheral nerves.
  - *a.* Accurate apposition of ends.
  - *b.* Removal of all scar tissue between and around the ends.
  - *c.* Perineural not paraneural transfixion.
  - *d.* Protection of point of union with local soft tissues or animal membranes. (Cargile membrane; egg membrane; tape saturated with paraffine.)
  - *e.* Asepsis.
- Clinical results

The nervous system is the most recent field of surgical invasion, and this only in a desultory manner. We feel that the nervous system offers sufficient inducement for surgical intervention to demand the surgeon's most careful, systematic, and analytic efforts, both experimental and clinical. Indeed, enough has already been accomplished to establish principles which must ultimately lead to great results. Modern investigations in the embryology, histopathology, and physiology of the spinal cord and nervous system have demonstrated the processes of degeneration and the potency of nerve tissue in repair, so that its surgery can now be based on definite histologic knowledge. Physiologic axioms are well grounded on established laws and principles. The surgery in the preantiseptic period was limited to unions of divided nerves and the extirpation of neuromata. We feel it can, in the logical course of events, be predicted that the lesions of the peripheral nervous system will become more and more amenable to surgical intervention. The foresight of Billroth, in his expression, "Die innere Medizin muss mehr Chirurgisch werden," impresses us forcibly. We may justly now include in this category, traumatism, epilepsy, facial paralysis, anteropoliomyelitis, atrophy and dystrophy of the extremities, neuritis, and other nerve lesions. The neurologic field has a great future. Autogenetic regeneration is creating a revolution in neurology. The controversy between the central neurone, and peripheral schools is waged by the most careful, analytic, and searching investigators; it has thrown much light upon the process of regeneration, which is the key to neurologic surgery. Two diametrically opposing principles cannot survive in science.

ANATOMY OF NERVOUS SYSTEM

Divisions

1. Central nervous system. Upper motor neurone.

2. Peripheral nervous system. Lower motor neurone.

Peripheral Nerves. A nerve-trunk is a collection of axis-cylinders originating from cells or neurones, situated either in the cord and brain or in the spinal ganglion. The axis-cylinders are protected by sheaths and connective tissue, and supplied by nervi nervorum, blood-vessels, and lymphatics.

Protective Sheaths. (*a*) Sheath of Schwann. (*b*) Myelin.

Sheath of Schwann. In embryonal life, the sheath of Schwann is represented by elongated cells which apply themselves end-to-end and precede the formation of the axis-cylinders. By the end of the first month, they form a sheath which completely surrounds the axis-cylinders. The sheath is not continuous. Ranvier has shown that it is interrupted at intervals on distinct segments, each of which represents the *parent* connective-tissue cell, and contains a nucleus which is the original nucleus of the parent connective-tissue cell.

Myelin. Myelin is a homogeneous substance, perfectly transparent in healthy nerves. It stains black with osmic acid and silver nitrate. It has been said by some that the myelin is a product or secretion of the sheath of Schwann.

Axis-cylinders. Déjerine (France) describes the axis-cylinder as an aggregate of fibrils. Leydig, Nansen, and others are of the opinion that the reticulated aspect of the axis-cylinder is nothing else than a continuation of the reticulum of the cell-body. Schafer describes the axis-cylinder as a bundle of distinct tubes, each preserving its identity. Lenhossek believes it is composed of a homogeneous substance, in contrast to the reticulated aspect of the body-cell.

Physiology of Nerves. The "nerve impulse," a very vague expression, has been discussed by the greatest physiologists, without any possibility of a definite comprehension of what it is or what it means. Déjerine believes that impulses are equal, identical in all neurones, but that differentiation depends entirely upon the structure to which the arborizations of nerve fibrils go ultimately.

Donaldson: "Nerve impulses are the result of chemical changes in the cytoplasm of the cell."

Schäfer and Horsley showed that neurones have a rhythmic discharge at the rate of ten times a second.

Distinction is made between *irritability* and *conductivity* of a neurone or its axis-cylinder.

A fiber, either divided or crushed, loses its power to respond to stimuli, but retains the power to conduct impulses from the cell-body. In other words, loss of irritability does not imply loss of conductivity.

Wallerian Degeneration. By Wallerian degeneration is understood the disappearance of axis-cylinders after separation of a nerve from its respective ganglion-cells, either in the anterior horn of the cord or in the spinal ganglia. In addition to this peripheral degeneration, there is a retrograde degeneration of the trophic cell. As Marinesco has shown, the trophic cell, either in the cord, spinal ganglion, or cerebrum, undergoes atrophy after severance of a peripheral nerve. In young animals, the atrophy of the central cell may be complete, actual disappearance. The trophic energy is greatly disturbed, and finally compromised, if deprived of external stimuli.

Electricity—Electrotonus. Electrotonus is the response to electricity of the nerve-trunk. This response is subject to definite laws, and any deviation from these laws constitutes an abnormal pathologic electrotonus. The response of the nerve to electricity is different from that of the muscle.

Response to Faradic Currents. Muscular tissue itself is not excitable by faradic currents. The rigidity in which it is thrown when stimulated by an electrode is due to the stimuli or responses of the nerve which supplies the muscular fibers. The rapid interruption places the muscle in a sort of tetanic contraction. *The nerve-trunk stimulated by an electrode responds only when the current is broken.*

Response to Galvanic Currents. The law of Pflüger for serial currents, that is, response to alterations in current strength, is, "A nerve-trunk is stimulated when electrotonus develops or an electrotonus disappears, but not under reverse conditions."

A nerve responds to the negative pole when the current is closed, and to the positive pole when the current is opened. The negative pole increases the excitability of a neurone; the positive pole diminishes it.

Anatomy of Nerve Fibers. The nerve fibers can first of all be classified in: (*a*) Medullated —Neurilemmic; (*b*) Non-medullated —Aneurilemmic.

The medullated fibers are surrounded by a refractive substance, the myelin, which is divided by septa in small segments (Schmidt-Lanterman-Kuhnt's segments). The myelin dissolves in alcohol-ether on boiling. A portion, however, remains as a fine network, and resists the action of trypsin. This resistant substance, similar to horn, is called neuro-keratin (horn sheath of Ewald and Kühne). The myelin is interrupted every 80–90μ at the constriction or node of Ranvier. The myelin is protected by a fine sheath, the neurilemma or sheath of Schwann. The inner surface of the sheath has a nucleus surrounded by protoplasm and situated midway between the two nodes; these are the neurilemma cells. The neurilemma or sheath of Schwann is thick and contracted at the node of Ranvier. At the point of constriction, the axis-cylinder presents a biconic swelling. This, being horizontal, forms, with the longtitudinal axis-cylinder, a cross, which is called the Latin cross of Ranvier.

The size of the medullated fibers is from 2 to 20μ. These fibers do not branch until they reach the periphery.

The fibers of the spinal cord and brain are medullated, but aneurilemmic.

Non-medullated fibers are devoid of a medullary sheath. The sympathetic nervous system, with very few exceptions, is composed of Remak's fibers.

NEURONE DOCTRINE

"Central School." According to Waller, Ranvier, Kölliker, Waldeyer, His, and others, it is generally accepted that, after division of a nerve, new axis-cylinders grow downward from the central segment into the degenerated peripheral nerve, replacing those which have undergone degeneration. The concept of the central school —with Waldeyer at its head —is, that the nervous system consists of innumerable and anatomically independent neurones, the relationship of which is that of contiguity. In support of the neurone theory, I will mention the following points:

1. His and others have found that an axone is an embryologic outgrowth of a nerve-cell.
2. Ramón y Cajal, Lenhossek, using Golgi's and methylene-blue stains, have seen that the different neurones are anatomically independent; that is, that the arborizations of the cells do not anastomose or communicate; they terminate in end-knobs.
3. An axis-cylinder separated from its trophic cell degenerates, but the process of degeneration does not involve neighboring neurones.

Opposing the neurone theory, there are the following:

1. Held, Dogiel, Bethe, admit the relation of contiguity of cells only in embryos and in the lower animals.
2. The knobs of the arborization described by several histologists belonging to the central school are simple artefacts.
3. Numerous clinical observations force us to the conclusion that peripheral regeneration, or regeneration independent of the central nerve system, is possible. In other words, the cells in the anterior horn of the spinal cord, and those of the spinal ganglion, are not necessarily trophic centers of the axis-cylinders.

The neurone theory, as it applies to the peripheral nerve system, considers it as made up of chains of cells set end to end, and axis-cylinder processes fused together to form continuous paths.[1]

[1] Ballance and Stewart. The Healing of Nerves, p. 109.

In an article published in *Archiv für Psychiatrie und nerven Krankheiten*, 1901, bd. xxxiv, p. 1066, Albrecht Bethe endeavors to prove the fallacy of the neurone doctrine. He says it construes the neurone to be an anatomic unity or entity composed of ganglion-cells, dendrites, and axis-cylinders. The relation of the neurones is that of contiguity; a neurone is a functional and pathologic unit. Thus, the pathologic condition of one does not involve a neighboring neurone.

The neurone is an embryologic unit originating from a single embryonic cell or neuroblast. After enumerating the essential elements of this doctrine, Bethe considers the various points. The first is contradicted by Apathy. He shows that the nervous system is in a relation of continuity and not of contiguity, the contact being established by primitive fibrils, or, as he calls them, "neurofibrils." Bethe has shown that in *Carcinus moenas* the nervous system can perform its function without the ganglion-cells. According to the neurone school, this would not be possible if the ganglion-cells were destroyed. It is true, says Bethe, that the protoplasm of a central ganglion-cell exerts a trophic, nutritive influence upon a peripheral axis-cylinder, but it is not true that its regeneration after division requires an outgrowth from the center. Peripheral regeneration is possible, and takes place by the proliferation of the nuclei of the sheath of Schwann.

Bethe has divided peripheral nerves and prevented them from uniting. The principal findings after such a procedure were:

(*a*) Degeneration of the peripheral fibers.

(*b*) Proliferation of the protoplasm of the sheath of Schwann.

(*c*) Differentiation of the protoplasm after six to nine months in adult dogs and rabbits, in an axial strand and surrounding sheath, deprived, however, of myelin.

At first there is no electric response. In the course of time, this reappears, and the nerves are restored. In young puppies and rabbits, the regeneration is more pronounced and much more active. At the time of publication, Bethe had five cases — four puppies and one young rabbit. In the three most successful cases, regeneration was perfect, both anatomic and physiologic, even though reunion was prevented. In his experiments, Bethe always selected the sciatic nerve, the main reason being the absence of anastomoses along the thigh. The regenerated nerves were electrically excitable. The respective muscles responded to the stimulation of these nerves by weak currents, which were not able to stimulate the muscle itself; in other words, the peripheral end and muscle formed an independent motor unit; among fibers without myelin, there were fibers with myelin, the histology of which was perfect. Furthermore, he showed that if a regenerated nerve is again divided, the proximal segment does not degenerate, although it is disconnected from the center; the distal end, however, degenerates as usual. Bethe, in his researches in chicken embryos, has found that before the nerve fibers have departed from the spinal column there are between the spinal cord and the myotom a series of cells along the lines of the future nerves.

According to Bethe, therefore, the first arrangement of the future peripheral nerves consists of bipolar cells. Second, the fine fibers may be just as numerous, or more so, in the myotom as in the group of fibers which branch out from the spinal cord. In other words, the primitive nerve fibers grow simultaneously with and independent of the fibers originating from the spinal cord. The primitive bipolar cells of the periphery in the vicinity of the spinal cord send a central outgrowth toward the ganglion-cells. The periphery is connected with the central by a series of juxtaposed cells. The peripheral cells multiply by karyokinesis. The differentiation in primitive fibrils takes place between the seventh and ninth day. The axis-cylinder originates only after the occupation of the entire fiber by myelin. The multicellular or pluricellular origin of the axis-cylinder, as shown by Balfour, Dohrn, Beard, Kupfer, and others is true — in opposition to His's view or theory of the unicellular origin of the axis-cylinder.

Recently, Langley and Anderson[1] have discussed the possibility of errors in investigations of those who believe in peripheral regen-

[1] Autogenetic Regeneration in the Nerves of the Limbs. Jour. of Physiol., 1904, vol. xxxi, p. 418.

eration. Harrison[2] has shown that in frogs there is a definite wandering or amœboid tendency toward the periphery on the part of the axis-cylinder. In other words, the periphery exerts some influence, possibly an affinity, similar to a chemical affinity, upon the central end. This, according to Harrison, does not necessarily imply that the periphery has power of independent or autogenetic regeneration. Again, it has been noticed by many investigators that even when an attempt to prevent a reunion of two divided ends has been made, union takes place in spite of the effort. We know that in extensive dissections of the neck for tuberculous glands, many nerve branches are sacrificed. Motion and sensation are, however, restored in time, even though an attempt to suture the injured nerves has not been made. This does not mean that the divided branches have been regenerated independently of the central branches. According to Harrison, a reunion of the divided branches takes place in the course of time.

Peripheral or Autogenetic Regeneration. The neurone doctrine has reigned for many years, and the clinical conceptions of the surgery of the nervous system have been based on and retarded by this doctrine. Of late, several histologists and physiologists have endeavored to reject this old doctrine. Bethe, Nissl, Dohrn, Schultze, and others are all strong opponents of the neurone theory. The most enthusiastic of all these investigators seems to be Albrecht Bethe, who explains his views in a monograph entitled "Ueber die Regeneration peripheren Nerven" (*Neurolog. Centralblatt*, 1901, pp. 720–725).

"Peripheral School." According to this school, which is called the "Peripheral School," in opposition to the "Central School," axis-cylinders are not the outgrowth of cells in the central nervous system. Their trophic centers are not within the cord or ganglion, *but arise independently in the periphery from chains of cells which ultimately represent the sheath of Schwann, and which only secondarily become united with the central cells.* The "Peripheral School" has found partisans in Italy and England. Among them are Tizzoni and Cattani of Italy, and Ballance and Stewart of England.

According to the "Central School," regeneration is a late process, and does not begin before degeneration has been completed. Followers of the "Peripheral School" believe that regeneration is simultaneous and independent of the process of degeneration. This view is based on histologic pictures, which show that attempt at repair is simultaneous with the decay of myelin and axis-cylinders. As to the time of regeneration, opinions vary considerably. It can be easily understood that, according to the neurone doctrine, regeneration is late. Vanlair has gathered statistics concerning the time required for regeneration: The repair of a divided facial nerve in the parotid gland required about eight months; the vagus regenerated in eleven months. Segments of from one to three centimeters required from thirteen to twenty and even twenty-two months. If more than $3\frac{1}{2}$ centimeters were excised, Vanlair did not obtain good results; regeneration was entirely absent.

If the return of sensation is slow or imperfect or entirely lacking after injury, it must be considered as an indication that there is an obstacle preventing the reunion of new fibers, which must be removed by operation. The degree of sensory restoration and its rapidity are of great diagnostic and prognostic value.

While the process of regeneration is considered by the neurone school as being governed principally by the central nervous system, the influence exerted by the peripheral segment upon the growth of the fibers from the central segment has not been denied even by the partisans of this doctrine. We noted before that Harrison speaks of the chemical affinity between the peripheral and the central segments. Long ago, Forsman remarked that a downward growth of the axis-cylinder from the central cell was not only a mechanical process governed by the "locus minoris resistentiæ," but that other factors must be considered. He believes that the decayed substance of the peripheric end "exerts a positive attraction" upon the new fibers growing from the central end. His views are well sustained by his ingenious experiments.

[2] Neue Versuchungen und Beobachtungen über die Entwickelung der peripheren Nerven der Wirbelthiere. Bonn, 1904.

Forsman divided the nerve and included the central and peripheral ends in a straw tube. In a second case he did the same thing, but connected the two ends by a thread, and in a third case one end was left outside the tube, while the other end was within the tube, and both were connected by a thread situated within the lumen of the tube.

In case 1, the growth of fibers is very slow; the same in case 3; the growth, however, is very rapid in case 2. This does not mean that the thread is a conducting element, thus increasing the rapidity of the growth, but simply favors the advancement of fibers. In another experiment he included between the two ends a small quantity of brain substance. He had for control another straw tube with segments of nerves at each end. There was less resistance in the empty straw tube. The growth, however, was very slow; while in the other tube, in which the fibers encountered the resistance of the brain substance, they grew very rapidly. This experiment showed that the mechanical element is of secondary importance, and that the brain substance exerts a certain attraction upon the fibers of each end. Forsman considers the power of attraction a sort of chemotropism, which he calls "positive neurotropism." The same experiments were repeated by Schiff and others. Partisans of the neurone doctrine, considering the fact that the "Peripheral School" see the possibility of an independent regeneration between two disconnected ends, suggest that if new fibers arise in the peripheral segment independently of the central segment, Why is it necessary that these two ends should be united at all? To this the "Peripheral School" answers, that it is perfectly true that regeneration can take place independently of any communication with the center, but that these new fibers are ready to carry impulses, *which originate from the center.* From many striking clinical examples we have learned that sensation, and sometimes even motion, has been restored in a short time after the operation. The "Peripheral School" explains the phenomenon by stating that new fibers were regenerated in the peripheral segment and were ready to carry impulses, but that this was not possible as long as the two ends were disconnected. In the words of Ballance and Stewart, anatomic perfection is necessary for physiologic activity.

Robert Kennedy, in an article published in the *Lancet*, July 22, 1899, discussing the secondary suture of nerves, declares himself a partisan of the "Peripheral School." The microscopic findings in his experiments of division and secondary suture were:

(*a*) Disappearance of axis-cylinders and myelin in the entire peripheral segment, and in a small portion of the central segment.

(*b*) Young nerve fibers developed simultaneously in the peripheral and central segment, even when no connection existed between the two ends.

(*c*) Young nerve fibers regenerated from the old sheath of Schwann from the protoplasm and nucleus of the interannular segment.

(*d*) The development of the new nerve fibers was imperfect as long as conductivity was not established.

(*e*) The cicatricial mass formed between the ends of nerves may be penetrated by new young nerve-fibers, but function is not established if the cicatricial tissue is able to produce sufficient pressure to prevent the traveling of impulses. These findings explain the permanent paralysis following nerve contusions without division, as stated in my article, "Osmic Acid Injections for Trifacial Neuralgia." [1]

Ballance and Stewart are also exponents of the "Peripheral School." They have strengthened their views by clinical and histologic researches in both South Africa and England. These authors are of the opinion that a divided peripheral segment of a nerve can regenerate independently of the central segment, "*from pre-existing elements in the periphery.*" Division of the nerve is followed by degeneration and regeneration. Regeneration, according to them, begins before degeneration is complete.

Changes in the Peripheral Segment of the Divided Nerve Which has not been United

Shortly after division, the end of the peripheral segment has a swollen appearance, to which is given the name of "primitive end bulb," and is nothing but a curling back of the divided nerve-fibers upon themselves. The neurilemma-cells produce small islands of axis-cylinders and

[1] Jour. Amer. Med. Assoc., Oct. 1–8, 1904.

medullary sheaths; these islands elongate, range themselves side by side, fuse together, and finally form long fibers which unite with those situated above. In the peripheral segment of a divided nerve which has been united to the proximal end, the neurilemma-cells range themselves side by side in longitudinal order, and are separated by connective-tissue cells. The former are the regenerators of the future axis-cylinders and medullary sheaths. Infection, neuritis, and interposition of scar tissue are great hindrances to the restoration of function.

The earliest clinical evidence of regeneration may be observed at the end of three weeks. At this time, the peripheral segment contains a great number of elongated spindle-cells, which shoot out axis-cylinders from their poles. The scar tissue between the ends contains some spider cells; their ends interlace in an irregular manner and form a network of new axis-cylinders. The anastomosis of the axis-cylinders of the two ends is accomplished at the end of four weeks. In a cat, Ballance and Stewart observed new fibers in the peripheral segment, which was disconnected from the central, "but they were embryonal." Full maturity is obtained only by connection with the central end, and is due to the stimulus from the center.

Mott, Halliburton, and Edmunds have made many experiments on dogs. They also observed neurilemma-cells elongating in a disconnected peripheral segment, assuming the aspect of a new fiber, but, as they state, "a strand of tissue may look like a nerve, but unless it can be shown to be both excitable and capable of conducting nerve impulses, it cannot be regarded as such."[1] In several of their experiments, a peripheral segment, divided from 100 to 150 days previously, was not excitable; the muscles showed marked wasting. This does not correspond with our clinical observations, as ours had the faradic response. If the nerves are permanently united under aseptic conditions, the peripheral segment will respond to electrical excitation. Sections made below the point of union of the two ends showed that regeneration was more perfect near the point of union, and less so at a distance. This observation is not in favor of peripheral regeneration, and shows distinctly that regeneration takes place by downward growth, and for its accomplishment and progress, time is necessary. I do not consider that this observation justifies the conclusion, as a peripheral regeneration may begin in the proximal end of the segment first, and extend from there to the distal.

[1] British Med. Jour., May 21, 1904, p. 1214.

Process of Degeneration

After division of a nerve there is a degeneration of the entire peripheral segment and of a small portion of the central end. In this article we will follow closely the accepted descriptions of F. Marchand[1] and Ballance and Stewart.[2] The modus of division has no influence upon the process of degeneration. According to some, decay takes place at the same time in the entire peripheral nerve (Stroebe, Howell, and Hubert). The degeneration of the central end extends to the first constriction of Ranvier. The process of degeneration is given by Marchand as follows: The second day after the section, the medullary sheath breaks down in the form of cylindrical fragments, which continue subdividing during the next few days. These fragments are still included in the sheath of Schwann. The axis-cylinders also fragment, and are either swollen or fibrillary in aspect; they are included among the myelin fragments. At the same time, a regenerative process begins in the nuclei of the sheath of Schwann. The nuclei enlarge, become richer in chromatin, and penetrate between the fragments of myelin. Simultaneously with this process, the protoplasm increases in volume and fills up the empty space between the drops of myelin. The latter are surrounded by protoplasm and form round nucleated lumps. The nuclei of the sheath multiply by karyokinesis on the third and fourth days, and subsequently become very abundant in number. The decaying medullary sheath and axis-cylinders, which are in the form of small round drops, are often mistaken for fat. A great part of these nuclei, surrounded by protoplasm, transform themselves into long spindle elements, the importance of which is variously interpreted. We will consider this feature when taking up the process of regeneration.

According to Ballance and Stewart, the vari-

[1] Der Prozess der Wundheilung (*Deutsche Chirurgie*)
[2] The Healing of Nerves.

ous steps in the process of degeneration are as follows:

1. Cellular changes.

2. Changes in the axis-cylinder and medullary sheath.

Cellular Changes

In the first sixteen to eighteen hours after the division, there is a diapedesis of leucocytes in the entire distal segment, and also in a small portion of the proximal segment. In from three to fourteen days, connective-tissue cells replace these leucocytes. Similar to the invasion of the nerve by leucocytes, on the second day after the injury there is a marked proliferation of the connective-tissue cells, invading the entire distal segment and a small portion of the proximal. The fourth day, the fatty substance of the myelin fragments is included within the substance of the connective-tissue cells. The exact time in which the absorption is completed has not been determined, according to the authors. It is estimated, however, that the myelin disappears in about five weeks. After the complete absorption of the myelin, the connective-tissue cells diminish, and are outnumbered by the gradually increasing neurilemma-cells. The remaining connective-tissue cells become spindle-shaped, and their destination is the formation of connective-tissue fibers separating the chains of neurilemma-cells. On the second day after the injury, the neurilemma-cells proliferate. At first their proliferative activity is greater than that of the connective-tissue cells, and during this period they exert an absorptive action upon the degenerating medullary sheaths. Later, their proliferative activity is surpassed by the connective-tissue cells, the former continuing to proliferate in columns. "The young neurilemma-cells preserve the original longitudinal direction of their parent cells, and from their opposite poles they shoot out fine protoplasmic processes which gradually increase."

Changes in the Axis-cylinder and Medullary Sheath

The histologic observations of Ballance and Stewart are summarized as follows: Decay of the axis-cylinder begins on the fourth day, and is very pronounced on the seventh. Fine fibers have greater resistance to distintegration than coarse fibers. (This view is contrary to the view entertained by Flemming.) The fragments of axis-cylinder are absorbed by the connective-tissue cells principally, and partly by the neurilemma-cells. *The degenerative process is not influenced by immediate suture, as it takes place uniformly.* The muscles supplied by the divided nerve rapidly diminish in weight.[1] A muscle supplied by a divided peroneal nerve weighed 4.77 grams twelve days after division, while the muscle on the other side weighed 6.98 grams.

Return of Sensation and Motion after Division of Nerves

Clinical reports concerning restoration of sensation and motion have been so varied that no definite conclusion can be drawn from the contradictory and chaotic statements of clinicians. This state of affairs has been rendered still more confusing by the new school, "peripheral autogenetic, regeneration." If, after the division of a nerve, regeneration begins "immediately," and independently of any influence from central ganglionic cells, then the element of time in regeneration of the peripheral axone after division is practically of no great importance, as the errors in recording too early return of motion have been recognized long ago. Létievant, for example, emphasizes the fact that after division of the musculospiral the abduction of the thumb is still possible, but this motion is due to the contractions of the extensor ossis metacarpi pollicis. Many errors have been recorded in experiments upon animals. From our experiments we have come to the conclusion that in order to appreciate exactly the extent and nature of motions in animals, it is necessary to use the forelegs, and to observe the animal while going up stairs. If, for example, we divide the sciatic nerve in a dog, the animal will have an apparently fairly good motion. This, because of the anatomic peculiarities of the posterior leg of quadrupeds. The hind leg is so suspended by ligaments and muscles which are not supplied by the sciatic, that the paralyzed limb, being attached to the ilium, will have a certain tonicity, thus conveying the false impression of a functionally normal leg. Furthermore, the lack or absence of motion cannot

[1] P. B Henriksen. Lancet, April 11, 1903.

accurately be estimated if the animal walks on a perfectly horizontal plane.

The errors have been still greater when recording the return of sensation after injury to the nerves. The important researches of Henry Head and James Sherren[1] throw a new light on the restoration of cutaneous sensibility after division of nerves. The fact that the patient has some sensation, tactile, or even caloric, after division of a nerve, followed by immediate reunion, does not necessarily mean, according to the above investigators, that there is immediate restoration of sensation. This sensation can be explained by collateral anastomosis, transmission of mechanical vibrations to intact neighboring nerve-ends, and by the "deep sensibility." This, however, is produced only by grosser stimuli, as severe pressure and extremes of heat and cold. After division of a nerve there may be some tactile sensibility left. The patient may be able to feel the pricking of a pin, may be able to feel some degrees of temperature, but he will not be able to discriminate between 22° and 40° C., locate the points of a compass, and will not experience pain. These patients feel temperature below 18° C., or above 45°; they feel either point of a compass, but they are unable to locate either when they are applied close together.

The remainder of sensibility after division of a nerve is called by Head and Sherren "deep sensibility." If the median, ulnar, or radial nerves are divided below the point where these nerve-trunks give off their muscular branches, then the deep sensibility will always be present. The cutaneous impressions are carried by efferent fibers, which, from the skin, travel along the plantar and palmar fasciæ, then along the tendons, and finally join the muscular branches of these motor nerves. These deep (efferent) fibers enter the spinal cord via posterior roots. According to this anatomic explanation, division of nerves in the plantar or palmar regions are not necessarily followed by absolute impairment of sensation. The latter would be present if, for example, we divided the brachial plexus, lumbosacral plexus, or posterior roots, which give the sole supply to same motor and sensory zone of body. Head and Sherren divide the efferent fibers into three systems:

"1. Those which subserve deep sensibility and conduct the impulses produced by pressure. The fibers of this system run mainly within the motor nerves, and are not destroyed by division of all the sensory nerves of the skin.

"2. Those which subserve *protopathic*[1] sensibility. This system of fibers and end-organs responds to painful cutaneous stimuli, and to the extremes of heat and cold. It also endows the hairs with the power of reacting to painful stimulation. If two divided ends of protopathic sensation are successfully connected, then the fibers regenerate, and a superficial sensation begins to return in from seven to ten weeks.

"In peripheral nerves the distribution of the protopathic fibers usually overlaps the area supplied by the fibers of the adjacent cutaneous nerves."

"3. Those which subserve *epicritic*[2] sensibility. The fibers and end-organs of this system endow the part with the power of responding to light touch with a well-localized sensation."

The existence of this system enables us to discriminate between two points, and to appreciate the finer grades of temperature, warm and cool, and finer grades of touch, as of cotton-wool. These fibers regenerate more slowly, so that the epicritic sensation returns in from six months to two years; although they may never be restored, even after union.

Since we have no histologic facts to explain why sensation is restored sooner than motion, we will consider the results of clinical experience. If a nerve be divided, motion and sensation disappear simultaneously. Without surgical reunion, sensation may be restored in from six weeks to several months, while motion returns very much later, or not at all. Where motion was restored without surgical approximation of the ends, it is plain that the separation of the ends was not maintained. Where restoration of function does not take place, a considerable gap must exist between the ends, or a muscle sheath or connective-tissue mass, etc.,

[1] Brain, 1905, pp. 116–338.

[1] Proper ability of skin to experience tactile and painful sensations.

[2] Proper ability of patients to discriminate, after reunion of divided nerves, various degrees of temperature, slight touch, points of a compass.

is interposed between the segments, which interferes with the penetration of nerve-fibers into the peripheral segment. There are cases on record where sensation and motion were restored very early. The explanation given for this (though not satisfactory to us) is, that this was due to collateral anastomosis with the nerves in the vicinity. From my repeated clinical observations, and from those of many others, it can be distinctly stated that the invariable order in which restoration takes place is:

(*a*) Restoration of the trophic energy of the tissues.

(*b*) Sensation.

(*c*) Motion.

(*d*) Material increase of the substance of muscles which for months have been inactive, except where muscular tissue has degenerated beyond repair.

The order is so definite that apparent exceptions have little value. If, during the first few days, primary union takes place after suture, the patient himself calls attention to the fact that his limb is not so blue, nor is it so cold. A favorite expression of the patient is, "My limb is not nearly so heavy." In other cases, if primary union of a divided nerve takes place under the same conditions, the patient will feel a pricking sensation in the paralyzed member, especially in the more distant parts. In some cases, however, this sensation is experienced very late; though always before there is any indication of muscular restoration. Why sensation should be restored in some cases in a few hours or days, and in others in a few weeks, months, or years, we are unable to say. The trifacial regenerates completely after division in from eight to eighteen months. Motion, as I stated before, is restored relatively late. An approximate estimate of the average time required for the restoration of motion is impossible. From the cases recorded we see that motion has been restored in from eight weeks to three and a half years. We cannot account for these enormous diversities of time in the restoration of motion in cases of primary reunion of nerves. We can easily understand why, in cases of secondary union of divided nerves, motion may return very late or not at all.

Historical Data concerning Nerve Union and Nerve Anastomosis

The first experimental attempt at nerve anastomosis was made by Cruickshank.[1] It was Haughton, however, who expressed the view that union of divided nerves leads to restoration of function.

In 1824, Flourens[2] divided and crossed the two principal trunks of the brachial plexus of a cock. The drooping of the wing ceased after several months, and the cock was able to use it as before. An exposure of the nerves showed a perfect union. Pinching of the nerve above the point of union gave the fowl pain, and the wing moved. The same thing happened if this was done below the point of union.

Philippeau and Vulpian,[3] in 1864, crossed the central end of the vagus with the peripheral end of the hypoglossal in dogs, and *vice versa*. The union was good, but the hypoglossal was "but an instrument at the command of the functional center of the motor fibers contained in the cervical part of the vagus."

E. T. Reichert,[4] in 1885, sutured in five dogs the central end of the vagus with the peripheral end of the hypoglossal. The author found that certain parts of the tongue would contract rhythmically with each inspiration; in other words, the motor fibers of the vagus have united with the motor fibers of the hypoglossal, which resulted in restoration of motor function over other terminal axones.

The same year, Rawa[5] operated on many cats. He came to the conclusion that "after the peripheral portion of a nerve supplying certain muscles has united with the central end of a nerve that supplies another muscle, the function of the former muscle becomes restored." "The direction of the voluntary motor impulses may be altered as one pleases, and the impulses will always accommodate themselves to the peripheral nerve ends."

Schiff[6] experimented about the same time. He crossed the vagus with the hypoglossal, and was surprised to find, at the end of four months,

[1] Philosophical Transactions of the Royal Society of London, 1795, vol. xvii, p. 335.

[2] Recherches expérimentales sur les functions du système nerveux, 1824, p. 272.

[3] Journal de Physiologie, 1864 p. 421.

[4] The American Journal of Medical Sciences, Jan., 1885.

[5] Archiv für Physiologie, 1885, p 296.

[6] Archives des sciences, phys. et nat., Genève, 1885

EXPERIMENTAL CASES OF CROSSING NERVES

Author and Publication	Operation	Immediate and Remote Results	Remarks
Flourens. Recherches expérimentales sur les propriétés et les functions du système nerveux, 1824, p. 272.	Division of two principal trunks of brachial plexus of cock and crossing.	Pinching of crossed nerves above the point of union produced movements of wing. The same result if pinched below. After several months, cock had no drooping of wing.	Union perfect. Objection to Flourens's experiment that union may have been defective, enabling nerves to unite in their normal continuity.
Philippeau and Vulpian. Journal de Physiologie, 1864, p. 421.	Anastomosis between central end of vagus and peripheral end of hypoglossal and *vice versa* (dogs).	Union perfect. The hypoglossal was "but an instrument at the command of the functional center of the motor fibers contained in the cervical part of the vagus."	
Reichert, E. T. Am. Jour. Med. Sciences, Jan., 1885.	Anastomosis between central end of vagus and peripheral end of hypoglossal (dogs).	"Certain" portions of the tongue contracted with each inspiration.	
Rawa. Archiv für Physiologie, 1885, p. 296.	Many anastomoses in cats of synergic, rhythmic with arythmic nerves, etc.	Restoration of function in domain of crossed nerves.	"The direction of the voluntary motor impulses may be altered as one pleases, and the impulses will always accommodate themselves to the peripheral nerve endings." (Rawa.)
Schiff. Archives des sciences, phys. et nat., Genève, 1885.	Crossing between vagus and hypoglossus (dogs).	After four months, return of motion in muscles of tongue.	Reichert, who examined the dogs of Schiff, did not find voluntary motion. He found, however, synchronous, rhythmical contractions in the tongue with each inspiration.

return of motion in the tongue. Reichert, who examined the dogs of Schiff, did not find voluntary movements of the paralyzed half of the tongue. He found, however, synchronous rhythmic contractions of the tongue with each inspiration.

A year later, A. Stefani[7] obtained perfect restoration of function by reuniting peripheral nerves. Recently, Robert Kennedy[8] of Glasgow found that anastomosis of nerves supplying synergic and even antagonistic muscles gives perfect restoration of function with the adaptation of cortical centers according to the peripheral necessity.

Calugareanu and Victor Henri crossed the pneumogastric and hypoglassal nerves three times in dogs. The results of the experiments are varied. In the first experiment the hypoglossal regained function; the vagus did not. In the second, there was perfect restoration in the hypoglossal and only partial in the vagus. In the third — the most interesting of all — there was perfect restoration of the vagus; the excitation of the nerve stopped the heart and diminished the blood-pressure. The experimentors concluded that the fibers of the vagus do not carry *specific* impulses, and that similar effects, impulses of a similar nature to that of vagus, may originate from the nuclei of the hypoglossal.

Similar experiments were made by N. Floresco. He operated on cats, crossing the sympathetic with the vagus. After from 86 to 425 days, electric stimulation of the peripheral stumps showed that the sympathetic had assumed some of the functions of the vagus. The phenomenon is explained either by a regeneration of fibers or by an inhibitory process. Microscopic examinations of the specimens show regeneration of new fibers at the point of suture.

The fact that muscles have been degenerated for a long time — ten or fifteen years, for example — is no contraindication to reunion or anastomosis of nerves. The muscles atrophy only after the division of a nerve, but do not degenerate completely. In two of our cases — one of birth paralysis, and the other of Erb's type of paralysis, due to a railroad injury — the volume of the muscles was fairly good, notwithstanding that the paralysis existed for 24 and 16 years, respectively. If a nerve is reunited after a long period of time, and function is restored, the respective muscles will increase in volume and resume their activity, notwithstanding their long period of inaction. In Hackenbruch's case of facial paralysis of eight years' standing, nerve anastomosis was followed by restoration of function.

Jamin studied the condition of muscles after division of nerves in dogs. He reached the following conclusion: The principal changes in muscles after the division of their respective nerves are characterized by the diminution

[7] Arch. für Anat. u. Physiologie, 1886, p. 438.

[8] Philosophical Transactions of the Royal Society of London, 1901, series B, t. cxciv, p. 127

ANASTOMOSIS OF PERIPHERIC NERVES

CLINICAL CASES

Author and Publication	Condition	Nature of Injury. Cause	Operation	Result
1. Robert Kennedy.	Paralysis.	?	Implantation of central ends of median and ulnar into the radial.	Negative.
2. Després.	Paralysis of median nerve.	Injury.	Implantation of median into ulnar.	"Fine result."
3. Moses Gunn.	Paralysis consecutive to removal of neuroma.	Operation.	Implantation of distal end of ulnar into the median.	Return of sensation. No report as to motion.
4. Sick.	(*a*) Paralysis of radial. (*b*) Paralysis of musculospiral.	? ?	(*a*) Eighteen months later. Implantation of radial into median. (*b*) Implantation of paralyzed musculospiral into median.	(*a*) Good motion and sensation. (*b*) Perfect motor control in both groups of muscles.
5. Neugebauer.	Paralysis of peroneal nerve.	Operation. Excision of a scar for contracture.	Anastomosis between peroneal and internal popliteal.	Negative after 8 months.
6. Dumstrey.	Paralysis of ulnar.	?	Implantation of ulnar into median.	Partial return of motion and sensation.
7. Trzebicky.	(*a*) Paralysis of external popliteal. (*b*) Paralysis of ulnar.	(*a*) ? (*b*) Operation. Resection of ulnar for sarcoma.	(*a*) Implantation of both ends of external popliteal to internal popliteal. (*b*) Implantation of ulnar into median.	(*a*) Satisfactory. (*b*) Primary result, although patient died from metastasis.
8. Codevilla. Clinica Chirurgica, 1900, Nos. 9–10.	Paralysis of peroneal nerve.	Crushing of knee.	Implantation of peripheral end of peroneal into internal popliteal.	No improvement.
9. Kölliker. Münch.med.Woch., 1901, No. 50.	Paralysis of radial.	Central origin.	Distal end of radial implanted into median (lateral implantation) combined with stretching of ulnar and median.	No improvement of paralysis, but disappearance of muscular cramps.

With the exception of cases Nos. 4 and 9, the rest are summarized by Charles A. Powers (*Annals of Surgery*, 1904, p. 641).

of the contractile substance of the muscles, but there are no degenerative changes. The experimentor could not find that the nervous centers exert a special trophic influence upon the muscles. The "reaction of degeneration" and the changes in the electric response are no evidences of *degenerative changes in the muscles*, and merely represent an absence of nerve stimulus.

"As soon as the communication of a peripheral nerve is established with the center, muscles previously degenerated (atrophied) regenerate almost completely" (Koch).

Changes in Nails

The changes, shape, thickness, color, etc., in nails after division of nerves were first systematically described by Weir-Mitchell. Head and Sherren do not believe that changes in the nails are due to the division of sensory branches, but that they are due to a "want of movement" of muscles and tendons.

Psychic Derangements Following Injury to the Nerves

Among the number of psychic derangements which may follow injury to the nerves, Krafft-Ebing[1] quotes: 1. Tetanus and tetany, meaning general tetanic spasms. This can be easily understood in case of injury to the nerves with a small wound where there is a possibility of infection, but there are cases where "tetanus" follows an injury without solution of continuity of the skin. It is not a true tetanus, and recovery usually follows. In 1886 I saw a severe case of this type (tetanoid spasm) following a severe trauma to the supraorbital nerve. It lasted for many hours, and was relieved only by the administration of chloroform. On cessation of its administration the tetanic convulsions would recur. Large doses of chloral and morphine finally controlled the spasms, and the patient recovered.

2. Epilepsy. In one case a traumatism of the occipital nerve was followed by reflex psychosis without injury to the central nervous system. In a case cited by Wendt with injury of the auriculo-temporal nerve by a bullet, patient developed epilepsy and delirium.

[1] Krafft-Ebing. Lehrbuch der Psychiatrie, 1893, p. 180.

EXPERIMENTAL SURGERY OF PERIPHERAL NERVES

Introduction and Generalities

From our personal experimental and clinical observations, and from those of many others, we accept peripheral regeneration. (In another part of this article will be cited the clinical

NERVE SUTURE

PERSONAL EXPERIMENTS ON ANIMALS

Animal, and Anæsthetic Used	Nature of Operation	Post-operative Course	Immediate and Remote Result	Reoperation or Post-mortem
Case 1. July 5, 1905. Dog, white; puppy. Chloroform and ether.	Division of 2 branches of brachial plexus and end-to-end crossing of same. Union with catgut sutures passed through perineurium, and *not* through nerve substance. Protection of point of anastomosis with axillary fat.	Primary union.	July 7, 1906: When walking up stairs, marked limping; this not pronounced when dog walking on level. Aug. 21st: Dog walks on all four legs; very little limping. Oct. 1, 1906: Perfect gait.	Oct. 1, 1906. Exposure of point of anastomosis. Perfect union. Point of crossing marked by enlargement of nerve-trunk. No perineural adhesions.
Case 2. July 12, 1905. Dog, white; puppy. Chloroform and ether.	Same operation, except that point of anastomosis was protected with muscle instead of fat.	Slight superficial suppuration.	July 16, 1905: Dog crawls on three legs. Sept. 5th: Walks on four legs. Perfect gait, Oct. 15th.	Dog killed by gas. Union perfect. No perineural adhesions.
Case 3. July 13, 1905. Gray rabbit. Chloroform.	Same operation. Point of anastomosis surrounded by fat.	Primary union.	The character of gait could not be well recorded, as rabbit does not walk, but hops.	Killed by gas. Union perfect.
Case 4. April 1, 1905. Dog, black; young. Chloroform and ether.	Crossing of 2 branches of brachial plexus, without protection of point of anastomosis by soft tissues.	Primary union.	April 5, 1905: Dog walks on three legs. May 1st: Same condition. July 1st: Same condition.	July 8, 1905: Reoperated. Wound exposed. Neuroma at point of anastomosis; all four ends imbedded in a bulk of scar tissue. Removal of connective tissue and small portions of each end. Reunion without protection of anastomosis. Sept. 20, 1905: Dog killed by gas. Four ends imbedded in similar bulk of connective tissue.
Case 5. April 4, 1905. Dog, red; ½ years old. Chloroform and ether.	Crossing of 2 branches of brachial plexus, without protection of point of anastomosis by soft tissues.	Primary union.	April 8, 1905: Dog walks on three legs. May 15, 1905: Same condition. Oct. 15, 1905: Same. July 15, 1906: Dog walks on three legs; marked atrophy of muscles; pain on pressure upon the axillary space.	Not reoperated.
Case 6. July 14, 1905. Dog, white; puppy. Chloroform and ether.	End-to-side anastomosis. Peripheral ulnar inserted into a niche of median. Anastomosis made at a very obtuse angle.	Primary union.	The defect in gait hardly noticeable.	Oct. 4, 1905. Dog reoperated. Union good; ulnar excitable below point of anastomosis; shows that the ulnar excitability returned in three months.
Case 7. August 1, 1905. Dog, white. Chloroform and ether.	End-to-side anastomosis. Peripheral ulnar into a slit of median.	Primary union.	The defect in gait hardly noticeable.	Oct. 6, 1905. Ulnar not excitable below point of anastomosis. Latter defective and marked by adhesions with surrounding tissues. This absence of faradic excitability shows there was no regeneration of peripheral axonal ends, as it was a slit approximation, and no end-to-end axonal contact secured by the operation.

instances in its support). Until a few years ago no one had the courage to defend such a theory, and clinical facts belong rather to the domain of history. The neurone theory, which was almost universally accepted in all countries, considered as erroneous any work concerning the possibilities of peripheral regeneration. The works of Ballance and Stewart, Bunger, Kennedy, and especially the work of Bethe, have greatly changed our views.

Schede is of the opinion that results obtained in animal experiments cannot be justly applied to man, as the general biologic law is that the regenerative power of tissues diminishes as the animal ascends the zoölogic ladder.

The object of our experiments was to investigate:

(*a*) The truth of peripheral regeneration independent of the spinal and cortical centers.

(*b*) The advantages and disadvantages of the various procedures of nerve anastomosis, and the determination of its efficiency and application to clinical conditions.

(*c*) Process of repair in primary and secondary sutures of the nerves.

(*d*) The value of interanastomosis of fasciculi of the cauda equina, and its effect on transmission of motor and sensory impulses — its feasibility in anteropoliomyelitis.

Most of the experiments on animals were conducted by Dr. Victor L. Schrager in the anatomical laboratory of the University of Chicago.

Peripheral Regeneration

Bethe tells us that for these experiments it is desirable to use young animals. We endeavored to use young dogs for our experiments, but their use was not always possible. A large percentage of young puppies gathered in a poorly ventilated room are subject to a peculiar disease affecting the mucous membrane of the nose, throat, and lungs, which kills the animals in a short time. At the time these experiments were begun, we did not have access to Bethe's treatise. Our work has been independent of his procedures.

One of our first experiments was to divide a nerve in its continuity and prevent any possibility of union between these two divided ends. As Forsman has shown that there is a powerful attraction on the part of the peripheral ends to the fibers of the central end, or as he calls it, "neurotropism," we put the two ends in such a position as to prevent any chance of this attraction. Some experimentors have found that after division of the nerve in its continuity, collateral nerve anastomosis was established, thus enabling the anastomotic branches to conduct impulses from the neighboring nerve into the divided peripheral end. In other words, in order to be able to judge of the possibility of peripheral regeneration, we had to avoid, first, the influence of neurotrophism, and second, that of collateral anastomosis. We generally divided a nerve of rather considerable size, excised from 2 to 3 cm., and then imbedded the peripheral and central ends into two different muscles; the terminals were then placed in such a way that the central end was directed toward the spine, while the peripheral end faced the extremity of the limb. In this way we were confident that by imbedding the nerve into the muscle there was no possibility of an outgrowth from the central end reaching the peripheral end, or that of collateral anastomosis.

Anastomosis Generalities—Technique—Results

The best region for neuro-anastomosis is in the axilla. While rabbits, dogs, and cats serve equally well, in our experiments only dogs and rabbits have been used. There are sufficient nerve-trunks to anastomose. While the axilla is, as stated, an excellent region for anastomosis of nerves, it is a very undesirable one in the hands of an inexperienced sugeon, especially if he neglects to protect the anastomosed nerve by soft tissues. In a recent article I have given the technique for preventing scar formation and subsequent pressure upon the nerves and vessels.[1]

The technique given in that article, as applicable to axillary excavation in cases of carcinoma mammæ, may be applied here; that is, after the nerves in the axillary space have been divided and sutured, a flap of the pectoral muscle, or of any one of the thoracohumeral muscles, should be used to cover the anastomosed nerves so as to prevent scar formation at the point of anastomosis, which will greatly compromise the final results. In several experiments, where this procedure had been neglected, we frequently had either a neuroma between the united ends, or the point of anastomosis was surrounded by very firm connective tissue. Histologic examinations of such failures have proved that there was a distinct attempt on the part of the central fibers to penetrate the scar, and that it was done imperfectly. I should like, also, to call attention to the fact that in experimenting upon the axilla it is necessary to expose the whole brachial plexus at the time of division, as otherwise we are liable to mistake one branch for another. Many methods of anastomosis have been tried. It can be made, as we know, end to end, end to side, and side to side. By "end-to-end anastomosis" is understood the end-to-end union of the peripheral with the central axones. Some experimentors have endeavored to ascertain what takes place when two central or two peripheral ends are united, but, generally speaking, the expression "end-to-end anastomosis" means the union of the central with the peripheral end. By "end-to-side anastomosis" is meant the implantation of one end into the whole end or a niche in another nerve, which is always advisable. This niche may involve one fourth, one third, one half, or even more of the diameter of the receiving nerve. We believe that the implantation of one end through a slit in another nerve is unscientific. The fibers of the nerve in growing will not penetrate another nerve if the perineurium is intact. The interfascicular connective tissue, neurilemma, and medullary sheaths prevent axonal contact, and therefore functional anastomosis. The implantation of a nerve-end into a transverse niche made into another nerve is further advis-

[1] New York Medical Journal, Jan. 6, 1906.

able, because in case of failure of restoration of function in the implanted portion the function of the receiving trunk is not compromised, as one half or two thirds of its fibers have been preserved.

In making an end-to-side anastomosis I have found it is desirable that the implanted nerve shall form with the fibers of the other nerve an obtuse angle. This can be readily obtained by fixing the anastomosed branch to the side of the trunk of the healthy nerve.

"The Union of Different Kinds of Nerves," by J. N. Langley and H. K. Anderson (*Journal of Physiology*, 1901, vol. xxxi, p. 365), "Summary of Results":

1. "The union of two central nerves is not followed by functional intertransmission; impulses set up in one nerve cannot pass across to the other. This confirms the results of Schiff, Stefani, also the recent work of Bethe."

2. "Union of two peripheric ends may be followed by restoration of function (Bethe)." Authors did not obtain functional union of peripheral ends in rabbits and cats.

3. "When the central end of a nerve has an opportunity of joining with two peripheral nerves, stimulation of one peripheral nerve after complete severance of the conjoined nerves from the central nervous system may cause contraction of the muscles innervated by the other. We consider this to be an axone reflex, set up in fibers from the central end, one branch of which has joined one peripheral end, and the other branch joined the other peripheral nerve."

4. "When one of the peripheral nerves consists of afferent fibers, an axone reflex may still be produced by stimulating it. Since efferent fibers do not, so far as is known, unite with afferent fibers, the axone reflex obtained supports the view that regeneration takes place by a down growth of axis-cylinders."

5. "The crural nerve can make a functional union with the sciatic, and so be artificially lengthened. This is in harmony with Bethe's observation that the sciatic can be elongated by joining it with the sciatic of the opposite side. The cervical sympathetic can also be elongated by joining it to the cervical sympathetic of the opposite side."

6. "We do not find that the internal saphenous nerve when joined to the sciatic acquires a motor effect."

7. "The phrenic nerve, when united with the cervical sympathetic can cause the usual effects produced by stimulating the latter nerve."

8. "We confirm Mislavski's result that the cervical sympathetic can unite with the recurrent laryngeal nerve and cause movement of the vocal cord. This movement is due to contraction of the thyroarytenoid muscle, and to unilateral contraction of the arytenoideus muscle. We find also that after functional union has taken place, section of the cervical sympathetic causes degeneration of the medullated fibers in the recurrent laryngeal, except for a few which arise from the superior laryngeal nerve."

9. "The cervical sympathetic can unite with the spinal accessory nerve and cause contraction of the sternomastoid muscle."

10. "The cervical sympathetic can unite with the phrenic nerve and cause contraction of the diaphragm."

11. "We do not find that either (*a*) the afferent fibers proceeding centrally from the ganglion of the trunk of the vagus, or (*b*) the afferent fibers proceeding peripherally in the great auricular nerve, or (*c*) cutaneous afferent fibers, can unite with the peripheral end of the cervical sympathetic in such a way as to transmit nervous impulses to it or to the superior cervical ganglion."

12. "We do not find that the cervical sympathetic, after excision of the superior cervical ganglion, can make functional connection with the fibers leaving the anterior ends of the superior cervical ganglion. We consider that the *pre*-ganglionic fibers cannot make functional connection with the *post*-ganglionic fibers."

13. "We do not find that the post-ganglionic fibers leaving the anterior end of the superior cervical ganglion can form functional connection with the pre-ganglionic fibers of the opposite cervical sympathetic." (One experiment only.)

14. "We do not find that the post-ganglionic fibers leaving the anterior end of the superior cervical ganglion can make functional connection with the hypoglossal fibers supplying the tongue muscles, although they do this with

the vasomotor fibers of the hypoglossal." (One experiment only.)

Langley undertook a more complicated anastomosis, as, for instance, that of sympathetic and pneumogastric nerves. By crossing these nerves, he succeeded in making the vagus take up the vasomotor activities of the cervical sympathetic.

Nerve Anastomosis for the Cure of Facial Paralysis

In French, and even in English literature, Dr. Faure of the Hôpital Laennec of Paris is claimed to be the originator of nerve anastomosis for the cure of facial paralysis. As we have seen, the procedure of anastomosing a paralyzed nerve with a histologically intact nerve is of older origin, and even though Faure may have been the originator of spinofacial nerve anastomosis, the credit should be given to those who conceived the principle of nerve anastomosis. After a careful search of the literature on the subject, we are convinced that the credit for spino-facial and hypoglosso-facial anastomosis belongs to Charles Ballance and Purves Stewart.

In an article published in the *British Medical Journal,* May, 1903, these authors state that they were the first to perform the spinofacial anastomosis for a facial paralysis, and that this was done in 1895. In 1898, Dr. Furet of Paris suggested the possibility of nerve anastomosis to Dr. Faure. In 1898, Dr. Faure united the facial with the trapezius branch of the spinal accessory, end to end. He divided the peripheral branch of the facial nerve near the stylomastoid foramen, and united it end to end with the trapezius branch of the accessorius. He reported the case in March, 1898, at the séance of the Académie de Médecine. His communication did not receive sufficient consideration. About the same time, Paul Manasse conducted, in Munk's laboratory in Berlin, researches along the same line. His results, which were mainly experimental, were published in the *Archiv für klinische Chirurgie,* 1900, bd. lxii, p. 805. Manasse used dogs for his experiments. He obtained restoration of function in five cases of experimental facial nerve anastomosis by uniting the peripheral end of the facial with the central end of the accessorius by lateral anastomosis. Results were satisfactory in all five cases. Two of them, however, were not definite, as fibers from the central end of the facial nerve grew downward and united with its peripheral segment. Furet reported his suggestion of nerve anastomosis for facial paralysis to Dr. Barrago Ciarello, who carried out the experiments at the Otorhinological Institute at Naples. The Italian doctor united the peripheral end of the facial nerve with the central end of the accessorius nerve in two experiments, and once with that of the vagus. The results were quite satisfactory. Electrical excitability and voluntary motion were restored in a few months. Of course there is some doubt as to the manner in which the function was restored, "as there is a possibility that re-establishment in continuity of the facial fibers may have occurred in these dogs."[1] In 1900, a successful nerve anastomosis was performed on a man by Robert Kennedy of Glasgow. He united the peripheral end of the facial nerve with the incompletely severed spinal accessory. The operation was followed by complete restoration of function in both groups of temporarily paralyzed muscles. Bréavoine advocates anastomosis of the facial with the branch of the spinal accessory which supplies the trapezius.

Harvey Cushing performed the same operation for traumatic facial paralysis in 1902, and function was restored in six months. As to the time that may elapse between injury to the nerve and the operation, nothing definite can be said. However, if the muscles will not respond to galvanic stimulation, our hopes of recovery are limited, otherwise delay of operation does not influence the favorable prognosis of the case. There are several cases on record where anastomosis was performed years after the division, yet function was restored. Nerve anastomosis Cushing considers very promising, both in facial paralysis and anteropoliomyelitis.

Nomenclature. Some surgeons call the spinofacial anastomosis the "Ballance-Stewart procedure"; others call it the "Körte-Ballance procedure." In my opinion, the paternity of the procedure belongs to Ballance and Stewart, but it should be called the spinofacial operation. The spinal accessory has been most frequently chosen, though other nerves have

[1] Annals of Surgery, 1903, p. 644.

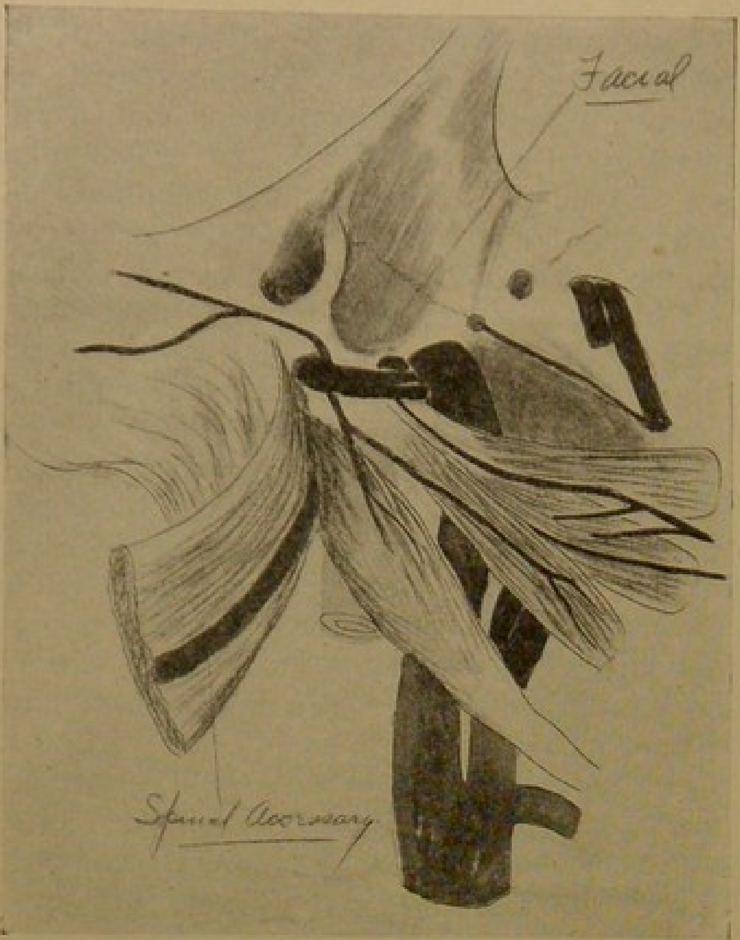


Fig. 41. Dissection. Showing facial and spinal accessory nerves.

been suggested and used for anastomosis; for instance, the hypoglossal or glosso-pharyngeal. Charles Ballance once anastomosed the facial with the hypoglossal, end to side. James H. Nicholl resorted to the same procedure. Körte, Frazier, and others have used the hypoglossal.

The reasons for preferring the hypoglossal to the spinal accessory are: 1. It has a larger trunk; 2. If the union is perfect and new fibers from the hypoglossal penetrate into the facial, then the associated movements cannot be observed if the patient keeps the mouth closed; 3. The vicinity of the cortical centers of hypoglossal and facial; and 4. According to Gowers, Tooth and Turner, Brugia, and Matteuci, the fibers of the facial supplying the muscular fibers which close the mouth may originate from the nucleus of the hypoglossal. Those who do not advocate the use of the hypoglossal refer to the fact that the spinal accessory can be easily switched upward, and lay particular stress upon the fact that the accessorius supplies muscles of less importance than the hypoglossal. A temporary paralysis will not disturb the patient much, nor will a complete paralysis be serious. Prof. E. A. Schäfer of Edinburgh suggests the anastomosis of the facial with the glossopharyngeal because of the close proximity of the motor nuclei of the two nerves.

Technique of Operation. There are four different types of anastomosis in facial paralysis:

1. End-to-end anastomosis. (Faure.)
2. Implantation of the facial into the slit of the spinal accessory.
3. Implantation of the facial into a niche in the spinal accessory, the latter being partially and transversely divided.
4. End-to-side implantation.

The length of time of the facial paralysis bears no relation to the rapidity of the return of function. Furthermore, there is no definite time at which the beginning of restoration of function can be observed. In the case of Cushing, the first return of motion was observed after thirteen days. In Kennedy's case, patient was able to move the upper lip after seven days. These two observations, it appears to us, are erroneous, in that they imply a functional axonal restoration of divided motor-fibers in thirteen and seven days, respectively, which we know is a histologic impossibility. The motion was evidently from collateral impulses. It is estimated, however, that the average time which must elapse between anastomosis and the first evidence of return of motion is about six months.

Mrs. E. L. H., widow, aged 21, clerk.

In 1904, patient was operated on for a right suppurative mastoiditis. Recovering from the anæsthetic, interne in hospital noticed right facial paralysis. Two weeks later, an effort was made by a surgeon to reunite the divided nerve. He stated to the patient that the facial nerve had been divided during the first operation, and that he believed the defect could be repaired. There was no improvement after the second operation; the facial paralysis remained unchanged, and, in addition to it, there was pain in the right supraclavicular fossa and in right shoulder. Patient was given electrical treatment for three months after the second operation.

Our examination of patient showed: (*a*) Atrophy of muscles of right half of face; (*b*) Right corner of mouth higher than left one; (*c*) Impossible to close right eye; (*d*) Cannot contract right half of forehead; (*e*) Cannot whistle. Pressure behind the ear causes pain. Pain along a longitudinal scar running from apex of mastoid to middle of clavicle. Partial atrophy of external sternomastoid. Paralysis of trapezius. Cannot elevate shoulder. When arm is passively elevated, there are associated contractions in muscles of right half of face.

Diagnosis. Paralysis of right facial nerve. The facial was probably anastomosed with spinal accessory, considering that there were associated contractions; that is, muscles of face contract when shoulder is elevated. Operation advised.

Operation. May 10, 1906. It was quite difficult during the operation for us to find the point of anastomosis of facial with spinal accessory. After a dissection of one hour in cicatricial tissue, it was located. The union was excellent, *but the point of union was imbedded in and compressed by scar tissue,* which caused the failure of functional restoration. There was a false neuroma the size of a bean, at the point of anastomosis. This was freed from the scar tissue, and faradic stimulation proximal to the scar tissue produced diffuse facial movement, showing axonal union. *The point of anastomosis was then imbedded in a muscle belly.* Faradic excitation of proximal portion of spinal accessory produced contraction in muscles supplied by facial.

June 29, 1906. The asymmetry of the two halves is less marked. The paralytic aperture of the right eye is not so great as before the operation. She is able to close the eye almost completely. The muscles of the face have somewhat gained in volume. The angle of

Fig. 43. Spinofacial anastomosis, showing the end-to-side implantation. (Original).

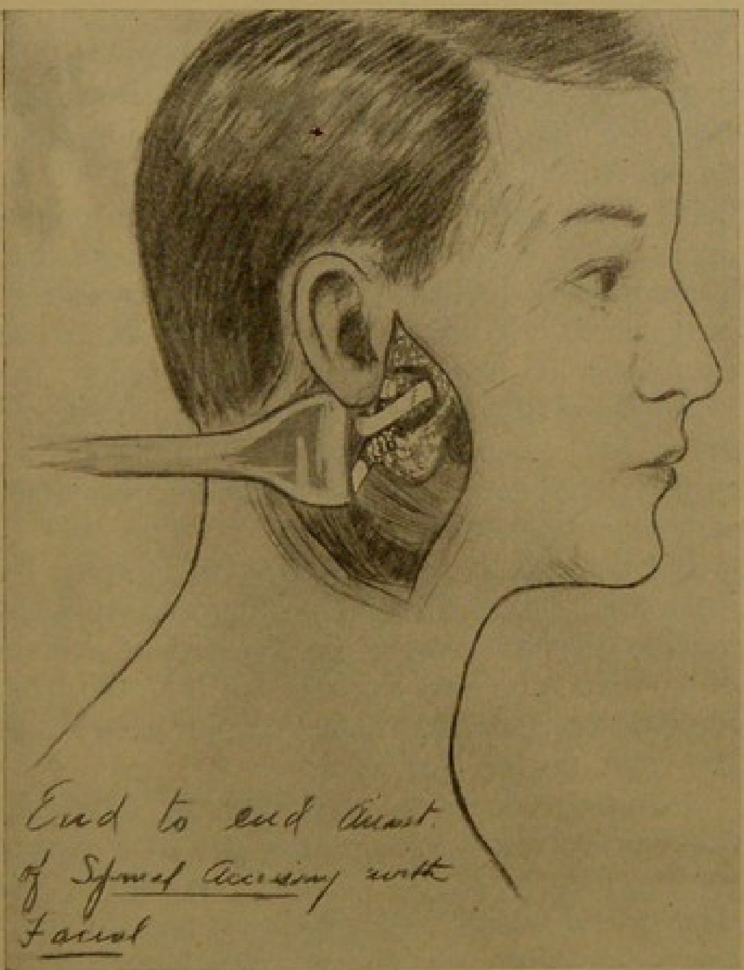


Fig. 42. Correct type of anastomosis (facial with spinal accessory). (Original).

mouth is still drooping, and patient cannot whistle. *Can elevate the shoulder; something she could not do before. Furthermore, has no associated movements.* No electrical response (faradic currents) as yet in the domain of upper and lower facial.

Miss M. H., aged 25. Admitted to Mercy Hospital, June 13, 1906. Referred by Dr. J. B. Walkinshaw, Wellsburg, West Virginia.

Physical Examination. In the region of left mastoid there are two vertical scars. Palpation reveals a depression instead of the usual convexity of the mastoid region. Hypersensitive to digital pressure and very painful at a point corresponding to the apex of the mastoid. The pain is almost characteristic of the neuroma pain usually present after accidental division of the nerves.

Inspection of the face shows at first glance a marked asymmetry; the left half is smaller than the right; the left corner of the mouth is lower than the right, so that a line running from corner to corner is oblique instead of horizontal.

The left palpebral aperture is one third wider than the right. There is an excessive lacrimal secretion in the lower palpebro-ocular groove, overflowing the border of the lower lid. The latter is drooped; there is, however, no ectropion. The muscles of the left half of the face are considerably atrophied. Application of faradic current upon the muscles on this side gives no electric response; even the application of very strong currents is not followed by muscular contraction. Intensive currents, however, produce pain at the point of application of the electrode. Patient is unable to perform voluntary movements with the muscles of left half of face supplied by the facial nerve. She is unable to

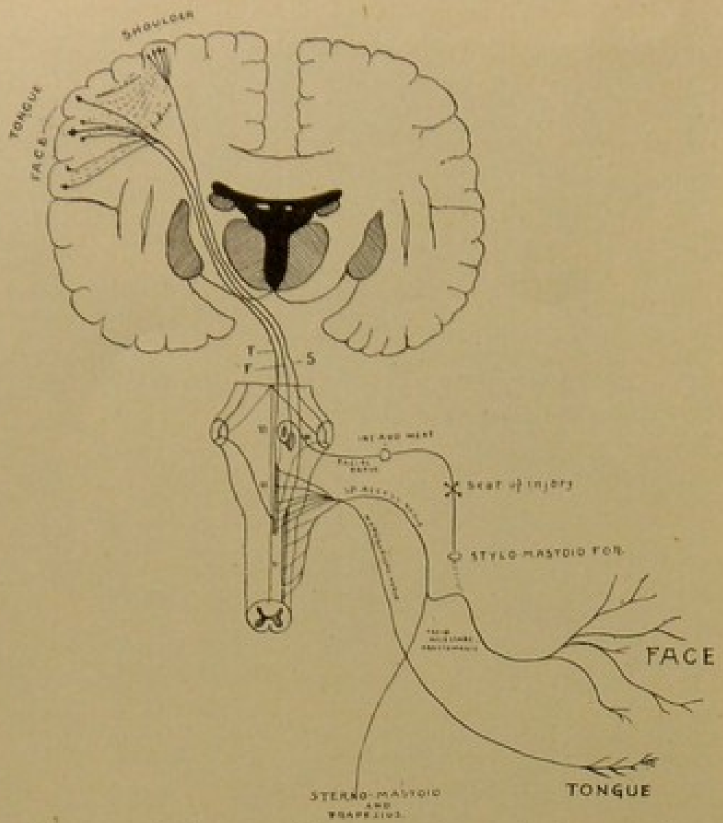


Fig. 44. Schema to illustrate the cortical connections, between the movements of the face, tongue, and shoulder respectively, and the relations of the corresponding nerves in the medulla. (Charles A. Ballance, Hamilton A. Ballance, and Purves Stewart. *Brit. Med. Jour.*, May 2, 1903, p. 1012).

whistle, to elevate the left nostril, to wrinkle the left half of the forehead, etc.

Sensation in left half of face is perfect. Hearing is impaired in left ear; she cannot hear a watch unless applied to the external auditory meatus.

Operation. June 30, 1906. An incision was made from the anterior border of the mastoid process parallel to the belly of the digastricus down to the level of the hyoid bone. The deep fascia was incised, hugging close to the anterior border of the sternocleidomastoid muscle. A blunt-scissors dissection was then made between the digastricus and the stylohyoid muscles close up to the base of the cranium. At the upper angle, passing across the field over the tip of the transverse process of the atlas, the spinal accessory was easily found. Just beneath the tip, and running parallel with the digastricus, the hypoglossal was seen. Passing up higher in the angle, and still hugging the mastoid closely, we arrived at the stylomastoid foramen, with its facial nerve, as a single trunk. The parotid was not disturbed; this can only be avoided by hugging close to the sheath of the sternomastoid. The facial was divided at the foramen, its end swung downward and joined to the end of the completely divided spinal accessory by a paraneural catgut suture. The connected point was then imbedded in the belly of the sternohyoid muscle and completely hidden from view by a few muscular stitches of catgut. The wound was closed without drain.

In a recent report which I received from Dr. Walkinshaw he states that the patient can now move practically all of the muscles of the face. The eyelids can be brought into apposition, but there is still a little drooping of the angle of the mouth.

In the removal of cervical glands the spinal accessory is not infrequently divided. It gives a paralysis of the trapezius, some of the sternomastoid, the former being the most noticeable, as it results in the drooping of the shoulder. A still more serious accident to the nerves, however, occurs in the operations for cervical adenitis. In the removal of the three or four outer lymph-nodes of the superficial set which are situated just above the middle of the clavicle and external to the jugular vein, two nerves are injured: 1. The rhomboidus; and 2. The levator anguli scapuli. Both of these are branches of the fifth cervical. When these are divided, either with or without the division of the spinal accessory, there is a swinging or tilting outward of the angle of the scapula, producing great deformity of the back and shoulders. If their division is recognized in the operation, they should be, like the spinal accessory, reunited with suture and the line of union imbedded in the muscle.

1. *The Nerves in Anteropoliomyelitis*

The histologic changes in the nerves have been but lightly treated in recent monographs on poliomyelitis, and are generally dismissed as being "the usual ones," or, "as one might expect them," etc., and for the fine changes one is referred to articles on nerve degeneration in general, the changes resembling those of traumatic or experimental degeneration by being primarily atrophic in type. In the literature on muscular changes in anteropoliomyelitis, however, several fairly accurate descriptions are found.

Prévost and David[1] describe an increase in interstitial tissue in the nerve filaments supplying the affected muscles, in the case of a man of 60 years who had the relics of infantile paralysis in the form of atrophy of the muscles of the right thenar eminence.

Schultze's[2] patient had infantile paralysis followed by paraplegia at the age of 3 years; the right leg was most affected; he died at 22. The sciatic nerves were of almost normal size; the perineurium and endoneurium were thick-

[1] Archives de Physiol. Norm. et Path., 1874, p. 595.
[2] Virchow's Archiv, 1876, bd. lxviii, p. 128.

NERVE ANASTOMOSIS FOR THE CURE OF FACIAL PARALYSIS, TIC, OR SPASM

A. Experimental

Author and Publication	Operation — Technique	Results	Remarks
Case 1. Paul Manasse (Munk's Laboratory.) Arch. f. klin. Chirurgie, vol. lxii, p. 822.	Seven dogs operated on, but only five considered in final results. Spinal accessory freed from its implantation into sternomastoid muscle to attas; end-to-end anastomosis of central spinal accessory with peripheral facial divided at the stylomastoid foramen. Four silk sutures passed through quarter of the thickness of spinal accessory.	Clinical results not sufficiently satisfactory. In first 3 months there was no faradic response; the latter obtainable after 4 months. Electric stimulation of central spinal accessory was followed by response in peripheral facial. Histologically, new axones penetrated the facial from the central spinal accessory.	Manasse thinks that "months" is an insufficient time for voluntary motion and disappearance of atrophy. "Years" would lead to perfect restoration.
Case 2. Barrago Ciarello. Policlinico, 1901, No. 3.	In two dogs. (*a*) Anastomosis between central end of spinal accessory and peripheral of facial. (*b*) Anastomosis between the central end of vagus and peripheral end of facial.	Restoration of function commenced after 4 months, and was almost completely restored in six. There was still some muscular atrophy left. Reaction of degeneration disappeared. Same results.	Ciarello believes that spinofacial and vagofacial anastomosis is promising in man in recent facial paralysis.

B. Clinical

Author and Publication	Condition	Nature of Injury — Cause	Operation	Results	Remarks
Case 1. Charles A. Ballance, Hamilton Ballance, and Purves Stewart. Brit. Med. Jour., May 2, 1903.	Facial paralysis.	Following operation for suppurating mastoiditis.	Spinofacial anastomosis. (Operation by C. A. Ballance.)	After several years, muscles responded to galvanic and faradic currents. Still some drooping of angle of mouth and widening of palpebral aperture. Movements always associated; voluntary independent movements of muscles of face not possible.	
Case 2. Ballance and Stewart.	Facial paralysis.	Otorrhœa of 20 years' standing.	Spinofacial anastomosis performed. (Operation by C. A. Ballance.)	Some improvement after 10 months.	
Case 3. Ballance and Stewart	Facial paralysis.	Following operation for otitis media.	Spinofacial anastomosis performed (after six months). Anastomosis of central end of spinal accessory with side of facial. (Operation by H. Ballance.)	All muscles of face responded to faradic currents after 4 weeks. Three months later, some voluntary motion.	Three sutures of French silk used.
Case 4. Ballance and Stewart.	Facial paralysis.	Following operation for otitis media.	Spinofacial anastomosis, 4 months after the initial paralysis. (Operation by C. A. Ballance.)	Faradic response after 9 months. Associated movements only. After 21 months, voluntary independent elevation of inner end of eyebrow.	
Case 5. Ballance and Stewart.	Facial paralysis.	Fracture of skull, due to a fall of 15 feet.	After 35 months. Spinofacial anastomosis. (Operation by C. A. Ballance.)	Slight voluntary movements in muscles of face if shoulder was elevated under resistance.	
Case 6. Ballance and Stewart.	Facial paralysis.	Following operation for otitis media.	Spinofacial anastomosis 6 months after initial paralysis. (Operation by C. A. Ballance.)	No improvement; no electric response; no return of independent voluntary or associated movements.	
Case 7. Ballance and Stewart.	Facial paralysis.	Following the removal of a cystic tumor of lower jaw.	Operation 12 months after initial paralysis. (By C. A. Ballance.) Anastomosis of peripheral end of facial with side of hypoglossal.	No improvement at the time of operation. Atrophy of half of the tongue.	
Case 8. Faure. Jan. 28, 1898. (*a*) Gazette des Hôpitaux, 71ème année, 1898, p. 259. (*b*) Revue de Chir., 1898, t. xviii, p. 1098.	Facial paralysis.	Injury	Eighteen months later, anastomosis of facial with accessories.	Slight improvement some time after the operation. "Half successful."	
Case 9. Robert Kennedy. Phil. Trans. of the Royal Society, series B, vol. cxciv, p. 127.	Facial tic (facial hemispasm).	?	Spinofacial anastomosis. End to side.	Cured. After 470 days, patient able to close eye. Muscles regained volume, and contracted voluntarily.	

B. CLINICAL — *Continued*

AUTHOR AND PUBLICATION	CONDITION	NATURE OF INJURY — CAUSE	OPERATION	RESULTS	REMARKS
Case 10. Hackenbruch (Weisbaden). Verhandl. der deutschen Gesellschaft für Chir., 1903, bd. xxxii, p. 231.	Facial paralysis.	Sudden paralysis of facial, 3 months' standing.	7¾ years after appearance of paralysis. Spinofacial anastomosis.	Improvement after 4½ months. After 9 months, patient could draw the angle of the mouth downward.	
Case 11. Körte. Deutsche med. Woch., April 23, 1903.	Facial paralysis.	Following operation for ostitis of mastoid process.	Hypoglosso-facial anastomosis.	Good after 1½ years, but associated movements present.	
Case 12. W. W. Keen. Phila. Neurol. Soc., Feb. 24, 1903. Jour. of Nerv. and Ment. Dis., Feb. 24, 1903, p. 366.	Facial paralysis.	Struck by a glass bottle.	Five weeks after accident. Spinofacial anastomosis.	"Perfectly smooth recovery."	
Case 13. Harvey Cushing. Ann. of Surg., May, 1903.	Facial paralysis.	Revolver - shot behind ear, involving mastoid.	Operation 43 days later. End-to-end spinofacial anastomosis.	Complete restoration of function.	
Case 14. Hackenbruch. Arch. f. klin. Chir., 1903, bd. lxxi, p. 631.	Facial paralysis of 7 years and 9 months' standing.	?	Spinofacial anastomosis by implanting two thirds of spinal accessory into facial.	Electrical response in paralyzed muscles. Voluntary contraction *independent* of those of trapezius.	
Case 15. James H. Nicoll. Lancet, 1903, p. 956.	Convulsive tic.	?	Anastomosis of facial with hypoglossal.	No improvement at the time of communication.	
Case 16. Szeyner. Abstracted in Fortschritt der Mediz., 1903, p. 1060.	Facial paralysis.	Shot; attempt at suicide.	Facial anastomosis with a flap taken from spinal accessory.	Voluntary contractions after 3 months; able to close eye, and whistle.	
Case 17. Charles H. Frazier. Univ. of Pa. Med. Bull., 1903–04, vol. xvi, p. 306.	Facial paralysis.	Gunshot wound.	End-to-side anastomosis between peripheral facial and side of hypoglossal.	Too recent to report conclusively on the case.	
Case 18. Glück. Berl. Gesell. f. Psychiatrie, May 11, 1903.	Facial paralysis.	Division of facial during mastoid operation for otitis media.	End-to-side anastomosis between peripheral facial and side of spinal accessory.	Regeneration of facial. Associated movements.	
Case 19. A. I. Bouffleur, Chicago. Personal communication.		Facial paralysis due to gunshot wound (attempt at suicide), June 14, 1904. The tip of mastoid was cut off by the bullet. Division of lower branches as well as of common trunk of facial.	End-to-end anastomosis between spinal accessory and facial, performed 4 weeks after injury.		Case lost sight of.
Case 20. Dr. O.	Facial paralysis.	Following operation for otitis media.	March 10, 1904. End-to-end spinofacial anastomosis. Dr. O.	Muscular atrophy not improved. Patient has associated movements only in muscles of face.	
J. B. Murphy.	Facial paralysis.	Same case as above.	March 10, 1906. Freed nerve from scar tissue and imbedded it in muscle.	Muscular atrophy improved, and voluntary motion of face is manifest. The associated movements continue.	Patient was reoperated on by the author.[1]

[1] The point of anastomosis found surrounded by great mass of connective tissue.
Electrical irritation of central end of spinal accessory produced contractions in muscles of the face, showing that new fibers had grown from the spinal accessory into facial, but that impulses could not travel, on account of surrounding connective tissue. Point of anastomosis was freed and protected by soft tissues. Good result.

AUTHOR AND PUBLICATION	CONDITION	NATURE OF INJURY — CAUSE	OPERATION	RESULTS	REMARKS
Case 21. Löhlein. Communication, Vereinigung der Chirurgen, Berlin, July, 1904.	Facial paralysis.	Otitis media.	Anastomosis of facial with spinal accessory.	"Excellent result." No associated movements.	
Case 22. Bardenheuer. Münch. med. Wochenschr., 1904, Nr. 28.	Facial paralysis of ten years' standing.	?	End-to-end anastomosis between peripheral facial and side of hypoglossal.	"Very good." Tongue paralyzed. No associated movements.	
Case 23. Bardenheuer and Lambeth. Festschrift zur Eröffnung der Akademie für praktische Medizin zu Köln, 1904, p. 219.	Similar.	Similar case.	Same procedure.	Good result.	

B. Clinical — *Continued*

Author and Publication	Condition	Nature of Injury — Cause	Oxeration	Results	Remarks
Case 24. Mintz. Zentralbl. f. Chir., 1904, No. 22, p. 684.	Facial paralysis.	Trauma during mastoid operation for otitis media.	Anastomosis of facial with spinal accessory.	Complete restoration of motor function. Good closure of eye.	
Case 25. Villar. Gaz. hebd. des Sc. Méd., Bordeaux, Feb. 7, 1904.	Facial paralysis.	Trauma to facial during mastoid operation for otitis media.	Anastomosis of facial with spinal accessory.	"The results were astonishing, as patient was able to move his lid on next day after operation. This rapid improvement, however, did not remain permanent."	
Case 26. Alexander. Wien. med. Wochenschrift, No. 11, 1904.	Facial paralysis.	Otitis media paralysis of 5 years' standing.	End-to-side anastomosis between divided facial and side of hypoglossal.	Paralysis of half tongue. No improvement at time of communication.	
Case 27. Alfred S. Taylor and L. Pierce Clark. Jour. Am. Med. Assoc., March 24, 1906.	Facial paralysis.	Suppurative mastoiditis.	The peripheral segment of facial inserted into a slit of hypoglossal.	Improvement after 25 months.	The anastomosis was protected by Cargile membrane.
Case 28. Same.	Facial paralysis.	Paralysis followed a severe earache.	Same operation.	Improvement.	
Case 29. Same.	Facial paralysis.	Suppurative mastoiditis.	Same operation.	Improvement.	
Case 30. Same.	Facial paralysis.	Bell's palsy. (Paralysis 12 years' standing.)	Same operation.	Improvement.	Although paralysis existed for 12 years, facial nerve was normal in size and aspect.
Case 31. Same.	Facial paralysis.	Suppurative mastoiditis.	Same operation.	No improvement.	
Case 32. A. I. Bouffleur, Chicago. Personal communication.	Facial paralysis resulting from fracture of mastoid, concomitant with fracture of skull (1903).		Nerve-trunk intact, as far as continuity is concerned. End-to-end anastomosis between spinal accessory and facial. Performed September 11, 1906.	Some improvement shortly after operation, inasmuch as fluids did not run out of the mouth from paralyzed side. Time too early to give definite results.	
Case 33. J. B. Murphy. Present paper.	Facial paralysis.	Division of facial during mastoid operation for otitis media.	End-to-end anastomosis between peripheral facial and central spinal accessory.	Evidence of returning motion pronounced. See history. Too recent for complete recovery.	

ened and rich in nuclei. The neurilemma-sheaths within the bundles were not thickened, but showed slight cellular increase. The nerve-fibers were thinner than normal, but not as thin as those of the anterior roots.

Déjerine[3] reports on a specimen from a child who died when 5 years old. At 2 years had anteropoliomyelitis followed by left talipes. He found disappearance of nerve-fibers in the intramuscular nerves involved. There were many empty neurilemma-sheaths, with increase in number of nuclei.

Similar loss of fibers, with corresponding increase in fibrous tissue, is described by Friedländer.[4] Von Leyden[5] and Stadelman,[6] also, describe atrophy and abnormal thinness of nerve-fibers with increase in interstitial fat, and emphasize the absence of inflammatory changes.

Sahli,[7] in an old case of anteropoliomyelitis with atrophy of right upper extremity, found no changes in the large nerve-trunks in the upper arm, except slight increase in interstitial tissue in the ulnar nerve; very marked fibrosis of the branch of the median nerve going to the atrophic thumb, and of the deep end-ramifications of the ulnar nerve. Here the endoneural sheaths were five or six times their normal thickness, and abnormal fibrous septa were seen within single nerve-bundles. There was no proliferation of neurilemma nuclei of the remaining nerve-fibers, which appeared normal.

Damaschino[8] briefly alludes to the nerve changes as atrophic, while a detailed histologic description, based on the study of 75 cases, is given by Rissler.[9] The first changes are found in the axones. In 3 out of 100 slides examined,

[3] Le Progrès Med., 1878, t. vi, p. 423.
[4] Virchow's Archiv, 1882, bd. lxxxviii, p. 84.
[5] Arch. f. Psych., bd. vi.
[6] Deutsch. Arch. f. klin. Med., 1883, bd. xxxiii, p. 125

[7] Deutsch. Arch. f. klin. Med., 1883, bd. xxxiii, p. 360.
[8] Gaz. des Hôpitaux, 1885, lviii, p. 625.
[9] Nordisk Med. Arkiv., 1888, xx, Nr. 22.

he found degenerated and swollen axones within normal hyalin-sheaths. More commonly the axones are broad, granular, and ill-defined from their myelin-sheaths, the latter being also in beginning degeneration. At the next stage we have the well-known picture of myelin lumps sometimes inclosing axone fragments within the neurilemma-sheaths. When the degeneration has reached this stage, the cells of the neurilemma also show distinct changes as follows: About the nucleus (or nuclei, as there are often two) the protoplasm is increased and sends processes inward, which not seldom penetrate between the inclosed myelin fragments. Myelin droplets often lie within the protoplasm of neurilemma-cells.

Eisenlohr[1] examined two cases, one dying fourteen months and the other three years after the acute stage. He carefully describes the atrophy of nerve-fibers, both in the muscular branches and trunks of the nerves going to the affected muscles. He emphasizes the simple atrophic nature of the nerve changes in anteropoliomyelitis as compared with the changes of multiple neuritis.

More recently, Bullen[2] has described atrophic changes with great increase of fibrous tissue, and in places with fatty change, in the nerves of an adult who died nine years after a traumatic poliomyelitis.

Leri and Wilson[3] describe and picture the nerve changes in an adult, who died seven and a half years after the acute attack.

Lövegren,[4] in a child who died two years after the attack, found atrophic nerve-bundles with fat deposited in the interstitial tissue between them. Other changes in the interstitial tissue were not detected.

From this report of the histologic changes in the nerve-trunks it appears that they are similar to those of degeneration of simple nerve division. Accepting this as true, they promise much for restoration by nerve transplantation.

The muscle changes in anteropoliomyelitis do not at first look at all promising for restoration with the return of nerve-supply. The opinion, however, changes when we consider that the nuclei are preserved to a great extent indeed, if not uniformly, in the atrophied muscle. These nuclei, from clinical observation, must be capable of regenerating and producing functionating muscles. The details of these muscle changes are as follows:

They are clearly stated in the article by Lorenz in *Nothnagel's Spec. Path. u. Ther.*, bd. xi, p. 2, where a good review of the literature is also found. (Many of the statements in the following brief review have been taken from Lorenz without consulting the original articles.)

From the literature the conclusion may be drawn that the muscle changes, like those in the nerves, chiefly consist of a simple disappearance of parenchyma with subsequent replacement by fibrous tissue, occasionally with fatty infiltration and other regenerative changes in the latter. In the old case of Prévost and David (loc. cit.) the atrophic muscles had been largely converted into fat, and only few transversely striated muscles were left.

Schultze (loc. cit.) describes the remaining muscle-fibers as thin, with plain transverse striation; muscle nuclei narrow, atrophic, not increased in number; increase in interstitial fibrous tissue, which at times entirely displaces the muscle-bundles. Déjerine (loc. cit.) found the muscle-fibers to be only three-quarters of their normal size, with intact longitudinal and transverse striation. There was no trace of fatty or albuminous degeneration in the interior of the primitive fibers. The nuclei were multiplied, but not larger than normal; in places, the muscle-bundles were entirely converted into fibrous tissue; interstitial tissue much increased.

Stadelman (loc. cit.) found the muscles atrophic, with fatty infiltrated but not increased interstitial tissue. Muscle-fibers very thin, cloudy, transverse striation only faintly visible. Many muscle-fibers contain deposits of fine granules, and also small deposits of fat. Empty sarcolemma-sheaths with fairly numerous nuclei are not rare, though there is no general increase of these nuclei. The longitudinal striation is even less marked than the transverse.

In the case of Sahli (loc. cit.) the greatly atrophied thenar muscles were largely transformed into fibrous tissue, poor in elastic fibers,

[1] Deut. Arch. f. klin. Med., 1888, bd. xxvi, p. 543.
[2] Jour. Ment. Science, 1902, vol. xxxviii, p. 71.
[3] Nouvelle Iconographie de la Salpêtrière, 1904, t. xvii, p. 433.
[4] Jahrb. f. Kinderheilk, 1905, bd. lxi, p. 269.

and rich in fat. Very few muscle-fibers remained, but these were normal. The lumbricales were entirely converted into fibrous tissue. Only in the palmaris brevis was an increase in elastic fibers present.

Damaschino (loc. cit.), in a case fatal three weeks after the onset, found the striation indistinct and proliferation of sarcolemma nuclei. At a later stage he describes replacement of the muscle by fat, which, according to him, had also been noted by Duchenne.

Eisenlohr (loc. cit.) described narrowing of the muscle-fibers, with preservation of the transverse striation. The increase in nuclei and perimysial fibers is in proportion to the atrophy. The most markedly atrophied fibers resemble connective-tissue fibers, and appear as homogeneous bands, with suggestion of transverse striation here and there, and in the oblong nuclei. He points out that in this form of muscular atrophy, as compared with that due to neuritis, little fatty change is found, the essential change being narrowing of the fibers, with retention of transverse striation and with nuclear proliferation. On the other hand, the changes in the perimysium in the two cases are similar.

Aufrecht[1] describes granular, fatty, and waxy degeneration in the muscles, and also nuclei fibers, which are pale, band-like, and rich in nuclei; these he takes to be regenerating fibers, while the fibers in the atrophied muscles usually are smaller then normal. Hypervoluminous fibers have been described by Oppenheim,[2] Strümpell and Barthelmes,[3] and Lövegren.[4] Bielchowsky,[5] in addition to changes like those described by Aufrecht,[6] found hyperplasia of the media of the blood-vessels.

Jagic[7] found no muscle changes in a case of acute anterior poliomyelitis of five days' standing. In a case of ten days' standing, Redlich[8] found advanced fatty degeneration without atrophy. This is considered a prodrome of atrophy.

[1] Deut. Arch. f. klin. Med., 1878, bd. xxii, p. 33.
[2] Arch. f. Psych., 1888, bd. xix, p. 381.
[3] Deut. Zeit. f. Nervenheilk, 1900, bd. xviii, p. 304.
[4] Jahrb. f. Kinderheilk, 1905, bd. lxi, p. 269.
[5] Zeit. f. klin. Med., 1899, bd. xxxvii, p. 1.
[6] Deut. Arch. f. klin. Med., 1878, bd. xxii, p. 33.
[7] Wien. med. Wochensch., 1899, Nrs. 9, 10.
[8] Wien. klin. Wochensch., 1894, p. 287.

Bullen (loc. cit.) found the muscles replaced by connective tissue rich in nuclei. Leri and Wilson (loc. cit.) describe chronic inflammation, atrophy, and fatty degeneration of muscle.

Lövegren (loc. cit.) found advanced muscle changes. "Here and there, groups of muscle fibers are met with, which are of normal size and polygonal on transverse section. Most of them are rounded and much reduced in size; however, here or there hypervoluminous fibers are seen. The muscle nuclei generally are enormously increased, so many fibers are largely covered by them. Not rarely, heaps of nuclei wholly surrounded by fibrous tissue are the sole remains of nuclear fibers. The transverse striation is frequently preserved, but in many fibers partly or entirely lost. Some fibers are split longitudinally and others are transversly broken up into lumpy masses. In places the muscle is converted into a mass of detritus. The fibrous septa are generally broad. Everywhere a large amount of adipose tissue is deposited; frequently it occupies the greater part of the section, so that only here and there small muscle-bundles are found imbedded in the fat. The walls of the intramuscular vessels are, in part, considerably thickened, the thickening effacing both media and adventitia. The intramuscular nerves show considerable loss in fibers, but also retain not a small number of apparently normal fibers.

From the above observation it can be seen, that the nuclei or regenerative factors are fairly well preserved, the changes being those incident to non-use (as atrophy), rather than to degeneration of the nuclei themselves. Clinical observations show that these muscles have regenerative potency, and on that basis, as well as on our clinical experience already at hand, promise good results for collateral nerve anastomosis.

Nerve Anastomosis for the Cure of Infantile Paralysis

Acute anterior poliomyelitis was considered until recently a medical disease, so far as the treatment was concerned. Since it has been demonstrated that a paralyzed nerve which is functionally separated from its motor center is still able to carry impulses if anastomosed with a functionally intact neighboring nerve, as previously shown in spinofacial anastomosis, several surgeons have seen the possibility of restoring motion in a set of muscles paralyzed as a result of anterior poliomyelitis by nerve anastomosis. The main points to be considered in surgical intervention for acute anteropoliomyelitis are: (*a*) The time of operation; (*b*) The extent of the paralysis; (*c*) The condition of the nerve — is it capable of regeneration? (*d*) The condition of muscle — is it capable of restoration? However, as Young states very correctly, "each case of spinal palsy is a study in itself"; therefore no general rule can be outlined.

As to the time for operation, according to Cushing it is probable that after it has become evident what the extent and what the seat of the residual palsy is likely to be, the earlier the operation is carried out, the greater will be the success. It is a well-recognized clinical fact that some of the members primarily paralyzed in an attack of anterior poliomyelitis regain their function, while others remain permanently paralyzed. Therefore it is necessary, according to Spiller and Frazier, to ascertain with exactitude before operating whether the full limit of nature's repair has been attained. As a rule, the muscles which do not show reaction of degeneration after three weeks will in all probability regain their motion. Spiller and Frazier recommend surgical intervention after six months, as until that time we are unable to ascertain which muscles will recover their functions and which will not. My experience is that infantile paralysis improves to a period of from eighteen to thirty months after the initial paralysis.

According to James K. Young,[1] the most suitable time for operation is between six months and three years. For surgical purposes we have classified the paralyses of the lower extremities into two nerve groups,[2] the lumbar and sacral. The former is composed of the second, third, and fourth lumbar roots: (*a*) The large branches continuing to form the anterior crural; (*b*) The smaller branches, to form the obturator; (*c*) The smallest branches from the first and second lumbar make up the genitocrural. The anterior crural partially supplies the extensor muscles on the anterior surface of the thigh, which are the quadriceps group and the sartorius, and sends some fibers to the abductors. The obturator supplies the adductor muscles of the thigh and the obturator externus. The sacral group supplies: (*a*) The flexor muscles in the thigh and the external rotators; (*b*) The internal popliteal, tibialis anticus, extensor muscles of the ankle, and flexor muscles of the toes; (*c*) External popliteal, anterior tibial, and extensors of the toes, and the musculocutaneous supplying the peroneal group of ankle extensors. In the upper extremity we will classify the paralyses into three nerve groups: (*a*) Those of the outer cord of the brachial plexus, including the musculocutaneous and median; (*b*) Those of the middle cord, the musculospiral and circumflex; (*c*) Those of the inner cord, including the ulnar and a portion of the median, involving their respective muscle groups.

Spiller and Frazier recommend for operation only those cases in which a few muscles are paralyzed. Cushing is of the same opinion. He says: "More complex problems arise when the paralyses are diffused and scattered." In other words, the operation is advisable when the paralysis involves only a certain set of muscles.

It seems from the literature that the operation for the cure of infantile paralysis was performed for the first time by Peckham.[1] This case was of ten years' standing. Considering the duration of the paralysis and the results obtained by the operation, I think his case is remarkable. Six weeks after the operation, the patient was able to extend the toes, which was absolutely impossible before. This is a histologic impossibility, according to either school, and it appears that there was either an error in the diagnosis or in recording the result.

The second case was that of James K. Young, who was inspired by the successful nerve anastomosis performed by Cushing for the cure of a facial paralysis. The case was presented before the Philadelphia Neurological Society, February 24, 1903.[2] The anastomosis was made between the branch supplying the anterior tibial muscle and the mus-

[1] International Clinics, 14th series.

[2] J. B. Murphy. Practical Medicine series, Surgery, 1901.

[1] Providence Medical Journal, Jan., 1900, p. 50.

[2] International Clinics, vol. iv, 14th series, p. 154.

culocutaneous. The former was inserted into a slit made in the latter, and secured to it by fine catgut. The following is, in brief, the result of the case: "The gait is fair, and the child can draw up the inner side of the foot almost with normal power."

The third and fourth cases were those of Spiller and Frazier.[1] The paralysis was limited to the anterior tibial muscle. Anastomosis between the upper division of the anterior tibial nerve and the musculocutaneous nerve was made. "The branches of the nerve which supply the anterior tibial muscle at its upper part were pushed through the incision in the musculocutaneous nerve without any attempt being made to separate the nerve-fibers from the sheath."

At the time of publication, "the gait is nearly normal and the child can draw up the inner side of the foot almost with normal power."

The fourth case was that of paralysis of two years' standing, confined to the peroneal muscles. Anastomosis between the anterior tibial muscle and the musculocutaneous was performed. No improvement was noticed after four weeks. No change for the better in the electrical reaction of the muscles was observed.

The fifth case is ours.

Becky H., schoolgirl, aged 10. Admitted to Mercy Hospital, January 23, 1905; operated on, February 1, 1905; left, February 28, 1905.

Diagnosis. Anterior poliomyelitis.

Present Illness. In 1899, when patient was 4 years old, played out-of-doors for several hours. Coming home, she complained of being tired. Mother put her to bed, and next morning patient awoke with a *high fever*, and *loss of motion* in both limbs, below the knees. History of previous trauma or chill cannot be obtained. The fever remained high for two to three days. The left limb gradually regained its power, but the right one never fully. The muscles of the latter atrophied, and the foot was in a condition of talipes. For past two years she has worn a brace reaching to the knee.

Pathology. Paralysis of all muscles supplied by external popliteal nerve.

Operation. February 1, 1905. Plastic operation on tendons and nerves. *Tendons:* Tenotomy: tendo Achillis elongated by splicing. Posterior ligaments stretched. Incision below internal malleolus. Tendons of flexor communis digitorum elongated. Flexor longus pollicis elongated. Plantar fascia, inner two thirds divided close to attachment to os calcis. This was done by subcutaneous incision. Tibialis anticus exposed, tendon shortened, and right half of it reattached in normal position, and the other half attached to middle cuneiform, displaced more to middle line by putting it in another sheath. Tendon of extensor digitorum exposed and shortened.

Nerve Anastomosis. External popliteal nerve completely divided and implanted into a one half division niche of the internal popliteal nerve. The union was accomplished by kangaroo-tendon sutures. The same suture material was used to unite the nerve-sheaths side to side for additional mechanical support. Fascia closed around the point of union with catgut. Skin closed with horsehair.

February 9, 1905. Stitches removed. Primary union of all wounds. Flexor longus pollicis and gastrocnemius act.

Post-operative Report. One year later, January 15, 1906. (*a*) The circumference of the leg has increased ½ cm. (*b*) The axis of the foot is on the same level with the axis of the leg (no inversion of foot). (*c*) Slight faradic response in muscles supplied by external popliteal. (*d*) Voluntary extension of the foot. (*e*) Patient cannot evert the foot. (*f*) Patient can run and jump. This result shows a restoration of function of the tibialis anticus and extensor longus digitorum; but up to this date the peronei have not been restored.

The sixth case. Hackenbruch of Wiesbaden reported a case of infantile paralysis in which nerve anastomosis was used. He implanted one third of tibial nerve into the peroneal nerve. At the time of communication, some improvement was made.

The seventh case is ours.

G. N., aged 7, schoolboy. Admitted to Mercy Hospital, November 24, 1905.

Present Illness. Patient was born at full term. Commenced to walk at the usual time, and walked normally up to the age of 3½ years. At that time he had an attack of: (*a*) Vomiting; (*b*) Fever; (*c*) Constipation. Was in bed for a long time, and when he was able to leave bed, parents noticed an impairment in gait to the extent of his requiring help when walking. There was a paralysis of the right leg from the knee down. In the course of time there was return of motion in most of the muscles, and finally patient commenced to walk and run, although imperfectly. He walked on the outer border of the right foot.

Patient has been in good health since the attack of 3½ years ago. Appetite is good; no gastro-intestinal symptoms. Is affected with enuresis. Child is of nervous temperament.

Previous Illness. Had smallpox at the age of 2. No other infectious disease. Two aunts are affected with epilepsy.

Examination. Patient is in good health. General appearance good; well developed for his age. Left leg normal. Left foot: The arch of the inner side of the foot is excavated; moderate degree of pes cavus.

[1] Jour. Amer. Med. Assoc., Jan. 21, 1905.

Right leg: The circumference, taken 2½ cm. from the apex of patella, shows a difference of 1 cm. as compared with the left. Circumference taken 1 cm. above the articular line of ankle-joint shows a difference of ½ cm. to the advantage of the right. Patient walks on the outer border of his foot. When he walks, the left half of the body is higher, the right half being thrown on the right foot, and especially on its outer border. The attitude of the foot is such that it forms an obtuse angle, with the opening inward. The foot is in forced adduction. Flexion and extension of toes is perfect. Voluntary abduction limited. Voluntary adduction exaggerated. Patient has a paralysis of the peroneus longus and brevis; or in other words, the nerves which supply these muscles are paralyzed. They are branches of the musculocutaneous, a subdivision branch of the external popliteal. Operation has for its purpose anastomosing the paralyzed branches supplying the peroneal with that supplying the anterior tibial; also implanting the distal peroneal tendons into the tendo Achillis.

Pathology. Paralysis of the peroneus longus and brevis muscles.

Operation. November 26, 1905. Transplantation of two fifths of tendo Achillis into split tendon of long and short peroneus. Implantation of musculocutaneous into a niche made in the trunk of anterior tibial. Kangaroo tendons used. Time is too short for any alteration in condition of muscles.

Nerve Anastomosis. A new nerve-supply for the paralyzed group of muscles. Generally, either one of the three groups usually paralyzed can be secured by transplanting the external or internal popliteus at the upper angle of popliteal space. In paralysis of the extensors, the external popliteal is paralyzed. It should be completely divided and inserted into the ends of a one third niche division of the internal popliteal nerve, so there will be a direct end-to-end contact of normal proximal fibers to the diseased distal fibers of the external popliteal. We combine the nerve anastomosis with tendoplasty, elongation of the flexor tendons, and shortening of the extensors, which overcomes the talipes.

When a muscle is paralyzed, and contraction of its opponent has taken place, the opponent tendon should always be elongated beyond its normal length, so as to give the paralyzed muscle the leverage advantage in the joint-fulcrum.

W. R., Cazenovia, Wisconsin, female, aged 9, schoolgirl. Admitted to Mercy Hospital, February 18, 1907.

Family History. Negative.

Personal History. Born and always lived in Wisconsin.

Previous Illnesses. The usual diseases of childhood, including an attack, at the age of 3, diagnosed as scarlet fever; recovery from the disease without complications. No history of injury.

Present Trouble. According to mother's statement, it dates back to March, 1902. One evening the patient retired apparently in good health. When she awoke the next morning she called for assistance, because she was unable to get up and unable to walk. Her hands were so weak that she was unable to hold anything in her hands, moreover, the lower limbs were paralyzed. For a week previous to the trouble, the patient was somewhat irritable and peevish. She felt "cross," as the mother chooses to describe the situation, and vomited several times during that week, mostly at night. Toward noontime of the same day she experienced severe pains in all four extremities, and the slight motion in the toes and in the fingers disappeared towards evening, so that all four extremities were completely paralyzed. The pains gradually subsided the next day, and finally disappeared in about 48 hours. The child was very sick the day of the trouble, and for four or five days afterwards, but the mother is unable to say whether fever was present. The patient remained paralyzed for three months, at the end of which period she began to creep. In the course of time she gradually regained power in both lower and upper extremities, and after six months she was able to eat without assistance, and commenced to walk. The left limb recovered almost completely, while the right one remained "weak." The patient wore a brace up to the present time, and was able to walk, run around, and go to school. She has been in very good health all this time. Her appetite is good; has no gastro-intestinal symptoms; no urinary symptoms.

Examination of the Patient. The child is well nourished and fairly well developed for her age. Lungs negative, heart negative, abdominal organs negative, on physical examination.

Upper Extremities: The limbs are well developed and shaped; skin normal in appearance; circumference of forearm and arm equal on both sides. No sensory nor motor disturbances. Muscles all active.

Lower Extremities: The right leg is smaller in circumference than the left one; the right foot is in forced adduction. There is paralysis of the peroneal group of muscles; weakness of the tibialis anticus, but no paralysis; there is some weakness of the extensor communis digitorum. The posterior muscles of the leg are apparently normal. There is some weakness of the similar group of muscles in the left leg.

Operation and Pathology during Operation. (Right foot.) Contrary to our expectations, faradic stimulation of muscles supplied by the musculocutaneous, and also the peroneal group of muscles, showed that the muscles were not completely paralyzed, and that they were only in a state of paresis. The reason why, clinically, it appeared as though there was a complete paralysis of muscles is explained by the fact that the antagonistic muscles overcame the weak muscles. In other words, the defective leverage gave the impression of paralysis. This shows that, clinically, we cannot always judge the exact condition of a certain set of muscles, and that the only definite and positive means of judging the degree of potency is the direct stimulation of either muscle or its respective nerve-supply. It also urges the physician to use orthopædic measures in every case of weakness of a certain group of muscles, so as to balance the powerful antagonistic muscles, in every case where operation is not performed.

Operation. Tendo Achillis was split and cut, so as to increase the length two and a half inches.

Half of it was detached, with a fragment of bone from the tuberosity of the os calcis. It was then split, carrying the part with the fragment of bone out to the tuberosity. The bone was freshened and was united with kangaroo-tendon sutures. The extensors of the foot were then exposed. The tendon of tibialis anticus was split; half of it was attached to the external cuneiform with kangaroo tendon, and the rest was shortened by plication. All tendons on anterior aspect of the foot were shortened.

The case is one of anterior poliomyelitis, and was intended to be treated by nerve anastomosis, the distal end of external popiteal to be inserted into a niche made in the internal popliteal. The absence of paralysis in the domain of the external popliteal changed the plan of operation, and tendoplasty was substituted for nerve anastomosis.

As has already been suggested by Young, nerve anastomosis can be supplemented by tendon implantation. Tenoplasty, to be successful, should involve three elements:

1. The implantation into a potent muscle tendon of a tendon of the paralyzed muscle.

2. The elongation of contracted opposing muscle tendons, if such exist; as the effect of a transplantation is often nullified by the contracted condition of the opposing muscles.

3. The adjustment of these forces, so that the pressure on the articulation is balanced over the central or pivotal zone of the joint.

TECHNIQUE OF OUR OPERATION

1. Plastic Operations on Tendons

(*a*) Tenotomy of tendo Achillis; tendon spliced.

(*b*) Posterior ligament stretched or divided.

(*c*) Incision below internal malleolus; tendon of flexor communis digitorum elongated; flexor longus pollicis elongated.

(*d*) Plantar fascia divided by subcutaneous incision close to its attachment to the os calcis.

(*e*) Tibialis anticus exposed; tendon shortened and displaced more to the middle line, attaching half of it to the periosteum of the middle cuneiform.

(*f*) Tendon of the extensor longus digitorum exposed and shortened.

2. *Nerve Transplantations*

External popliteal nerve completely divided and implanted into a niche in the internal popliteal. The union is accomplished by fine kangaroo-tendon sutures. The same suture material is used to unite nerve-sheaths. The operation was performed by us February 1, 1905, and the stitches were removed February 9th. Primary union of all wounds.

A point to which insufficient consideration and thought have been given concerning the principle of nerve anastomosis is the rôle played by the interfascicular and intrafascicular connective tissue of nerves.

Anatomy teaches us that between the several fasciculi which constitute the nerve-trunk, and among the fibers themselves, there is connective tissue which separates and *insulates* the neurones and nerve fasciculi. According to my conception, connective-tissue neurilemma and medullary sheath do not perform the function of carrying nervous impulses, so that it may be said that the axones included within a nerve fasciculus are independent longitudinal units. On this premise I believe that the connective-tissue, neurilemma and medullary sheaths separating the fasciculi and surrounding the axones

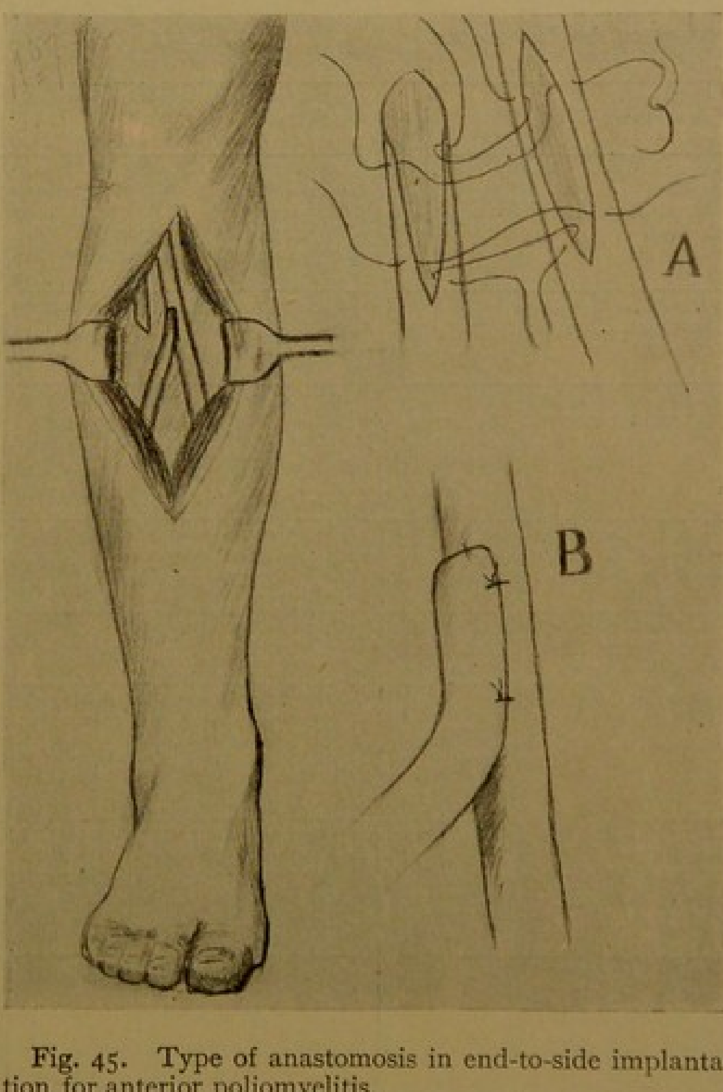


Fig. 45. Type of anastomosis in end-to-side implantation for anterior poliomyelitis.

and nerve-fibers act as insulators, which must be taken into consideration in nerve anastomosis. The technique of Frazier and Spiller, which consists in making a longitudinal slit into the substance of the nerve and carrying the transplanted nerve-end through this slit to the opposite side and attaching it there, does not seem, histologically speaking, correct, but the final solution of this must come from practical results. I believe, in order to get the best results in nerve anastomosis, that the neurones should be placed in close end-to-end apposition, so as to bring the surface of the peripheral segment in contact with and directly facing the fibers of the central segment. This, of course, can take place only in end-to-end union, or end-to-side (niche) (equivalent to end-to-end of axones) union, as in most of the cases it is not desirable to sacrifice the whole trunk of a healthy nerve. The anastomosis should be made by a transverse niche in the healthy trunk, into which the distal segment is fixed. The transplanted trunk should join its recipient at an acute angle. If a lateral apposition were sufficient, and impulses could be transferred to neighboring healthy neurones, the external and internal popliteal should carry the respective impulses of their neighbors in their close apposition in the sciatic nerve. This they do not do, as each fiber is effectually insulated. It is possible that by harrowing the sciatic in its middle portion with fine needles, after the plan of Réclus, that this insulation can be sufficiently disturbed to permit of alternating impulse transmission. End-to-side implantation does not imply apposition of exposed nerves.

In the Spiller and Frazier cases, the branches supplying the anterior tibial muscle (the par-

NERVE ANASTOMOSIS FOR THE CURE OF ANTERIOR POLIOMYELITIS, BRACHIAL PALSY, BULBAR PALSY, ETC.

Author and Publication	Condition and Extent of Paralysis	Operation	Result
Peckham. Providence Med. Jour., 1900, p. 5.	Anterior poliomyelitis; 10 years' standing.	Section of healthy fibers and anastomosis with diseased fibers.	Extension of toes 6 weeks later.
W. G. Spiller and Charles H. Frazier. Jour. Am. Med. Assoc., Jan. 21, 1905.	(*a*) Paralysis of anterior tibial muscle (limited paralysis, anterior poliomyelitis). (*b*) Paralysis confined to the peroneal muscles; 2 years' standing.	Branches supplying tibial muscles pushed into musculocutaneous. Anastomosis between anterior tibial nerve and musculocutaneous.	(*a*) "Gait nearly normal, and the child can draw up the inner side of foot almost with normal power." (*b*) No improvement after 4 weeks. No change in the electrical reaction.
James K. Young. Internat. Clinics, 14th ser., vol. iv, p. 159.	Paralysis limited to anterior tibial muscle only.	Anastomosis between the branch supplying anterior tibial muscle and musculocutaneous by inserting the former into slit formed in the latter.	Improvement at time of presentation of patient.
John B. Murphy. (Present paper.)	Paralysis, limited to anterior tibial muscle and extensor communis digitorum; 5 years' standing.	Nerve anastomosis combined with tendon-plastic. (*a*) Anastomosis between external and internal popliteal. (*b*) Elongation of flexors, shortening of extensors.	Great improvement. Extension voluntary. Electrical response in extensors. Patient walks — runs.
Kader (Cracow, Poland). Quoted in Chipault's État actuel de la chirurgie nerveuse, vol. ii, p. 202.	Four cases of infantile paralysis with equino-varus (paralytic)	Anastomosis between external and internal popliteal; also, tendon plastic, elongation of flexors, and shortening of extensors.	One success; one failure; one improvement; one in convalescence at time of publication.
P. Hackenbruch (Wiesbaden). Proc. Germ. Surg. Cong., 1903, vol. xxxii, p. 238.	Infantile paralysis.	Implantation of one third of tibial nerve into the peroneal nerve.	Some improvement at the time of communication.
John B. Murphy. (Present paper.)	Infantile paralysis. Paralysis of peroneus tertius and brevis.	(*a*) Transplantation of two fifths of tendo Achillis into slit tendon of long and short peroneal. (*b*) Implantation of two thirds of peroneal branch of musculocutaneous into anterior tibial.	Too early for muscular return.
Willfred Harris and V. Warren Low. Communication, Neurological Society, London. (Brain, 1903, p. 465.)	Paralysis of left arm, due to fall on shoulder. Muscles paralyzed: Deltoid, supraspinatus and infraspinatus, supinator longus, both radial extensors of wrist, pronator radii teres.	Fifth branch cut across one third inch above junction with sixth root, and distal end "sutured into a nick made in the upper border of the sixth root."	Recovered sensation in thumb perfectly, in 3 months. No return of motion at the time of publication.
Same authors. Brain, 1903, p. 466.	Infantile paralysis of shoulder.	Fifth root split into halves longitudinally Then upper half of fifth root divided one inch above junction with sixth root; distal end turned downward and sutured into a nick made in upper border of sixth root.	At the time of communication it was too early to report conclusively.

alyzed segment) were pushed through an incision in the musculocutaneous nerve, "without any attempt being made to separate the nerve-fibers from the sheath."

According to Young, the ideal method of anastomosing nerves would be to place the axis-cylinders of the divided nerve in relation to and parallel with the axis of the sound nerve.

In our case, a new nerve-supply for the paralyzed group was secured by implanting the external popliteal nerve into a niche in the internal popliteal, thus securing an end-to-end union of the neurone. The nerves were readily exposed at this point. The external popliteal was completely divided and inserted into a one third transverse division niche of the internal popliteal nerve, so that there was direct contact of the normal proximal fibers with the distal fibers of the diseased popliteal. Merely drawing the diseased ends through a slit of a healthy nerve is not sufficient.

Brachial Plexus — Birth Paralysis. I do not intend to go into the anatomy of the brachial plexus. I wish, however, to emphasize a few points which may be of value in the diagnosis of its lesions. The circumflex nerve is given off just at or a little below the clavicle. An injury above the latter will not only involve the circumflex individually, but also the musculospiral.

The pectoralis major has a double nerve-supply. The external cord gives one branch and the internal cord the other. If, therefore, the pectoralis is completely paralyzed, it indicates that both cords are involved in the lesion; one from the upper and the other from the lower brachial plexus roots.

In endeavoring to establish whether the roots of the brachial plexus are divided at the foramina or beyond it, the integrity of the diaphragm on the corresponding side is of positive diagnostic value. The movements of the diaphragm can readily be observed with the fluoroscope. If it acts on the side in which a lesion of the brachial and cervical plexuses is suspected, then the cords are not divided in the foramina, but a considerable distance outside. In a recent operation we found the phrenic had branches of origin from four cervical roots. The faradic

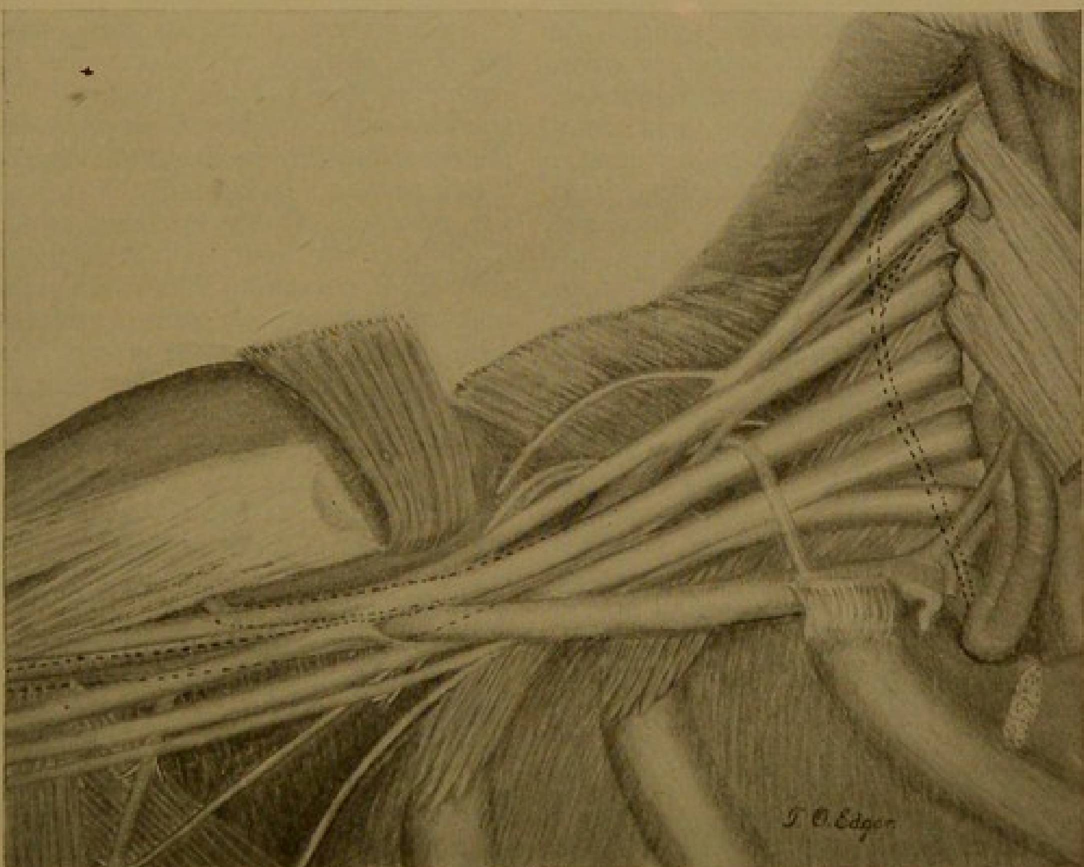


Fig. 46. Brachial plexus. The dotted vertical branch is the phrenic nerve. (Original drawing. From a dissection made by Dr. White at the College of Physicians and Surgeons, Chicago).

exciter applied to any one of these produced spasm of the diaphragm.

Nerve anastomosis has been used in several instances, seldom, however, for the cure of birth paralysis. If we are not mistaken, the first attempt was made by Robert Kennedy,[1] who regards birth paralysis the result of trauma to the brachial plexus from forcible depression of the shoulder during delivery. Some cases recover without treatment, others never recover. In his case the muscles affected were deltoid, infraspinatus, biceps, and brachialis anticus. As to the treatment, this author is of the opinion that no intervention is necessary for about two months. It is very desirable to test the muscles at that time by faradic currents. If they respond, showing that the axones are not broken, the case will, in all probability, recover, and no surgical intervention is necessary. If they do not respond to faradic currents, but do so to galvanic, an operation is indicated.

The following case of birth paralysis was treated by us at Mercy Hospital:

Miss K., aged 24 years, housekeeper. Admitted to Mercy Hospital, October 12, 1904.

Present Illness. A few days after birth, the mother noticed that her baby could not move her right forearm and hand; this condition has continued ever since. At present she cannot extend the arm, forearm, or wrist, nor can she extend the fingers nor supinate the forearm. She can flex the fingers fairly well. She cannot, however, flex the forearm nor put her hand to her mouth. The deltoid was paralyzed. The musculature of the right arm, forearm, and hand is atrophied. It cannot be ascertained whether there was any obstetrical accident at her birth. All of the muscles supplied by the posterior cord of the brachial plexus are paralyzed, and those supplied by the inner and middle cord are atrophied.

Diagnosis. Birth paralysis, involving the sixth and seventh trunks of the brachial plexus. It was my belief that the trunks had been contused or divided, and that the best result would be obtained by transplanting the distal portion of the musculospiral into the proximal end of the completely divided ulnar; that we should then have flexion through the nerves supplied by the median, and extension by the muscles supplied by the musculospiral, thus making a fairly serviceable hand out of a now practically useless one. This, however, left all of the shoulder muscles supplied by the circumflex, the axillary, the subscapularis, and the anterior thoracic still paralyzed.

Operation. October 14, 1904. Complete division of the ulnar and musculospiral; the distal end of the musculospiral was united to the proximal end of the ulnar, and the distal end of the ulnar was inserted into a niche in the median.

[1] Brit. Med. Jour., Feb. 7, 1903. "Surgical Treatment of Birth Paralysis of Upper Extremities."

I am indebted to Dr. Church for the careful examination of the case before the second operation.

Report of Dr. Archibald Church. Examination January 14, 1905, four months after the first operation, which accounts for the response in some of the musculospiral group through the ulnar root:

"Your patient, Miss K., came to the office this morning, and I made an electrical examination of the arm, which resulted, briefly, as follows:

"All muscles below the elbow in the forearm and hand refuse to respond to any kind of electrical current, except the supinator longus. This shows partial reaction of degeneration. Very slight response to faradism with the negative and positive contractions; to galvanism, about equal. The biceps and triceps and posterior scapular group respond fairly to faradism, and about equally to both the positive and negative pole of galvanism. The pectoral muscles respond to all forms of current, and are practically normal, although in a weak manner. Sensation seems to be natural throughout the extremity, except the tips of the four inner digits, especially on the palmar surface. The vasomotor condition of the hand is defective, the parts being cyanotic."

Four months after this examination, on going over the case more closely, I decided that a second operation should be performed, which was done August 30, 1905.

I determined to find the site of the original injury to the cervical roots, which occurred 24 years previous, supplying the brachial plexus. I feared, from the teaching which we had received on nerve atrophy, that the cord distal to the line of division would be so small that it could not be located. To my surprise and pleasure, I found it of fully developed adult size. The point of division was easily found, as there was a proximal and distal neuroma. The bulbous ends were excised and an end-to-end suture approximation secured. Line of junction imbedded in muscle to prevent connective-tissue intervention. The seventh cervical trunk was found imbedded in dense scar, but not divided. It was freed from connective-tissue bindings, and protected by muscular flap. I then undid the work of the first operation, re-established the musculospiral and ulnar trunks in their normal anatomic relations. At the end of the operation there was complete paralysis of all of the arm and forearm.

She remained in the hospital for four months, and at the end of that time she was able to grasp a glass and carry it to her mouth. Her power of extension was yet very feeble. She could not supinate the forearm nor extend the hand. The strength of the flexors was fairly good. She has returned to her home in Europe, and I have not been able to receive a recent report of her condition.

Division of the roots and cords of the brachial plexus are not uncommonly the seat of trauma

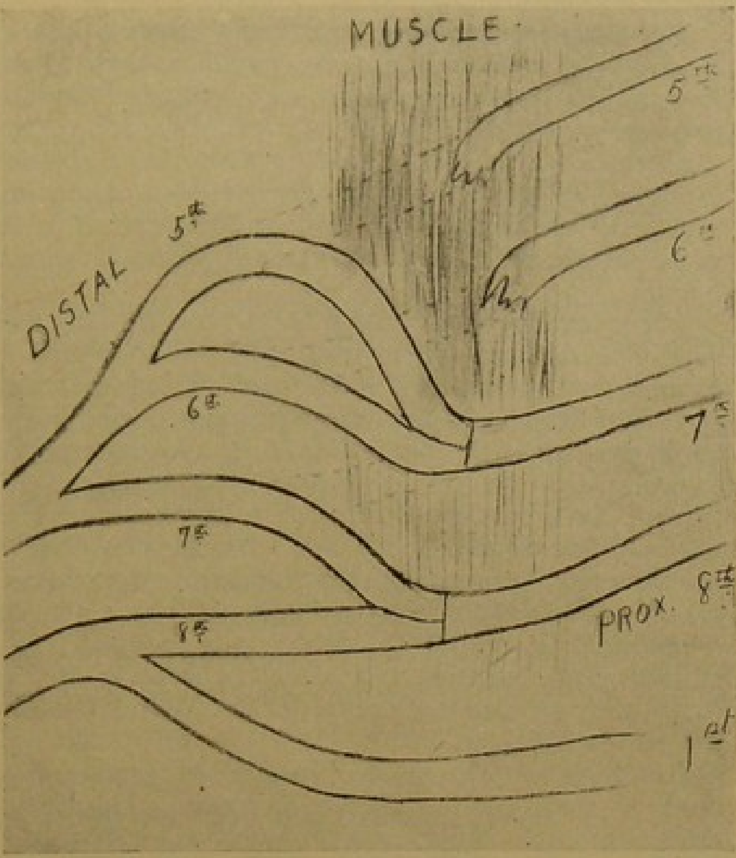


Fig. 47. Type of approximation of branches of brachial lexus.

resulting from blows or severe pressure on the angle of the neck. I have seen three cases of complete paralysis — two the result of falling trees, not operated on; the other the result of a railway accident, details of which are given in the following history. In all three cases there were two symptoms for which the patient demanded relief; foremost, the intense neuralgia, and second, the paralysis; the first the result of the neuroma, and the second the sequence of nerve division in cicatricial compression. The following case illustrates the pathologic conditions and our technique of operation.

Mrs. W. H. C., aged 44, housewife. Admitted to Mercy Hospital, September 1, 1905.

Present Illness. Seventeen years ago, patient was hurt in a railroad accident. (She is unable to give details.) She states, however, that after the accident she was unconscious for 22 hours, and when she recovered she noticed that her left arm was completely paralyzed. The arm was an inert body, and was of no service whatever. A few days after the accident, she noted an ecchymotic area on the tip of left shoulder. It was evident from the flaccid paralysis that the roots of brachial plexus had been injured near the foramina by a blunt object, which did not cut the skin. The first three weeks after the accident, patient had no pain. At the end of this period, she suddenly experienced pain from the shoulder to the tip of the fingers. She states that the pain was very severe, and she could only compare it to a burning and at times to a severe toothache. From that time on, the arm remained completely paralyzed, and up to the present the patient has never been free from pain. At times it was paroxysmal, very severe, almost unbearable, and not relieved by medication. For nearly a year after the accident, electricity was applied; daily massage and passive movements were resorted to for thirteen months. Three years later, she was operated on, two operations being performed at intervals of three weeks; the first operation in the axilla, the second one in the neck. The patient cannot state the nature of these; she thinks, however, that the surgeon "stretched the nerves." Neither of these operations helped her. The arm was still paralyzed, and the severe pains were experienced just the same as before the operations.

The general health of the patient has been good during this period, but was very nervous. Has been able to do her household work with her right arm.

Previous Illnesses. Patient denies any acute illness.

Obstetrical and Family History. Negative.

Examination. Medium stature. General nourishment, fair. Has a neurotic aspect. One can easily read in her face worry and suffering. Thoracic and abdominal organs negative. Patellar reflexes slightly exaggerated. Right arm normal. Left arm lies against the thorax like an inert body; no voluntary motion in the left shoulder, arm, or forearm. The tissues are flaccid; the muscles are atrophied. The adipose tissue is very abundant. The skin is smooth, warm, very fine, and whiter than that of the other hand. The fingers are conical in shape, and the nails convex and small. The patient is unable to extend or flex the fingers of left hand. She cannot pronate or supinate the hand, is unable to flex the forearm upon the arm. Pronation of the forearm is impossible. Supination of the arm and forearm from the shoulder slightly possible; however, by closely observing the movement, it was discovered this is not performed by the supinator of the forearm, but is brought about by some active effort of the biceps, combined with a voluntary elevation of the shoulder. In other words, the supination is only apparent, not real. She cannot lift the elbow, nor project it forward or backward. She is able to elevate and lower the shoulder voluntarily. Spinal accesssory intact. The electric reaction of muscles could not be used in this case, as patient is extremely neurotic and cannot stand even a weak current. After a few tests, she refused further use of electricity.

Sensation. Patient cannot feel the touch of a needle along the arm and forearm. She has no tactile sensation in the thumb and index finger. Very little sensation in the other three fingers. It was found, however, that on touching certain areas of the dorsum of the hand with the point of a needle, the patient asserted that she was faint. There were very few areas which, being touched by the needle, caused great local pain or diffuse and severe pain through the entire arm. The elbow reflex is absent. From what we observed, it appears that the only voluntary motion is the elevation of the left shoulder. The deltoid muscle seems to take little part in this action.

Pathology and Operation. Complete division of left fifth, sixth, seventh, and eighth roots of brachial plexus about two inches from the spine; divided ends were matted into a firm, cicatricial mass, which was smaller distally than proximally. The distal portion of the mass was beneath the clavicle. The striking feature is that the cords below the line of division were apparently normal in size, and that the application of the electrodes to the distal portion of median, musculospiral, and circumflex nerves was followed by immediate response. The ulnar responded tardily.

Operation. September 1, 1905. Cicatricial mass dissected out, and proximal and distal bulb-ends of the four roots exposed. The neuromata excised, and the two distal portions of the seventh and eighth roots were attached to the proximal end of the eighth, and the distal portions of the fifth and sixth were approximated to the seventh. The lines of union were protected by the scalenus muscle and deep fascia. Kangaroo tendon material was used. The faradic response we interpreted as being due to a peripheral regeneration of the nerve, but the fact that the first dorsal segment had not been divided may account for the muscular contractions. Primary union was obtained. Clavicle, which was divided during the operation for the purpose of gaining space, was wired.

Result. April, 1906, seven months after operation No sensitiveness on pressure in the supraclavicular fossa. The latter has filled out almost equal to the opposite side. The head of the humerus is somewhat prolapsed. The arm has increased in size, and is about equal to the other. The skin has lost its glossy appearance. Sensation to light touch and to the faradic current is absent below the elbow. The pain, which was such a prominent feature before the operation, has entirely disappeared. There is a muscular hyperæsthesia in the arm. The patient makes use of the following expression: "The arm is progressively becoming more and more like a tin arm from the shoulder downward." *There is no return of voluntary motion up to this date, which is exactly what should be expected from our knowledge of regeneration of nerves.*

Mr. S. S., Kenton, Michigan, laborer, aged 17. Admitted to Mercy Hospital, January 21, 1907.

Family History. Negative.

Personal History. Born and always lived in Michigan. Farmer by occupation. Habits regular.

Venereal History. Negative.

Previous Illnesses. Most of the diseases of childhood.

Present Trouble. Six weeks ago, while at work, his right arm was caught in a pulley, and arm was carried over pulley. Patient was unconscious for some time, and when he recovered he was told that he had a fracture of the skull, that the right arm was fractured at the elbow, and that the right clavicle was also fractured. The arm was kept in a cast for three weeks. Upon its removal the patient noticed that the arm was totally paralyzed. He has not recovered sensation or motion since.

Examination of the patient shows absolute loss of all forms of sensation from the shoulder down to the fingers, except the cutaneous areas supplied by the first and second dorsal; that is, the inner portion of the arm and forearm, and a great portion of the anterior aspect of the shoulder. All muscles of the forearm, arm, and hand are completely paralyzed, except the flexor carpi ulnaris, which responds partially to faradic stimulation.

These findings led us to the belief that all the nerves supplying the arm and forearm were either divided or compressed, except a few fibers of the ulnar which remained intact, and that this division occurred below the formation of the brachial plexus. A dissection of the neck above the clavicle showed that the plexus was intact. On a subsequent dissection below the clavicle, we found, as we assumed, that all trunks were divided and imbedded in considerable connective tissue, which changed the anatomic relations to such an extent as to make it impossible to identify the trunks. Only after careful dissection and extensive removal of scar tissue were we able to identify the trunks and re-establish their continuity.

Pathology and Operation. In none of terminal portions of nerve could response be gotten. Distal portions of musculocutaneous were found to be separated, and did not respond to faradic current. Musculocutaneous exposed and freed from connective tissue, was then cut, and lacerated portion removed, and freed ends came together with little tension.

The median nerve was badly traumatized and strangulated in several places. The traumatized portion, including neuroma, was resected, and ends approximated by traction of ends. The appearance of excised portion of nerve was strikingly characteristic. The middle of neuroma showed on cut surface a uniform whitish surface without bundles, while at a point where it was separated from healthy nerve, bundles showed up very conspicuously. Ends were approximated with catgut.

The ulnar had a depression and thickening above and below; no real neuroma at the point of injury. The strangulated portion was excised and ends approximated with catgut. The excised portion had a dumbbell appearance. There was no response on placing electrode on musculospiral. Multiple depression was found on the nerve. At one point it was completely divided and continuity of the nerve held by connective tissue. One and a half inches of musculospiral were removed. The traumatized portion had the shape of an S, and there were numerous nodules along it.

The nicest apposition was secured with musculospiral, as suture fell in position without traction. The ends of the line of approximation showed healthy cable bundles, indicating that type of approximation was anatomically ideal. The point of line of union was imbedded in muscle. The deltoid muscle was stitched to the biceps muscle, so as to imbed the line of anastomosis. Pectoral muscles, both major and minor, were divided in the first step of the operation, and were reunited by catgut suture No. 3.

In this operation the greatest care was exercised to use perineural transfixion with the sutures, and to protect points of union with soft tissues, so as to prevent subsequent invasion of connective tissue. The time is too short to report anything about the subsequent result of this operation.

TECHNIQUE OF KENNEDY'S OPERATION

A long incision at the outer border of the sternomastoid was made, exposing the roots of the brachial plexus, followed by a division of the fifth and sixth nerves proximally; division of the suprascapular nerve, the branch to the outer cord and the branch to the posterior cord distally; then suture of the three peripheral ends to the two central ends, with chromicized catgut passed through the nerve-trunks. After the operation, the arm was raised and the dressing left on for twelve days. Kennedy had four similar cases. The time for recovery was sufficient in only one case before the publication of the article, and in it the results were quite satisfactory. Birth paralysis was also considered by Tubby.[1] He discusses the paralysis of the upper root of the brachial plexus (Erb-Duchenne) treated by operation. He considers the palsy due to different causes; among others, fracture of the clavicle, difficult and prolonged labor, infantile paralysis, injury, etc. While Tubby thinks *that nerve-grafting in these cases would be of some value*, yet he prefers muscle-grafting, which he used with success in two cases. Tubby has evidently confounded anterior poliomyelitis with its nerve degenerations with an Erb's birth paralysis; the latter is a traumatism of a nerve-trunk, resulting in its division and separation of the ends, as clearly shown in our preceding cases. In other cases the nerve is not divided, but contused to such a degree that the nerve-fibers are destroyed and the trunk changed to a dense mass of connective tissue, which does not permit the penetration of axis-cylinders, or the transmission of impulses, as mentioned and illustrated by cases in section on "Regeneration." The proper treatment for this condition is exposure of the injured nerve zone with re-establishment of the continuity of the nerve with implantation into a healthy trunk.

That the treatment of the various palsies by nerve anastomosis is destined to a broader application, we firmly believe, as the principle underlying this treatment is physiologically and histologically established. In Spiller's and Frazier's recent article we were impressed by the application of the method of nerve anastomosis to palsies of cerebral origin and to athetosis. This operation for cerebral palsies is original with them. From clinical observations we know that in cases of hemiplegia or paraplegia, after a certain length of time, which varies from two to ten years, there may be some return of motion in a limited group of muscles. Spiller and Frazier noted that, generally speaking, the return of power was in the flexors of the upper limb and the extensors of the lower. The nerves supplying the extensor muscles were anastomosed with those supplying the flexor muscles in which power was wholly or partially restored. The procedure has been applied clinically in but one case. They anastomosed half of the median with the musculospiral in the axillary space in a case of hemiplegia of five years' standing. The time is not sufficient to enable the surgeon to speak definitely of results. In this case the paralysis was in the extensors absolutely, and only partially so in the flexors.

Rawa states: "The direction of the voluntary impulses may be altered as one pleases, and the impulses will always accommodate themselves to the peripheral nerve-endings"; "that the central nerve mechanisms can enervate organs that formerly did not connect with them, as soon as those organs become connected with them by nervous conductors." Cunningham does not agree with Rawa. He did not find, in his experiments, that the cortical centers adjust themselves after crossing, "to suit the altered peripheral innervation, and, by practice, supply exactly what is required of them by the peripheral organs with which they become connected." The recent experiments of Robert Kennedy, though, favor the views of Rawa. Spiller and Frazier seem also to believe in the possibility of re-education of centers.

Experimental work has been carried out on dogs by Frazier and Spiller. They anastomosed the nerves of one group of muscles with that of another; for instance, the sciatic with the crural. Animals have a transitory paralysis. In the course of time they gain motion,

[1] British Med. Journ., Oct. 17, 1903.

but in some instances they are somewhat confused in their movements. Later, by auto-education of the cortical centers, this was overcome. It is now a well-established principle that a proximal nerve-trunk that formerly supplied flexor muscles will with equal efficiency, if changed in its distal attachment, carry impulses to extensors; i. e., the impulse is the same, but, applied to a different set of muscles, produces an opposite action.

There will be many technical difficulties to overcome in the surgical treatment of anterior poliomyelitis, particularly where large sets of nerves are permanently paralyzed; for example, all of the branches of the brachial plexus on one side, or those of the lumbar or sacral. Nerve anastomosis is neither impossible nor impracticable in any of these regions. In the brachial region, one nerve-root is usually not diseased, and the other roots can all be grafted to that one, as was done in my case, with four roots of the brachial plexus divided traumatically. In paralysis of the lumbar roots we can anastomose to the sacral roots, or to the external cutaneous. While the latter is classed as a sensory nerve, it has motor fibers, and can be attached to the cords of the anterior crural.

The anterior crural and sciatic cannot be readily anastomosed. There is no nerve except the obturator which can be transferred to the crural with ease; the obturator originating from the second, third, and fourth lumbar is frequently paralyzed with the crural, but not often with the sciatic. Therefore its anastomosis with the crural would have no advantage.

There is a zone, however, in which the lower lumbar and sacral nerves are in close proximity, and that is in the area known as the cauda. Here the lumbar and sacral fibers parallel each other and are in close proximity. They are, however, within the dura, and in order to effect the suture it would be necessary to open the latter. This is no more difficult or hazardous in infantile paralysis, than it is with syringomyelic spina bifida, or the removal of tumors from the cord. When the dura is open, the fibers leading to the paralyzed muscles can readily be picked out with the electric excitor, as was done in my case of excision of the spinal cord, with end-to-end suture. When the fibers are located, they can then be readily approximated to neighboring fibers, or even transferred to roots at the opposite side of the cord, so that a left-sided paralysis may be treated by implantation into the right motor roots. This was easily accomplished experimentally on the dog. (The results of these experiments will be given later.) It was also technically demonstrable in my case of resection of the cord. (See article on spina-bifida.) The spinal canal can be opened and intradural transplantation made in the sacral region. The distal fibers of the cauda can be implanted into the sacral peripheral roots. Technically, these operations seem almost as uninviting as the Kraske operation, but practically they are not difficult or distant.

EXCISION OF THE SPINAL CORD WITH SUTURE

Experimentally, in the higher vertebrates there never has been a successful division and suture of the cord with restoration of function. Clinically, there is one reported successful suture of the cord after resection; that of Dr. R. H. Harte for a bullet wound completely dividing the cord (detailed in full under "Surgery of the Spinal Cord"). The contused ends were excised, the surfaces freshened, an end-to-end suture accomplished, and motion was restored to a considerable degree. It is not definitely known how much of the cord was divided in this case. The improvement was controverted by the universal failures of experimental spinal cord suture.

The restoration of functions of the cord after complete paralysis from pressure from neoplasms is supported by numerous clinical results. While we know that a cord can be restored after a pressure sufficient to produce complete paraplegia or paralysis, the restoration of the cord never has been observed after division. The restoration of function in division of a cord in the *cauda zone* has not been experimentally demonstrated. It is fair to assume that as union of the sciatic after division gives functional perfection, there is no anatomic reason that we now recognize to prevent the re-establishment of functionating neurones after division of the same nerve-fibers within the spinal canal in the cauda zone, as the caudal fascicular axones have a neurilemma similar to the extraspinal axones. It was on this basis that the author made the resection of the segment of cord involved in a case of syringomyelic spina bifida. The electric excitor applied to the terminal segments in the cord produced vigorous response in the muscles of both lower extremities, notwithstanding they had not moved voluntarily from birth; this we interpreted as indicating regeneration of the peripheral ends, regardless of the prenatal separation of the distal from the proximal end by the pathologic conditions. There are, already, three months after the operation, pronounced favorable trophic changes in both lower extremities. It is too soon for the restoration of motion. Should motion finally be restored in this case, it will establish the principle that end-to-end union of the spinal cord in the cauda zone is technically feasible and histologically and physiologically correct. Eight months after, there is slight faradic response in the muscles of the legs, although there is no voluntary motion. This is not in consonance with the usual order of restoration, as voluntary motion precedes faradic reaction. In this case, however, we have the fact that the muscles preceding the operation never voluntarily responded, so they must require a special training.

The nerve-supply and overlapping of segmented spinal zones, in their relation to the individual muscles, may readily be estimated from the charts of Purves Stewart, and the best point of anastomosis can be elected by comparing these charts with the anatomic charts of the cervicobrachial plexus and the lumbosacral plexus from Kocher.

NERVE-STRETCHING — A SURGICAL PROCEDURE FOR THE TREATMENT OF VARICOSE ULCERS AND NEUROTROPHIC CONDITIONS OF THE EXTREMITIES

Historical Data concerning Nerve-stretching

In 1869, the method was applied to man by Billroth. The patient was a man of 25, who fell from a ladder; the thigh struck the sharp edge of a table; generalized spasms and tremors followed the injury. Billroth made an incision and explored the injured region, with the hope of finding some anatomic reason for the spasms. To his surprise and disappointment, he found that there was no damage to the nerves, muscle, or bone. Empirically, or perhaps by intuition, he decided to stretch the sciatic nerve. This relieved the patient of pain and spasms.

After the example of Billroth, Nussbaum[1] stretched the branches of the brachial plexus, as well as those of the cervical plexus, in a case of contractures of the arm following a chronic abscess. The result was disappearance of contractures and return of sensation. These gratifying results were followed by many similar surgical attempts. In 1879 there was a spontaneous enthusiasm concerning stretching of nerves. Langenbuch[2] applied the methods to tabetic patients, and claimed good results. The enthusiasm proved to be entirely sporadic and fallacious. The method received a final blow at a meeting of the Verein für innere Medizin in 1881, and at one of the medizinische Gesellschaft. (Records of the meeting in *Verhandlungen der medizinischen Gesellschaft*, bd. xiii, I, pp. 45–64. Vienna). A quiet period followed this, and the method was revived by Möbius, Benedict, Stintzing, and others, who advocated the "unblutige Dehnung" (bloodless stretching).

Similar conclusions were reached by Alfred Lewandowski,[3] who states that after stretching a nerve there is immediate sensory, motor, trophic, vasomotor paralysis, the degree of which is proportionate with the force applied to the nerve. There is, however, perfect regeneration in time. Speaking of "unblutige Nervendehnung," he says that regeneration is possible if the force applied for stretching is less than half the weight of the body.

It seems to us that the revival of trophic energy produced by nerve-stretching is due to the vasomotor paralysis, which causes a local hyperæmia acting, in principle, as Bier's treatment, but of a much more prolonged duration.

CLINICAL AND EXPERIMENTAL

Preliminary Report

For a long time, elongation or stretching of nerves was resorted to for the relief of intractable neuralgias. Some of these clinicians have observed that neuralgias were occasionally accompanied by trophic disturbances, the most

1 Deutsche Zeitschr. f. Chir., 1872 vol. i, p. 450.
2 Berliner klin. Wochenschr., 1879, Nr. 49.
3 Therapie die Gegenwart, 1904, p. 209.

common being the ulcer. These ulcers healed very slowly, or not at all, even after the severe neuralgia had subsided. Experience has shown that where stretching of the nerve was performed for these neuralgias, both of the conditions were frequently benefited. While the observation was made, it did not attract sufficient attention until Chipault of France emphasized the relationship between the stretching of nerves and that of the spontaneous healing of these trophic ulcers. He concluded that the ulcers accompanying neuralgias or neuritis are trophic, and that the stretching of the nerve or nerves has for result a stimulation of the respective nerve-supply. This observation was purely empirical. Nevertheless, it led to this treatment of many trophic conditions of the extremities. The observation of Chipault was first applied clinically by Duplay in 1894, where a malperforant was treated successfully by nerve-stretching. The treatment of neurotrophic conditions of the extremities by nerve-stretching has since been practised by Delbet, Marchand, Réclus, Tuffier, in France; by Quervain in Switzerland; Halley in England; Sick in Germany; Rose, Roncali, Marini, in Italy; Bardesco in Roumania; and by the author. It is still applied empirically.

Conditions for which stretching of nerves has been resorted to:

Varicose ulcers (with or without pain).

Gangrenous ulcer of the foot and leg.

Gangrene of the foot, with osseous necrosis and pain.

Senile gangrene.

Dry and painful gangrene of the foot.

Symmetric gangrene of the hands (R. de Bovis).[1]

Malperforant of the extremities.

Malperforant of the lower gum (Chipault).

Scleroderma along the external saphenous nerve.

Angioneurotic changes of the lower extremity (pain and cyanosis).

Severe neuritis (Billroth and Nussbaum).

Traumatic infectious neuritis (Roncali).

Trophic œdema (Navarro).

Raynaud's disease.

Hysteric œdema.

Progressive hemiatrophy of the face.

[1] Sémaine médicale, 1904, vol. xxiv, No. 6.

Ununited fractures of the clavicle.

Arthropathy of the big toe (tabetic).

Chronic tumors below the hip (Bardescu).[1]

Ligation of saphenous vein and stretching of nerves.

Tuberculosis of bones and joints in the extremities (Murphy).

Trophic ulcers of dorsum of hand (Codman).

Experimental Observations

The following experiments have been performed by Chipault:[2]

1. Several abrasions and ulcerations were made on the plantar surface of both hind feet of a guinea-pig; the sciatic nerve was stretched on one side only. The result was rapid and complete healing of the ulcerations and abrasions on the side where the nerve was stretched, and incomplete or absent effort at healing on the other side.

2. Guinea-pig presenting callosities on the plantar surface of both feet. The sciatic nerve was stretched on one side only, while the other side was left undisturbed. Fifteen days later, the callosities had disappeared on the side where the sciatic nerve was stretched, while the condition of the other side remained unchanged.

3. Sciatic nerve was stretched in guinea-pig, presenting some ulcerations on the lower extremities. No improvement, however, after stretching.

4. Applications of ice and salt on feet of a guinea-pig produced an ulcer on one and a falling of the hair on the other. Bilateral stretching of the sciatic was followed by healing of the ulcer on one side and rapid growth of hair on the other.

5. Aqueous solution of cocain injected into the trunk of the sciatic nerve produced callous ulcers of the plantar surface of the feet in the guinea-pig. Stretching of the sciatic resulted in disappearance of the callous ulcers in three weeks.

Personal Experiments

It was very difficult for us to produce a permanent ulcer in a healthy animal, even in the most unsatisfactory surroundings, as far as sepsis was concerned. We were unable to find a guinea-pig presenting callouses or callous ulcers, as mentioned by Chipault. The experiments undertaken consisted of shaving the lower extremities and then stretching the sciatic nerve on one side only. The hair on the side where the sciatic was stretched grew much more rapidly than on the other. Unilateral stretching of the sciatic was followed by a

[1] Spitalul, 1902, Nrs. 18, 19. Bucharest, Roumania.

[2] Monographies cliniques, sur les questions nouvelles en méd. et chir en biologie, Nr. 33, March 23, 1903.

similar result in the growth of the nails. This is only a preliminary report. (Additional experiments which I have outlined will be published in a future paper.)

Varicose Ulcers — Treatment — Technique of Nerve-stretching

Text-books on surgery teach us that varicose veins of the lower extremities are always of mechanical origin, and pressure, either in the pelvis or in the thigh, is responsible for the gradual dilatation of the veins of the lower extremities. If the intravenous pressure is beyond the resistance of the wall, trophic changes occur, producing ulcers which are frequently very painful, and are surrounded by œdema and infiltration. Caloric changes, algias, and hyperalgias are frequently noticed in the teguments surrounding the ulcer, so we must admit that mechanical obstruction is only one of the ætiologic elements of the varicose ulcer, and that there must be some other factors responsible for its production. We do not know what disturbs the trophic equilibrium of the tissues, but we do know from clinical observations by Chipault and others, that a varicose ulcer is purely dystrophic, due to a lack of nerve stimulus of the respective area.

Chipault, considering a varicose ulcer as a pure dystrophy, conceived the idea of treating the ulcer in the same manner as he treated another and more pronounced dystrophy, malperforant. The method of nerve-stretching for varicose ulcers was performed by him in thirteen cases. The ulcers healed either primarily or secondarily. In Chipault's monograph above mentioned, he collected more than fifty cases of varicose ulcers, treated by this method.

The technique consists in stretching the external or internal popliteal nerve, the saphenous major, peroneus communis, the cutaneus dorsalis or pedis lateralis. Some surgeons curette and clean the ulcer before stretching the nerve, others make no local treatment of the ulcer. The essential point in nerve-stretching for varicose ulcer, according to Chipault, is to traumatize it, not too far from and not too near the seat of the ulcer. The experience of Chipault and others in using this method has been very gratifying in most instances.

The following is my procedure:

1. Disinfection of the leg from the knee-joint up. No curetting or disturbance of the ulcer at surface.

2. A longitudinal incision is made over the popliteal space, thus exposing the lower part of the great sciatic and its two divisions, the external and internal popliteal; this requires very little dissection. In some cases I stretched the trunk of the sciatic only; in others, both the sciatic and its branches, and in the majority I stretched only the external and internal popliteal. The reason for stretching the sciatic at the beginning was, that I thought the trauma would be less likely to affect injuriously the stronger trunk. I found later that the external and internal popliteal were sufficiently strong to resist great traction without danger of rupture.

The stretching of the nerve was done either on Kocher's blunt dissectors or on the fingers. I prefer the latter; they stretch the nerve uniformly and do not damage the trunk. How many pounds of pressure a nerve will withstand

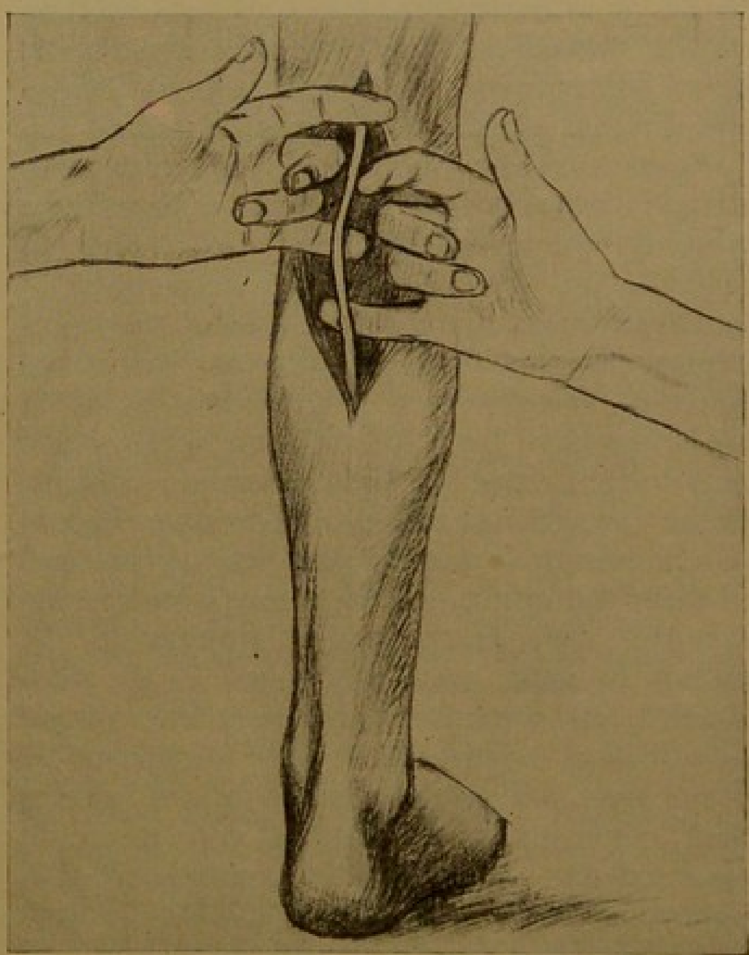


Fig. 48. Showing technique of nerve-stretching by digital traction.

before rupture I have not experimentally studied. The traction is kept up for from one to two minutes, and is intermittently applied. Striking changes were observed in the nerve-trunks. In a case of malperforant, the external and internal popliteal were œdematous, resembling myxoma, or the funis at birth. The nerve-fibers could not be discerned in the œdematous nerves showing a gross change in the histology of the trunk, as will be noted in report of case.

Malperforans Technique

Chipault divides the operation into two steps:

(*a*) The curettement and carbolic cauterization of the ulcer.

(*b*) The stretching of the nerve. This is best effected in the lower portions of the popliteal space. It should be very forcibly stretched, so as to rupture the myelin sheath. Some surgeons are satisfied with stretching the posterior tibial. The leg is dressed in an elevated position; local treatent of the ulcer is not necessary. Réclus of France resorted to neurotripsis or to harrowing; that is, the dissociation of the nerve-fibers by a metallic instrument. From Schlessinger[1] we learn that in some cases the internal plantar nerve was stretched.

Results of Nerve-stretching. Chipault's monograph includes 79 cases of malperforant treated by nerve-stretching. Of these, 69 healed — 14 primarily and 55 secondarily — while 10 failed to repair. Some of these improved temporarily. Of the 69 immediate healings, there were 7 recurrences. Some of the cases have been followed for seven years, and no recurrence has been observed. In most of the cases of malperforant, there was a history of alcoholism. In some, diabetes was present; in these the results, on the whole, were unsatisfactory. An element which appears to the author to have been overlooked is the endarteritis, as announced by the intermittent claudication. This was readily demonstrable in our case, and was a marked feature in cases that came under observation and were not operated on. These arterial changes are probably the result of alcoholism. On account of the extent of the circulatory disturbance, we believe that the nerve-stretching in these cases would be effective if made in the popliteal space. In our case, in six weeks the entire appearance of the foot and leg had altered. On this basis it appears to us that the stretching of the sciatic and crural nerve might benefit the cases of intermittent claudication where no ulcer exists.

[1] Indikationen zur chir. Eingriffen, teil i, p. 110.

Therapeutic Effect of Stretching the Nerves. The principle of the beneficial effect of nerve-stretching is not definitely known; as stated, the method has been employed after empirical observations. According to Chipault, the stretching of a nerve has an indirect influence upon the sympathetic system, thus increasing the trophic energy of the tissues.[1] D. de Buck-Gent considers the trophic changes of the extremities to be due to a lack of nerve stimulus, and that the artificial irritation of the nerve during the stretching supplies this element.

Pathologic Changes in Stretched Nerves. The fear of untoward results from stretching the nerve has deterred surgeons from resorting to it as a remedial agent. Clinical observation proves that their apprehension is ungrounded. While in some cases there is a temporary paresis of sensation and motion, yet both are transitory. The changes following a stretched nerve are both peripheral and central.

Peripheral Changes. Histologic examinations of stretched nerves have been made in Italy by Consentino and Nirnichi, and in this country by Dr. Victor L. Schrager, who has carried on these experiments for the author in the laboratory of the Chicago University. According to Consentino, there were marked changes at the point of stretching for a short distance above and for a considerable distance below. The observations of Consentino, Nirnichi, and Schrager harmonize in this respec.t The histologic findings of Consentino were the disappearance of some axis-cylinders for a considerable distance below the point of stretching; high swelling of some, and very faint reaction to stain. The myelin was fragmented, and the nuclei of the sheath of Schwann were in a state of multiplication. At the point of stretching he found interstitial hemorrhages, hyperæmias of the capillaries, and a thickening of the intima

[1] Medisch Weekblad. Voor-en Zuiderland, Oct. 27, 1901.

and media; also, a preponderance of intrafascicular connective tissue. Nirnichi found within the sheath of Schwann myelin, granular in aspect, and the axis-cylinder interrupted. Schrager used dogs for most of his experiments, as the gait can be better observed after the operation.

How much traction a nerve can stand has not been ascertained experimentally, but it can be safely estimated that it will stand a traction of a weight equal to that of its body. The sciatic of man resists a traction of from 100 to 120 pounds. Angular instruments should never be used for nerve-stretching. The first few hours after the operation the gait of the animal was impaired. In some animals the impairment was very slight — almost imperceptible — and very marked in others. Schrager never observed complete paralysis of motion. Sensation cannot be easily ascertained in animals. It can be stated, however, that the animals responded to the touch of a needle. In rabbits, Schrager always obtained a primary union. He was not so successful with some of the dogs. The animals were rejected in every case where aseptic wound-healing failed. Histologic examinations were made from 60 to 72 hours after the stretching; in one case, on the fifth day. Sections were made from the portion corresponding to the point of stretching. In all cases Schrager found degenerative changes, which, generally speaking, are always proportionate to the amount of injury that has been done to the nerve.

From an examination of several specimens, Schrager's conclusions are, that myelin fragmentation after nerve-stretching is common. The histologic appearance of a stretched nerve is similar to one on the third day after division.

(*a*) The myelin appears in small cubical or prismatic fragments.

(*b*) Interstitial hemorrhage, especially where trauma was directly applied to the nerve.

(*c*) The axis-cylinder, as a rule, did not disappear, as was found by Consentino. It is true that some axis-cylinders disappear; most of them, however, are only interrupted in their course. Below the point of stretching, the degenerative changes are present for a considerable distance, and are similar to those above mentioned. The histopathologic changes, however, are of a lesser degree above the point of stretching. Schrager never saw disappearance of axis-cylinders above the point of stretching; the myelin was granular in aspect, but seldom fragmented. In one case the sciatic of a puppy was stretched, and not examined until six weeks later. Schrager was then unable to find any pathologic changes in the nerve.

Mr. H. Z., aged 26, tailor. Admitted to Mercy Hospital, June 9, 1906.

Family History. Mother has articular rheumatism; otherwise negative.

Personal History. Born in Russia. Resided in Chicago last six years. Habits regular.

Previous Illnesses. Diseases of childhood. Does not remember of ever having had typhoid. Five years ago, while bathing after 9 P. M. he got a chill, and about 1 A. M. felt pain in both feet. Fell asleep again, and awakened in the morning feeling all right and went to work. Two months later, unable to move legs. This disappeared as suddenly as it came on, and two days later patient was able to get out of bed and walk to work. About four months later, had a similar attack. Made a good recovery, and was well for a year. At the end of that time he noticed his legs growing weak, and when walking, tired easily. He gradually became worse for four months, when he was compelled to remain in bed for five weeks because his feet were swollen and painful. Does not remember whether or not he had a fever. At the end of this time he felt much better, went to Mt. Clemens, Michigan, and took the baths for a month. These seemed to do him good. Six months after having been at Mt. Clemens, middle finger of left hand became cold and painful, and about the same time he noticed that his finger was blue from tip to last phalanx. While treating this, the index finger of same hand became involved. Last winter, third finger of right hand and ring-finger of left hand became involved and sloughed off. A doctor cut off third finger of right hand. Patient's appetite fair. No gastric distress, no incontinence of fæces or of urine.

Present Trouble. Sixteen weeks ago, patient had an ulcer form on fourth toe of left foot; very painful. This gradually spread until it covered the whole toe. It was very sensitive to pressure for two or three months prior to the time he first noticed the ulcer, and nine weeks ago a surgeon amputated it. After leaving hospital, patient says second toe became ulcerated on side proximal to big toe. The ulcer has increased to size of dime in a week. This toe was painful and sensitive to pressure two months before he noticed ulcer forming. He comes to the hospital for this trouble. Patient says that about Christmas, 1905, his face was swollen; no pain, redness, or glossiness of skin, but his mouth and tongue were so swollen that he could not eat. About a week after, patient had a dull, aching pain in left calf of leg, and in three or four days felt pain in foot. The day patient says that his face was swollen he had a slight chill and some (he does not know how much) fever. Cannot state whether he lost weight in the last six months.

Diagnosis. Obliterating endarteiitis with Raynaud's disease.

Operation. June 12, 1906. Both popliteal nerves were stretched. There was primary wound-healing. The pain was very much relieved at once, with distinct trophic improvement in four weeks; in five weeks the ulcer healed, but the claudication was yet quite pronounced.

Mrs. Anna D., aged 53, housewife. Admitted to Mercy Hospital, June 27, 1906; discharged July 22, 1906.

Family History. Negative.

Personal History. Born in Russia; came to this country 18 years ago; married at 23; habits regular.

Previous Illnesses. Has always enjoyed good health.

Menstrual History. Menopause five years ago. No discharge since.

Obstetrical History. Thirteen children; seven living and well. All normal labors; no miscarriages.

Present Trouble. Two years ago, had two small ulcers on inner and lower third of right leg, since which time veins of legs have been large. They were smaller than a dime, and disappeared after two or three months' treatment with salves. A year ago, patient was thrown from a wagon, and wheel passed over lower third of leg, denuding skin off area that is now ulcerated. This has not healed, and patient has doctored since without getting any relief; ulcer has gradually increased in size. Patient suffers a great deal of pain, and ulcer smarts and burns. Appetite good; no distress after eating. Has lost fifty pounds in last year. Says she has not had typhoid.

Diagnosis. Indolent varicose ulcer with great œdema.

Pathology. No œdema or swelling of nerve.

Operation. Excision of short saphenous vein. External and internal popliteal nerve stretched. Varicose ulcer curetted. Wet dressings continued, 1-3000 formalin.

Ulcer healed rapidly and completely before patient left the hospital, less than four weeks. Keloid developed in popliteal scar; removed ulcer remains completely healed 4½ months after.

Mr. B., aged 41, day-laborer. Admitted to Cook County Hospital, July 9, 1906.

Family History. Negative.

Personal History. Born in Ireland; married at 33; wife living and well; no children; habits regular.

Previous Illnesses. Diseases of childhood. Never had typhoid. Always enjoyed fairly good health. Denies venereal disease of any kind.

Present Trouble. Says that veins of both legs have been large as long as he can remember, but have increased gradually in size for last twenty years. Swell considerably when patient is on feet, but toward morning (after patient has been lying down) veins decrease in size. On lower third and anterior part of right leg, patient has a discoloration, and says he was injured 18 years ago by striking against a box. Says this area becomes abraded with the least bit of trauma, but has always healed, as seen at present. Never given any pain until about five or six weeks ago. (Patient says his legs feel tired.) Appetite good; sleeps well; no distress after eating; very constipated; says he never has a bowel movement without taking a cathartic; has lost ten pounds in last two years.

Pathology. Extensive crust formation and dermal atrophy over anterior surface of right leg. An angle-worm formation of veins just beneath skin.

Operation. Right leg: External and internal popliteal nerves stretched. Long and short saphenous veins excised. Left leg: External and internal popliteal nerves stretched; short saphenous vein excised. Rapid improvement in trophic condition of skin.

Mr. W. G., aged 45, barber. Admitted to hospital, June 25, 1906.

Family History. Negative.

Personal History. Born and always lived in Illinois. Married at 22; habits regular. No venereal disease.

Previous Illnesses. Diseases of childhood. Attack of appendicitis twelve years ago. In bed over two months. Was not operated on.

Present Trouble. Shortly after getting up after the attack of appendicitis, patient says he noticed an ulcer about the size of a dime on the lower anterior part of right leg. This gradually grew to size of silver dollar. (As soon as it grew to that size, patient put on an elastic bandage, which he has worn continuously for about eleven years.) First ulcer did not disappear for about four years. Then a similar ulcer appeared on inner ankle. He was operated on in New York a year ago last May; some of the veins of leg were ligated and part of long saphenous in thigh exsected. The ulcer healed in two weeks, but recurred in same place two months later. Has since grown to its present size. Ulcer is painful, sharp, and cutting in character; burns and smarts. Appetite good; no distress after eating; some loss of weight.

Pathology. External saphenous was much larger than normal.

Operation. External popliteal was smaller than normal; the internal popliteal larger and œdematous. Both were stretched. Rapid repair of ulcer.

Mr. John B., aged 44, American; married 22 years. Admitted to Mercy Hospital, November 27, 1905.

Present Illness. Five years ago, patient had a callus on inner border of right foot. He made an attempt to extirpate it and cut too deep. The exposed area became painful, and suppurated to the bone. A surgeon operated on the patient, the wound closed and was covered with normal skin. Patient was in good condition, able to walk, and had no pain. In the summer of 1904, patient noticed that the callus recurred. The entire indurated area came off and left an ulcer, which was painful, deep, and discharged pus. September, 1904, patient was again operated on. The surgeon "curetted the bone and closed the wound." It healed nicely, and patient was again able to walk. In the spring of 1905, callosity recurred. Patient cut it, and ulceration followed. After the first operation, however, the foot and the leg between the ankle and knee commenced to increase in size; the skin became violaceous, infiltrated, and pachydermal. Since the spring of 1905 the foot and leg have considerably increased in size. Lost fifteen pounds in last fifteen months. General health, however, is good.

Previous Illnesses. Denies syphilis. Had no typhoid fever. Abuses alcohol. Moderate smoker. No Raynaud's disease in the family. No malperforant. No dystrophies. Enormous foot and leg, reminding one of elephantiasis. Right foot twice as big as the left one. The sole of the foot has three holes, two of which extend down to the bone. The plantar teguments are very thick, enormously infiltrated. The openings are surrounded by callous margins. Pressure upon the foot causes some pain; also gives impression of emphysema in the tissues.

The main clinical points are:

(*a*) Elephantiasis-like appearance (right foot and leg up to the knee).

(*b*) Three ulcers on the sole of the foot.

(*c*) Gaseous infiltration of tissues of foot.

(*d*) Slight circulatory disturbance (diminution of heat). Arteries are not easily compressed. The tension is great. No evidence of periodic claudication in the other leg or foot, but he cannot tell when the foot is cold. Advised operation of nerve-stretching and removal of necrotic bone.

PERSONAL CASES

CONDITIONS FOR WHICH STRETCHING OF NERVES HAS BEEN PERFORMED

Where Operated	Condition	Clinical Features	Operation	Immediate Result	Remote Result
Case 1. Mercy Hosp. Mrs. S. April 4, 1905.	Varicose veins. Two varicose ulcers.	Pain. Brown pigmentation of skin around ulcer. Infiltration.	Stretching of external and internal popliteals. Ligation and exsection of short and long saphenous veins.	No sensory paralysis. No motor paralysis.	Complete healing of ulcer. The tegument covering the ulcer is on a level with normal skin. Normal resistance of tegument. Very rapid healing.
Case 2. Mercy Hosp. Mr. F.	Trophic ulcers of the leg.		Stretching of sciatic proper.	No sensory or motor paralysis.	Marked improvement in trophic energy of tissues. Ulcers greatly diminished.
Case 3. Mercy Hosp. Mr. J. B.	Malperforant of left sole. Necrosis of bones associated with it.	Pain. Elephantiasis. Diminution of heat. Infiltration in derma; gaseous œdema; brownish pigmentation. Three ulcers, surrounded by callous margins.	Stretching of sciatic proper, also external and internal popliteal nerves.	Slight sensory paralysis for 4–5 days.	Foot considerably diminished in size. Pain disappeared. Equal local temperature in both feet. Openings closed. Skin not infiltrated.
Case 4. Mrs. Anna D. Mercy Hosp. June 27, 1906.	Large indolent ulcer with much œdema.	No evidence of repair in margin of ulcer.	Varicose ulcer curetted. Short saphenous vein excised. External and internal popliteal nerves stretched.	Ulcer rapidly and completely healed before patient left the hospital; less than four weeks.	Keloid developed in popliteal scar, which was removed. Ulcer remains completely healed. Trophic condition of skin excellent.
Case 5. Joe T. Cook Co. Hosp.	Varicose ulcers. Moderate degree arthritis deformans.	Arteriosclerosis. Dark skin of lower extremities. Contraction of toes. Feet cold.	Stretching of external and internal popliteal.	Transitory sensory paralysis for a week.	All ulcers healed three weeks after operation. One ulcer reopened lately, but is less extensive and not deep. Some improvement in the trophic energy of tissues.
Case 6. Henry C. Cook Co. Hosp.	Varicose ulcer of left leg; varicose veins.	Sloughing ulcer of left leg. Surrounding skin cyanotic, infiltrated.	Idem.	Idem.	Ulcer not healed.
Case 7. Mike B. Cook Co. Hosp.	Large ulcer right leg and few small ulcers (varicose).	Sloughing ulcers, surrounded by infiltrated black skin.	Stretching of external and internal popliteal.	No sensory paralysis. No motor paralysis.	Ulcer healed; skin less infiltrated.
Case 8. Mr. McD. Cook Co. Hosp.	Varicose ulcer.	Skin œdematous around the ulcer.	Idem.	Idem.	Ulcer healed.
Case 9. James D.	Varicose veins. Ulcer.	The ulcer closed and opened a great number of times. Present ulcer of two years' duration.	Idem.	Idem.	Ulcer healed.
Case 10. H. Z. Mercy Hosp. June 9, 1906.	Raynaud's disease. Trophic ulcer between the second and third toe of left foot, associated with severe pains.	The ulcer is very dirty, covered with offensive pus. Pains are terrific.	June 12, 1906. Stretching of external and internal popliteals in popliteal space.	Slight sensory paralysis for 3 days only. Slight motor paresis.	After five weeks, ulcer healed completely. No pain.
Case 11. Mr. B. Cook Co. Hosp. July 9, 1906.	Varicose veins. Atrophy of skin.	Crustation and atrophy of skin.	Right leg: External and internal popliteal nerves stretched. Long and short saphenous veins excised. Left leg: External and internal popliteal stretched. Short saphenous excised.	Rapid improvement in trophic condition of skin.	Cured.
Case 12. Mrs. W. G. June 25, 1906.	Varicose veins, with large trophic ulcer.	Edges indurated and very painful.	Excision of veins. Stretched popliteal nerves.	Rapid repair of ulcers.	Cured.

Operation. On foot: Removed carious bones and cicatricial tissue. Little hemorrhage. Leg: Stretched external and internal popliteal nerves; both were enlarged to three times the normal size; were œdematous, looked like myxomatous ropes or umbilical cord; no nerve fibers could be recognized in them. Also stretched the sciatic.

Result. Rapid healing of ulcers; great subsidence of infiltration of skin on foot and lower half of leg. All pain disappeared.

This extreme œdema of the nerves I have never observed before, and it seems to me that there must have been a relation between the trophic change in the skin and the great œdema of the neuroglia in the popliteal and sciatic nerve, and therefore the question arises in my mind, Is elephantiasis a trophic change in the skin, due to alteration in the nerve-trunks, or is the œdema and myxomatous condition similar to and due to the same causes as the pathologic changes in the skin? In our case, at least, the nerve lesion extended high above the skin changes and to about a level which would correspond to a position in the nerve-trunk, where the cutaneous branch was given off.

Mrs. S., widow, aged 45, German. Admitted to Mercy Hospital, April 4, 1905.

Present Illness. Patient noticed varicose veins of the left leg about fifteen years ago. She had pains in the calf, mainly after walking. There was swelling of the feet, especially during pregnancy. December, 1904, patient had a slight contusion of the anterior surface of the leg, which was followed by an extensive ulceration, which, in January, 1905, invaded a vein, giving rise to a profuse hemorrhage, controlled by pressure.

Previous History. Patient has given birth to seven children.

Family History. Negative.

Examination. Varicose ulcer the size of a quarter situated two fingerbreadths above the tibiotarsal joint, and occupies the outer side. Under it there is a second small ulceration.

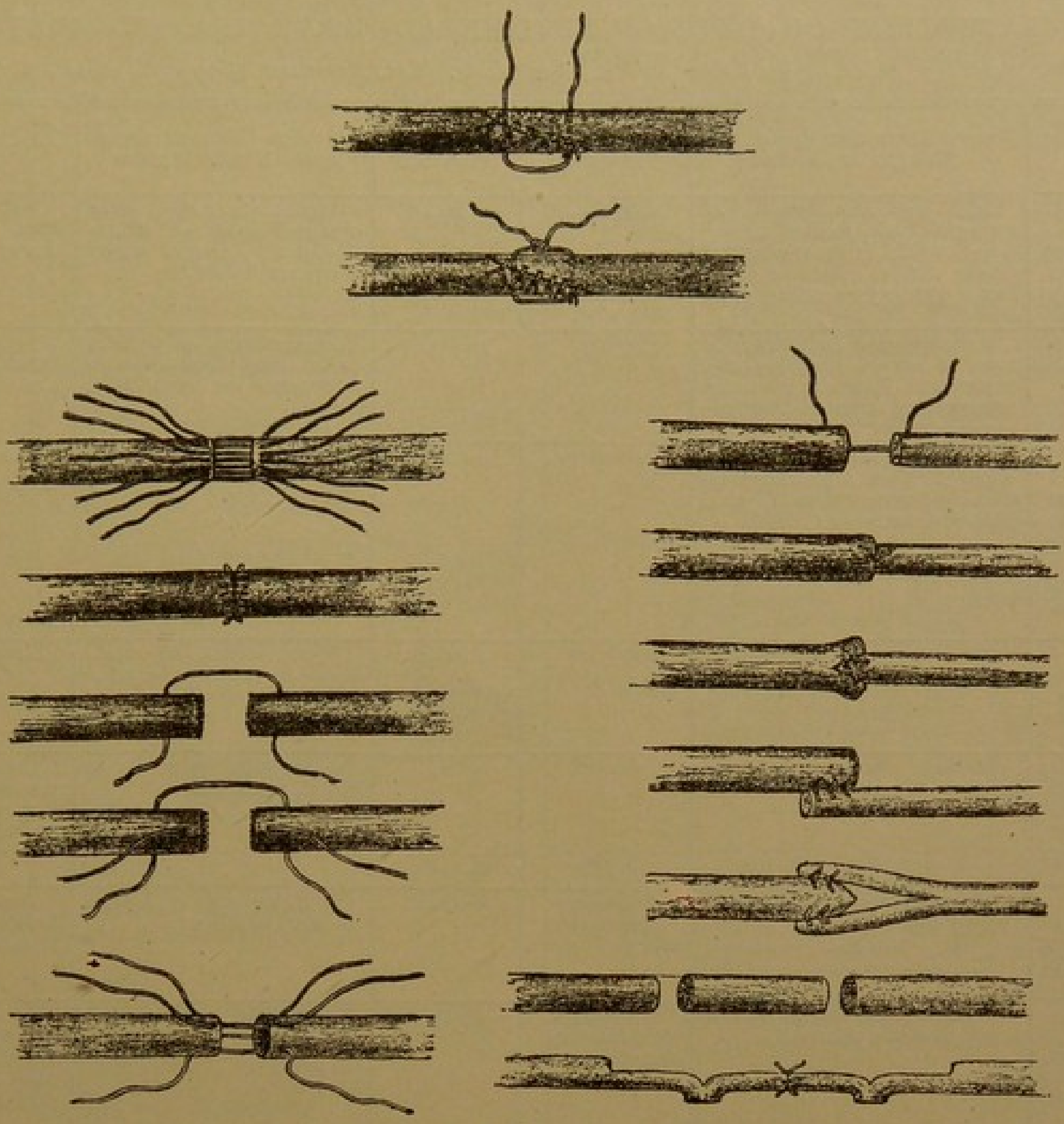
Operation. Curettement of ulcer; ligation of short and long saphenous veins; stretching of internal and external popliteal nerves. Very rapid healing.

SUTURE OF NERVES.

Either primary or secondary. By "primary suture" is understood suture of the two divided ends immediately after the accident or within the first twenty-four hours. Secondary suture is the approximation of the two divided ends within several months or years after the injury. The older surgeons base their practice on the theory that a peripheral segment which has been disconnected from the central for a long period of time is permanently degenerated, and an attempt at union would be futile. The most modern surgeons, better informed on the histology and physiology of the peripheral nerves, and especially those who believe in peripheral regeneration, are of the opinion that new nerve-fibers regenerate in the peripheral segment, even if it has remained disconnected for a considerable period of time. The fibers are imperfect, and as soon as they are united with the central end, under the influence of central impulses they develop to anatomic perfection and to normal physiologic function. The limitations of time in which this restoration may take place has not been definitely determined, but restoration of function has been secured many years after division.

In any operation upon the nerve-trunks, one must, first of all, identify the nerve; otherwise the operator is in the dark, and the final result will undoubtedly be compromised. This identification can easily be accomplished by making use of the nerve-excitor. It is composed of a metallic cylinder which can be attached at one end with the poles of a faradic battery, while the other end is provided with two platinum needles or wires, three centimeters in length, and situated a distance of half a centimeter from each other. These tips can be easily sterilized, and should always be at hand when operating upon the nerve-trunks. The importance and the value of this instrument can be appreciated when we think of the conditions of the tissues in an old and extensive injury in which they are all matted and bound together by connective tissue. The structures have lost their natural color, and the anatomic relations and identification of nerve-trunks becomes an impossibility without the use of the excitor.

Operative Procedure. The first essential is the removal of all connective tissue that caps the nerve-endings to a degree sufficient to expose clearly the fasciculi and neurones, even though this shortens the nerve beyond the possibility of direct end-to-end suture. The axis-cylinders or nerve-fibers will span a considerable distance under favorable conditions, to be mentioned later, but they will not penetrate connective-tissue masses, so that it is absolutely necessary to have all of the cicatricial or new-formed connective tissue removed from the



Types of Nerve Suture.
Fig. 49. (*International Text-book of Surgery*, vol. i, p. 855).

nerve ends. The failure of this removal has contributed more than any other element to the failure of restoration of function after nerve suture. This, of course, has reference to secondary suture. The needle used for suturing should be a round intestinal needle, without cutting edges. Suture material should be fine linen, silk, kangaroo tendon, or slowly absorbable catgut. The suture should not be passed through the substance of the nerve, but through the perineurium, and also through the surrounding connective tissue. This point is very essential for success. The trauma produced by passing the needle through the substance of the nerve, and especially if a slight infection takes place, causes a proliferation of connective tissue which compromises the restoration of function. Above we give a table showing the various methods of suture.

If the nerves face each other, and are not separated by a considerable gap, and a slight traction established when the muscles are completely relaxed, the ends can be easily approximated. A little stretching of the ends can be easily made without any prejudice to the ultimate result. If the distance is considerable between the two ends, the nerves can be united in several ways. Létievant is the originator of "suture à lambeaux," or flap-suture. Foreign bodies, nerve segments from other animals, portions or segments of spinal cord from small animals, have been interposed between distant segments. In the last ten years, in cases of considerable distance between the ends, the divided peripheral end was implanted into a neighboring healthy nerve, which was only partially divided. In several instances, especially where the trauma has compromised the nerve and bone, in order to be able to approximate the two ends a segment of bone was resected, that

TABULATED LIST OF CASES OF PERIPHERAL NERVE TRANSPLANTATION

(*Reuben Peterson, M.D., Ann Arbor, Mich., Am. Jour. Med. Sciences, 1899, vol. cxliii, p. 398*)

Operator, and Where Reported	Nerve Operated upon, and Nature of Injury	Prim. or Sec. Operation	Dist. Bet. Segments	Method Used to Overcome Nerve Defect	Results
Case 1. Albert. Einige Operationen an Nerven. Wien. med. Presse, 1885, xxxix, 41.	Man, aged 40. Removal of sarcoma from median nerve. Good results from paraneural catgut suture. Recurrence of tumor required secondary operation.	Primary.	3 cm.	Implantation of a nerve obtained from an amputated foot. Catgut suture above and below.	Patient observed only 10 days. *No* return of sensation or motion.
Case 2. Albert. Ibid.	Farmer, aged 61. Enlargement of nerves, probably sarcomatous. June 11, 1881, an enlargement removed from ulnar near elbow.	Primary.	10 cm.	Implantation of a piece 10 cm. long taken from an amputated leg (post-tibial nerve).	June 17, 1881. The segment became necrotic and was eliminated.
Case 3. Kaufmann. Eine nerven Transplantation. Corresp. Blatt. f. Schweizer Aerzte, March 15, 1882. Revue des Sciences Médicales, 1884, t. 24, p. 305.	Necrosis of humerus. Operation. Section of musculospiral during operation. Nerve exposed later, ends vivified.	Secondary.	4 cm.	Dog's sciatic implanted. Silk suture.	Observation incomplete.
Case 4. F. Lange. Verhandl. der deut. Gesellsch. f. Chir., Berlin, 1882, xi, p. 55.	Musculospiral. Neuroma.	Primary.	4 inches.	Four inches of dog's sciatic inserted between the ends.	Result negative.
Case 5. Gersuny. Rep. by Damar Harrison. Trans. Clin. Soc., London, 1892, vol. xxv.	Median. Removal of neuroma just above annular ligament.	Primary.	6 cm.	Engrafting of sciatic of rabbit, 6 cm. long.	Sensation returned in 2 months. No return of motion recorded. No further tendency to neuromatous degeneration.
Case 6. Vogt. Mittheilungenaus der Chir. klin. z. Greifswald, p. 122.	Musculospiral in upper arm. Gunshot wound.	Secondary. 1½ years after injury.	8–10 cm.	Bridging by 2 sciatics (12 cm.) of a dog.	Suppuration. No impulses conducted after 2 months.
Case 7. Mayo-Robson. Clin. Soc. of London, June 5, 1889. Brit. Med. Jour., 1889, vol. i, p. 244.	Paralysis of median nerve due to removal of a neuroma.	Secondary. 48 hours after operation.	2½ inches.	Post-tibial from an amputated leg.	Sensation returned after 36 hours. (Abductor and flexor brevis pollicis diminished in size, but not paralyzed.)
Case 8. Dr. H. Landerer Einheilung eines Kaninchen Nerven in einen Defect der nerven Radialis. Deutsche Zeitschr. f. Chir., 1888, vol. xxviii, p. 604.	Woman, aged 18. Radial injured (operations on a phlegmon, long-standing).	Secondary. ½ year.	3½ cm.	4½ inches of sciatic of rabbit implanted.	Electric stimulation, followed by contraction of muscles above and below, *after three weeks*. Patient could elevate the arm after 10 weeks.
Case 9. Mr. Ward. Remarks on Nerve-grafting. By Edward Atkinson. Brit. Med. Jour., Sept. 13, 1890, vol. ii, p. 624.	Laborer, aged 42. July, 1888, a tumor shelled out from median. Jan. 1, 1889, a second tumor and 1¾ inches of the nerve were removed.	Primary.	1¾ inches.	2½ inches of median from amputated arm.	
Case 10. Mayo-Robson. Remarks on Nerve-grafting. By Edward Atkinson. Brit. Med. Jour., Sept. 13, 1890, vol. ii, p. 624. Later report, Brit. Med. Jour., Oct. 31, 1896.	Ulnar and median. Nerves cut (patient fell on a scythe).	Secondary. 7 months after injury, Jan. 30, 1890.	2 inches. (Median.)	Ulnar nerve sutured. Internal cutaneous was also found divided. It was sutured to ulnar. Interposition of *spinal cord* of rabbit between the ends of median.	March, 1891, resumed work. Feb., 1896, motion and sensation good.
Case 11. Edward Atkinson. Ibid.	Sciatic nerve. During operation for gluteal abscess. One inch of the sciatic was removed.	Primary.	1 inch.	Same piece implanted.	Five days later, sensation returned in the toes. (Nothing said about motion.)
Case 12. Edward Atkinson. Ibid. (?)	Ulnar nerve injured (laceration wound)	Primary.	2 inches.	2½ inches taken from the 2 sciatics of a rabbit implanted and sutured with a single suture above and below to the ulnar; muscles sutured and wound closed.	Sensation almost complete and some motion after 3 months.
Case 13. Damar Harrison. A Case of Nerve-grafting. Trans. Clin. Soc. of London, 1892, vol. xxv.	Boy, aged 13. Median.	Secondary. 11 weeks.	2 inches.	Two inches of a sciatic taken from a kitten, sutured with catgut between the ends.	Sensation returned (in part). Motion returned, but not perfect (8 months).

Operator, and Where Reported	Nreve Operated upon, and Nature of Injury	Prim. or Sec. Operation	Dist. Bet. Segments	Method Used to Overcome Nerve Defect	Results
Case 14. Mitchell Banks. Brief Report Given by Damar Harrison, loc. cit.	Ulnar nerve. Excision of neuroma.	Primary.	4 inches.	Four inches from sciatic of a dog, grafted.	Thirty-six hours after operation, sensation returned.
Case 15. Heath. Lancet, 1893, vol. i, p. 1104.	Jan. 6, 1893, tumor removed from ulnar. Following operation, motor paralysis.	Secondary. 4 days after injury.	2½ inches.	Transplantation of a piece from post-tibial of amputated leg.	Imperfect and limited return of sensation. No return of motion.
Case 16. Moullin. Lancet, June 24, 1893.	Male, aged 28. Operation for ununited fracture of humerus. Motor and sensory paralysis. Section of musculospiral nerve removed.	Secondary.	2 inches.	Two inches of dog's sciatic implanted between resected ends of musculospiral.	Sensation nearly normal after 5 days. Voluntary power did not return. Wasting of muscles marked.
Case 17. Bradley. Med. News, Feb. 8, 1896. Lancet, 1896, vol. i, p. 1592.	Soldier. Musculospiral (fracture of humerus). Operation June 22, 1895. Tumor at seat of injury involved nerve. Removed (tumor). Loss of 5 cm. of nerves.	Secondary. 2 months after injury.	5 cm.	Five cm. of sciatic of dog implanted.	Unsuccessful.
Case 18. J. William White. Not published. (Notes received by Dr. Peterson from Dr. White, April 8, 1898.)	Female. Musculospiral divided during removal of tumor. First operation: Flap operation, ends united. Second operation: Excision of a piece of nerve.	Secondary. Late after injury.	?	Sciatic nerve of dog inserted between ends of musculospiral. A tube of decalcified bone of chicken was used to keep continuity.	Immediate result encouraging; no permanent improvement.
Case 19. A. H. Ferguson. Unpublished.	Female, aged 18. Neuroma removed; 1 inch of median removed above wrist; gap bridged with catgut. (Sensation imperfect, grasp of hand imperfect; 3 months later, atrophy of ball of thumb.) Operation again, later on.	Secondary. 3 months after bridging with catgut.	1½ inches	1½ inches of sciatic nerve of young dog inserted between fragments of median.	Three days after operation, some return of sensation. Two years later, hand still lame, but very useful; sensation entirely returned for objects larger than fine thread or horsehair.
Case 20. R. Peterson. Amer. Jour. of Med. Sc., 1899, cxliii, p. 378.	Male, aged 24. Circular-saw cut of right wrist. Median and ulnar severed. Operation, Oct. 24, 1896, 5 months after injury.	Secondary. 5 months.	3½ cm.	Four cm. of sciatic nerves of a dog inserted between the incised ends of both median and ulnar nerves; fragments sutured with kangaroo tendon above and below.	Sensation returned gradually within 21 days. Went to work after 2 months; "could chop wood." Functional repair probably due to tendon restoration; and not to nerve regeneration.
Case 21. Durante. R. Accad. Med. di Roma, April 24, 1892.	Removal of an osteosarcoma of the head of the fibula; compelled to resect a portion of external popliteal nerve, on account of impossibility of approximating the ends.	Primary.	5 cm.	Five cm. of dog's sciatic inserted between the ends.	Movements of toes and foot notably improved after operation. Sensation also improved. However, recurrence of malignant tumor necessitated amputation.
Case 22. Charles A. Powers. Annals of Surgery, 1904, vol. xl, p. 636.	Man, aged 18. 1896. Run over by train; crushing of the toe; lacerated wounds of gluteal region; also, extensive lacerated wound of outer aspect of right knee, with a compound comminuted fracture of the head and of the neck of right fibula. Anæsthesia and paralysis in parts supplied by external popliteal nerve. Operation, but no attempt to suture severed nerve, as ends were not located.	Secondary. 47 days later, June 27, 1896.	?	Four cm. of dog's sciatic implanted.	Aug. 18, 1896 (51 days later): All forms of sensation normal. April 13, 1897: *No* improvement in motion. Aug. 31, 1903: No improvement in motion.

the divided ends might be brought in contact (Keen, Trendelenburg). Büngner interposed between the segments of the peripheral nerve in a dog a piece of human brachial artery. After 43 days the space between the two divided ends of the sciatic was filled with new nerve-fibers. Büngner ascribed to the artery the rôle of a director and medium along which the nerve-fibers can travel in a straight direction. Nothaft interposed a segment of a rabbit's aorta between the two ends of the sciatic, from which he had excised one half centimeter. When the gap between two ends is considerable, the contact can be established by foreign material. In a case of injury to the radial, Glück, in 1888, interposed catgut bundles between the ends, which were separated by a gap of 5 cm. The gap was filled in six months, or, as he says, there was a neurotization of the catgut bundles. The result was good.

On the strength of Forsman's observation on "positive neurotropism," several surgeons and experimentors have inserted the two ends into a tube of decalcified bone, previously sterilized; this has enabled a more rapid union of nerve-fibers.

"Suture à Distance." This procedure originated with Assaky (Roumania). By this is meant the interposition of catgut fibers between the two ends of a divided nerve where a considerable gap exists. In Assaky's experiments (4 dogs, 2 rabbits), in the dogs favorable results were obtained in from 67 days to 4 months. The greatest segment successfully excised was 3 cm. Once he resected 6 cm. and interposed catgut fibers between the ends; no regeneration occurred after 39 days. If the distance is considerable between the segments, a piece of muscle may be interposed, or the two segments may be imbedded in a muscle from the vicinity in such a manner as to place the cut surface of each end face to face. This has been done by Levings of Milwaukee and several times by myself. The ideal results in nerve suture are obtained, as in the repair of all other tissues, by an exact juxtaposition of similar histologic elements, end to end. This requires the least interposition of new-formed connective tissue, and in nerve union it is of especial importance.

Protection for United Nerves. The ends of the nerves which are either in perfect apposition or face each other from a distance may be protected from the invasion of connective tissue from the side by sterilized tubes. Decalcified bones were used by Vanlair. Payr used tubes of magnesium. Lotheisen[1] prefers to protect the nerves with tubes of gelatin previously hardened in a two-per-cent solution of formalin. The gelatin-tubes, after being treated by this solution, are preserved in 96-per-cent alcohol; the lumen should be from 4 to 9 mm. Gelatin-tubes can be sterilized for two hours by dry heat of from 110° to 120°. It has been my practice to protect the nerves from invasion of connective tissue by covering the point of suture with fascia, muscle substance, or fatty tissue. In the wrist or ankle areas, where muscle and fat are not available, gelatin or decalcified bone make good materials. The blood-vessels of animals, always at hand, are good for this purpose. One of the best and always available materials is a mass made of equal parts of oil of sesame and paraffin, with a melting-point of 107°. This can be flattened out with a spatula almost as thin as paper and wrapped around the point of union, completely sealing the ends so that the neuroglia can supply the only immediate agglutinative material, which is readily penetrated by the axones.

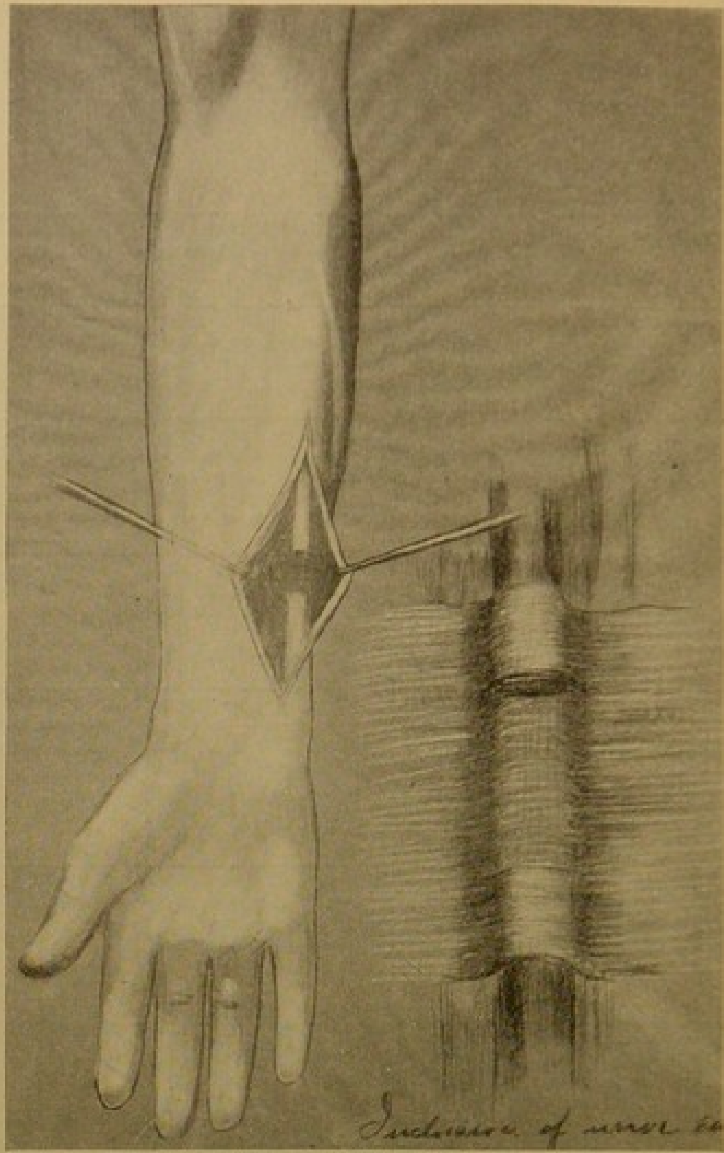


Fig. 50. Ends of ulnar nerve separated by a considerable gap. On account of impossibility of approximation the ends were fastened to fascia in such a manner as to place them in same longitudinal axis.

Foramitti protected the points of anastomosis by surrounding them with arteries of calves. The union is always perfect, and the point of anastomosis entirely free from the connective tissue in the vicinity. There is an insignificant adhesion between the intima of the artery and the sheath of the nerve. The arteries are prepared for this purpose in the following manner: A segment of artery from a calf is placed on a glass rod and hardened in 5 to 10 per cent formalin solution for 48 hours, then it is washed in water for 24 hours, afterwards boiled for 20 minutes in water, and finally preserved in 95-per-cent alcohol.

[1]Langenbeck's Arch. f. klin. Chir., bd. lxiv, h. 2.

An ever-available material for this purpose is the membrane lining the inner surface of an egg-shell. The Cargile membrane may be used for the same purpose, as may also the peritoneum of a rabbit. Recently I have conceived the idea of wrapping around the point of anastomosis a roll of paraffin tape or a mixture of paraffin and wax, or simply a wax tape. In our work, however, these foreign materials were never used when a muscle or fascia could be obtained either by flapping (as in the protection of the brachial plexus in our operation for the prevention of axillary cicatrices, following excavation of the axilla after removal of mammary carcinoma), or splitting the muscle and inserting it where a normal muscle belly is directly available. The use of heterologous organs or inorganic material is always somewhat hazardous.

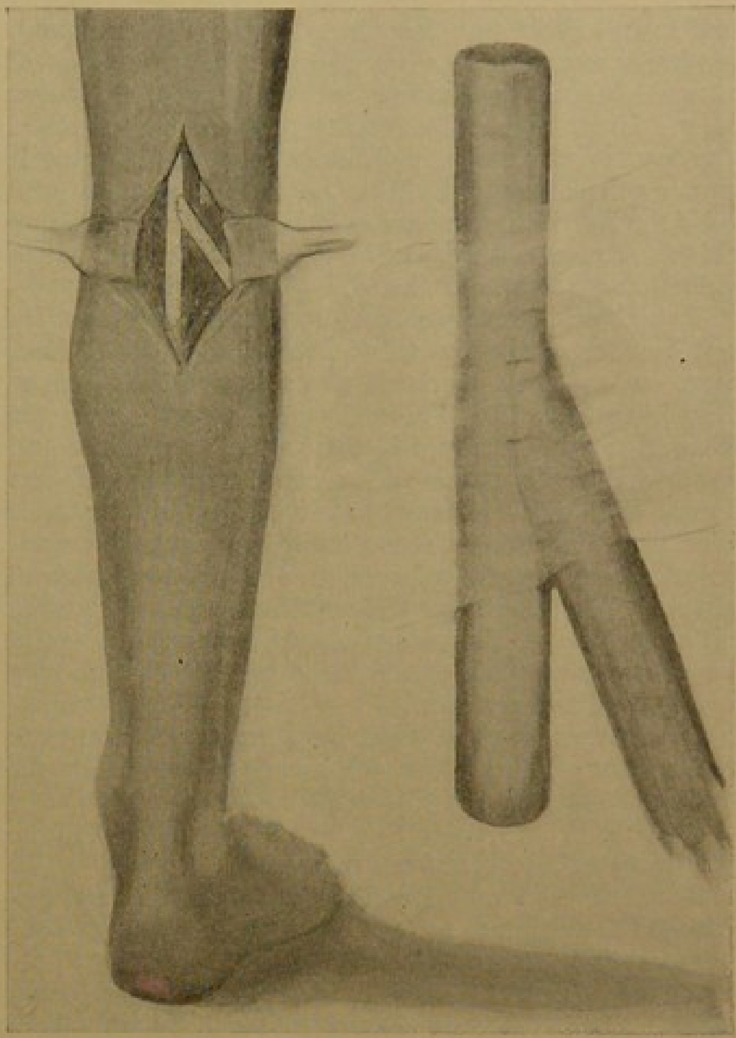


Fig. 51. End-to-side implantation and protection of point of anastomosis by fascia.

P. M., aged 43, laborer.

Diagnosis. Musculospiral paralysis from callus on left arm.

Admitted to Mercy Hospital, June 14, 1902; discharged July 6, 1902.

Operation June 24, 1902.

History of Present Illness. Ten weeks ago, while hoisting up timbers to the roof of a building, a piece fell and struck him on the left shoulder and arm, fracturing the clavicle in the middle third and the humerus in the lower third; both simple fractures. Patient was treated by a doctor, who kept the arm and the shoulder bandaged for four weeks, and when the bandages were removed, patient found that he could not use his hand, having a drop-wrist. On examination it was found that at the point of fracture there is an enormous callus, the same corresponding to the point where the musculospiral winds around the humerus.

Operation and Pathology. Constriction on arm. Incision five inches long from bend of elbow upward externally to the tendon of biceps. The muscle pulled to one side and musculospiral nerve exposed, lying on and firmly adherent to exuberant callus, which formed marked protuberance at this point. Nerve dissected free from callus, above which point it was not adherent. The nerve-trunk at site of callus was fibrous in character. The callus chiseled away (the bone was osteoporotic). Muscle pulled outward across the bone surface, and sutured to external-internal muscle septum. Nerve allowed to fall in place over muscle; constriction removed. Skin closed, dry dressing applied with arm elevated.

Mr. F. L., aged 35, engineer.

Diagnosis. Paralysis, musculospiral.

Admitted to Mercy Hospital, September 25, 1904; discharged October 8, 1904.

Operation September 28, 1904.

History of Present Illness. October 19, 1903, patient had a simple fracture of shaft of left humerus, the arm having been caught in belt. The limb was in a splint with elbow flexed. Also had burn on forearm and back, which was skin-grafted. Was in hospital 82 days and had splint on 8 weeks. When the splint was removed at the end of 8 weeks, the arm was quite strong. About seven weeks after the fracture, patient noticed tingling along arm and into hand, and could not raise the hand. The muscles of the back of hand were powerless. The hand always rested on the chest. When the splint was removed, had decided wrist-drop, slight atrophy, and grip very weak. Forearm muscles are developing some and seem to be getting strong. Was operated on in April, 1904, four months after injury, but had no effect on wrist-drop.

Family History. Negative.

Operation. Incision between brachialis anticus and triceps on posterior surface of left lower arm. Musculospiral nerve exposed and dissected out up to its entrance into a callus on left humerus, the result of the fracture eleven months previous. Dissection made over callus, and the nerve found emerging from an opening in the upper end of the callus, which was chiseled transversely down along canal, in which the nerve was found greatly reduced in size. Nerve lifted out of canal, and portion which was imbedded in callus excised. Proximal and distal ends drawn together without tension, and sutured end-to-end with fine catgut and with fine kangaroo tendon through entire coats of upper and lower ends of nerve. Incision following nerve was about seven inches long. Cicatricial end of upper segment was resected previous to suturing. A drainage-tube was inserted at upper part of wound. The fascia was then sutured over the callus and the nerve imbedded in a muscle on this to prevent subsequent involvement of nerve in the callus, and the wound closed.

Pathology. A simple fracture of center of shaft of left humerus caused a callus formation at site of fracture. A previous operation was followed by suppuration in the wound. The left musculospiral nerve was imbedded and destroyed in this callus. An upper and lower opening in callus with a connecting channel was found, in which the nerve was imbedded in adhesions. The upper portion of nerve, after leaving upper canal in callus, was greatly enlarged for one inch by cicatricial tissue. When this end of nerve was resected, it showed, on cross-section, a large amount of cicatricial tissue, in which the nerve bundles were imbedded, though they could be distinctly seen.

In eighteen months, there was practically complete restoration of function in the muscles supplied by the musculospiral nerve.

Mrs. G.

Diagnosis. Laceration of ulnar nerve.

Admitted to Mercy Hospital, October 5, 1904; discharged October 22, 1904.

Operation October 8, 1904.

Present Illness. June 1, 1904, patient fell down stairs. She went immediately to the hospital, where she was told the arm was fractured above the elbow. The arm was put in acute flexion at elbow. Shortly after that, she experienced great pain and a stinging sensation along the area supplied by the ulnar nerve. She was kept in this position for eighteen days, when the dressing was removed. At the end of this time it was found that the little and ring fingers had lost sensation, but not completely. She stayed at home for one month; then she went to another hospital, where an operation was performed, the nature of which cannot be ascertained. Patient adds that passive motion was instituted after the operation. The limitation of motion in the elbow improved somewhat after the administration of passive motion, but the numbness of two fingers remained permanent, and there was inability to flex the distal phalanx of the little finger.

Operation. An incision was made on the internal aspect of the arm. The ulnar nerve freed from the periosteal fibrous tissue and perineurium repaired. The nerve was covered with fat and fascia.

Pathology. Right ulnar nerve partially divided over the olecranon. Much of the divided fibers was included in fibrous tissue. Eleven months after, sensation was completely restored, and flexion of the distal phalanx of the little finger was perfect.

M. M., aged 3½ years.

Admitted to Presbyterian Hospital, October 15, 1905; discharged November 21, 1905.

Diagnosis. Separation of flexor tendons of left wrist, following cut.

Present Trouble. Patient enters hospital complaining of inability to flex left hand on wrist.

History of Trouble. August 4, 1905, the patient fell, cutting wrist on glass. Was taken to a doctor immediately, who sewed the skin. Wound healed by primary union. Since this time, patient has been unable to flex the hand. On examination, it was found that there was a transverse scar on left wrist (flexor surface) and that patient was unable to flex wrist, except somewhat by action of flexor carpi radialis and pronator radii teres. The proximal phalanges can be moved more than distal.

Operation. Three external superficial flexors and three deep flexors divided. Distal ends retracted to middle of palm; proximal ends of deep flexors replaced by a fibrous stump on deep muscles. Ulnar nerve was partly divided, and presented a bulbous neuroma at site of injury. An incision external to middle of palm was made; distal tendons were freed from fibrous mass. The deep tendons were united to fibrous stumps on proximal ends of muscles. The superficial ones were sutured to retracted ends. Neuroma removed, and nerve sutured over the muscles as well as possible. Wound closed without drainage; hand put in extreme flexion and cast kept on for three weeks.

There was a primary union. On examination seven months after, the flexion of the fingers was completely restored. The sensation zone of the ulnar was reestablished. It could not be determined that the adductor pollicis brevis was restored to function.

Mr. S. D.

Admitted to Presbyterian Hospital, December 2, 1905; discharged December 20, 1905.

Present Trouble. Patient enters hospital with a stab injury in outer side of lower third of left arm. On December 1, 1905, was struck in arm with a knife. Immediately afterward, discovered he could not extend left wrist He had no chill.

Physical Examination (29 hours after injury). Patient is a young, able-bodied man; good color and flesh. Walked into hospital. In lower third of humerus, on outer aspect of left arm, there is a stab wound three fourths of an inch long and about one inch deep. Extensors of forearm paralyzed. Pain sensations greatly diminished over thenar eminence of left hand, also along index fingers. Tactile sense retained.

Operation. Musculospiral nerve was exposed; it was found completely divided two inches above the elbow, and the ends separated one half inch. They were freed, their surfaces freshened and an end-to-end suture made with fine catgut. Line of union imbedded in the brachialis anticus. Primary healing.

December 15, 1905. The area of absence of sensation is very greatly diminished. This merely emphasizes the overlapping of sensory conducting nerves, which give an impaired impulse; it must not be interpreted as indicating that there was a restoration of function from reunion at this early period.

G. B., aged 17, machinist.

Admitted to Presbyterian Hospital, January 4, 1906; discharged January 19, 1906.

Present Trouble. Patient enters the hospital complaining of loss of sensation down ulnar side of left wrist, hand, little finger, and ulnar side of ring-finger. Inability to completely extend or flex the fingers of left hand.

History of Trouble. Forearm in good condition up to September 23, 1905. At this time got caught in a milling-machine, suffering considerable laceration of left forearm, with bones. Forearm was put in a cast and tubular drainage established. Kept in cast for one month and splints for one week. When hand was first taken from splint, could flex only two distal phalanges of fingers. Since this time, by passive motion, has become able to flex proximal phalanges of fingers slightly.

Examination. Upper left extremity, scar at upper end of lower third of forearm, running from ulnar side downward and outward across flexor surface. Fingers of this hand "clawed." Is unable to extend or flex fingers entirely; palm of hand flattened. Distal to scar is a triangular area anæsthetic to pain and sensation.

J. N., aged 20, barber.

Diagnosis. Musculospiral paralysis.

Admitted to Mercy Hospital, August 8, 1905; left September 16, 1905.

History of Present Illness. In April, 1905, patient, while oiling a sawing-machine, had his right arm caught. He states that it was fractured in the middle, and the skin of the posterior surface of the lower third of the forearm was lacerated, and the bones of the forearm were fractured and perforated the skin. According to patient's statement, the accident caused a simple fracture of the middle third of the humerus, and a compound fracture of both bones of the forearm in the lower third. The day after the accident the patient had fever of a moderate degree and manifested symptoms of shock. The fever continued for eight days, when patient developed a "pneumonia." The treatment of the arm was expectant. The tendons, which protruded through one of the openings, were replaced without suturing them. On the ninth day, the physician replaced the splint by a plaster-of-Paris cast, which was kept in place for three weeks. The pain continued while the arm was in the cast, and did not disappear until a month and a half after the accident. He can state nothing definite concerning his paralysis of sensation in the forearm. He only states that the feeling was gone a few weeks after the accident in the little finger and one half of the ring-finger, also on the thumb side of the hand. The sensation has slightly improved in the last five or six weeks. It is a gradual but slow return

Examination of Patient. The attitude of the affected upper extremity is the following: The elbow forms an obtuse angle; the forearm is in moderate pronation; the fingers are flexed upon the palm, so they give to the hand the aspect of a claw-hand. Palpation of the arm reveals a deformity of moderate degree in the middle third of the humerus. The deformity is angular, mostly on the inner side of the arm; on the corresponding outer side we detect a moderate depression. No pain on pressure; no subjective pain. The axis of the humerus is not visibly changed. Sensation, caloric and tactile, normal. Elbow: No deformity. The elbow, as a whole, seems to be of normal conformation. Extension of the elbow is almost perfect; flexion not painful, but incomplete. Forearm: The dimensions taken from the most prominent part of the olecranon to the extremity of the styloid process of the ulna show nine inches on the right side and 9½ inches on the left side. The circular measurements of the forearms, taken 3 inches from the epicondyle, show a difference of 1½ inches. The circumference of the left wrist is 7½ inches, while that of the right is only 6½.

Inspection. There is a marked muscular atrophy of the forearm. This is even more marked in the thenar and epithenar regions. The dorsum of the right hand is covered with more hair than the left one; the muscles of the fingers are more round; the nails are more convex and the shape is somewhat different from those of the corresponding left ones. Extension almost normal; supination impossible; pronation free and painless; rotation normal; flexion of the wrist very limited and extension impossible; extension of finger also impossible.

Operation. Bone chiseled and nerve freed; neuroma resected to normal nerve fasciculi; end-to-end union of nerves secured with kangaroo tendon. Point of approximation imbedded in muscle.

Pathology. Musculospiral nerve passed through canal in line of union in right humerus extending about one third of diameter of bone. Neuroma where nerve entered bone larger distally than proximally. Nerve not completely divided. A small shred of nerve-tissue maintaining continuity of nerve through the center of the bone remained. No electrical reaction distal to neuroma.

Operation. On wrist for removal of dead bone. Incision of tissue, cutting out necrotic bone.

Pathology. Chronic osteomyelitis of right radius, lower end.

Primary union.

The following letter was received from his physician January 17, 1906. I have been unable to locate the patient since that time.

MOUNT HOREB, WIS., January 17, 1906.

J. B. MURPHY, M. D., Chicago.

My dear Doctor: Mr. J. P., that you kindly referred to me for electric treatment for an injury of the arm and forearm, has, in my opinion, so far recovered, that I think he is now fit and in shape to undergo the operation of suturing the muscles and nerves. The regeneration of the radial and ulnar nerves is quite good, together with the musculospiral.

He is very anxious to get through as soon as possible, and any suggestion from you will be gladly received by him.

If, in your opinion, you think it best for him to come down to see you, you will kindly let me know and I will send him to you for further procedure. I am,

Yours fraternally,

N. C. EVANS, M. D.

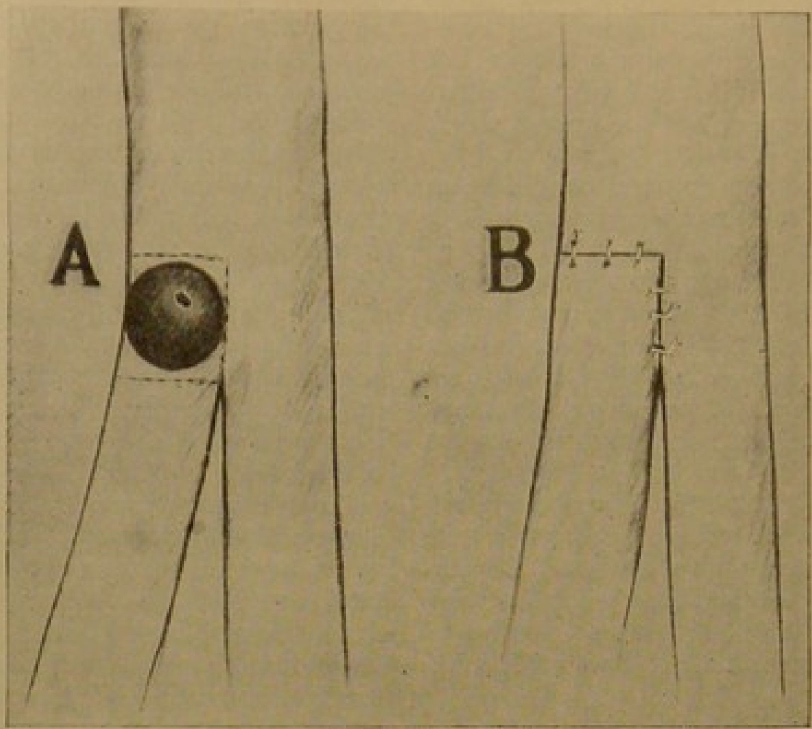


Fig. 52. A. Bullet in external popliteal nerve. B. Aspect of nerve after removal of bullet.

W. W., aged 41, police-officer.

Diagnosis. Paralysis of muscles supplied by right external popliteal nerve.

Admitted to Mercy Hospital, November 16, 1905.

History of Present Illness. October 6, 1905, patient was shot. The bullet struck the outer aspect of the right thigh, at the lower part of the upper third. The direction of the bullet was diagonal, from the point of entrance to below the articular line of the knee, on the inner side. Immediately after the accident, patient experienced considerable pain in the right ankle, the outer border of his right foot, and in his right heel. The pain in the right thigh was moderate, and less by far than that in his ankle. He was taken to Cook County Hospital, where the bullet, which was palpable under the skin, was extracted. The pain in the ankle and heel was very acute the night of the accident, and patient was unable to sleep. For seven days, patient remained in bed, suffering considerable pain, diminishing in intensity every day. While in bed, patient was able to move his right leg, and the pain was not increased by active movements. The eighth day, patient left his room. He was able to walk, but was lame. The pain in his right ankle was more accentuated when he walked than when he rested.

Examination of Patient. At first glance, there is no difference in attitude or in circumference between the two legs. The point of entrance of the bullet is marked by a small circular crust. There is no muscular atrophy of the muscles of the thigh; no pain on pressure of the thigh at any point; no interruption in continuity of femur; no difference in circumference of the knees; no deformity; no pain on pressure of the outer and lateral regions of the knee. In the lower part of the popliteal space, there is some tenderness and slight pain on pressure. Pressure along the origin of external popliteal nerve causes great pain. Pain and tenderness along his right calf. Numbness from the knee to the ankle, and on the outer side only. The contour and circumference of right ankle is similar to the left one. Pain on pressure around the external malleolus.

Sensory and Motor Tests. Patient does not feel touch of needle on right half of the calf, nor at several points on the outer aspect of the leg from the knee down. Has several zones of anæsthesia in the right half of right foot and three toes. Patient has no absolute paralysis of sensation in the regions mentioned, for zones of anæsthesia alternate with zones of hyperæsthesia. The paralysis of sensation is limited to the posterior and right half of the calf. Patient feels the touch of a glass of cold water on the regions mentioned, but states that the water is warm.

Motion. Flexors below the knee, normal. Patient is able to flex and extend his right knee. He is unable to extend the right foot; also unable to extend his toes. Adduction of the foot is almost normal. Abduction of the foot is impaired. Paralysis of extensors of the foot and toes.

Pathology and Operation. November 16, 1905. One half inch above the division of great sciatic nerve into popliteal, it was cut by bullet, dividing only the external popliteal portion, and in its place is a dense fibrous, cicatricial mass as hard as bone. This was resected and specimen sent to pathologist. After division, there was spouting of the central artery of the nerve. A double resection was made, about one inch in all. Bulbous end was harder on distal than on proximal end. No electrical reaction found in lower end of external popliteal nerve. The external half was completely separated from the internal popliteal, so that the ends could be brought together; the contused surfaces excised, and end-to-end union made with paraneural kangaroo-tendon sutures. Lateral support was given to the distal end by suturing the perineurium of the external to the internal popliteal. Point of union covered with muscular flap from the vastus externus.

Primary union.

Mr. J. S., aged 43, single, shoemaker. Admitted to Mercy Hospital, June 21, 1906.

History of Present Illness. January 1, 1906, patient, while hunting, slipped, and the gun, which was at his side, exploded. The shot glanced along his forearm and then struck a watch, a piece of which was torn off and implanted in the external aspect of left elbow. The shot produced a long oblique wound on the antero-external aspect of the forearm, which bled profusely for about three hours. At the hospital, where he was under the care of physicians, an hour later, he had his wound closed by sutures. No attempt was made to remove the foreign bodies. As soon as the patient was able to inspect his arm, he noticed a wrist-drop and loss of sensation in the thumb and index finger. There was practically no sepsis after the closure of wound. Two weeks later, a piece of the watch and numerous shot were removed. Patient left hospital at the end of six weeks. There was no improvement of sensation; the wrist-drop was pronounced.

Record of Positive Findings. Impairment of sensation on the external aspect of forearm; loss of sensation in thumb and external half of index finger. Paralysis of the following muscles: (*a*) Extensor carpi radialis longior; extensor carpi radialis brevior. (*b*) Extensor minimi digiti. (*c*) Extensor communis digitorum. Paralysis of radial nerve. There is a very painful point situated on the antero-external aspect of the forearm, on a level with the head of the radius, a little below the division of musculospiral into posterior interosseous and radial. The bulbous end of the nerve can be distinctly felt at the upper angle of the scar, but the lower end cannot. Advise operation. If less than an inch and a half of the nerve is destroyed, he will fully recover; if more, he may recover.

Operation. June 23, 1906. The cicatricial tissue was excised, the bulbous proximal end of the radial readily found. The distal had a smaller bulb, both of which were excised, leaving a gap of about three fourths of an inch. By flexing the forearm they were readily brought in apposition, united, and surrounded by healthy muscle. Primary union.

Result will be reported in later paper.

J. F. H., aged 41, salesman.

Diagnosis. Pathologic fracture of right humerus; inclusion of musculospiral nerve in callus. Patient enters the Presbyterian Hospital complaining of crepitation at junction of lower and middle thirds of right humerus on movement. Periodical muscular pain, incontinence of urine and inco-ordinate gait. Loss of weight and weakness.

History of Trouble. About four or five years ago the characteristic symptoms of tabes dorsalis appeared. Two months ago, while shaking hands with a friend, felt and heard humerus fracture. Had no pain at the time, but found function of arm was gone. Was taken to the County Hospital, where he has since been treated with splints, extensions, etc. Gained use of arm, and was discharged July 3d. Yesterday, one day after leaving hospital, while trying to put on his coat, he felt the bone separate again. No pain at this time. Arm began to swell afterward and now shows marked œdema. Has had but very slight use of fingers since first accident, but not because of pain. Has lost twenty pounds in weight in past two months. Easily exhausted.

Personal History. Takes alcohol freely. Measles when a child.

Operation. By Dr. James M. Neff, my associate. Incision over the callus. Same chiselled away with care. Nerve found imbedded in the imperfect callus. The neuroma at the seat of injury was removed and the ends, which showed the cable bundles, approximated.

DEEP ALCOHOL INJECTION FOR THE TREATMENT OF NEURALGIA

The injection of a nerve-trunk with substances of a destructive character for the relief of neuralgia originated with Pitres and Vaillard in 1887. For a long time, osmic acid was employed in the treatment of facial neuralgia by Eulenburg, Jacobi, Schapiro, Nicoladoni, and many others. It seems that chloroform preceded the use of osmic acid, and was employed by Bartholow, Mattison, and Fothergill. Normal salt solution is used by Dr. A. H. Ferguson of Chicago.

The injection of alcohol into a nerve-trunk for the purpose of relieving neuralgia originated with Pitres and Verger. Schlösser, however, was the first one to inject alcohol into the nerve-trunks at the base of the skull. The method was introduced in France by Ostwalt and further promoted and modified by Lévy and Baudouin. In the United States, as far as I am able to learn, the method has been tested and employed by Drs. Hugh T. Patrick and D'Orsay Hecht. Dr. Louis H. Hauck of St. Louis, after having learned the method from Professor Schlösser of Munich, successfully treated a severe neuralgia of the dental branch by injecting into the nerve ten drops of an eighty-per-cent solution of alcohol at the mental foramen.

The most enthusiastic advocates of the method of deep alcohol injection are Schlösser of Munich, Ostwalt, Lévy, and Baudouin of Paris, and Allacys of Antwerp. While the injections have been limited, to a great extent,

PERSONAL CASES

INJURIES TO PERIPHERAL NERVES — OPERATION — CLINICAL RESULTS

Name and Date	Diagnosis	Symptoms and History	Operation	Pathology and Result
M. P. Age 43. Laborer. June 14, 1902. Mercy Hospital.	Musculospiral paralysis from callus inclusion.	Fracture of clavicle and lower third of left humerus, from fall. After removal of cast, musculospiral paralysis found. Large callus at the seat of injury.	June 24, 1902. Incision lower third of humerus external to biceps. Nerve imbedded in callus. Nerve transformed into fibrous cord. Callus chiseled away. Fibrous portion of nerve excised and ends approximated.	Complete restoration of function.
F. L. Age 35. Oct. 25, 1904. Mercy Hospital.	Musculospiral paralysis from fracture.	Fracture of left humerus, division of musculospiral. Operated on in April, 1904, by another surgeon, without result (because nerve became imbedded in callus).	Sept. 28, 1904. Reoperated Sept. 28, 1904. Incision over lower third of humerus between brachialis anticus and triceps. Nerve imbedded in callus. It was freed from callus and found to be fibrous and much swollen. Callus chiseled away. Fibrous portion of nerve excised and ends united by kangaroo tendon. Fascia sutured over callus and beneath the nerve, to prevent subsequent trouble. Drainage.	Eleven months after operation, patient had full restoration extensor muscles.
Mr. J. N. P. Age 20. Sept. 8, 1905. Mercy Hospital.	Musculospiral paralysis following fracture of humerus.	Marked paralysis of musculospiral. Humerus and bones of forearm fractured while being caught in a machine.	Aug. 10, 1905. Incision on course of nerve. Nerve found in a groove over line of bony union. Neuroma found, involving three fourths of nerve. Neuroma and nerve ends excised; end-to-end suture with kangaroo tendon made. Point of union surrounded by muscle and fascia.	Perfect restoration of function.
Mr. D St. Age (?) Dec. 2, 1905. Presbyterian Hospital.	Musculospiral paralysis from stab wound of arm.	Stab wound on lower third of left arm. Musculospiral paralysis.	Dec. 1, 1905. Ends of divided nerve reunited, and point of anastomosis protected by soft tissues.	Dec. 15, 1905, pain sensation returning in index finger and thenar eminence. May, 1906, full functional restoration.
Mr. James F. H. Age 41. Aug. 4, 1905. Presbyterian Hospital.	Tabes dorsalis. Pathologic fracture. Musculospiral paralysis.	Symptoms of tabes; spontaneous fracture of right humerus followed by musculospiral paralysis due to division by bone.	Dec. 1, 1905. End-to-end union, kangaroo tendon. Point of union surrounded by fascia. (Operation by associate, Dr. J. M. Neff.)	Patient lost sight of.
Mrs. G. Age 35 Oct. 5, 1904. Mercy Hospital.	Laceration of ulnar nerve.	Ulnar paralysis following fracture of humerus Patient has had previous operation, but no definite history obtainable as to what was done.	Oct. 15, 1904. Ulnar nerve found adherent to surrounding tissues and also imbedded in considerable connective tissue. It was freed from adhesions; perineurium repaired and nerve covered with fat and fascia.	Good functional restoration.
Mr. M. M. ——— Oct. 15, 1905. Presbyterian Hospital.	Laceration of ulnar nerve and injury of flexor tendons of forearm.	Arm cut with glass previous to Oct. 15, 1905.	Oct. 18, 1905. Incision on anterior portion of forearm flexor tendons. Nerve exposed. Flexors repaired by suture. A neuroma of ulnar nerve removed and nerve sutured over the muscles as well as possible.	Good result.
Mr. George B. Age 17. Jan. 4, 1906. Presbyterian Hospital.	Paralysis of ulnar nerve.	Injury to forearm.	Jan. 7, 1906. Neuroma of ulnar resected and ends united, point of anastomosis being protected by soft tissues.	Restoration of function.
Matthew H. Age 42. April 17, 1906. Cook County Hospital.	Compression paralysis of ulnar nerve, due to scar tissue, the result of a cut, just above the annular ligament.	Paralysis of the adductor pollicis and all of short muscles supplied by the ulnar.	April 17, 1906. Exposure of nerve above and below scar tissue, and then a division of the cicatricial fibrous mass which bound it down; it had not been divided.	Faradic excitation proximal to the scar tissue before division showed no muscular response; distant to scar tissue, showed muscular response, which indicated that nerve had not been divided. After removal of cicatrix, there was faradic response to both proximal and distal from the point of previous constriction. Repeated examinations made afterwards by Drs. F. G. Dyas and V. L. Schrager show "perfect restoration of function."

Name and Date	Diagnosis	Symptoms and History	Operation	Pathology and Result
Willy D. Age 10. July 9, 1906. Presbyterian Hospital.	On the day previous, fell and skinned his elbow on a piece of glass; incision just above elbow-joint and between internal condyle and olecranon	Paralysis of long flexor of little finger, and loss of sensation over ulnar side of forearm and hand, and also paralysis of muscles of palm supplied by ulnar nerve	Ulnar nerve exposed and found completely divided; it was secured, cleansed, and freshened, and end-to-end union with kangaroo tendon (by paraneural transfixion) established. Point of union surrounded by fascia.	There was faradic reaction in all of the muscles di stant to the point of division, as would be expected, considering that one day has elapsed since the injury. Dec. 2, 1906. Slight atrophy of hypothenar eminence. There is a numbness of sensation of the inner side and palm of hand. He can flex both little and ring fingers completely, but the force is less than that of the opposite side. Some paresis remains five months after operation.
John S. Age 43. Mercy Hospital.	Division of radial, with paralysis of extensors of hand, forearm, and the supinators.	Shotgun wound. Piece of watch and shot imbedded in left elbow. Destroyed musculospiral radial at elbow.	June 22, 1906. Nerves united end to end, and line of union surrounded by muscle. Primary union.	Dense cicatricial mass resected. With arm flexed, we were able to secure a good apposition. Too early for return of function.
William S. Age 41. Nov. 16, 1905.	Paralysis of muscles supplied by right external popliteal.	Injury of right sciatic following bullet wound of thigh. Oct. 6, 1905. Bullet cut off external popliteal portion of sciatic one half inch above division.	Excision of traumatized zone of nerve, and end-to-end operation.	Partial return of sensation. Too early for return of motion.

to the branches of the trifacial, Ostwalt and others have employed it in neuralgias of other nerves; for example, the sciatic. The method can be applied to affections of both sensory and motor nerves. Facial spasm, tic, has been benefited by the injection of alcohol into the facial nerve as it leaves the stylomastoid foramen. As a rule, there is no facial paralysis after the injection.

Ostwalt injects 1–1.5 c.c. of an eighty-per-cent solution of alcohol (to which he adds 0.01 cg. of cocaine or stovaine) into each of the affected branches at the point of exit from the skull. Lévy and Baudouin inject one or two cubic centimeters of alcohol at the time. They use first a seventy-per-cent solution, then eighty per cent, and finally ninety per cent, with or without cocaine. Four to five drops of chloroform are then added to each cubic centimeter, and injection repeated.

In my case I used a seventy-per-cent solution of alcohol without any additional anodyne. The reason for employing this strength in preference to eighty, ninety, or one hundred per cent is based on the experience of bacteriologists, who found that in this percentage the alcohol has the greatest bactericidal power. In the technique of the injection I have followed the directions of Lévy and Baudouin. As these investigators have dissected a great many heads and measured about seventy-six skulls, their anatomic *data* are instructive. I shall give below, as closely as possible, the contents of their article published in the *Presse Médicale*, Feb. 17, 1906.

The inferior maxillary branch can be found by passing a needle 2.5 cm. from the descending bifurcation of the longitudinal root of the zygoma and pushing it gently and slightly backward a depth of 4 cm. This will strike the inferior maxillary nerve as it comes out of the foramen ovale. The tissues through which the needle passes are skin, subcutaneous tissue, zygomatic insertion of the masseter muscle, posterior part of the temporal tendon; the needle finally crosses the superior border of the external pterygoid muscle. One passes in front of the capsule of the temporomaxillary articulation. The organs to be avoided are the transverse facial artery, the internal maxillary artery and corresponding veins, and particularly the middle meningeal artery. The avoidance of injury to these organs is accomplished by using a special needle. It consists of a metallic tube 10 cm. in length and 1.5 mm. in diameter. One end is pointed; the needle is graduated from the point in five subdivisions of 1 cm. each. The pointed extremity is rendered blunt by passing into the lumen of the needle a wire with a rounded dull end. The puncture of the skin is made with the point of the needle, without the use of the guide, and pushed a distance of 1.5 cm. Then the blunt wire is inserted and the needle pushed on 2.5 cm. more until a depth of 4 cm. is reached.

Superior Maxillary Branch. A vertical line is drawn from the posterior border of the orbital process of the malar bone to the lower margin of the zygoma. The needle is inserted 1 cm. behind this point, tangentially to the zygomatic process; it is then directed slightly upward and pushed a distance of 5 cm., when the needle strikes the trunk in the pterygomaxillary fossa. If the needle does not advance beyond a distance of 2 cm., it probably strikes a bony plane, usually an abnormally developed coronoid process, or the external pterygoid plate. If this be the case, the needle should be gently inclined forward. In so doing, one must be very careful, as there is danger of puncturing the eye. On the other hand, one must keep well above the sphenopalatine foramen, which leads into the nasal fossæ.

The tissues through which the needle travels are skin, cellular tissue, anterior fibers of the masseter muscle, anterior border of temporal tendon, which can be avoided by opening the patient's mouth. The needle then reaches the fat in the pterygomaxillary fossa, and finally strikes the nerve.

The Ophthalmic Branch. Lévy and Baudouin state that one cannot claim to inject the trunk directly, as the ophthalmic branches are within the cranium. The first of the three branches, the nasal, is not accessible, being situated among the motor nerves which surround it. The frontal and lacrimal branches are reached via the orbit.

These writers state they keep close to the external wall of the orbit at the level of the inferior extremity of the external angular process of the frontal bone. The needle, being inserted at this point, passes beneath the lacrimal gland and follows the periosteum without injuring the globe of the eye, or any of the important organs. The blunt guide is withdrawn at a distance of 35 or 45 mm. and the trunk injected. Some difficulty is experienced in penetrating the external layer of Tenon's capsule, which is very dense. It is advisable that the patient be placed in a recumbent position, with the head slightly elevated, when the superior and inferior maxillary nerves are injected.

The results with this method have been very gratifying. In the first place, the use of the alcohol renders the procedure aseptic; furthermore, it has the peculiarity of relieving pain without giving sensory or motor paralysis, or, if any, it is slight and temporary. The paresis may persist a few hours, or a few days; exceptionally, a few weeks, and rarely a few months. The neuralgic pains are sometimes relieved instantaneously while the needle pricks the nerve. The rule, however, is, that the first injection gives considerable relief, but that in severe neuralgia, two, three, or more injections must subsequently be given.

Ostwalt of Paris practised several hundred injections, the patients being affected with very severe neuralgia, dating back 6, 7, 9, 10, 13, 16, and even 20 and 30 years. In one third of all patients there was a return of symptoms in four to five months. A few subsequent injections, however, relieved the neuralgia permanently.

Dr. Louis Hauck states that Schlösser of Munich had 100 per cent of cures since 1902. Almost all the cases treated by Lévy and Baudouin were cured, although in some cases repeated injections had to be made.

My experience with this method is very recent, and the patient on whom I have used the method is still in the hospital, so I am unable to draw any conclusions as to the value of the method.

The following is a history of our case:

N. L., aged 50, laborer.

Family History. Negative.

Personal History. Born in Germany; has lived in Iowa past 18 years. Habits regular.

Previous Illnesses. Diseases of childhood, including an attack at age of 10, diagnosed as smallpox; recovery without complications. Rest negative.

Present Trouble. Began 15 years ago with sudden, sharp, stabbing pain, seeming to be in lower maxilla on right side. Consulted dentist, who removed two teeth. Following this the pain became greatly exaggerated. Continued this way for a year, there being intervals of a few days to a week, when patient was free from pain. Patient then went to doctor, and says "nerve-stretching" operation was performed. Following this, patient was entirely free from pain for next six months. Then pains recurred in same area, also including the ear, the right side of the nose, and supraorbital and infraorbital region. The patient then began using cocaine, and for the next two years he was able to control the pain to great extent. Massage and electricity were employed, but this did no good. Patient then began using atropine, which helped him for about two months; following this he used morphine for the next two years (two or three grains per day). February, 1906, he was operated on by Dr. Murphy, and affected branches injected with osmic acid. Four weeks later, was again injected. Following this, had some soreness over right cheek, even when pain was not present. Patient states that antrum was opened and drained. From then until June, 1906, pain returned; patient also began to complain of marked tenderness over right cheek; a yellowish purulent material discharged from right nostril, especially noticeable when he held his head forward. Four weeks ago, right cheek became acutely swollen, and suffering was marked. Patient had chill and considerable fever at onset, followed in few days by marked flow of yellow pus.

At present, complains of pain in supraorbital and infraorbital region, over cheek, and over inferior maxilla. Comes in paroxysms, sharp and stabbing, which nearly take patient off his feet. Some constant tenderness over right cheek and a constant discharge of pus through right nostril. Appetite good; bowels constipated.

Pathology and Operation. Incision above the orbit. Nerve exposed and injected with seventy-per-cent solution of alcohol. An opening was made into antrum and opening packed with iodoform-gauze. Infraorbital nerve had regenerated to full size. Discoloration of previous injection very evident, and around this could be seen the white regenerated nerve-tissue. No injection of infraorbital. Inferior dental injected with same solution of alcohol.

From August 1, 1906, to February 1, 1907, Dr. Hugh T. Patrick had treated six cases of trifacial neuralgia with deep injections of alcohol, according to the method of Lévy and Baudouin, each patient having received from three to eight injections. On February 1st, all of the patients were entirely comfortable, the time since the last injection having been from six months to a few days. In every case there had been immediate relief of pain, but in some the pain had returned with less severity before the series of injections was completed. In no case had there been any untoward result of any moment.

Dr. D'Orsay Hecht has studied the method for some time. He has measured a number of skulls and figured out the exact anatomical relations and anomalies. His experience will be published in detail at some future date. From his cadaver work he is convinced that it is entirely feasible to approach the foramina either by an external of internal (buccal) puncture. He tested the value of both needles — the straight one of Lévy and the bayonet-shaped one of Ostwalt — and concluded that the straight needle is much easier to handle.

Dr. Hecht treated two patients. The first one, aged 42, had a neuralgia of the left middle branch dating back eight years. Injection performed November 20, 1906, under ethyl-chloride anæsthesia; there was immediate relief; recurrence of pain in two weeks; second injection; slight recurrence of pain after eight days; third injection December 24th; perfectly free from pain to date; undisturbed by hot or cold drinks, washing face, or drafts of air. The second case was an aged patient affected with neuralgia of the right inferior branch. He had severe paroxysms. Injected March 11, 1907, "too recently for comment, except to say that considerable relief was had about four hours after puncture. I imagine this patient will require several injections."

CONCLUSIONS

The gray and white matter of the spinal cord down to the cauda is made up of:

(*a*) Intrinsic ganglionic cells (gray matter).

(*b*) Centrifugal cerebral axones (white matter of motor columns).

(*c*) Centripetal sensory axones from the spinal ganglia in which is located the trophic cell-body of these axones, making up the sensory columns. The centripetal and centrifugal axones within the spinal cord are aneurilemmic.

(*d*) Intercommunicating or associating axones, which connect the various segments of the intrinsic ganglionic cells of the cord, and form the anterior ground bundle of white fibers, are aneurilemmic.

(*e*) Direct cerebellar centripetal axones from ganglionic cells situated in the posterior internal region of posterior horn, and close to the commissure leading to the ganglionic cells situated in the vermis superior of the cerebellum, are aneurilemmic.

(*f*) Gowers' tract, the axones of which originate in the posterior horn, close to the commissure, are also aneurilemmic.

(*g*) Neuroglia.

(*h*) The bulb or medulla oblongata is a continuation of the same ganglionic zones of gray matter in the cord, and has all of the centrifugal and centripetal axones of the spinal cord. And, in addition, it has its own peculiar anatomic elements.

Nerves arising from the medulla are, surgically, essentially spinal nerves. Therefore all of the "cranial" nerves, except those of special sense, are, from a surgical standpoint, essentially spinal nerves. The extramedullary motor and sensory axones are medullated and neurilemmic.

Nerves of special sense should be called cerebral nerves, as their trophic cell-bodies are outside the cranium, and in the organs of special sense. The centripetal axones of these nerves, with the exception of the olfactory, are all medullated and all aneurilemmic.

The extraspinal roots, both centrifugal motor and centripetal sensory, are medullated and neurilemmic.

The peripheral nerves of the bulb and cord (cranial and spinal) are medullated and neurilemmic to their nerve-endings. The terminal dendrites are medullated and aneurilemmic. They possess, however, a limited potentiality of outgrowth of the axones beyond their neurilemma.

The cauda is, from a surgical standpoint, an intradural collection of extramedullary spinal axones, and is made up of axones with a medullary sheath and a neurilemma.

The sympathetic system is a chain of ganglia and nerve-trunks. Their axones, except a few of the abdominal, are neurilemmic and non-medullated.

When an axone is divided, the entire portion distal from the trophic ganglionic cell-body degenerates; proximally, it degenerates for a short distance, and for a longer distance it atrophies.

When a ganglion-cell is once destroyed, the degeneration of its axone or axones and dendrites is complete, and the restoration of motion and sensation in its peripheral zone can only be obtained by anastomosis of its axones to other axones originating from healthy ganglionic cells, as in operations for anterior poliomyelitic paralysis.

Ganglionic cells and aneurilemmic axones, once destroyed, never regenerate. All neurilemmic axones, centrifugal (motor) and centripetal (sensory), are capable or potent of both anatomic and physiologic regeneration under favorable conditions. *The key, therefore, to the surgery of nerves is surely the neurilemma; i. e., nerves with a neurilemma are all capable of regeneration under proper surgical conditions; those without a neurilemma, i. e., aneurilemmic axones, are incapable of regeneration.*

Ganglionic cells in the gray matter of the cord never regenerate to functional potency.

Centrifugal motor axones within the cord never regenerate, as they are aneurilemmic.

Centripetal sensory axones within the cord never regenerate, as they, also, are aneurilemmic.

It can therefore be seen that there is no regeneration of the white columns or gray matter of the cord after destruction or division; it has never been demonstrated experimentally nor authoritatively observed clinically.

All these principles equally apply to the bulb or medulla oblongata.

The spinal, cranial, sensory, and sympathetic ganglion-cells once destroyed, never regenerate.

The caudal portion of the cord is made up of neurilemmic centrifugal axones from the spinal-cord ganglion-cells, and of centripetal sensory axones from cells in the spinal ganglia, all being neurilemmic from the place they leave or enter the conus. They are therefore all capable of regeneration, the same as peripheral axones.

Spinal Roots. The extracordal or extramedullary and extrabulbous spinal and cranial axones are neurilemmic and potent for regeneration. Therefore divided roots within the cranium and spinal canal regenerate.

Peripheral nerves, cranial and spinal, are composed of neurilemmic axones, and are therefore capable of regeneration.

The nerves of special sense — the optic, olfactory (?), gustatory, and auditory — are composed of aneurilemmic axones, and are incapable of regeneration if once destroyed. In this particular they resemble the columns of the cord. Their trophic ganglion-cells are situated in the retina, Schneiderian mucosa, taste organs, cochlea, and vestibule, respectively. When these membranes and ganglion-cells are destroyed, the cells and axones are incapable of regeneration.

The sympathetic nerve-trunks are non-medullated, but neurilemmic and capable of regeneration.

Surgery. Hemorrhage, concussion, and contusion of the cord without laceration may offer the same immediate symptomatic picture as that of division, so that a positive differential diagnosis may be practically impossible.

There is no direct relationship between the severity of the trauma and the degree of lesion in the cord. The element of time and the order of appearance of symptoms are of great importance, and may be the only guide in the differential diagnosis.

Absence of paralytic symptoms immediately after spinal trauma does not justify the surgeon in assuring the patient that such symptoms will not appear. They may set in within a few days, or even some weeks, after the injury.

If paralysis is due to hæmatorrachis, the condition may be relieved by early spinal puncture.

Intra-arachnoidal or periarachnoidal hemorrhage from bullet or stab wound may produce complete paralysis resembling that of a division of the cord. If the pressure be relieved, the patient will survive and the paralysis will be temporary.

The majority of cases of transverse or incomplete traumatic irregular paralysis following fractures recover without operative treatment, which signifies an absence of spinal-cord division. Wherever there is immediate and complete circular paralysis, operation does not benefit the patient in the least, as there has been a division of spinal neurones which never regenerate.

Surgical intervention in injuries to the cord should be resorted to only in cases in which the spinal cord is not completely divided, except in the caudal zone. Immediate intervention will be of benefit when the cord is compressed above the cauda, or compressed and divided in the cauda. If operation is at all indicated, there is no reason for delay, as degenerative changes may take place in the cells and neurones of the cord, which would be as irreparable as its division.

In fractures of the spine without considerable displacement, we are justified in assuming that the cord is not suffering continued compression, regardless of the degree of paralysis; operation is contraindicated. If this paralysis is due to laceration, it will not be improved by operation. If it is due to contusion, it will recover without operation.

Immobilization is most important in favoring the repair of the spinal cord and lessening the likelihood of connective tissue and callus compressions at the site of injury.

In gunshot and stab wounds with immediate paralysis, operation is contraindicated, except in the caudal zone, as the cord is probably severed, and its reapproximation will avail nothing.

After division or crushing of the nerves of the caudal zone, there is a positive indication for an end-to-end suture of the various fibers, the same as in peripheral nerves.

In spina bifida centralis (paraplegia in the caudal zone), resection of the atrophied portion of the cauda with end-to-end union is indicated.

Upper ependymal and true cord central spina bifida may be treated by ependymal arachnoidal drainage.

In all non-malignant tumors of the cord, laminectomy should be performed at the onset of paresis. Delay in completion of paralysis is unpardonable, not to use a more forcible expression. Operation after complete paralysis from compression with degeneration is contraindicated.

In tuberculoma compression of the spinal cord, the operation should be done at the onset of the symptoms of paralysis. Late operations, that is, after pressure necrosis of the cord has taken place, are worthless.

Surgery of the spinal cord, like surgery of other parts of the body, must be timely; i. e., the operation must be performed before the pathologic condition is advanced beyond the possibility of repair. In our present positive though limited knowledge, timely action means conservatism, while delay must be interpreted as timidity or inefficiency.

In our citation of cases we do not include the great bulk of cases of fractures which recovered without operation prior to our present knowledge of the cord, nor tumors or granuloma which had the symptoms of paralysis and which improved without surgical intervention, nor the great number of cases of fractures of the spine treated by laminectomy which did not recover. A detail of these histories would have no value, as they did not have back of them a knowledge of the potency or impotency of degeneration of the various portions of the cord; nor do we place under this heading the histories of cases of nerve anastomosis, or peripheral nerve union for anterior poliomyelitis, as we feel they should come under the heading of surgery of the peripheral nerves. We cite only exemplary cases to enforce or elucidate the text.

All of the peripheral nerves, except those of special sense, are neurilemmic, and capable of regeneration under favorable circumstances.

The nerves of special sense are aneurilemmic, and, once divided, never regenerate. The neurilemma is therefore the index for surgical intervention.

The peripheral axones in nerve-trunks are completely insulated; axonal contact cannot be secured unless the axis-cylinders are freed from their insulation laterally or brought into end-to-end approximation.

Functional regeneration is not accomplished until the distal end of a divided axone is brought in contact with a proximal axone, which extends uninterruptedly from a ganglion cell-body. Lateral apposition or end-to-side approximation of nerves gives only occasional axis-cylinder contact, and only a small per cent of the axones regenerate. All nerve reunion should be made by end-to-end contact or fixation.

In old divisions, a sufficient resection of the bulb ends must be made to expose the normal nerve fasciculi before the union is attempted. It is useless to approximate bulbed ends covered with connective tissue. The nearer to the spinal cord that a nerve-trunk is divided, the longer the time required for its functional regeneration.

The most favorable results are obtained where the cement substance of the union comes exclusively from the neuroglia or neurilemma. One of the greatest barriers to axonal functional regeneration is the interposition of connective tissue. Primary union is therefore a *sine qua non* to success.

Every nerve union should be surrounded by a guard against the intervention of collateral connective tissue.

The results of nerve suture without infection and without the interposition of connective tissue can be made as uniformly successful as bone unions under similar conditions.

The earlier the nerve union is re-established, the more rapid and certain the restoration of function.

It is therefore as incumbent on the surgeon to produce an immediate aseptic end-to-end approximation of a nerve after division as it is to replace a fractured bone. The period of time necessary for a restoration of function varies materially, and only an approximate estimate can be given with our present knowledge. Sensation is restored, at the earliest, in six weeks, and motion in ten to twelve weeks.

Whenever there is tension on the line of union, the nerve should be given support by suturing it to the neighboring structures. When the distance is too great for end-to-end contact, the gap should be filled with a fresh nerve-trunk from animals, incased in a slowly absorbable animal material, preferably an artery hardened by formalin. During the process of repair the muscles should have massage, passive motion, and mild *galvanic* current.

The order of functional regeneration after union is: 1. Trophic; 2. Sensory; 3. Motor.

I wish to express my appreciation of the services of Dr. Victor L. Schrager in the preparation of this paper.

Dr Burkhart
Gloucester
Eng