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LABORATORY NOTES OF TECHNICAL METHODS FOR THE NERVOUS SYSTEM.

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METHODS OF OPENING THE BRAIN AT AUTOPSIES AND FOR GENERAL DISSECTION OF THE BRAIN.

1. *Meynert's Basal Section of the Brain.*—The ordinary method of opening the brain at autopsies by slitting open the corpus callosum, laying aside the hemispheres, and then making longitudinal and transverse incisions, renders it difficult to handle the brain without tearing it, and, after hardening, the pieces can not be fitted together well, so that the lesions may be accurately localized in conjunction with the microscopical examination. Meynert's section is much more valuable for the purposes of localization after hardening, and is recommended for general use at autopsies and as an instructive method for demonstrating to students the coarser anatomy of the fresh brain. With some modifications of Meynert's original plan, the section is made as follows:

Resting the brain upon its hemispheres and lifting up the cerebellum, the pia mater and vessels are cut above the corpora quadrigemina and around the crura and inner

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margins of the temporal lobes on either side until the middle cerebral arteries are reached. The incision of the pia then follows the middle cerebral arteries into the Sylvian fissures, and passes to the terminations of the posterior branches of the Sylvian fissure. As the incision of the pia follows in this way, the boundaries of the temporal lobes, the temporal convolutions should be gently separated from the neighboring convolutions and from the base.

The apices of the temporal lobes are then lifted up, and, resting the knife flat on the base, their junction with the base is cut, until the descending horn is opened. Then inserting the knife into the descending horn, the incision passes outward and backward to within an inch of the apex of the posterior horn, or even to its extremity, severing the junction of the occipital and temporal convolutions on the lateral surface of the brain. During this manipulation the cornu Ammonis and posterior pillars of the fornix should not be cut. The temporal lobes are thus freed from the base and folded outward and backward over the occipital lobes, giving access to the island of Reil (Fig. 1). The operculum is then pulled well outward to completely expose the convolutions of the islands, and a slightly curved transverse incision (*a*, *a*, Fig. 1), with the convexity directed frontally, is made connecting the anterior sulci of the islands on both sides. This incision should be deep enough to pass through the anterior horns of the ventricle to the corpus callosum.

The cerebellum is then lifted up with the left hand, and placing the knife in the ventricle at *c*, Fig. 1, and passing it beneath the rounded posterior extremities of the optic thalami, the internal capsules on either side and the anterior pillars of the fornix and septum lucidum are cut from behind forward without injuring the basal ganglia. As the internal capsules are being cut, the cerebellum is gradually

rolled over forward on the base, in order to lift up the basal ganglia from the corpus callosum. A square piece—"the

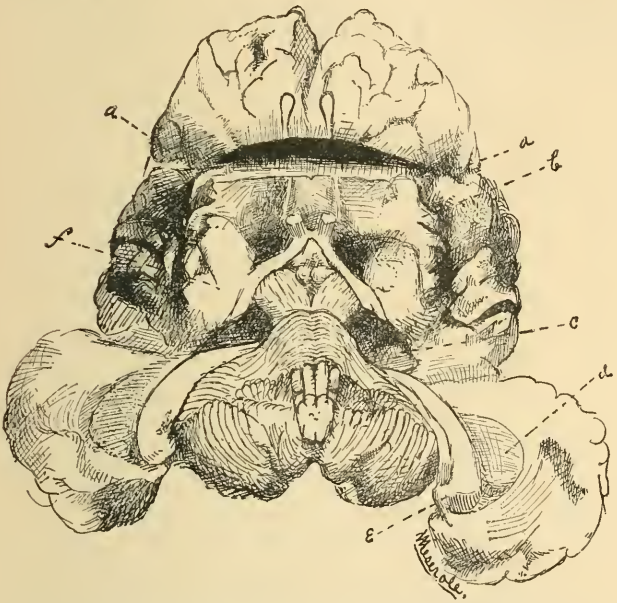

Fig. 1.—Drawing of the brain, showing how the brain axis is exposed before it is cut out. *a*, anterior sulci of the islands of Reil; *b*, convolutions of the island; *d*, descending horn in the reflected temporal lobe; *e*, cornu Ammonis, with the posterior pillar of the fornix passing from it into the ventricle; *f* passes across the convolutions of the operculum to the cut surface of the junction of the temporal lobe with the base.

basal piece" (Fig. 2)—is in this way cut out of the base. The fornix is left lying on the corpus callosum.

When the brain is soft, and in children's brains and in cases where the basal ganglia are injured by hæmorrhage, the above-described method of removing the basal piece is modified by putting the index and middle fingers of the left hand through a transverse incision of the base (Fig. 1, *a*, *a*)

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into the anterior horns of the ventricles and raising the basal piece, while the internal capsules, anterior pillars of the fornix, and septum lucidum are cut from before backward. This plan of cutting out the base is not recommended ex-

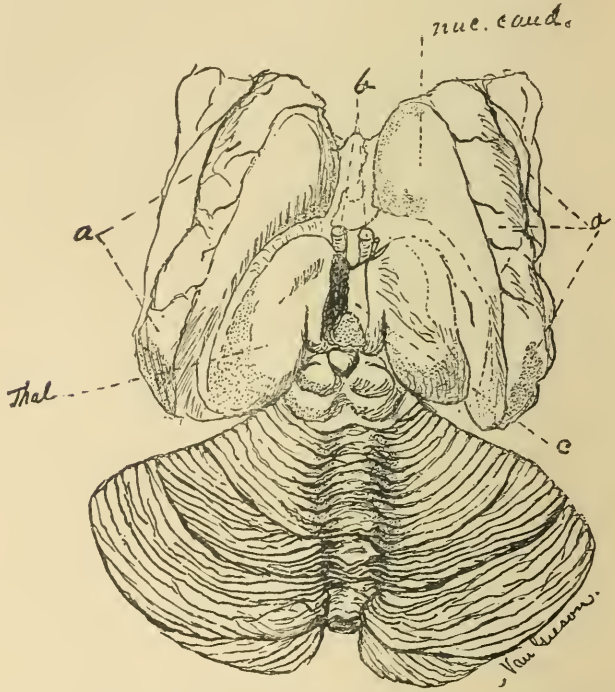

FIG. 2.—Ventricular surface of the basal piece or brain axis. *a*, *a*, fibers of the internal capsule cut transversely as they enter the basal ganglia; *b*, anterior pillars of the fornix cut across; *c*, third ventricle; *nuc. caud.*, caudate nucleus; *thal.*, optic thalamus.

cept when the brain is very soft, because the introduction of the fingers into the ventricles is apt to damage the ependyma.

The advantage of this plan of opening the brain is that it uniformly separates the organ into two great anatomical subdivisions. The square basal piece—the *brain axis* (Fig. 2)—includes the island, basal ganglia, internal capsules, crura, pons, medulla, and cerebellum. The remaining piece —*the brain mantle of Burdach* (Fig. 3)—includes the con-

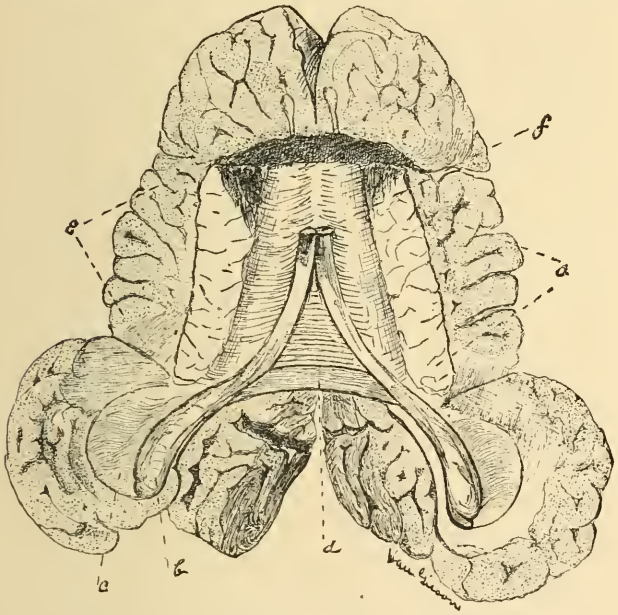

FIG. 3.—Ventricular aspect of the brain mantle. *a*, internal capsule ; *d*, posterior border of the corpus callosum ; *b*, cornu Ammonis ; *c*, descending horn ; *e*, operculum ; *f*, anterior pillars of the fornix cut transversely.

volutions, corpus callosum, fornix, and cornu Ammonis. This method also exposes the internal capsule (Figs. 2 and 3) advantageously for the localization of lesions in the internal capsule, because its fibers are cut transversely between their entrance into the basal ganglia and the centrum ovale.

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Both these pieces may be hardened *en masse* by making transverse incisions about one half to three quarters of an inch apart in the basal piece, and both transverse and longitudinal incisions into the white matter of the larger piece not quite reaching to the pia mater, which serves to hold the convolutions together for the purposes of localization after hardening. The incisions should be kept open with absorbent cotton, and each piece should be suspended in gauze or laid on absorbent cotton in a large volume of Müller's fluid (five or six times the volume of the pieces), which is kept cool and changed on the second, fifth, and eighth days, and again in the third week. It is best to remove the cerebellum from the rest of the brain axis at the autopsy unless the integrity of its pontial relations is desirable, so that in hardening the fluid may penetrate into the floor of the fourth ventricle. The cerebellum is removed by cutting away its crura close to the pons. The practice of inspecting the fourth ventricle by splitting the cerebellum in the median line is apt to distort the floor of the fourth ventricle by the traction of the two divergent cerebellar hemispheres.

2. *Division of the Brain into Transverse Segments for Examination at Autopsies.*—This method is used quite frequently at the laboratory in Heidelberg, and consists simply in making serial transverse vertical slices of the brain about three quarters of an inch thick, first having removed the cerebellum and pons by cutting the crura cerebri transversely. When the slices have been hardened they may be easily fitted together for the detailed localization of the lesions. This method is good for the preparation of certain museum specimens and for lantern demonstrations of tumors or cortical lesions by stained sections of the entire brain, made with the Gudden microtome.

3. *Preparation of the Brain for the Dissection of the*

Association, Commissural, and Projection Fibers, by the Cleavage Method.—The pia mater is carefully removed from the convolutions, and the brain—suspended in gauze in forty-per-cent. alcohol, which is renewed on the third day—is turned on a new surface each day to avoid flattening. No incisions are made except a small one through the infundibulum to let the fluid into the lateral ventricles. At the end of the first week of hardening, the alcohol is replaced by sixty-per-cent. alcohol; at the end of the fourth week this is replaced by eighty-per-cent. alcohol, and then, for three weeks or a month, the brain is left in strong alcohol. With this treatment the bundles of fibers of the white matter become tough and elastic, and a particular tract may be isolated by pushing and cleaving aside the surrounding structures with a scalpel handle. The same method of hardening may be used if dissociation of the tracts is to be practiced in some particular portion of the brain, such as the brain axis or brain mantle.

As an example of how a system of fibers may be dissected out when the brain is successfully hardened in this way, the path of the motor tract in the brain axis is selected. The brain axis (Fig. 2) is hardened as described above, and the pyramids of the medulla are loosened from the olivary bodies, and from each other in the median line to their decussation, with the handle of a scalpel. The superficial transverse fibers of the pons are cut in the median line and pushed away from the pyramids with the scalpel handle inserted in the groove between the inferior pons border and medulla, until they are reflected as two flaps on the middle cerebellar crura. The pyramids are now exposed between the two sets of transpontial fibers, and, when freed from a few interlacing pons fibers, they may be picked up with the forceps and followed up through the middle thirds of the crura into the internal capsules. This method

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is exceedingly valuable for teaching purposes, and a series of these cleavage dissections is the best way of giving students a clear idea of the various association, commissural, and projection systems. Plates 18 to 22 of Meynert's "Psychiatry," translated by Sachs (Putnams, New York, 1885), and Plates 28 and 30 in Edinger's "Zehn Vorlesungen über d. Bau d. nerv. cent. Organe," 1885, may be used as guides for other dissections of the different systems composing the white matter.

NOTES ON THE HARDENING OF THE BRAIN.

Müller's fluid does not preserve the ganglion cells as perfectly as bichloride of mercury or alcohol, but it preserves the nerve fibers and neuroglia well; it gives the proper consistence for cutting sections, and all the important methods of staining the nervous system depend upon bichromate hardening. The obstacle in getting perfect hardening results with Müller's fluid is the great impermeability of the cerebral tissues. To get the best results in hardening the pieces should not be more than one to two cubic centimetres in volume, and should be left in the fluid at the temperature of the room until they have a dark-brown color, and sections cut by hand do not wrinkle when placed in water. This may take four or five months or longer, and the blocks should be frequently tested to guard against their becoming brittle. Rapid hardening in the thermostat is not often used in this laboratory; it is not so good as slow hardening at low temperatures, or at the ordinary temperature of the room. In hardening with heat, the superficial portions of the specimens become hardened too rapidly, and become dense and prevent the fluid from gaining access to the central portions of the blocks. If rapid hardening is necessary, it is much better to renew the fluid frequently after the first two or three weeks of the

hardening, and at each renewal to gradually increase the strength of the bichromate-of-potassium solution up to five per cent. *Erlicke's solution* hardens the superficial portions of the blocks more rapidly than Müller's fluid, and consequently does not preserve the central portions so well. It also has a greater tendency to make the specimens brittle. The bichromate solution should always contain camphor (a piece of the size of a pea to each pint of the fluid) to prevent the development of micro-organisms.

When specimens have become brittle it is very difficult to get them into good condition for cutting. A prolonged immersion of the specimens in equal parts of glycerin, alcohol, and water reduces the brittleness somewhat, but the best results have been obtained by soaking the blocks for twenty-four or forty-eight hours in a fifty-per-cent. or stronger aqueous solution of ammonia. A twenty-four-hours' immersion in peroxide of hydrogen also tends to make the specimens less brittle. When very large segments of the brain are to be preserved for microscopical examination it is best while hardening to keep the bichromate solution cold for the first month or two in a refrigerator.

If specimens are not thoroughly hardened in Müller's fluid, the subsequent permanent preservation in strong alcohol does them considerable injury. The alcohol slowly dissolves out the chrome salts and changes the myelin. In the course of a year or two cholesterin crystals form in the alcohol, and minute cavities and vacuoles appear in the specimens and they do not stain well. If for any reason it is necessary to examine specimens before they are thoroughly hardened, it is best to keep the material for permanent preservation on the shelves in water containing camphor, or dilute, thirty to forty per cent., solutions of alcohol. In the laboratory even thoroughly hardened material is not kept permanently in strong alcohol. Seventy or eighty per cent.

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is used, and in certain cases water. Well hardened material kept in water has a tendency to become brittle in the course of years. The structure of the neuroglia never shows so well, and carmine staining is never so successful in sections of material which has been placed in alcohol as in sections cut from specimens while in Müller's fluid, or kept in water slightly tinged with Müller's fluid.

DIVISION OF THE SPINAL CORD INTO ITS ANATOMICAL SEGMENTS FOR MICROSCOPICAL EXAMINATION.

The portions of the cord from which each pair of spinal nerves arise are termed the corresponding segments of the cord, and it is desirable to use this well-defined anatomical segmental structure of the spinal cord as a basis for the localization of lesions in the microscopical examination. We can then know exactly from what part of the cord a given section is taken.

The cord may be hardened with the dura mater attached, but is distorted less if the dura is removed. At intervals of little less than half an inch the cord should be traversed by transverse incisions not completely severing the cord, in order that the pia mater may hold the pieces together in their serial order. The cord is then rolled up in a loose spiral and laid in Müller's fluid on absorbent cotton. After the subsequent alcohol hardening the attachments of the nerve roots form a guide for the division of the cord into its proper segments.

To distinguish the spinal segments after they have been taken from their serial order in the hardened cord, I have found it most convenient to mark them by sticking a hot teasing needle in the white matter of one half of the upper surface of each segment. This burns a permanent little hole in the segment, and, by making notes of the different positions of the needle-holes in the different segments, a num-

ber of blocks from the cord may be carried through the celloidin imbedding process together and easily identified. If imbedded on pieces of white wood, the number of the segment or portion of the spinal segment to which the imbedded specimen belongs may be written on the wood in pencil. In the sections the needle-hole also indicates the right or left hand side of the cord. When it is necessary to examine the spinal nerve roots, these may be reflected against the corresponding segment and tied in place with thread, and then the segment with its roots is imbedded in celloidin, so that transections of the upper level of the segment will include transections of the corresponding nerve roots. The lumbar and sacral segments are so short that the entire segment may be mounted on the block of wood; but in the cervical and dorsal regions it is more convenient to use only the upper thirds or quarters of the segments for mounting.

OSMIC-ACID STAINING OF LARGE NERVE TRUNKS.

Most of the human peripheral nerve trunks are so thick that osmic acid does not penetrate into them when stretched out in the ordinary way on a bridge, as advised by Ranvier. To stain the human posterior tibial or sciatic nerves affected with peripheral neuritis, the nerve trunks are cut with sharp scissors into segments a little less than half an inch long. One extremity of the segment is held with a forceps, and with a second forceps the protruding funiculi at the other extremity are gently pulled out of their lamellar sheaths and laid in the one-per-cent. osmic-acid solution for twenty-four or forty-eight hours. This manipulation cracks the myelin sheaths transversely somewhat, but the osmic acid penetrates well among the fibers, and stains them quite uniformly. Osmic-acid nerves are kept best in glycerin.

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NEW STAINING METHODS FOR THE PERIPHERAL NERVES.

1. *Dilute Aqueous Solution of Basic Fuchsin for staining the Connective Tissue of the Peripheral Nerves in the Fresh Condition.*—A slender funiculus of a human nerve, pulled out as described above, or a bit of the sciatic of a frog or mouse (not over half an inch long), is laid upon a slide for twenty to thirty seconds, until its surface begins to stick to the glass. (If the bit of nerve is very wet, it is moved about on the slide until some of its surfaces begin to adhere to the glass.) Then, with two teasing needles, the nerve is rapidly rolled or pulled away from the adherent portion, so that it is spread out into a thin film on the glass. Before it dries—this is to be avoided by breathing on the specimen—a drop or two of an aqueous solution of basic fuchsin, such as is used for bacterial staining (one drop of a saturated alcoholic solution of basic fuchsin in eight or ten c. c. of distilled water), is placed on the specimen. The slide is inspected with the low power, and in two or three minutes the nuclei of the endoneurium will be stained. The specimen may be mounted and studied in the staining fluid, or the dye may be washed with water; the stained nerve is dried in the air on the slide and permanently mounted in balsam. In drying in the air, the endoneural cells are not perceptibly distorted. Sometimes the dye penetrates through the constrictions of Ranvier for a slight distance between the axis-cylinder and periaxial sheath. I have found this method of service in studying the endo neurium in normal nerves, in experimental nerve lesions, and in peripheral neuritis in the human nerves.

2. *Acid Fuchsin for staining Isolated Hardened Nerve Fibers.*—Nerves are hardened from three to five weeks in Müller's fluid, and then, after washing out some of the Müller's fluid in water, for a week in strong alcohol. A

slender strand of fibers, about half an inch long, is teased very finely in water on a slide. The water is then removed, and the specimen covered with a drop of a saturated aqueous solution of acid fuchsin (Grübler's) for two to five minutes. The teased fibers are washed in water, then thoroughly in two alcohols, cleared in oil of cloves, and mounted in balsam. The axis-cylinder, neurilemma, constrictions, incisures of Schmidt, neurilemma nuclei, and branching cells between the fibers have a distinct red color. In successful preparations the incisures show very distinctly. This method is so simple and efficient in demonstrating all the details of the nerve fiber, except the medullary sheath, that it is recommended for staining specimens for classes in normal histology.

The most convenient nerves to prepare for this method are the spinal nerve roots, because they can be teased more readily than the peripheral nerves, which contain more connective tissue. When nerves have been hardened for a short time (three to eight days) in Müller's fluid and subsequently in strong alcohol, or when they have been placed in ether and then hardened in alcohol, or when they have been entirely hardened in alcohol, the myelin is coagulated in the form of a network—the neuro-keratin network. This method also stains the neuro-keratin network distinctly. Fibers stained by this method may be kept for months in oil of cloves.

3. *Peroxide of Hydrogen for bleaching Peripheral Nerve Fibers stained with Osmic Acid.*—Osmic acid preserves nerve fibers more perfectly than any other reagent, but very often the medullary sheath is stained so dark that the other details are obscured. Peroxide of hydrogen removes the black color from the myelin and makes the fibers transparent, so that they may be examined with high powers. The stained fibers are teased in water, and a few drops of hy-

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drogen peroxide are flowed under the cover glass, while the specimen is being examined with high-power lenses. Sections of osmic-acid nerves may be bleached in the same way, but it is difficult to stain them afterward with the ordinary dyes. If the fibers are treated with strong alcohol after they have been bleached in the peroxide solution, the myelin coagulates in the form of a network. This method is good for studying the incisures of Schmidt and the structure and size of the axis-cylinder, which does not shrink in careful osmic-acid hardening.

ACID FUCHSIN AND PICRIC-ACID MIXTURE FOR STAINING SECTIONS OF THE PERIPHERAL NERVES AND CENTRAL NERVOUS SYSTEM.

Sections which have been properly hardened in Müller's fluid and then in alcohol are stained rather deeply with hæmatoxylin—preferably Delafield's solution—to color the nuclei. They are then washed in water, and left for three to five minutes in acid fuchsin and picric-acid mixture prepared as follows: A few drops of a saturated aqueous solution of Grübler's acid fuchsin is added to one hundred c. c. of a saturated aqueous solution of picric acid, until the mixture has a dark-garnet color. The sections are then rapidly washed in water and in two volumes of alcohol, cleared in oil of origanum, and mounted in balsam. This stain selects the ganglion cells, neuroglia, blood-vessels, and sclerotic areas, distinctly giving them a garnet color. The axis-cylinders are stained red and the myelin is stained yellow. This stain is used considerably in the laboratory in place of carmine.

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METHODS OF STAINING NERVOUS TISSUE.

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DURING the past year a number of new staining methods have been discovered which enable the histologist to differentiate distinctly various parts of the nervous system. These methods have been published in different foreign journals, having originated for the most part in German laboratories. For the benefit of those who are studying nervous histology or pathology they are brought together and reproduced here, with a few additions which experience has suggested.

I.—WEIGERT'S HÆMATOXYLIN METHOD.

This is at present the most satisfactory of all the methods of staining, and is therefore given the first place. It brings out in strong contrast, even to the naked eye, the difference between gray masses and white fibres, and under the microscope differentiates so perfectly each fibre that it stands out like a stiff black hair in the field. The gray matter is colored a brownish yellow, the nerve cells being more deeply stained than the basis substance, and their nuclei and nucleoli coming out with great clearness. The network of fine fibres in the gray matter is demonstrated satisfactorily;

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hence the method is of use in studying both the spinal cord, medulla, and brain cortex. Connective tissue does not retain the dye, and therefore in pathological specimens which contain sclerotic tissue this appears unstained and white in the dark field of normal substance. The sharp limitation visible to the naked eye makes class demonstrations of the location of system diseases easy, and under the microscope it is possible to contrast varying degrees of sclerosis, and even to count the number of axis-cylinders which have escaped destruction in the hardened area. The method is as follows: The specimen to be stained should be hardened in Müller's or Erlicki's fluid, and then cut into sections. If it has already been removed from the Müller's fluid and kept in alcohol it may be cut, but the sections must be placed in Müller's fluid for twenty-four hours before staining. In cutting the sections in either case the knife is to be wet with alcohol, and the sections are to be transferred to Müller's fluid and not to water or alcohol, as is customary. Having been placed in the Müller's fluid, and allowed to become thoroughly impregnated with it, the excess of fluid is removed by dipping the section for a moment into water, or by pouring a stream of water over it. The section must not, however, remain more than a minute in contact with water. It is then placed in the following solution of hæmatoxylin:

Hæmatoxylin (Merck's crystal.) . .	0.75
Alcohol (97%)	10.
Water	90.

This solution is prepared by boiling the hæmatoxylin in the alcohol and water until all the crystals are dissolved. It is then allowed to stand exposed to the light and air for four days before using. In placing the section in the hæmatoxylin it is well to spread it out flat upon a piece of tissue paper, as it becomes very brittle after absorbing the dye. The solution may be poured into a glass box, and the sections on their separate papers laid one upon another in it, but there should always be an excess of the dye. If the sections are large, the papers may be held apart by small bits of wood laid across their sides, in order to secure free

access of the dye to all parts of the section, else the staining will be deeper at the edges than in the middle. Very large sections should be stained singly to prevent this.

The glass box containing the sections is then covered and placed in an oven, whose temperature is kept at a point between 35° and 45° C. (95°–112° Fahr.). It remains there an hour. If the temperature rises above 45° the process is not spoiled, but the specimens are liable to swell; if it remains below 35° the sections are not clearly stained. After being heated in this way the sections become very brittle, and therefore it is a good plan to immerse the box containing them in a large basin of water after pouring off the excess of dye. They can then be floated out of the box on the paper in the water, and washed by renewing the water in the basin. The washing should be continued until no more dye can be extracted from the section. It then appears uniformly black. It is then transferred to the following solution:

Ferricyanide of potash (red prussiate) . .	2.5
Borax	2.
Water	100.

in which it remains until a portion of the hæmatoxylin is removed. The length of time differs with different sections. They may be ready for removal in half an hour. They may have to remain for twenty-four hours.

The solution of potash has the power of dissolving the dye out of the gray matter and connective tissue rapidly, but acts very slowly upon that which has been taken up by the axis-cylinder and medullary sheath. The desirable degree of staining for the gray substance is a matter of personal taste. The longer the section remains in the potash solution the lighter will be the staining. For thin sections of the cord or medulla the time required in the potash is not more than an hour. The exact stage of decolorization can be watched if the specimens lie in white porcelain dishes. When they are sufficiently decolorized the sections are to be removed and washed in water until no more of the potash solution can be extracted. They may then be dehydrated by absolute alcohol, cleared up with oil of

origanon, or cloves, or cedar, and mounted in Canada balsam. If a double stain is preferred they may be placed in a solution of alum-carmine before being dehydrated. This gives a pink color to the nerve-cells, but does not make them any more distinct than is done by the yellow potash. The only objection which can be made to this process is that the sections are rendered brittle by it, but if they are removed from solution to solution upon paper or a spatula, and not grasped at all by forceps, they need not be broken. Both the dye and the potash solution can be used a second time. It is sometimes impossible to conduct a staining process from beginning to end at one time. It may be a matter of convenience to leave the specimens indefinitely before the process is completed, They may be left in the staining mixture covered, or in the water after decolorizing with the potash, or in the alcohol used for dehydration, for any length of time. While they are in water after being dyed, and while they are in the potash solution, they should be watched. If left too long in the water they swell, if too long in the potash solution too much of the dye is removed.[1]

II.

Weigert has recently published[2] a modification of this method. He recommends that, after hardening in Müller's fluid, the specimen be placed in a saturated solution of neutral acetate of copper, which has been diluted with an equal part of water, and allowed to stand for two days in a heating box at a temperature of 40° C. From this it is removed to alcohol, in which it may be kept indefinitely. After cutting, the sections are placed directly in the hæmatoxylin solution, previously described, which is modified by the addition of a cold saturated solution of lithium carbonate in the amount of one part to one hundred of the dye. They remain in this for several hours, not being heated. Sections of spinal cord will stain in two hours; sections of brain must be left in a day. After removal they are treated

[1] Weigert's original article is to be found in the *Fortschritte der Medicin*, Bd. ii., No. 6, 1884.

[2] *Fortschritte der Medicin*, April 15, 1885.

as in the other method by the prussiate of potash. Weigert claims that the pigment produced by the union of the dye with the copper salt is more distinct and more sharply differentiated than that produced with the chromic salt. The addition of the lithium carbonate changes the color from black to dark blue. I find that sections which have been cut may be placed in the copper solution for a day and then stained by the aid of heat. If this is done, the differentiation in the prussiate-of-potash solution occurs much more rapidly, and must be carefully watched. The advantage of this modification is, however, not very great.

III.—WEIGERT'S ACID FUCHSIN METHOD.

This method, like the preceding, has for an object a sharp definition of nerve fibres. It accomplishes the object equally well with the hæmatoxylin method, but has certain disadvantages. The gray matter is less deeply stained than in the first method, and there is no contrast between the color of the gray and of the white, except one of shade, both being a magenta. The process is also a more complex one, though it has the advantage of being a rapid one.

The nerve fibres are well stained when the process succeeds, the nerve cells are slightly stained, the connective tissue is hardly colored at all. In sclerosis of the cord the method has much value, as the line of demarcation between normal and degenerated columns is distinct. In general myelitis it is not satisfactory, little differentation between the pathological products being obtained. It is evident, therefore, that nothing can be obtained by fuchsin which is not better obtained by hæmatoxylin. The method involves the preparation of the following solutions :

Sol. No. 1. A saturated watery solution of acid fuchsin (Fuchsin, S. No. 130 of the Baden anilin factory).

Sol. No. 2. A saturated absolute alcoholic solution of caustic potash, diluted with an equal part of alcohol.

Sol. No. 3. A five-per-cent. watery solution of hydrochloric acid.

The section, however hardened, is after cutting, to be first washed in water, then placed in Sol. No. 1, in which it

remains one hour or longer, at the ordinary temperature. It is then removed and washed in water till the excess of the dye is taken off. It is then placed in Sol. No. 2, for a moment only, as it must be removed as soon as a distinction appears between the gray and the white matter. The gray matter will rapidly lose its color, while the section is in this solution, and the white matter will also be decolorized if the section is left in too long. A little experience will enable one to judge of the right moment for removal. The section is then placed in water, upon which it swims about as the alcohol evaporates, giving off red clouds of coloring matter. Here, too, it must be watched, and as soon as the clouds cease to be given off, it must be dipped in a fresh dish of water, and thence transferred to Sol. No. 3. This fixes the color definitely in the course of five minutes, when it is again washed in water, and then dehydrated with alcohol, cleared up with oil of cloves, and mounted in balsam.

If the section is thick it must remain several hours in the dye. If too much color is removed by Sol. No. 2, the section is to be washed in water and then replaced in the dye. If Sol. No. 2 has not taken out a sufficient amount of the color the section may be replaced in it at any time before it has been put in the oil of cloves. It is better, however, to have a specimen too deeply stained than too pale.

The sections do not become brittle in the dye, and no particular care is needed in the handling.[1]

Both of these methods of staining may be pursued with a specimen which has been embedded in celloidin. They have, therefore, some advantage over the carmine staining which has for so long been the favorite among neurologists. That staining is somewhat interfered with by the necessary soaking of the specimen in alcohol and ether, which precedes the embedding process. As no embedding mass can equal celloidin for convenience and cleanliness, this is an important consideration. It is to be remembered that celloidin is dis-

[1] The original article of Weigert is to be found in the *Centralblatt für medicin. Wissensch.*, 1882, Nos. 40 and 42.

solved by oil of cloves. Oil of origanon may be used to clear the section which is embedded, as it does not dissolve the celloidin.

I have secured a pleasing result by combining these two methods; using first the hæmatoxylin and then the acid fuchsin. The gray matter is by this means stained red, and contrasts well with the black lines of fibres. The nerve cells become somewhat more distinct than by the yellow stain of the potash salts alone, and the network of fibres in the basis substance is very distinct. It has a little advantage, therefore, over the combination of hæmatoxylin with carmine suggested by Weigert.

IV.—SEGUIN'S MODIFICATION OF THE ANILIN BLUE METHOD.

Anilin blue-black, or nigrosene, was used as a staining color by Sankey as long ago as 1872. Its use was revived by Bevan Lewis in 1882. He found it especially advantageous as a dye for the cortex of the brain. The solution which he used was a dark, concentrated one, and after immersing the section in the dye it could not be seen, and consequently it was occasionally broken in being removed. On account of this objection Seguin tried a weak solution of a color not too deep to be translucent. He recommends a solution of one to three or even four thousand parts of water. The section is to be first soaked in water after being cut, as alcohol interferes with the staining process. It is allowed to remain in the solution of aniline blue for six to eighteen hours, when it becomes a dark blue color. It is then washed in water until a distinct difference is apparent between the gray and white matter, or until no more of the dye will wash out. The gray matter is seen to be a deep blue, and the white is a lighter shade. The specimen is then dehydrated with alcohol, cleared up by oil of cloves, and mounted in balsam.

This method stains the nerve cells a deep blue, but does not enable one to distinguish the nucleus from the cell body. The fibres are only lightly stained and do not come out with great distinctness. Connective tissue is stained deeply;

hence sclerotic tissue can be contrasted with normal tissue very well. The process is a simple one and easily carried out. The result is, however, not as satisfactory as the old carmine method.

If a section first stained in blue is then colored with acid fuchsin the appearance is very striking, as the mixture of the dyes makes the gray cells very distinct, and the fuchsin at the same time colors the fibres. Such a combination, however, involves considerable time, and the result, when attained, is in no respects superior to that reached by the simpler hæmatoxylin process.

V.—SAHLI'S DOUBLE STAIN WITH METHYL BLUE AND ACID FUCHSIN.[1]

The specimen must be hardened in Muller's fluid or in a 3–4 % solution of bichromate of potash. It is to be stuck upon a cork with gum arabic, and put in alcohol only long enough to fix the gum. Specimens preserved more than a few days in alcohol cannot be stained without being first soaked for an hour in the bichromate solution after being cut. If the specimen is embedded in celloidin it must be cut very thin, or the celloidin must be removed after cutting. After cutting the specimen the sections should not remain more than ten minutes in water. They are then placed in a concentrated watery solution of methyl blue for several hours, until deeply stained. They are then washed in water to remove superfluous dye, and placed in a saturated watery solution of acid fuchsin for five minutes. After the excess of fuchsin is removed by washing in water, the section is placed for a few seconds only in a 1 % alcoholic solution of caustic potash, and then again in water, where the differentiation takes place. The gray matter is stained red, the white matter is stained blue. Dehydration with alcohol, clearing up with oil of cedar, and mounting in balsam complete the process.

Sahli claims that when this stain does not succeed the difficulty lies rather in some defect in the hardening process than in a defect in the staining process. The fuchsin stains

[1] *Zeitschrift für Wissensch. Mikroskopie*, ii., S. 1.

the axis-cylinder, the methyl blue stains the medullary sheath. In some fibres, however, the medullary sheath is stained with fuchsin instead of with blue, and Sahli hazards the conjecture that the difference of selective action of the sheath to absorb the blue or the red dye indicates a difference of function in the fibre which the sheath surrounds.

Repeated trials of this process have convinced me that the varying colors obtained is a matter of accident, depending upon the length of time the section is left in the various fluids. If after staining with blue they are left too long in water much of the dye is dissolved out. If after staining with fuchsin they remain a few seconds too long in the potash too much of the fuchsin is dissolved out. Thus a slight variation in the process gives results which differ greatly. The conjecture of Sahli seems therefore unwarranted.

VI.—SAHLI'S METHYL BLUE STAIN.

Sahli has also proposed[1] a single stain obtained by placing sections, however hardened, for ten minutes in the following solution:

Sat. sol. of methyl blue	24 parts.
5 % sol. of borax	16 "
Water	40 "

Mix. Stand for twenty-four hours and filter.

They are then removed, washed in water to take off the excess of the dye, and dehydrated with alcohol. As the alcohol also dissolves out the dye they must not be left more than five minutes in it. They are then cleared up with oil of cedar and mounted.

This method has the advantage of being rapid and simple. It gives a clear blue color, and the fibres appear quite distinctly differentiated from the surrounding substance. The only objection is the rapid fading of the dye when exposed to light, which prevents its use for any specimen which is to be preserved.

[1] In the same Journal.

VII.—THE SAFFRANIN STAIN.

This stain was proposed about a year ago by Adamkievicz,[1] and has been used by him in the study of pathological specimens. By means of it he claims to have observed a constant change in the vesicular column of Clarke in cases of locomotor ataxia, consisting in a disappearance of the fine reticulum of fibres which surrounds the cells of that column. The advantage of the stain is its power to bring out the finest fibres in the gray matter. The process is as follows:[2]

The specimen, however hardened, is cut, and the sections washed in water. They are then dipped in distilled water, which has been acidulated by a few drops of nitric acid. Thence they are transferred to a saturated solution of saffranin, where they remain from a few minutes to several hours, according to their thickness. Then the excess of the dye is removed by washing in alcohol, and the section is dehydrated by absolute alcohol. If a double stain is desired, it may now be washed again in water which has been acidulated with acetic acid, and then put in a concentrated solution of methyl blue. After a few hours it is taken out, washed in alcohol, dehydrated, and mounted. Specimens which have been hardened in alcohol show to better advantage by this method than those which have come in contact with chromic salts. The dye is taken up by the medullary sheath, and not by the axis-cylinder. The depth of color obtained will vary with the time the section remains in the dye, and the time it is allowed to stay in the alcohol subsequently to staining.

My own results with this method have not been satisfactory. So much of the dye is removed by the alcohol of dehydration that the specimen is very pale, and it is with difficulty that the fibres are seen. They certainly do not appear as distinctly as in the acid-fuchsin method.

These are the methods which, among a large number re-

[1] *Anzeiger der K. Akad, d. Wissensch.*, iii. Abt., 1884, No. 7.

[2] For the details of this method, as well as for many useful suggestions regarding technique, I am indebted to Dr. C. F. Freeborn, Instructor of Technique at the Col. of Phys. and Surg., N. Y.

cently brought forward and tested, have seemed of sufficient value to warrant mention. Each has its peculiar advantages, as already stated. The hæmatoxylin and fuchsin methods of Weigert are of the greatest importance, and should be adopted by all who are doing pathological work here, as they have already been adopted in Germany. In staining embryonic tissue they have been of great service, and a number of discoveries of new tracts and bundles of fibres have been made by their aid. The dye in both cases is taken up by a portion of the medullary sheath, called the chromoleptic substance. Hence, in a fœtal brain, where some tracts are medullated and others non-medullated, these dyes select the medullated parts and define them clearly. In staining normal specimens they are also of service, since the various constituents of nerve tissue are differentiated distinctly. In staining sclerotic tissue their advantage has already been noticed. These methods are superior in all respects to the older carmine method, and should take its place.

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FURTHER INVESTIGATIONS AS TO THE EXISTENCE OF A CORTICAL MOTOR CENTER FOR THE HUMAN LARYNX.*

By D. BRYSON DELAVAN, M. D.,
PROFESSOR OF LARYNGOLOGY IN THE NEW YORK POLYCLINIC.

At the Eighth International Medical Congress, held in Copenhagen in 1884, the writer presented a paper in which attention was called to the possibility of localizing in the brain the cortical motor center for the human larynx.

In support of the hypothesis that such a center existed, the histories of two cases, one of which was original, were related, and through them clinical evidence was for the first time brought to bear upon the question.

The study of these cases seemed to suggest the possibility of the following conclusions:

1. That there is a cortical center of motion for the human larynx.
2. That this center is in the course of the third branch of the middle cerebral artery.
3. That it is toward the proximal end of this vessel.
4. That it is in the vicinity of the convolution of Broca.

Although by no means sufficient evidence was furnished to prove the above, the subject was one of such unusual scientific interest that, with the intention of calling forth discussion and of inciting others to its more careful investigation, it was thought worthy of presentation. In this respect at least the effort has been successful, for since the appearance of the article several others upon the same topic have been published, and results of considerable value attained.

* Read before the American Laryngological Association at its tenth annual meeting, September 19, 1888.

In the recent work of Gottstein ("Krankheiten des Kehlkopfes," Leipsic, 1888) there is given such an excellent synopsis of the literature of the subject that it appears unnecessary to reproduce it here.

In the case reported by the writer in 1884 it was impossible then to verify the diagnosis. The patient having since died and an autopsy having been made, another instance is now added to the small list of those in which a full history has been obtained, and among which that reported by Garel ("Rev. mens. de laryngologie," May, 1886) is the only one in which, corresponding with a paralysis of the larynx, a cortical lesion has been found. The complete record of the writer's case is as follows:

Case I.—Male, aged sixty, retired merchant, family history excellent, except for rheumatic diathesis. Has always been very strong and healthy, of regular habits, and strictly temperate in the use of alcoholics, but an immoderate tobacco smoker. Since middle life has suffered much from rheumatism, and has rapidly accumulated fat, but gives no history of any other diathesis. Has also suffered from naso-pharyngeal catarrh. Is right-handed. In 1876 had a slight attack, attended with vertigo, partial insensibility, and numbness, but without any distinct paralysis. A year later was again attacked, this time with well-marked hemiplegia. Although complete insensibility was at no time present during the attack, there was intense pain in the back of the head and in the nose on the left side, numbness and impairment of motion of the left arm, side, and leg, almost total inability to swallow, and, finally, a remarkable change in the quality of the voice, which, from having been full, deep, and sonorous, was reduced to a cracked, piping, and uncertain tone, which rendered its use almost impossible. Articulation was also impaired, the patient for months afterward being obliged to pronounce each syllable separately, speaking slowly and with difficulty. There was at no time or to any degree aphasia.

There was distinct ptosis, with paralysis of the left side of the face and tongue. After continuing two or three days, the general symptoms began to subside, beginning with the leg, then the arm, and, last of all, the face, and slow but steady improvement continued for many months in all the above-mentioned conditions except the voice. In this there was little apparent change for some time, but by degrees it became more readily controlled and less discordant, although the high-pitched quality has continued up to the present time, together with the loss of power and inability to force it. In 1882 a laryngoscopical examination showed the existence of complete abductor paralysis of the left vocal band, the position of which was in the median line. The larynx was remarkably easy of demonstration, and the diagnosis was afterward confirmed by Dr. Clinton Wagner. Laryngoscopic examinations made at intervals since 1882—one on February 12, 1885, in consultation with Dr. George M.

Lefferts, and the last one in January, 1888—have demonstrated no change in the position of the cord. There was, however, a slight attempt at rotation on the part of the left arytenoid, due evidently to the action of the inter-arytænoideus muscle. Efforts made to determine the electrical "reaction of degeneration" proved unsuccessful. To summarize, in a case of common left hemiplegia, in which pharyngeal and laryngeal paralysis were especially well marked, all of the symptoms disappeared in regular order, except those relating to one set of muscles—namely, the laryngeal abductors of the left side, and these continued paralyzed for a period of ten years.

Death, due to valvular lesion of the heart and pulmonary œdema, occurred April 12, 1888.

An autopsy was made, twenty-four hours after death, by my friend Dr. Frank Ferguson, of the New York Hospital. The brain was examined sixty-two hours after death by Dr. M. Allen Starr, with the following result:

Arteries.—Left vertebral artery distended to the diameter of one fourth of an inch and very tortuous, being deflected forward and outward so as to lie upon the outer side of the medulla oblongata behind the olivary body and upon the ninth, tenth, and eleventh nerves at their exit (Fig. 1).

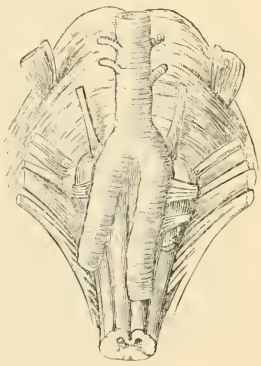


Fig. 1.

The right vertebral artery was less distended and thickened but very atheromatous, and occupied its normal position, being, however, constricted at its entrance to the basilar.

The basilar artery, also much wider than normal and intensely atheromatous, had become elongated so that it pursued a curved course, with the convexity toward the right upon the pons.

In both vertebral and basilar firm, calcified plates could be felt, producing such a rigid condition of these vessels that they could not be compressed by the fingers. At no point in their course, however, could any embolus or thrombus be discovered. The posterior cerebral arteries were also atheromatous, as were all the smaller branches of these larger trunks. The posterior communicating arteries were unusually small and apparently unaffected by the atheroma. Both carotids were equally atheromatous with the basilar, calcified plates being easily felt in their walls. The middle cerebral artery and all its branches over the island of Reil were extremely atheromatous, and this atheroma extended to the finer branches throughout the cortex, and was especially marked in some of the smaller arteries of the anterior perforated space.

The anterior cerebral artery of the right side was normal, as was the anterior communicating. On the left side it was very atheromatous.

Right Hemisphere.—The appearance of the pia mater was that of moderate congestion. It was neither œdematous nor opaque excepting at one point, about an eighth by a sixteenth of an inch in extent, over the posterior extremity of the second frontal convolution near to its junction with the ascending frontal convolution, and even here the pia was not adherent.

Left Hemisphere.—Pia congested; vessels atheromatous; no opacities; no adhesions. Cortex of the island of Reil and of the convolutions apparently normal in all respects.

Sections made in the frontal direction through the hemispheres from before backward, at intervals of one quarter of an inch, showed a normal condition of the centrum ovale, corpus callosum, and optic thalamus.

In the left hemisphere, at the summit of the internal capsule, in its anterior half and in the corresponding portion of the caudate nucleus, and extending backward, outward, and downward through the entire middle

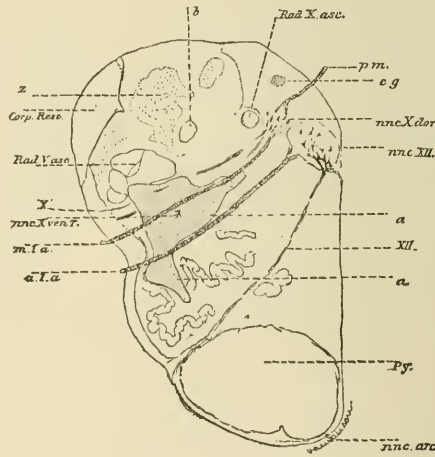


FIG. 2.—Section of the medulla near the lower level of the wedge of softening. *a*, triangular softened area; *nuc. arc.*, nucleus arciformis; *py.*, left pyramid; *xii*, hypoglossal nerve; *nuc. xii*, hypoglossal nucleus; *c. g.*, column of gelatinous substance of Rolando; *rad. x asc.*, ascending root of the vagus (respiratory fasciculus); *corp. rest.*, corpus restiforme; *rad. v asc.*, ascending trigeminus root; *x*, vagus strands; *nuc. x dor.*, sensory vagus nucleus; *nuc. x vent.*, site of the motor vagus nucleus (nucleus ambiguus) destroyed by the softening; *m. l. a.*, median lateral artery; *a. l. a.*, anterior lateral artery supplying the sensory vagus nucleus in conjunction with the posterior median artery, *p. m.*, entering the ventricular floor; * *z*, analogue of the posterior horn and posterior column nuclei.

* The vessels are represented schematically and are copied from the diagram in Ross's "Diseases of the Nervous System," vol. i, p. 761.

body of the lenticular nucleus, was a cavity lined with connective tissue and containing a small amount of clear fluid, the remains of a very old area of softening. Below and a little outside of this cavity was a small

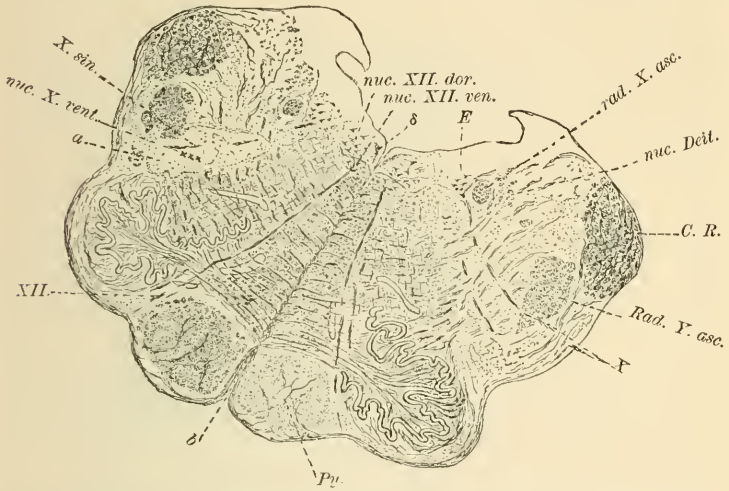


FIG. 3.—Section of the medulla near the upper level of the softened wedge. (The floor of the ventricle is from a higher level than the pyramids, and the right half of the section is higher than the left half.) δ, Decussation of the external arciform fibers at the ventral margin of the rhaphe; *py.*, partially degenerated right pyramid with some of the external arciform fibers running across it to the hilus of the olive; *x*, vagus strands; *rad. v asc.*, ascending trigeminal root; *c. r.*, corpus restiforme; *nuc. Deit.*, direct sensory cerebellar tract of Edinger (fascicular portion of Deiter's nucleus); *rad. x asc.*, ascending vagus root; *e*, common sensory nucleus of ix and x nerves; δ, vertical set of association fibers for the cells of the xii nucleus; *nuc. xii dor.*, dorsal group of cells of the xii nucleus; *nuc. xii vent.*, ventral group of cells of the xii nucleus; *x sin.*, vagus filaments of the left side passing through the softened area *a*; *nuc. x vent.*, site of the motor vagus nucleus; *xii*, hypoglossal filaments.

space with smooth walls, in which one of the basilar branches of the middle cerebral artery had lain.

In the right hemisphere, at a point in the upper portion of the internal capsule in its middle third opposite to the anterior extremity of the optic thalamus, a small area of softening was found. This involved the upper limit of the internal capsule for about a quarter of an inch from before backward, and the upper portion of the outer body of the lenticular nucleus.

Sections through the crura, pons, medulla, and spinal cord presented nothing abnormal to the naked eye.

In the right choroid plexus a fatty tumor five eighths by three eighths by two eighths of an inch was found in the descending horn of the lateral ventricle. In the right crus cerebri, in its middle third, a slight constriction seemed to indicate a descending degeneration in the motor tract.

The following report of the microscopical appearances was made by Dr. Ira Van Gieson, in the Laboratory of the College of Physicians and Surgeons, New York:

Sections of the lower termination of the two central convolutions and posterior one third of the third frontal convolution from both hemispheres show no abnormity in the number or arrangement of the nerve fibers or ganglion cells. Many of the latter are heavily pigmented. A focus of softening about 7 mm. in diameter, in the posterior arm of the right internal capsule, just behind the knee, involving slightly the optic thalamus, has produced a partial degeneration of the right motor tract.

The left side of a segment of the medulla corresponding to the upper three quarters of the olivary bodies contains a wedge shaped mass of softening (3 by 4 by 5 mm. in diameter) situated in the path of the intramedullary root strands of the vagus nerve. Near its lower level (Fig. 2) the softened mass is triangular in transection. The apex of the triangle (corresponding to the thin edge of the wedge) points toward the dorsal vagus nucleus and is 1½ mm. distant from it. The base (5 mm.) extends from the ascending root of the trigeminus to the olive and is about 1½ mm. distant from the lateral surface of the medulla. The ventral angle passes through the convolutions of the olive into its hilum. The dorsal angle involves a small portion of the ascending root of the trigeminus.

The wedge of softening gradually tapers at the expense of its base as it passes upward, so that in a section at the entrance of the uppermost vagus root bundles (Fig. 3) the ventral angle has retreated from the olive. The dorsal angle lies against the ascending trigeminal root, and the apex, pointing toward the sensory vagus nucleus, is 2 mm. from it. Thus the upper portion of the focus of softening has in section an oval outline instead of the triangular outline which it presents below.

The softened mass is not of recent origin, because it is composed largely of hyperplastic neuroglia. Many of the vagus root strands, the internal arcuate fibers, and some of the vertical fibers of the formatio reticularis, pass through the softened region undamaged. A few of the thickened anterior and median lateral arteries* running parallel to the intramedullary vagus strands also pass through the softened mass. The ventral vagus nucleus—nucleus ambiguus of Clarke—and the recurrent vagus root strands are present on the right side, but are absent on the left side.

Many of the extra-medullary root fasciculi of the left vagus are degenerated. In some places the normal nerve fibers occupy less than one half of the volume of the fascicle (Fig. 4). The right vagus root and the hypoglossal roots of both sides are normal. The spinal accessory nuclei and the first and second cervical segments of the spinal cord are normal.

The softening was probably produced by an obliterating endarteritis

* For a description of the blood-vessels of the medulla, Ross's "Diseases of the Nervous System," vol. i, p. 761, may be consulted.

of the median or anterior lateral arteries (see Fig. 2), which are branches of the diseased left vertebral. No area of secondary degeneration was found in the left ascending trigeminus root above the softened region.

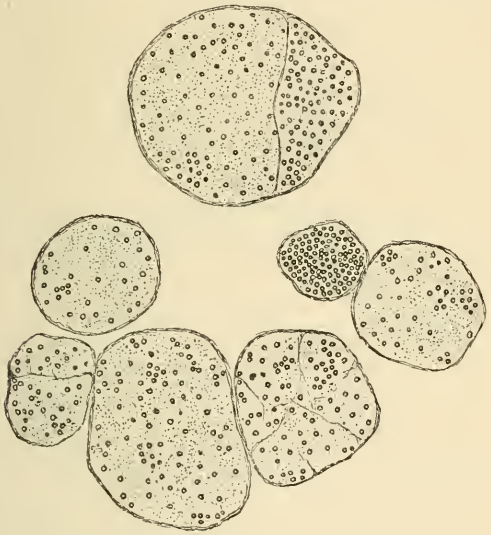


FIG. 4.—Transections of left vagus root-bundles, showing the degenerated areas.

The focus of softening in the medulla, occurring simultaneously with the lesion in the internal capsule of the opposite side, was probably more instrumental in the production of the permanent laryngeal paralysis, by the complete destruction of the ventral or motor vagus nucleus, than by interference with the root strands. This I infer from the fact that so many of the root strands pass through the old softened focus intact. The degenerated areas in the extra-medullary root bundles of the left vagus represent the paths of the motor fibers in them. The left glosso-pharyngeal filaments, although not directly involved by the lesion, seem to have been in such close proximity to the small upper pointed extremity of the softening that a temporary impairment of their functions ensued when the softening occurred. The trunk of the vagus was not preserved for examination. The results of the microscopical examination prove that the laryngeal symptoms were not due to a cortical lesion. The case is interesting in confirming clinically the motor character of the ventral vagus nucleus.

Thus it would appear that the theory maintained by Gottstein—namely, that paralysis of the larynx of central origin is due to bulbar lesion and not to disease of the cortex—receives from this case direct confirmation, and that the localization of the cortical motor center

for the human larynx still remains, so far as the above is concerned, an uncertainty.

[Since the presentation of this paper, the attention of the writer has been attracted to a case singularly parallel with the one herein described, reported by C. Eisenlohe, of Hamburg ("Archiv für Psychiatrie," Berlin, vol. xix, 1888, p. 314), in an elaborate article entitled "Zur Pathologie der centralen Kehlkopflähmungen." The patient was a laborer, aged thirty-three, in whom, attended with severe dysphagia and aphonia, there was found complete left recurrent paralysis, with loss of both motion and sensation. There was also paralysis of the left side of the velum palati. The post-mortem examination showed a condition of acute bulbar myelitis and thrombosis of the medulla, the location of which was apparently identical with that of the lesion described above by Dr. Van Giesen.]

On the other hand, collateral evidence to prove its existence continues to accumulate. Following the excellent experiments of Herman Krause upon the dog, Victor Horsley and Felix Semon, of London, have investigated the question in a series of carefully conducted experiments upon the monkey, with most interesting results. Through the kindness of Mr. Horsley the writer is permitted to publish the following *résumé* of facts as to "the representation of the vocal cord movements in the cerebral cortex":

1. "In the monkey there is a small area at the lower and anterior portion of the foot of the ascending frontal gyrus, excitation of which produces complete adduction of the vocal cords (bilateral action). We never, in this animal, observed anything but adduction.

2. "Around this area there is also represented adduction of the vocal cords, but only feebly and in association with other movements —*i. e.*, deglutition, etc.

3. "Unilateral extirpation of the whole region produced no appreciable paralysis of the glottis closers or openers.

4. "Krause's statements respecting the position of the center in the dog were confirmed by us.

5. "In the cat we found that, when the cortex was excited, adduction was observed very rarely, but abduction almost invariably."

The foregoing conclusions are extremely interesting, not only by reason of the additional proof which they give as to the actual existence of a cortical motor center for the larynx, but because of their support of, and correspondence with, the recently accepted theories of cortical motor activity for voluntary movement, namely:

1. That unilateral irritation of a given cortical center excites the corresponding bulbar center and causes bilateral movement.

2. That unilateral destruction of a given cortical center gives no result, as the influence of the opposite cortical center is sufficient to excite the corresponding bulbar center and thus to cause bilateral movement.

3. That bilateral destruction of a given cortical center causes paralysis.

These very discoveries, however, increase rather than diminish the difficulties in the way of solving the problem; for, admitting the foregoing, it is difficult to understand how any unilateral lesion of the cortex could cause a corresponding unilateral paralysis of the larynx. The hypothesis, therefore, upon which all the hitherto reported clinical observations relating to this subject have been made would seem to be wrong. In other words, paralysis of the larynx of central origin must, as a rule, depend upon bulbar lesion and not upon cortical injury.

In short, combining with the arguments of Gottstein, founded upon clinical and pathological evidence, the strong testimony of Mr. Horsley's physiological experiments, it can not be denied that, with the methods hitherto employed in the human subject, the results have been unsatisfactory, and to a certain extent misleading, and that the question is not likely to be settled until more successful means for its solution have been discovered. That in the human brain such a center does exist is made more certain with every succeeding group of experiments made upon the lower animals, and, well established in the dog and the monkey, it can be but a matter of time for it to be unquestionably located in man.

PLAN

OF THE

CEREBRO-SPINAL NERVOUS SYSTEM.

READ BEFORE THE BOSTON MEETING OF THE AMERICAN ASSOCIATION FOR THE ADVANCEMENT OF SCIENCE, AUGUST 29, 1880.

BY S. V. CLEVENGER, M. D., CHICAGO.

(*Reprinted from* THE JOURNAL OF NERVOUS AND MENTAL DISEASE, *October*, 1880.)

WE accept the motions of protoplasm as evidence of life, and yet ungrouped elementary atoms are subject to the play of physical forces which become known as modes of motion: sound, heat, light, electricity, etc., through the changes in place of atoms and molecules.

Inasmuch as sensations have for their ultimate expression motion in the living organism, cause and effect exchange places in the recognition that forces are manifest to us as sensation only in the molecular movements caused by forces. These molecular movements impress us as sensations which, of necessity, must be translated into some form or forms of motion.

Sensibility and motility, then, are sequentially convertible

terms, and we find it none the less true in the most complex than in the simplest forms of life.

There are certain fundamental considerations which should stand in axiomatic relation to all biological inquiries.

1st. Sensibility and motility are merely afferent and efferent terms to express the effects of force upon matter and matter upon force.

2d. In life a primary object of motion is for procurement of food.

3d. Growth depends upon proper nutrition (ingestion).

4th. Multiplication (as fission) proceeds from growth.

5th. Food is any material, gaseous, liquid or solid, which tends toward nutrition of the body.

6th. "Development is a process of differentiation by which the primitively similar parts of the living body become more and more unlike one another." (Von Baer.)

7th. "Higher sensory organs are special elaborations with one special function capable of response to stimuli of one special kind. They are developed from the lower kind of sensory organs, and often times still possess the essential structure of that lower kind." (Gegenbaur.)

As illustrative of undifferentiated faculties it may be mentioned that by the Gregarinæ food is taken in by endosmotic processes at the surface. Any place in the protoplasm can act as a digestive cavity by enveloping and absorbing nutritive matter.

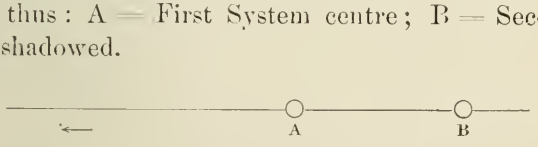
It is the simpler view, entertained by some (in opposition to the delamination precedence theory), that the form which preceded the gastrula was a one-layered vesicle which, by invagination, produced the endoderm from the ectoderm. While the ectoderm was undifferentiated, all parts of the cell were assimilative. In the gastrula stage the endoderm acquired specific ingestive faculties. Differentiation of the purely ingestive proceeds thus from the intestine, while the ectoderm remained in contact with the more variable conditions of the environment, and developed the greatest qualitative sensory and motor organs. The entire nervous organization, in its earliest condition, answers to that portion which, in vertebrata, presides over the vermicular motions of the intestines, and the

correlated respiratory and circulatory structures,—the sympathetic nervous system. This, therefore, we may entitle the First System. As soon as the enteron is created, by folding in of the ectoderm, qualitative development of this First System is restricted to such functions as are more clearly nutritive, as, when the blood vascular system is differentiated from the mesoderm, the vaso-motor nerves are derived from or added to the sympathetic, and exactly in the ratio of development of the viscera so does the First System differentiation proceed.

In high forms of invertebrata, but more pronounced in vertebrata, the viscera, and consequently the First System of nerves, occupy an inferior position, properly termed ventral, while as a broad rule the upper surface of the animal comes most in contact with varying molecular motions of the outer world. Hence, we may say that it comes to be a law, that from the dorsal to the ventral parts of the animal, ingoing impressions proceed, and, of necessity, progressive development must occur, by superimposition upon the ventral system. Then :

I. *Qualitative differentiation of the nervous organization proceeds dorsally, with a tendency toward the head end.*

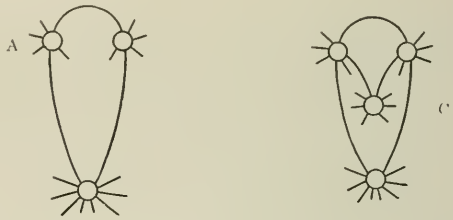
The first appearance of a Second System, equivalent to the spinal cord (segments coalesced) of vertebrata, is indicated in ganglionic enlargements upon the afferent nerves of the First System, thus : A = First System centre ; B = Second System foreshadowed.



This is apparent in the Oyster, whose anterior ganglia (A) are placed upon the fibres leading to the principal ganglion of the body. (In a typical embryonic, not phylogenetic sense, for the oyster is a degraded Lamellibranch.)

This appears to be a specialization of the tactile sense, with reference to its uses anteriorly in food discrimination and ingestion, involving ciliary prehension, and control of the valves. In Pecten further quantitative development of a Second Sys-

tem produces the pedal ganglion (C), also related to the touch sense.



The cilia of Protozoa subserve ingestive as well as locomotor purposes, and show the relationship of ingestive and general motions, and that the locomotor ability is often derived from the prehensile ingestive. In the free Rotifer this is quite apparent.

As the segments increase the sub-œsophageal ganglia multiply; the first set of ganglia become relatively ventral and preside over nutrition, while the second set, relatively dorsal, indicate progressive differentiation, as control of a pedal extremity or some special organ related externally. At the same time this dorsal ganglion is connected always with the ventral system. Fusion of these segmental ganglia with each other, or with ganglia of other systems, produce confusing appearances. This fusion of systems is most clearly seen in vertebrata.

The vibrating molecules which produce the undifferentiated impressions upon lower protozoa may be considered as causing purely tactile excitation. Just as the waves that dash the primitive animal about differ from the ripples that bring it food, only in degree, so the differences between impressions must be regarded. All sensation being related to molecular motions, and all special sense organs being derived from indifferent primaries, so we must regard it as a law:

II. *All senses are primarily tactile and differ from each other only in degree.*

Otocysts in their simplest form are connected directly with nerves, as are the pigment granules which eventually develop into eyes. Prof. Alf. M. Mayer shows that the fibres of the antennæ of the male mosquito vibrate sympathetically to the notes of the female mosquito, and that the vibrations of the

insect's antennæ may teach it the direction of sounds (thus allying this sense to the so-called "space sense" of the human labyrinth). Prof. Mayer also announced that the terminal auditory nerve-fibres vibrate half as often in a given time as the membrane of the tympanum and the ossicles.

In these instances there is a direct derivation of an auditory from the special tactile which, in turn, was evolved from the general tactile sense and does not seem to be lost even in man, as a property of sensory nerves.

A heat sense system of nerves developed from pigment terminals, by further elaboration could become ocelli and finally eyes.

A special series of nerves for heat appreciation would have necessarily *a general distribution throughout the body*, to viscera as well as to more external peripheries.

Nervous tissue appears at the same time as muscular, and affords a better path or course of less resistance for the molecular vibrations from without. The muscular is a definitely located expression of what previously belonged to all parts of the animal, contractile ability or motility for assimilative purposes.

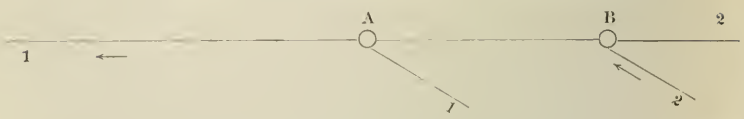
This assimilative faculty is essentially prehensile, and in the word prehension we may grasp the idea of a differentiation of such faculties as respiration, locomotion, deglutition, etc.

Carrying the comparison from Protozoa to Man, all that Man does or may hope to do has for its basis the single fundamental, though widely differentiated faculty of prehension.

Jaws and arms are prehensile, clearly. Ribs are prehensile in the sense that they assist in prehension of oxygen (food) for the lungs, morphologically and less physically in Man, while in Ophidia the ribs are locomotory prehensile, direct.

Legs are prehensile directly in quadrumana, and in Man in carrying him over ground in search of food.

As mentioned, the next step in development of the nervous system is when the ingoing general impressions become specialized and a secondary ganglion appears upon a sensory strand of the primary, which signifies that from among the general impressions some one sense, as sight, is being specialized. This is outwardly evidenced by formation of ocelli or eyes (Leech), which require a special projection.



(1 A 1—First system. 2 B 2—Second system from retina to musculus choanoides, as in Reptilia, and homologous or accompanying oculo-motor innervations in other forms. B to A becoming the motor projection to the first system from the second or inter-systemic commissural.) Here is an evidence of a sensory of the first becoming a motor nerve of the second. An afferent becoming an efferent though as between systems still afferent to the first system but efferent from the second.

By quantitative increase multiple eyes may form (Leech) and these become united into bilateral organs (pyramidal fusion in Crayfish).

The likeness between the chain of ganglia in the Leech and the spinal cord of vertebrata has led many comparative anatomists astray in homologizing. A nearly similar chain of ganglia obtains in vertebrata but situated ventrally from the vertebral column. This chain is a first system. The head ganglion, only, of the Leech, as in most invertebrata can be compared to a spinal. In Insecta and Myriapoda the superimposed secondary becomes more evident. An "unpaired system" runs in the median line between and connected with the paired or primary system, typifying the more definite appearance of the medullary grey and its commissures below or back of the head.

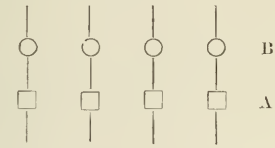
Todd and Bowman (pages 611 and 614, Vol. III.) use the following words, which indicate an early recognition of the anatomical fact without their having seen its connection or full import:

"In the Bee, the cerebral ('secondary') ganglion is very large; from its anterior portion is given off two nerves which pass forward to the base of the antennæ and have their origin well marked by a distinct ganglionic enlargement!"

Todd dwells upon the importance of recognizing this distinct ganglionary enlargement and repeats: "The sensory nerves have ganglionic enlargements in the Bee."

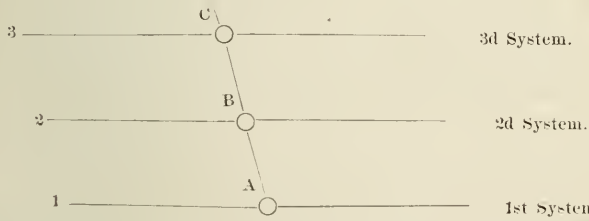
(This appearance of a third system is rare in invertebrata, though the Crab and Pterotrachea also may prove to be its possessors.)

The ganglionic swellings which on the sensory nerves of the Bee distinguished it from most Arthropoda and in fact all invertebrata, in vertebrate types from Cyclostomes upward become more markedly developed.



A—Secondary ganglionic segments. B—Third system (intervertebral) ganglia. Spitzka believes that the intervertebral ganglia and central nervous axis constitute a morphological unit, and to this I agree with the added consideration that the unification may be both embryological and phylogenetic, and still in accomplished development be as distinct physiologically as they are anatomically separate.

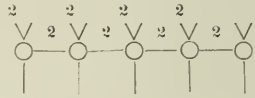
The identical procession of development of first into second and second into third systems may be seen in this diagram.



While both the first and second systems possess recognized afferent and efferent fibres, before being able to comprehend the relationships between systems or the process of projection formation we must consider whether some fundamental law does not underlie these series of relations which will better account for their creation.

The typical segment is an animal whose nerve centre lies midway between an afferent and efferent strand, thus: $\frac{1}{0}$. A series of such segments if ununited present this appearance:

These segments could be correlated by a second fibre 2, which instead of passing between peripheries as in the instance of non-union, unite the segmental ganglia by making another ganglion its motor projection, thus:



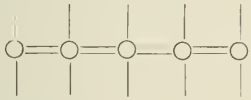
Carpenter (*Principles of Comparative Physiology*, p. 642) expresses this view: "When different organs are so far specialized as to be confined to distinct portions of the system, and each part consequently becomes possessed of a different structure and is appropriated to a separate function, this repetition of parts in the nervous system no longer exists; its individual portions assume special and distinct offices, and they are brought into much closer relationship to one another by means of commissures or connecting fibres, which form a large part of the nervous system of the higher animals It is evident that between the most simple and the most complex forms of this system there must be a number of intermediate gradations, each of them having a relation with the general form of the body, its structure and economy, and the specialization of its distinct functions. This will be found, on careful examination, to be the case; and yet, with the diversity of its parts as great as exists in the conformation of other organs, its essential character will be found to be the same throughout."

Segmental union, thus, is accomplished through efferent nerves no longer penetrating to primary organs, but passing to nerve centres of other segments, for the purpose of producing coördinated movements, and consequently to exert an inhibitory effect thereupon.

At this stage the so-called afferent commissure alone is established, but the same law of unification of segments in the construction of an individual from its component colonial members will also confer upon it an efferent commissural system.

While this is intended to represent the visceral nervous system of invertebrata, the same rule will apply in the union of vertebral ganglia segments in higher forms, beginning in such invertebrata as possess more than one secondary system ganglia (some Arthropoda).

Ganglionary fusions occur in parasitic insects and other forms, but this is secondary and does not interfere with the general application. By omitting the afferent part of the fibres that form the commissures the segmental union may be expressed thus, and confusion avoided:

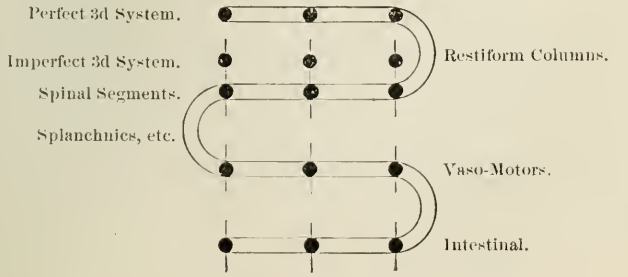


It may be stated, then, that

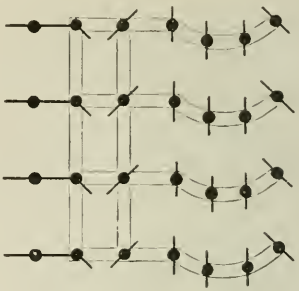
III. *Repetition of parts of a system, up to a certain point, ceases; and these parts become commissurally united before another system is perfected.*

These may be schematically expressed in diagrams which show the higher ganglionic series to be commissurally connected with the lower; each higher segment presiding over a lower system series and the commissures between forming apparently, direct projection systems.

This scheme would explain why the splanchnics have no inhibitory control over intestines (Ludwig and Hafftter), such control really pertaining to higher projections (Ott).



Each spinal ganglion segment presiding over a similar series would be thus represented:



While each and every ganglion preserves its primary projection functions, the commissures inter-relate the systems and are themselves projection fibres.

The third system in this diagram is incomplete in not being commissurally connected, hence it is but a ganglionic (intervertebral) swelling upon the secondary spinal afferent nerves, and attains its complete functional character within the cranium only.

In Trigla adriatica the brain and dorsum of the cord are marked by a double chain of well-developed tubercles on the secondary nerves just as they enter the cord. These ganglionic enlargements are exact reproductions of the cerebellar and cerebral enlargements, *en chatelaine*. These intervertebral ganglia constitute the third system, the difference between the spinal ganglia and those above being that the latter are commissurally united to one another, and in higher vertebrata fused longitudinally.

("Osseous fishes, as Dactylopterus, the small ganglionic risings upon the dorsal region of the myelon receive nerves of sensation from the free soft rays of the pectorals, and the homologous ganglia are more marked in other Gurnards [Triglæ], which have from three to five, sometimes six pairs, *e. g.*, in Trigla adriatica. Similar myelonal cervical ganglia are present also in Polynemus. Although many fishes (Bream, Dorsk) show a slight enlargement at each junction of the nerve roots with the myelon, the anatomical student will look in vain in the recent eel or lump-fish for that ganglionic

structure of the myelon which the descriptions of Cuvier might lead him to expect."—Owen, p. 271, Vol. I.)

The vermis of the cerebellum is probably a mere commissure compounded of different segmental heights, for the transverse striations are pronounced in birds and some reptiles.

According to Owen the cerebellum (vermis) retains its embryonic form of a simple commissural bridge or fold in the parasitic suctorial Cyclostomes and sturgeon, and in the almost finless Lepidosiren, while the cerebellum (still vermis, because centrally placed) is highly developed in the sharks. In the saw fish it even rests upon the "cerebrum."

The first well-marked posterior ganglionary mass which in higher vertebrata becomes a portion of the cerebellum is the vagus tubercle, placed posterior to and below the "cerebellum" of the fox shark. It might be safe to assume that subsequently this tubercle (third system ganglion) forms the flocculus or pneumogastric lobule of the cerebellum.

The Gasserian ganglion (unmistakably an intervertebral), where non-existent, must form a portion of the cerebellum.

The cerebellum then appears to be formed from fused hypertrophied intervertebral ganglia.

Stilling regarded the law of isolated conduction as inapplicable to the cerebellar lobes, owing to the very great commissural (fused) union which occurs there. Thus a coördinating function between cranial nerves on the one hand (the cerebellum acting as connected intervertebral ganglia for many cranial nerve fibres), and the general spinal system on the other, must follow in such vertebrata as are governed mainly by cerebellar supervision, while in higher forms it is brought directly into relation with the cerebrum itself.

Above this the cephalic intervertebral ganglia developed in some animals, atrophic or rudimentary in others, appears to be the posterior and anterior tubercula bigemina, epiphisis cerebri, eminentia mammillaria, olfactory lobes, cerebrum, which latter is itself composed of several lobes or ganglia, some of which, as the anterior, are undeveloped in most vertebrata and even in many mammals.

The posterior bigeminal lobe appears to be a third system ganglion related to special tactile sense (see Spitzka, *N. Y.*

Medical Record, March 13, 1880), while the optic lobes (anterior bigeminal) are third systems for the optic nerves. The primitive optic fibres were related to the grey matter above the chiasma, and even in man retain some primary thalamic connections. It is affirmed that there are cerebellar connections, but Spitzka doubts this.

The epiphisis cerebri (pineal gland), bilobed in the fœtus and devoid of sabulous matter in forms below man, attains quite a large size in some animals (Meleagris gallapavo, p. 260 *Huxley's Vertebrates*). It may with the mammillary eminence indicate a sense between sight and olfaction.

The mammillary eminences can be third systems, their positions and cinereal envelope weighing nothing against the idea, for the Teliost cerebrum itself drops to a comparably defective structure and inferior position.

These eminences are very large in monotremes, marsupials, and the horse. They stand related to the fornix, which in turn is connected to the olfactory lobe.

The olfactory lobe (another third system ganglion) appears to have been derived from a place lying in front of the mammillary eminences, according to Luys' sections, but Meynert is doubtless more correct in attaching the olfactory primitively to the optic thalamus.

The olfactory lobes, of more importance in some vertebrates than the cerebrum, in man became strangled, so to speak, by the preponderance of higher third systems.

("The olfactory lobe bore such important relations to the life history of early vertebrates that we are not surprised to find the *cerebral hemispheres* developing at first as mere appendages of the olfactory lobes."—Spitzka, "Architecture and Mechanism of the Brain," p. 37.)

The lobes of the cerebrum are related to the corpus striatum, which seems to be a part of the medullary grey second system, though formed after the hypophisis cerebri had become atrophic as the end of the spinal cord.

The hypophisis ended in the sella turcica and the corpus striatum (caudate nucleus) and subsequently lenticular nucleus developed in the scale of intelligence (Meynert).

In Teliost fishes the optic lobe (third system) occupies the

place of the cerebrum of mammals in point of mass development, and the inference is natural that this optic lobe contains the highest centres related to the psychic life of this division of vertebrates; the cerebrum proper being an undeveloped tubercle in front of the mammillary eminence with the infundibulum between them (Todd, p. 619, Vol. III).

In Amphioxus we have the culmination of the secondary ganglionic type with the foreshadowing, seemingly, of the tertiary, in the black pigmentary formation in the dorsal portion of the notochord. This vertebrate, so far from being anomalous, explains by its rudimentary organization what appears later in the Cyclostomi or above. Its second pair of nerves runs from the dorsal segmental nerves to the head end ganglion, thence to the ventral segmental nerves, typifying the medulla oblongata control over lower centres, without the intervention of a cerebellar or any other third system.

The optic ganglion (secondary) of the crab (Carcinus mænas) topographically precedes the antennal, from which may be inferred that the posterior bigeminal (tertiary) is related, as Spitzka claims, to the special tactile (fifth pair) sense.

The slight development of the superior ganglia in Brachiopoda is correlated with higher sensory organs, and Gegenbaur, p. 310, notices that the nerves for the arms are probably given off from the *ventral ganglia*, a condition which I suspect is more common than usually thought to be the case, due to the want of differentiation between alimentary and locomotor parts so far, at least, as central innervation is concerned. "In the Mollusca the visceral ganglia are not only of importance, as forming a part of the general nervous system, but they may also fuse with the cerebral ganglia, owing to the gradual shortening of their commissures. New and primitively peripherally placed parts are thereby added on to these central organs, and it becomes a matter of doubt whether or no these ganglia, which formerly belonged to the visceral nervous system should still be regarded as belonging to it."—Gegenbaur, p. 344.

The development of the nervous system appears to have proceeded as follows:

PRIMARY.

Intestinal—Circulatory and visceral, cardiac.

SECONDARY.

Respiratory—Special tactile locomotory, auditory, optic, or optic and next auditory.

Antennal special tactile from which auditory in some (olfactory not certain in invertebrata, possibly in cephalopoda. *In vertebrata originates highest secondary and tertiary).*

The progression of faculties intermingle and a branch sense appears often to develop indifferently from one or other trunk, as while respiratory may give rise to the tactile for locomotion, and audition follows upon this, the antennal for gustatory purposes may originate the auditory, while locomotor tactile may be developed separately.

NERVOUS ORGANIZATION OF INVERTEBRATA.

1. *Protozoa.*—Not perceptibly differentiated.
2. *Calenterata.*—Rudimentary primary.
3. *Vermes.*
4. *Echinodermata.*

Secondary appears and becomes highly developed. Often fused with primary.

5. *Arthropoda.*

Secondary well developed. In insecta the primary quantitatively developed.

Tertiary pronounced in bee.

6. *Brachiopoda.*—Degraded secondary.
7. *Mollusca.*

Resemble Vermes.

Secondary feeble lamellibranchiata.

Secondary well formed in gastropoda.

8. *Tunicata.*

Secondary well defined (extending by commissures dorsally (?) Copelata).

First appearance of extended secondary in invertebrata.

Anterior ganglia vesicularly developed.

Gegenbaur (p. 501) justifies this view of the central nervous system of vertebrata being homologous with the superior central ganglia of invertebrata "in an exceedingly high state of development."

The dorsal position of the central nervous system can be well made out in Tunicata. It proceeds from ectodermal differentiation.

An anterior larger mass divides into three consecutive (secondary) lobes, produced by unequal thickening of the walls of the central tube.

The anterior mass is in connection with the origin of the visual organs in Ascidiæ, Salpæ and Copelata.

A median dorsal nerve cord appears in ascidian larvæ, which prolongation Gegenbaur, p. 396, regards as noteworthy as being the only *dorsal* prolongation in invertebrata, and thus a medullary secondary central system appears stretching the length of the animal.

EMBRYOLOGICAL CONSIDERATIONS.

Notwithstanding the feeble development of the cerebral ganglia in most Mollusca the homology of these ganglia with the cerebral ganglia of Vermes and of Arthropoda has been clearly made out. There exist in Arthropoda and Mollusca cerebral (secondary) ganglia connected with nerves of special sense and visceral (primary) ganglia innervating, in Mollusca, the heart, branchial apparatus and generative organs, comparable to the "stomato-gastric nervous system" of Arthropoda.

The ventral chain of ganglia, so obvious in Crustacea and Insecta, partakes of primary or secondary characteristics, or both, depending upon the position of the metamera and the degree of development they have undergone. With concrescence of the anterior metamera into a more or less extended cephalothorax the anterior ganglionic masses are fused, as in Stomapoda, where a portion of this ventral chain innervates the anterior buccal and prehensile feet, while the six smaller ganglia of the abdomen still correspond to the segments and have more apparent primary than secondary significance. In Arachnida where nerves are given off to the enteron from both

the cerebral and ventral ganglia an appearance is presented of the vertebrate pneumogastric projection.

Recent embryological observations, as set forth by Balfour (*Comparative Embryology*, Vol. I., 1880) from monographs of Kowalevsky, Kleinenberg, Fol, Lankester and others, distinctly show that where the nervous system has been made out at all, as a rule it proceeds from epiblastic thickening and differentiation. There are many remarkable exceptions to this, however, and no particular class seems exempt from such deviations. The supra-œsophageal or cephalic ganglia arise from the head epiblast and the ventral cords from the ventral epiblast, but in Platyelminthes the cephalic originates from the prostomial mesoblast, and in Mollusca especially the epiblast does not afford the nervous origination. Fol thinks the pedal ganglión comes from the mesoblast of the foot, which Bobretzky denies. Lankester states that in Cephalopoda the various ganglia originate in mesoblastic tissue, each ganglion separately, and subsequently commissural cords unite them. The claim is made that the epiblast in Mollusca and Chætopoda always affords the supra-œsophageal.

Attention is called to the statements of Claus and Dohrn, that in Nauplius the second pair of antennæ is innervated from a sub-œsophageal ganglion.

The crustacean "mesoblast appears to be formed of cells budded off from the anterior wall of the archenteron (Astacus) or from its lateral walls generally (Palæmon). They make their first appearance soon after the invagination of the hypoblast has commenced" (Balfour, p. 427).

The ventral nerve cord of the crustacea develops as a thickening of the epiblast along the median ventral line; the differentiation of which commences in front and extends backwards. The ventral cord is at first unsegmented. The supra-œsophageal ganglia originate as thickenings of the epiblast of the procephalic lobes. The ventral cord divides by constrictions into as many ganglia as there are pairs of appendages or segments. The commissural tissue soon becomes continuous through the length of the ventral cord and is also prolonged into the supra-œsophageal ganglia. The commissural tissue also gives rise to the transverse commissures which unite the two halves of

the individual ganglia. The ganglia, usually, if not always, at first appear to correspond in number with the segments, and the smaller number so often present in the adult is due to the coalescence of originally distinct ganglia" (op. cit., p. 434).

While the epiblast, as a rule, supplies the main nervous organization and is the protective and sensory layer, and the hypoblast is essentially the digestive and secretory layer, the nerves answering to the sympathetic, as the visceral, may develop from the epiblast. But as the mesoblast is only found in fully developed conditions above Cœlenterata and originates the vascular and excretory system, it follows that a vaso-motor system can only appear subsequent to the intestinal innervation, and hence must proceed from the mesoblast as an intermediate between the secondary and primary nervous systems, though more closely related to the primary.

The junction between the two parts of the nervous system (supra-œsophageal and ventral) takes place comparatively late in Chætopoda, and in Insecta "the præ-oral portion of the nervous system consists entirely of supra-œsophageal ganglia which remain disconnected on the dorsal side till quite the close of embryonic life" (op. cit., p. 322). In Arachnida the abdominal ganglia fuse into two continuous cords united by commissures to previous ganglia, and the supra-œsophageal forms independently of ventral cords.

According to Kleinenberg (*Quarterly Journal of Microscopic Science*, Vol. XIX., 1879, "The Development of the Earthworm, Lumbricus Trapezoides"), a subsequent pair of prolongations *runs backward from the supra-œsophageal ganglia to meet the ventral cord.* This appears to represent a higher ganglionic series projecting afferent and efferent nerves, other than commissural, into a lower series as a periphery, which we shall see is evidently the case in vertebrate cerebro-spinal development.

THE FIRST SYSTEM

Arises from intestinal innervation, the ganglion of which affords in invertebrata locomotor nervous control. The respiratory, digestive, and excretory functions, as in larva of dragon

fly and fish Cobites, being performed, not only by the same sets of nerves, but the same organs (*vide* Darwin's *Origin of Species*, p. 170). We have seen locomotion to proceed as an accidental accompaniment of respiration (Branchipus), and the sub-œsophageal ganglion innervating the second pair of antennæ in Nauplius (*vide supra*).

The vaso-motor division of the first system is added when the mesoblast appears and the vascular is differentiated. The concentration of the fibres and ganglia of this system in certain areas, as solar plexus, renders any attempt at systematic classification of strands, etc., futile, but by studying the arrangement of the sympathetic system backward from the præ-vertebral ganglia, the warrant for the scheme I have adopted is more apparent. The præ-vertebral are united by longitudinal commissures, precisely as is the ventral chain of ganglia in Arthropoda; often as in the cervical region these ganglia coalesce to form larger nerve centres, precisely as in cephalo-thoracic formation from metamera, or as in the Leech; one ganglion may in the adult represent three of the embryonic segmental ganglia.

No matter how exalted the function or position pertaining to a ganglion *in any system*, it does not lose its identity as a simple centre from which afferent and efferent fibres proceed. The præ-vertebral chain presides directly as centres over the immediate vascular area with which it lies in contact, with its more or less obscure peripheral projections, while the commissural system binding it to the visceral plexuses lengthen and broaden out into such great fasciculi as the splanchnic and cardiac nerves.

SECOND SYSTEM GANGLIA.

By quantitative caudad development of the cerebral ganglia homologies of invertebrata, as supra-œsophageal, optic, auditory, pedal, or tactile, commissurally connected by afferent fibres posteriorly (columns of Goll and Burdach), and by efferent fibres anteriorly (columns of Türck and anterior fundamental tract), a view is obtained of the primitive spinal cord segments ununited. Spitzka records that the planes of junction of the original segments may be still made out by the

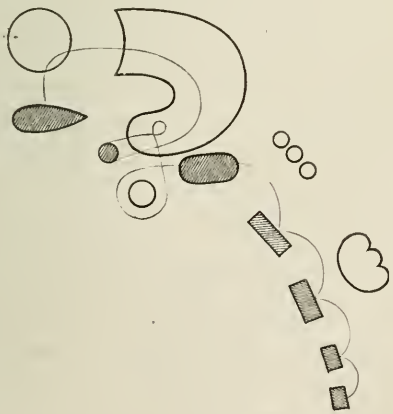
poorness in cellular elements of such areas under microscopic examination, while the centres of the spinal ganglia are determinable by their richness in these elements. Longitudinal fusion and consequent shortening of this chain forms the central tubular grey of the spinal cord.

The "medullary white" of Flechsig first appears in the columns of Burdach, in the fœtus of 25 c. m. Spitzka says it also appears in the processus cerebello ad cerebrum. This is reasonable, for the processus cerebello ad cerebrum is a continuation of the columns of Goll and Burdach, as will appear later in this description.

The second system ganglia consist of :

1. All the coalesced segments which form the spinal cord.
2. The medulla oblongata grey.
3. The grey masses in the pons Varolii.
4. The optic thalami and soft commissure.
5. The tuber cinereum.
6. The (doubtful) olfactory ganglion of Luys.
7. The caudate and lenticular nuclei of the corpora striata.

The hypophisis cerebri being the atrophied end of the cord, needs no numerical consideration.



SECOND SYSTEM COMMISSURAL AFFERENT POSTERIOR LONGITUDINAL FIBRES.

1. Spinal ganglia united by columns of Goll and Burdach.
2. Spinal and medulla oblongata by part of restiform columns.

3. Medulla oblongata segments by fibres of reticular field.

4. Medulla oblongata, grey of pons and optic thalamus by processus cerebello ad cerebrum and habenulæ to Luys' anterior centre of optic thalamus.

5. Optic thalamus to tuber cinereum.

Luys, pl. XXI., 18: "*Fibres réunnisant le centre anterieur à la substance grise du tubercule mammillaire* (*faisceau de Vicq d' Azyr*)."

6. Tuber cinereum to olfactory ganglion.

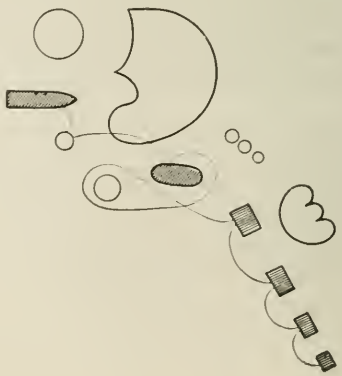
Anterior pillars of fornix, through fornix to olfactory ganglion, through "*corps godronné*."—Luys, pl. XXI., 8-20-7-16.

7. Olfactory ganglion to corpus striatum.

Luys, pl. XXI., 23: "*Substance grise du corps godronné en continuité en* 21, 21′ *avec les tractus de Lancisi, qui se perdent après avoir parcoura toute l' étendue du corps calleux, dans le sens antéro postérieur, dans la substance grise accumulée au niveau de la partie inférieure de la cloison, la où les fibres olfactives internes vieunent se distribuer en* 17 (*comparez avec pl. XV., fig.* 1, (6) *et pl. XXVIII., fig.* 2).

The longitudinal fibres of the gyrus fornicatus generally and tractus Lancisi fall in this division.

Gratiolet's surcingle addition to the caudate nucleus (see *Brain*, July, 1880) would connect Luys' olfactory ganglion with the caudate nucleus, but Meynert's proposed optic thalamus, secondary origin, for the olfactory afferent nerves, seems most reasonable.



SECOND SYSTEM COMMISSURAL EFFERENT ANTERIOR LONGITUDINAL FIBRES.

1. Corpus striatum to olfactory ganglion fibres near and in anterior commissure.—Luys, pl. XXVI. (Omission of the olfactory ganglion, and regarding an anterior portion of the optic thalamus as a secondary olfactory, simplifies the scheme and accords better with Meynert's views.)

2. Olfactory ganglion to tuber cinereum.

Luys, pl. XXVI., 11, 12, 10 : Tænia semicircularis or stria cornea (Meynert). Luys, pl. XV., 10 : From olfactory ganglion to anterior centre optic thalamus. Also, pl. II., 18, 18′.

3. Tuber cinereum to optic thalamus fibres of crus.

4. Optic thalamus to pons and medulla fibres through pons Varolii.

5. Medulla oblongata segments.

Luys, pl. XV., 14, 14′ : "*Fibres les plus internes et les plus supérieures des faisceaux spinaux antérieurs (fibres entre-croiseés).*

6. Medulla and spinal segments.

Internal anterior columns, spinal cord.

7. Spinal segments.

Columns of Türck and anterior fundamental tract.

SECOND SYSTEM TRANSVERSE COMMISSURES.

1. Transverse fibres near central canal spinal grey.
2. Fibres of the reticular field, medulla oblongata.
3. Posterior commissure of optic thalami.
4. Part of anterior commissure, corpora striata.

Transverse fusion of cord and pons grey and the soft commissure of the optic thalami are equivalent to commissural union.

SECOND SYSTEM PROJECTIONS.

We have thus far considered only the central tubular ganglia and their commissures. In passing to the enumeration of the afferent and efferent nerves, it will save repetition to include mention of the third system ganglia, which in vertebrata above Pharyngobranchii develop upon the sensory nerves near their junction with the spinal cord. The second system projections may be thus most conveniently considered with the

THIRD SYSTEM GANGLIA.

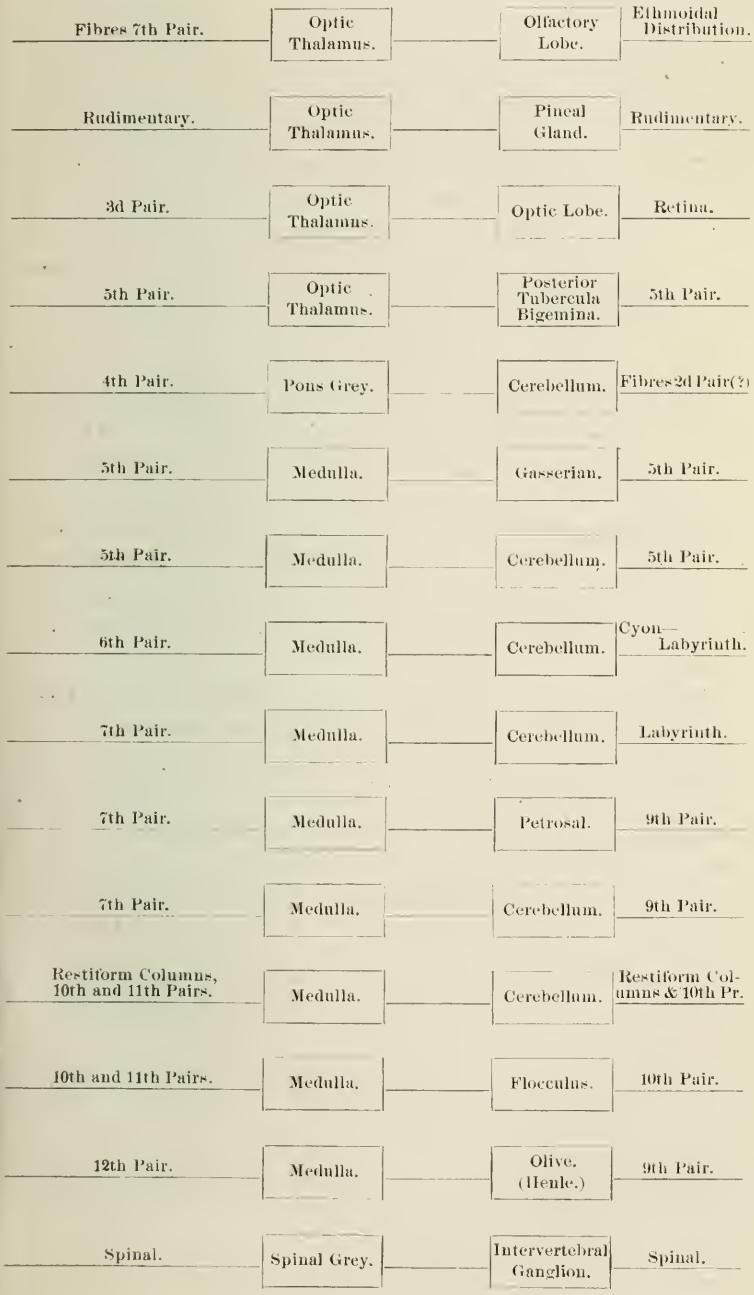
Turning again to the Amphioxus, we find that the second system ganglia, or spinal cord, give off afferent and efferent nerves dorsally and ventrally, *without intervertebral ganglia, cerebellum, or anything resembling a cerebrum.* The "second pair" of nerves of the head end, instead of passing ventrally and dorsally, as do those of the lower segments, run backward or caudally; those which run from the tail to the head along the dorsum are afferent, while those projected backward along the ventral portion of the body are efferent.

These sets of nerves resemble strikingly in many particulars the pneumogastric nerves *and the lateral columns of the spinal cord* of higher vertebrata. Confer ganglionic swellings upon all these afferent spinal nerves of the Amphioxus, proportioning their sizes to the nerve bundle sizes, and an appearance is presented like that which obtains in Trigla adriatica, a series of dorsal (intervertebral) ganglia from tail to head forming intervertebral ganglia, cerebellum, optic lobe (so-called cerebrum) and the higher series differ from the lower only in point of mass.

The crura cerebri and tegmental fibres thus become efferent and afferent nerves from the higher homologues of the central tubular grey; the corpora striata and optic thalamus, and these fibres with part of the restiform column project at different levels *from and to the spinal grey as peripheries* along the antero- and postero-lateral columns of the cord. But this does not comprise *all* of the projection series from these parts for the cerebro-spinal nerves have their primary projections as well.

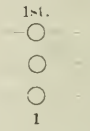
PROJECTIONS SECOND SYSTEM AND THIRD SYSTEM GANGLIA.

Efferent Nerves.	Second System Ganglia.		Third System Ganglia.	Afferent Nerves.
Crura cerebri.	Lenticular Nucleus.	Corona Radiata.	Frontal Lobe.	External & Internal Capsule.
Crura cerebri.	Caudate Nucleus.	Corona Radiata.	Occipital and Temporal Lobes.	Internal Capsule.
Rudimentary.	Tuber Cinereum.		Corpus Mammillarius	Rudimentary.

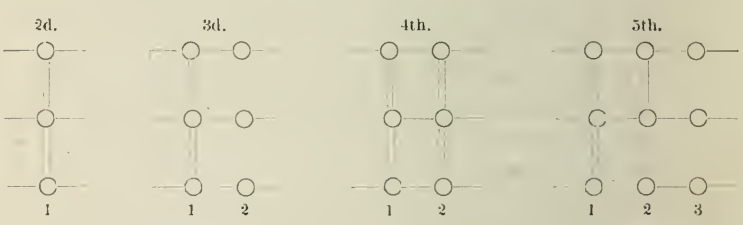

Fibres 7th Pair.
Optic Thalamus.
Olfactory Lobe.
Ethmoidal Distribution.
Rudimentary.
Optic Thalamus.
Pineal Gland.
Rudimentary.
3d Pair.
Optic Thalamus.
Optic Lobe.
Retina.
5th Pair.
Optic Thalamus.
Posterior Tubercula Bigemina.
5th Pair.
4th Pair.
Pons Grey.
Cerebellum.
Fibres 2d Pair(?)
5th Pair.
Medulla.
Gasserian.
5th Pair.
5th Pair.
Medulla.
Cerebellum.
5th Pair.
6th Pair.
Medulla.
Cerebellum.
Cyon—Labyrinth.
7th Pair.
Medulla.
Cerebellum.
Labyrinth.
7th Pair.
Medulla.
Petrosal.
9th Pair.
7th Pair.
Medulla.
Cerebellum.
9th Pair.
Restiform Columns, 10th and 11th Pairs.
Medulla.
Cerebellum.
Restiform Columns & 10th Pr.
10th and 11th Pairs.
Medulla.
Flocculus.
10th Pair.
12th Pair.
Medulla.
Olive. (Henle.)
9th Pair.
Spinal.
Spinal Grey.
Intervertebral Ganglion.
Spinal.

THIRD SYSTEM COMMISSURES.

Carpenter's law finds its highest realization in the cerebellar fusion; but in the cerebral connections it becomes masked, and only by referring to the first part of this paper can we be enabled to clearly see our way through the labyrinth. Let the first system ununited by commissures be the *first stage*, where each segment acts for itself.



The successive steps are: Second stage segments of ventral system united commissurally; third stage, second system appears, etc.

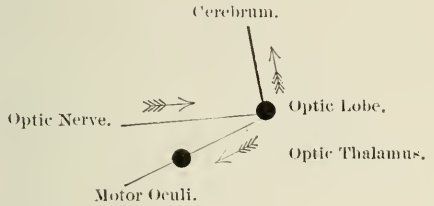


The fifth stage being the appearance of the intervertebral third system ununited commissurally.

When the cerebellum is formed, this occurs by fusion of higher third system ganglia. The sixth stage then is

Now, while the optic lobe predominates in fishes, any third system commissural strands from the cerebellum can go no higher than this optic lobe; but when other lobes, as the cerebral, develop, there must arise a system of commissural projections equivalent to those connecting the second system ganglia,

and exactly like the motor efferent projections, passing to lower third systems. These projections can be regarded as efferent nerves from the lower third systems projected into higher lobes, thus, as the corpora geniculata, external and internal:



"The internal corpus geniculata is a fasciculus of the corona radiata arising in the ganglion of the inferior corpus quadrigeminum. It develops with and is connected to the tegmentum.

"Both geniculate bodies are connected to occipital and temporal lobes."—(Meynert, pp. 409, 438.)

If these fibres pass from the optic lobe to the gyrus angularis in the cerebrum, the effects of lesions there are explained. The visual impression being afferently projected there excites the sensation of vision in a higher centre, and brings it into coördinate relations with other centres, as auditory, just below gyrus angularis (projected from cerebellum), olfactory, being also projected to cerebrum, temporal lobe. The arcuate fibres form the longitudinal series between lobes, and upon reaching the frontal region, are projected backward and downward, to exert an inhibitory effect upon posterior cerebral centres before descending through crus. The anterior lobe being the remoter from direct lower connections, paralysis does not follow injury to its extremity; while the middle frontal, being related directly to the crus, paralysis ensues quickly upon injury thereto.

As far as seem evident, the third system commissures are as follows:

AFFERENT.

1. Fibres from posterior column of spinal cord entering restiform column, passing to olivary body, thus beginning the connection between longitudinal commissural for second sys-

tem and those for the third. This transition may occur in the medulla oblongata, and form a portion of the arcuate fibres.

2. Fibres of the restiform column from the olive to the cerebellum.

3. Fibres of the lemniscus tract (which also has lateral projection fibres like the restiform, and seems analogous to it).

4. Pons fibres and tegmental fibres.

5. "External fasciculus of crus, which passes into the decussation of the pyramids into posterior column of cord."—(Meynert, p. 409.)

6. Arcuate fibres of cerebrum.

EFFERENT.

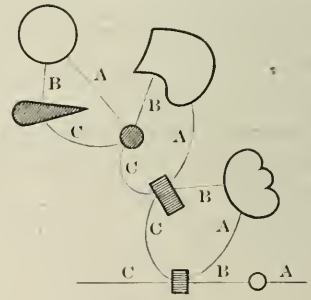
1. Arcuate fibres of cerebrum.

2. Fibres of crus and tegmentum.

3. Posterior longitudinal fasciculus.

4. Column of Türck and anterior fundamental column, as transition fibres commissural between second and third systems.

PLAN OF THE CEREBRO-SPINAL PROJECTIONS.



First Plane.

A. Sensory nerve to intervertebral ganglion.

B. Thence to spinal cord.

C. Motor nerve.

Second Plane.

A. Restiform column.

B. Brachium Pontis.

C. Antero-lateral columns of cord.

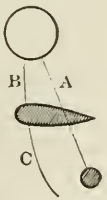
Third Plane.

A. Tegmentum.
B. Corona radiata.
C. Crus.

Fourth Plane.

A. Internal capsule.
B. Corona radiata.
C. Crus.

The fourth plane undergoes additional complication by the fibres of A (internal capsule) passing through the corpus striatum, thus:



The PLANES must be renumbered in accordance with the view that the tubercula quadrigemina, olfactory lobe, etc., are third systems:

1. There will be thirty-one segments of the first plane, consisting of all the spinal segments and their appendages below the medulla.
2. The cerebellum and medulla, olive, ganglion Gasser, etc.
3. The posterior tubercula quadrigemina and motor nuclei.
4. The optic lobes and their motor nuclei.
5. The epiphises and their motor nuclei.
6. The mammillary eminences and tuber cinereum.
7. The olfactory lobes and ganglia.
8. The occipital and temporal lobes and caudate process.
9. The præ-rolandic lobe and corpus striatum.
10. The frontal lobe proper and lenticular nucleus.

The afferent fibres A of the planes may then be classed in:

First plane: Sensory nerves to intervertebral ganglia.

Second plane: Restiform columns and cranial nerves.

Third plane: Longitudinal fibres of pons Varolii and cranial nerves.

Fourth plane: The optic nerves and fibres from basal optic ganglion and optic thalamus to optic lobes.

Fifth plane: Rudimentary.

Sixth plane: Rudimentary.

Seventh plane: Ethmoidal nerves.

Eighth plane: Corona radiata and internal capsule.

Ninth plane: Internal capsule.

Tenth plane: External and internal capsule.

B—Afferent fibres, from third to second systems:

First plane: Fibres between intervertebral ganglia and cord.

Second plane: Cranial nerves and fibres from cerebellum to medulla.

Third plane: Fifth pair and fibres from posterior tubercula bigemina to nuclei.

Fourth plane: Fibres from optic lobes to oculo-motor and lower nuclei.

Fifth plane: Rudimentary.

Sixth plane: Rudimentary.

Seventh plane: Fibres from olfactory lobe to optic thalamus (Meynert), olfactory ganglion (Luys).

Eighth plane: Corona radiata.

Ninth plane: Corona radiata.

Tenth plane: Corona radiata and fibres from frontal lobe to lenticular nucleus.

C—Efferent fibres from second systems to periphery:

First plane: All motor spinal nerves.

Second plane: All motor cranial nerves and restiform columns to the antero-lateral columns cord.

Third plane: Seventh pair fibres and antero-lateral columns spinal cord.

Fourth plane: Posterior part of tegmentum and antero-lateral columns cord (predominate in fishes).

Fifth plane: Rudimentary.

Sixth plane: Rudimentary.

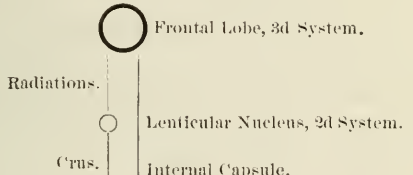
Seventh plane: Fibres from olfactory ganglion to optic thalamus, medulla, etc. (?)

Eighth plane: Tegmentum and crus to medulla and spinal cord.

Ninth plane: Crus.

Tenth plane: Fibres from lenticular nucleus to corpus striatum and crus.

Meynert (p. 416) shows the relations existing between the frontal lobe and lenticular nucleus, which, interpreted by our present scheme presents the frontal lobe as a Third System, with its internal capsular part as the afferent nerve, the radiations from the convex surface of the lenticular nucleus to the insula and frontal lobe, as the second part of the afferent system. The crus from the nucleus forms the efferent system of nerves. Thus:



Flechsig claims that the entire mass of the pyramidal fibres of pes pedunculi terminate in the præ- and post-central gyri. Spitzka says a large portion terminates thus, undoubtedly, but not the whole.

It goes to show how the upper lobes acquire a motor supremacy.

"Meynert asserts that the efferent fibres of a large portion of the nucleus lenticularis run inwards as the fourth stratum of the ansa peduncularis to become the most internal fibres of the pes pedunculi; these same fibres are supposed on very strong grounds to place the cranial nerve nuclei under the control of the higher centres. We judge the latter from the course of the raphe fibres and the greater thickness of the raphe in those regions where the hypoglossal facial and motor trigeminal are found."—Spitzka, J. N. AND M. D., Jan., 1879.

THIRD SYSTEM—TRANSVERSE COMMISSURES.

1. Vermis.

2. Transverse fibres between tubercula quadrigemina in some mammals.

3. Fusion of epiphises cerebri.

4. Transverse fibres, tuber cinereum connecting the corpora mammillaria.

5. Part of anterior commissure.

6. Corpus callosum.

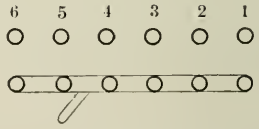
(Transverse fibres of pons Varolii intentionally omitted.)

MORPHOLOGY OF THE THIRD SYSTEM LOBES.

The position of the cerebellum and its recognizable phylogenetic changes may be easily traced through the vertebrata generally, but the lobes superior to it undergo a variety of distortions and changes of position for the solution of which we must resort to schematic views.

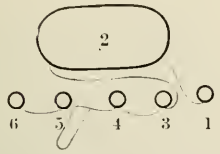
Given, a series of tubercles which shall from behind forward represent the lobes of the brain, as follows :

1. Posterior pair of tubercula quadrigemina.
2. Anterior pair of tubercula quadrigemina.
3. Epiphisis cerebri.
4. Mammillary eminence.
5. Olfactory lobe.
6. Cerebrum.

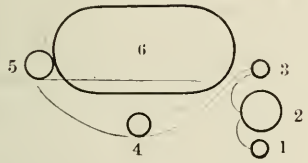


The grey secondary of each being united by commissures, the afferent and efferent. The first of these commissures it will be most convenient to follow through the developmental gyrations as apparently connecting the under surface of each lobe, but in reality connecting the secondary segments pertaining to each, as optic thalamus, tuber cinereum, olfactory ganglion and corpus striatum.

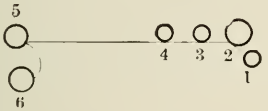
1 is connected to 2 and 3 by the upper end of the brachium conjunctivum, 3 to 4 by prolonged habenulæ, 4 to 5 by fornix, 5 to 6 by hippocampal fibres, tractus Lancisi and gyrus fornicatus (the latter principally). In the case of a fish with optic lobe (2) developed covering the other tubercles, the course of the commissures and relative mass appearance would be thus:



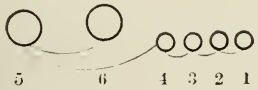
Bird, as pigeon, with cerebrum developed covering 1 to 5, the optic lobe being pressed to one side.



The following appears to be the arrangement of the brain of the fox shark, with lobes equally developed. I think the main mass must be the optic thalamus, with the quadrigeminal bodies fused on its surface (this latter feature not represented here).

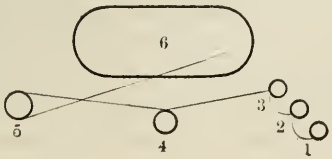


This form appears in mammal with large olfactory lobe and cerebrum.

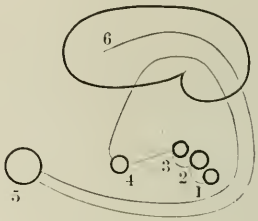


EVOLUTION OF THE AFFERENT LONGITUDINAL COMMISSURES, FISSURE OF SYLVIUS AND TEMPORAL LOBE.

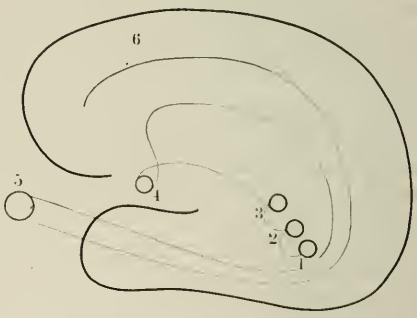
This condition is presented by an unconvoluted brain such as the beaver's, which is but faintly fissured.



An illustration of the gradual appearance of the Sylvian fissure with the hippocampal formation, is attempted below:



The last cut represents the Sylvian fissure formed as in man, with the accompanying fascicular distortions:



The growth of the frontal lobe in proportion to the intelligence of the primate individual augments this creation of temporal. Many of the longitudinal sulci of the quadrumana fold over and under with this advancement of the occipital into temporal, and the parieto-occipital fissure on the median face of the cerebral hemisphere is doubtless created directly by this bend, and the calcarine may also owe its origin to this change. A variety of causes combine, however, in fissure formation, aside from those mentioned.

OLIVE.—Schroeder van der Kolk tried to bring the olivary nucleus in relation to the hypoglossal nerves as coördinators of the tongue, and Spitzka sees new reasons why this investigation should be renewed. The latter, as do Deiters and Meynert, regards it as an internuncial station of the restiform columns and cerebellum.

This would bring the olive into the third system, and the hypoglossal being a motor nerve, in the main, its proper nucleus should not appear in

the olive, but its sensory nerves, if any, should alone do so. The olive is often correspondingly atrophied with the opposite cerebellar lobe. Deiters and Meynert claim that the olives stand in connection with the opposite cerebellar hemispheres through the restiform decussation (columns of Burdach). The parrot's olive is highly developed. "The superior olive in the trapezium is entirely different, the inferior being the olive *par excellence*, and development is opposite. When nucleus of trapezium is large the olive is small, and *vice versa*." (Spitzka.)

Optic Lobes.—In reptiles they exhibit a true cortical structure, and the cortical lamina is covered by white substance.

The outer consists of fibres entering the optic tracts; the inner of projection fibres which run parallel with contour of Sylvian Aqueduct, and in part decussate beneath its floor. This is the homologne of Forel's fountain-like tegmental decussation.

The innermost fibres of this mass pass into the oculo-motor nucleus of the same and of the opposite side, still others which fail to decussate leave their original direction and become longitudinal, constituting the post-longitudinal fasciculus, which in no animal can be traced farther than the posterior commissure.

Flechsig and Forel consider this fasciculus as a connecting band of the oculo-motor nuclei and the spinal grey substance. *It can certainly be traced with great clearness into the column of Türck, especially in lower animals.* It sends a distinct branch to the trochlearis nucleus and the abducens is entered by a similar nucleus. (Spitzka.)

In reptiles the (anterior pair of man) optic lobes are covered outside by expansion of optic nerves. Inside are layers of cells (described by Spitzka), the central tubular grey containing in inferior part the nucleus of origin of the oculo-motor nerves.

From this the post-longitudinal fasciculus continues with columns of Türck, terminating *directly and by anterior commissures in nuclei of origin of cervical nerves.*

It would then constitute a band of union between the visual impressions and the movements of the nucleus controlling the eyeball and rotation of the head and neck. It is the part by virtue of the existence of which the pigeon, deprived of its cerebrum, follows a candle light with its eyes and head.

The primitive eye vesicle is the first of the organs to appear in embryo, corresponding with its appearance in Invertebrata phylogenetically.

Spitzka accounts for the atrophy of the superficial grey in the optic lobes of man, whose visual sense is well developed, by stating that as the thalamic and cerebral projection of the retina gains in extent, that in the optic lobes diminishes, and probably no more extensive projection of the retina takes place in the human optic lobes than is necessary for the coördinating purpose. In the lower animals they are both coördinating, registering and receiving centres; higher ganglia usurp the latter function in man. This view is borne out by the fact that in the mole, whose retinal and oculo-motor innervations are almost *nil*, the superficial cortex and superficial white matter are absent and the deep grey atrophic, while the higher visual centres, as in other insectivora, are indeterminable.

"In Teliost fishes the optic lobes occupy the place of the cerebral, the latter being an undeveloped tubercle in front of the mammillary eminence, with the infundibulum passing between them." (Todd, Vol. III., p. 619.) If we are to regard the import of a centre as determinable by its preponderance in mass over its fellow centres, then we must consider the optic lobe as a centre of higher importance in the fish than in mammals, and as vicarating more or less the cerebral functions of the latter. (I am indebted for this expression of my own views to an eminent comparative anatomist. We found our differences of opinion reconciled in these words.)

POSTERIOR TUBERCULA BIGEMINA appears to be in relation to the fifth pair, and does not connect with anterior (Forel and Spitzka). Forel claims they are separate from anterior pair, though here we have an excellent example of subsequent fusion of systems where the two pairs are indeterminable and an evidence of the origin of the optic sense from the special tactile. Considering the fifth and seventh pairs as the primitive cranial nerves, amplification or refinement of tactile sense would differentiate the optic, auditory, gustatory and olfactory senses. As a rule, says Spitzka, the higher the animal range the more distinctly do the posterior tubercles become demarcated and developed. Lesion of posterior pair does not affect vision and only the anterior pair is related to retinal and oculo-motor innervations, the optic lobes alone of reptiles are analogous to anterior pair of man. The posterior pair is hypertrophic in animals with defective vision (the blind have touch sense augmented). Bats and moles rely upon touch sense.

CONVOLUTIONS in any animal usually signify that the brain is growing faster than the skull.

Something analogous to the fissure of Sylvius appears in the optic lobes of fishes, due to the same cause that produces this fissure in the cerebrum. The olfactory lobe of the cod is convoluted or crenated.

The cerebral hemispheres do not extend over cerebellum in marsupials, or any other animal having a larger brain case than brain. An overlap can occur when cerebral growth, generally anteriorly, presses the cerebrum backward, or when the small relative size of the skull compels the brain to pass backward and downward.

This relatively defective development is also accompanied with lesser development of trapezium or Pons Varolii.

Concerning the development of these centres and their connections, Spitzka ("Architecture of Brain") has thus excellently expressed it:

"We have found one striking feature in the elaboration of the projecting tracts, namely that, in higher developments, the fasciculi show a tendency to emancipate themselves from the interruptions offered by intermediate ganglionic categories; in short, that the tendency is to establish a direct communication between the cortex and the central tubular grey matter. It is the same tendency which led to the development of the longitudinal tracts of the cord, thus establishing a readier association than the circuitous route furnished by the fibrillary net-work of the grey substance.

"The course of this development is determined by two physiological laws; the first is, that, the greater the distance traversed, the longer will the

impression take to travel to its destination; the second is, that every ganglionic element to be traversed delays the transmission of the nerve current.

"Thus it is that the first ganglia preponderate over all the others as a *Brain.* And thus it is that the brain shows such complex relations in contrast with the relative uniformity found throughout the spinal cord.

"But it was not only the *ganglia* of olfaction, smell, taste, mastication, respiration and circulation that underwent such an increase in dimensions; superior as they were in functional importance to all the other ganglia, and exerting therefore a control over these others, it followed that the *associating tracts* joining the cerebral and spinal centres predominated over the associating tracts joining the spinal centres to their fellows.

"We have here a fundamental law of the cerebral mechanism: *The highest functions do not reside in any special centre, but in the functional union of several centres through associating tracts.*"

Fornix.—A concentrated projection system from the cornu ammonis grey to the superior tubercle of the optic thalamus. The fornix is drawn under the corpus callosum and forward by movements of the temporal lobe in its formation from the occipital by pressure backwards of the growing frontal lobe.

The mammillary eminences remaining stationary, the anterior pillars of the fornix fibres pull the posterior edge of the corpus callosum downward and forward. Wundt says the corpus callosum unites the fornix primitively. Spitzka has found traces of the callosal fibres in brains where they were not supposed to exist, the tendency being to create transverse union between symmetrical lobes at an early stage.

Insula.—While the hypoglossal fibres, etc., are united to the Island of Reil or its homologue, the anterior brain must guide it as it does other parts, commissurally through arcuate system. Hence the island and lenticular nucleus need not develop *pari passu.* The island is a lobe by itself, and is fused with the postero-frontal lobe, while the lenticular nucleus is the corpus striatum equivalent for the frontal region. As the frontal lobe develops it crowds the occipital backward, downward, and then forward, forming the temporal, which tends to lessen relatively in size to frontal brain preponderence, and necessarily the Sulcus Rolando is pressed farther back in the scale of intelligence, while the cranium *tends* to adapt itself to this change by the creation of a higher forehead. Changes in brain shape being readily effected as compared to skull modifications, several generations of tendencies in the latter direction may be required to render the effect of education thus visible, while the brain itself may undergo extensive alteration in the individual.

The hippocampal fornix fibres are twisted as the temporal passes forward and the upper cerebral mass weighs down upon the temporal, producing partial rotation of the latter.

Ontogenetic seldom repeats phylogenetic development, and this is why in the embryo we have the direct appearance of the Sylvian fissure without much of this folding-in process, though some of it is evident. Meynert (p. 379) says that when the elevation of the lenticular nucleus is low the fissure of Sylvius is reduced to a mere slit.

CORPUS STRIATUM.—In Echidna appears to be only the caudate process; in bats and rodents forms greatest part of hemispheres; appears large in monkeys, owing to small size of cerebrum. Appears in twelfth week in fœtus. United by small anterior commissure in Sauropsida. The lenticular nucleus preponderates in man more than the caudate nucleus, "the latter being continuous with what is in man an atrophied portion of the hemispheres, the olfactory lobes." (Meynert, p. 421.) This recognizes two points of origin for the olfactory lobe: one from the optic thalamus and the other from the caudate nucleus. This may have occurred and subsequent fusion into one lobe have taken place.

CORPUS CALLOSUM.—Absent in fishes, reptiles, birds, and monotremes; rudimentary in marsupials; broadest of all in man. All transverse commissures such as the corpus callosum can be considered only as of secondary origin and importance. I have observed a broad band of transverse fibres, resembling the corpus callosum, stretched between the tubercula quadrigemina in other animals—as the calf, in which it is quite distinct.

HIPPOCAMPUS MAJOR lies behind the corpus striatum in Echidna. Meynert calls it a defective structure, due to its passing out of use in man. Relatively larger in lower animals. Its rolled appearance is produced by rotation of temporal lobes in passing backward, downward and forward.

VASO-MOTOR CENTRES.—Budge and Waller fix the sympathetic control of the iris in the cilio-spinal centre in the lower cervical cord.

Claude Bernard places vaso-motor centres for vessels of head in the cord between sixth cervical and fourth dorsal vertebræ; genito-spinal centre, both dorsal to lower end of cord (Budge and Schiff), near which the ano-spinal fixed by M. Masius of Liege (See Jewell, Vol. I., p. 116, JOURNAL OF NERVOUS AND MENTAL DISEASE).

Luys and Jewell suppose that there is a continuous column of such centres—vaso-motor centres for whole body in medulla (Schiff, Salkowsky, Ludwig, Thiry). Tscheschichin fixes it at junction of point of medulla and pons. Brown-Sequard extends vaso-motor tract to cerebellum and brain proper. Kronecker, at Leipsic, confines it to floor of fourth ventricle (Vulpian, *Rev. Scient.*, '74, No. 35).

I regard vaso-motor control as inherent in every segment of all three systems. Primarily, the sympathetic projects the vaso-motor system; through this passes fibres to the dorsum, which causes the intestines and blood-vessels to be under the direct influence of external impressions. The secondary system interposes and thus inhibits and distributes sensations to a variety of points other than vaso-motor. So, thus, each and all would be correct in their views, and Brown-Sequard has given it the clearest expression. The propinquity of systems to the vaso-motor is in direct ratio of influence exerted upon vaso-motors.

See also Spitzka, "Architecture of Brain, Mixed System."

The forced hypnotism of animals and sleep in general may be accounted for as a withdrawal of vaso-motor cerebral control. Now, if the second and third systems be relieved from such excitation, as is usually experienced during waking hours, both these systems would, in a great measure, abandon their inhibitory control of the primary system, and the hypnotized

would be reduced to the lowest state of animation, with occasional interruptions from inner or outer stimulation, inducing dreams, restlessness, etc.

NUCLEI OF MEDULLA.—Gegenbaur (p. 520) shows that the whole vagus must be regarded as a complex of a large number of nerves which are homologous with spinal nerves. Darwin's view of the consolidation of segments to form higher series is quite compatible with the tenor of this paper as well as with the special point just referred to. "Natural selection accounts for it. In the vertebrata we see a series of internal vertebræ bearing certain processes and appendages; in the articulata we see the body divided into a series of segments bearing external appendages, and in flowering plants we see a series of successive spiral whorls of leaves. An indefinite repetition (as Owen has observed) of all low or little modified forms. *Therefore we may readily believe that the unknown progenitor of the vertebrata possessed many vertebræ*, the unknown progenitor of the articulata many segments, etc., and as the whole amount of modification will have been effected by slight successive steps, we need not wonder at discovering in such parts or organs a certain degree of fundamental resemblance retained by the strong principle of inheritance." (*Origin of Species*, p. 380.) Compare this with the fact that the Amphioxus possesses from fifty to sixty notochordal segments, and we may conceive that just as the sacrum preserves a certain resemblance to the vertebræ composing it, so the cerebellum in its laminated and lobulated appearance indicates its origin from the intervertebral ganglia which preceded it, and from which it has been formed.

There would be nothing in this to explain rhythmic respirations unless we consider the *somewhat*, not wholly, regular intervals of respiration from the standpoint of food ingestion (oxygen being a food). Most animals are compelled to eat when they can obtain food, and there can be nothing rhythmical about eating, but oxygen being always present as a rule, regular inhalations would follow and become impressed upon the nervous system in about the same manner as habitually regular three meals daily to the man whose habits in this respect are regular, become eventually a physiological necessity, and a meal missed would exert as comparable disturbance as a respiration missed. The only difference between the two as to rhythm becomes a question of time—respiration being simply at shorter intervals than food taking, and both are muscular involuntary acts from the pharynx downward. So also the vermicular pulsations of the arteries become rhythmical owing to the constant presence of an exciting cause for rhythmicality.

Nothing could be more natural than that the abducens nucleus should be primitively connected with an auditory nerve, for sounds reaching an ear from one side could thus reflexly draw the eye to that side.

The abducens, then, is part of the auditory reflex united commissurally to the oculo-motor and trochlearis by columns of Türck.

The primitive labyrinthine vesicle is second in appearing in the embryo, the eye appearing first.

(Dr. Spitzka convinced me that he has a prior claim to the auditory reflex suggestion made above, as he published his views on this point several years ago. The point I consider a good one, and I am pleased to know

that Dr. S. and I arrived at this conclusion from different points of view and in different connections.)

The auditory being derived from the pedal in Lamellibranchiata and Scaphopoda would account for respiratory and auditory nuclei in medulla oblongata. The respiratory and locomotor functions are identical in peripheral expression in Branchipus.

The antennary origin is directly in front of the optic in the Cockroach, according to Huxley, p. 357, and this either implies crossed nerve fibres or that the antennal sense of the Cockroach is different from that in Crustacea.

The fact that in the Teleostei the facial nerve enters into connection with the trigeminal, and in many sharks is fused with it, would indicate that the trigeminal is the sensory part largely of the seventh pair. Gegenbaur thinks, however, that this union was effected during their ontogenetic development, and in Urodela and above, these pairs being distinct suggests the idea that if we are to consider the trigeminal and facial as distinct the sensory part of the latter has been utterly or nearly lost in mammalia. Subsequent increase of commissural connections in the brain has given this nerve a wider range of reflex action than that merely afforded by the trigeminal, *as witness the facial muscular play under emotional or intellectual excitation.*

RÉSUMÉ.

1. *The primitive sense is tactile and all senses have proceeded from its differentiation.* For illustrative purposes let us consider energy as divided into molecular vibrations, from one ethereal pulsation in an eternity, to an infinite number of vibrations in one second. In such an undulatory series we may see, as a small division of it, all forces from sound to gravitation represented. While the protozoön may be visibly affected by every such undulation the homogeneity of its composition prevents any differential response; for instance, the tremor of a musical note, heat, light, electricity, alike produce contractions or expansions (motions) of its mass. In a higher form of life nerve tissue appears, which conveys only certain vibrations and rejects all others. Take one undulation in a second as the capacity of this nerve fibre. It is a tactile nerve. When a nerve-fibre conveys more rapid undulations differentiation begins. Sixteen to forty thousand per second begin and end the auditory vibrations. Quicker vibrations to four hundred and fifty billion per second we may view as heat appreciation, thence to eight hundred billion from red to violet light, above this fluorescent undulations, "chemical energy,"

electricity, to infinity. We may thus mathematically conceive an auditory sense derived from the general tactile or a special touch sense (like that of the fifth pair of nerves). An optic sense would arise from this same tactile, and we have seen it thus differentiated embryologically.

2. *Qualitative differentiation of the nervous organization proceeds dorsally, with a tendency toward the head end.* That portion of the animal which stands in most direct relation to the changing molecular movements of the environment develops the highest sensory and motor nerve-centres and projections.

3. *Repetition of parts of a system, up to a certain point ceases; and these parts become commissurally united before another system is perfected.*

The sympathetic nervous system, consisting of the intestinal and vascular or vaso-motor nerves, develops first. Schenck and Birdsall (*Archives of Medicine*, vol. I., No. 2) on the Embryogeny of the Sympathetic, consider this system as composed of masses originating in the central nervous system. This is a truth from one standpoint, and that a very narrow one. Blending the results of comparative embryology and anatomy, the sympathetic precedes the creation of other systems.

The second system to appear phylogenetically is the spinal, equivalent in the invertebrates to their "cerebral" ganglia.

The third system is the intervertebral, the swellings upon the posterior roots of the spinal nerves.

4. *The cerebellum is formed from fused hypertrophied intervertebral ganglia.*

Many sensory cranial nerves pass through this organ and by the fusion of these originally separate centres co-ordination occurs necessarily.

Excessive development on the one hand, or want of development on the other, places all the ganglionary tubercles and lobes of the encephalon in the third system category. Thus *the præfrontal lobe of the cerebrum, the occipital and temporal lobes, the olivary body, the olfactory lobe, the mammillary eminence, the epiphisis cerebri, the tubercula bigemina, the petrosal and Gasserian ganglia were originally intervertebral*

ganglia, and still maintain resemblance to these ganglia in many particulars.

5. *The præ-frontal lobe is the last intervertebral ganglion to develop.* It grows larger in the scale of intelligence and presses the occipital (see the brains of monotremes and marsupials) backward, downward and forward, thus forming the temporal (or what has been erroneously termed the middle) lobe.

6. The cerebro-spinal nerves, in some cases, preserve their original projections from and to muscles, but these nerves may also have not only a distribution to the viscera, as has the pneumogastric, but may also project into and from *other system-centres.* The lateral columns of the spinal cord, the tegmentum and crura cerebri in their main mass may thus be regarded as cerebro-spinal nerves of the highest series, having lower system-centres for peripheries. The præ-frontal lobes thus exert an inhibitory control over the highest centres, because such centres are peripheries for the nerves of these foremost ganglia.

THE SULCUS ROLANDO

AND INTELLIGENCE.

BY

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The Importance of the Position of the Fissure of Rolando, as an Index to the Intelligence of Animals.

By S. V. Clevenger, M. D.

IN studying the external configuration of the brain, I was struck some time since, by the variable position of the fissure of Rolando in the brains of different persons when compared together.

This fissure has acquired universal importance latterly, in the progress of research into the localization of function in the cerebral cortex. I began, accordingly, a study of the position of this fissure in different classes of animals, as well as in different specimens of the human brain, the results of which are given in the following paper.

In all animals lower than man, the sulcus Rolando or its homologue, occupies a region far in front, and as the frontal lobes are developed this fissure is thrown backwards toward the parieto-occipital part of the brain. I have noticed that in the brains of some idiots the position is similar to that observed in dogs, baboons and anthropoid apes, and the more abject has been the condition of the imbecility, the farther forward the situation of the sulcus.

The simplest method for examination is as follows: Divide the brain antero-posteriorly (on an imaginary semicircle) into ten parts from the orbital to the occipital extremities (exclusive of basilar measurements), along the upper arch of the longitudinal fissure. The summit of the sulcus Rolando ending in or near the great longitudinal fissure will be found to be six to six and one-half removes from the orbital extremity, four to three and one-half from the occipital in the average human brain. It is usually farther back on the left than on the right hemisphere.

Other methods may be suggested, but this is the least

difficult, and can be rapidly and accurately employed even during hasty autopsies.

"That the high, broad, and prominent forehead marked intellectual power, was a belief which the ancient Greeks entertained, and which has long been popularly held ; and the notion that lowness and narrowness of the forehead indicates intellectual inferiority is in harmony with the observations that in the negro, and more markedly in the Bosjesman, the anterior part of the hemispheres is narrower than in Europeans, and that the narrowing of the frontal lobes to a point is one character by which the brain of the monkey differs from that of man."*

That this was not absolutely correct has long been admitted. The skull may have a general tendency toward adaptation in shape to its contents, but in physiological as in ordinary affairs it may be incorrect to judge the value of a package by the size or appearance of the box in which it is contained. The endeavors of Gall and Spurzheim, but more particularly those of their followers, to trace cranial relationship to mental traits, have made a *reductio ad absurdum* of what is known as phrenology, and they have thus delayed the recognition of many useful points in craniology and brain studies. Many explorations have been made in the field of comparative anatomy to connect psychic peculiarities of animals with visible brain shapes. Gratiolet at one time suggested the possibility of the Sylvian fissure as an available landmark for anatomico-psychological purposes,† but failed to elucidate matters, as he seems often to have mistaken the frontal or crucial sulcus for that fissure, and was arbitrary in his estimates of the places corresponding to that fissure in brains where it did not exist. At one time, according to Pozzi,‡ it was suggested that the Rolandic sulcus would indicate the "race type" by its position, but Broca demonstrated that this was inexact.

The most surprising feature is that in all these discussions it should not have occurred to any one to seek for at least an

* Maudsley, *Physiology of the Mind*, p. 260.

† *Anatomie Compareé du Systéme Nerveux.* Leuret et Gratiolet, Atlas, pl. VII., et seq.

‡ *Dictionnaire Encyclopedique des Sciences Médicales*, tome XVII.

approximate boundary for the frontal lobe. As the brain was subjected to various distortions by cranial growth, influence of gravitation from the habitual postures of the animal, and compression by extra development of the temporal muscles in keeping with the large size of the jaw, the different shapes assumed by the anterior as well as all other parts of the brain have served to puzzle anatomists. But we now have the means of more clearly defining what should be considered frontal lobes and what posterior. The position of the "giant cells" of Betz in the cortex of the brain, as indicated by the place occupied by the crucial or Rolandic sulcus, approximately affords us the means of bounding posteriorly the frontal lobe, and if there is any truth in the generally accepted notions of the relativity of frontal lobe preponderance in the scale of intellect, we have the data necessary for fully demonstrating it.

Proportions of total brain weight to body, absence or presence of convolutions, preponderance of cerebral over other lobes, as the optic, etc., in animals, will not be discussed here, as not *directly* applicable, though having a general reference to the subject.

The foregoing is deduced from the preponderance, in lower animals, of sensory over motor cortical areas, and is based upon the proportionate development of the latter, exhibited in an ascending scale of intelligent animals. As it may not be necessary to conduct others over the route by which this conclusion was attained, I shall begin *in medias res*, and leave definitions and discussions for subsequent treatment.

The psycho-motor cells of the cortex cerebri and the nerve fibres with which they are connected, are greater relatively in mass and number than the sensory cells and their connections, in the same cortex, in proportion to the intelligence of the animal.

Assuming that the essentials of the modern doctrines of localization are established truths, and that the motor centres in the frontal and parietal lobes are capable of separation histologically from the sensory cells of the parietal, occipital and temporal lobes, it is evident that to demonstrate the proposition made, it is only necessary to separate such cells and fibres and com-

pare their areas and volumes in a series of animals. But as at present no records of such comparisons exist, I shall seek a means to approximate such measurements from the best available material at hand. The sulcus Rolando separates the posterior sensory and anterior motor regions somewhat closely, more nearly than at first sight might appear, for most of the centres back of this sulcus evoking motor phenomena by electrization, Ferrier concludes, must be sensory, since ablation of such parts does not produce motor paralysis.* The sulcus Rolando of man and the apes is physiologically comparable to the crucial sulcus of quadrupeds with convoluted brains.

At the third month of human fœtal life the hemispheres rise above the optic thalami and the fissure of Sylvius appears. Soon after movements of the fœtus in utero begin, the cerebrum divides laterally, by the formation of the sulcus Rolando, though at a very early stage of development of the cerebral vesicles there is a shallow fossa to indicate the future position of this sulcus.

In the following table, column I. indicates the estimated masses of the frontal lobes as bounded by the sulcus Rolando, 100 equaling the entire cortex; II., the distance of the summit of this sulcus back from the orbital extremity, the entire antero-posterior length to the occipital extremity equaling 100; III., the percentage of anterior lobe included by a line projected in the plane of the medulla oblongata axis; IV., the angular dimensions of the psycho-motor region measured from the basilar surface with the corpora mammillares as a centre, and gyrus paracentralis as the movable point, the posterior boundary of this gyrus being the median surface termination of the sulcus Rolando; column V., is an attempt towards a mean expression of the factors in the preceding columns by dividing the sum of the columns by the number of columns for each individual. Adding angles to numbers would be unjustifiable were this result better than an approximation. The angles, however, increase in the same direction toward 180° as do the numbers toward 100, hence there is nothing unfair in this treatment. An arbitrary set of figures is thus obtained whose only use is to illustrate the ten-

* *Functions of the Brain*, p. 166.

dency of these crude comparisons to form the series here indicated.

The greater part of the table was arranged, by me, from the excellent engravings of Leuret and Gratiolet's Atlas: *Anatomie Comparée du Système Nerveux, considéré dans ses rapports avec l'intelligence.* Paris, 1839–57. Huxley, Benedikt, Mivart and Pozzi afforded a few illustrations as indicated in the margin. In forming the means of the last column, where the information necessary to fill the four columns was lacking, I bestowed upon each individual numbers as closely characteristic of his class or species as possible, drawn from neighboring numbers or angles; as in the case of Benedikt's criminals, the two incomplete columns I supplied by conferring upon all, the figures obtained by measurements of the brain of "Charruas," his calibre seeming to resemble that of the criminals, though he was not a criminal, and had a far better brain than any of these malefactors.

FŒTI.	I.	II.	III.	IV.	V.	
14 weeks	25	45	25	40°	33.8	
4½ months	35	40	25	80°	45.0	*Dic. Encl. Sci. Médicales*,
5 months	30	30	30	75°	41.2	t. XVII., p. 381.
5½ months	35	45	45	80°	51.2	
6 months	40	45	45	80°	57.5	
6½ months	40	45	50	95°	58.8	
Age not stated	40	50	40	95°	56.2	
7 months	45	50	40	115°	61.2	
8 months	45	55	40	110°	62.2	*Op. cit.*, p. 385.
At term	50	61	45	115°	67.7	
At term	50	55	45	115°	66.2	*Op. cit.*, p. 384.
CHILDREN.						
White infant, age unk'wn	50	70	45	110°	68.8	
7 months old	50	50	45	110°	63.8	
Microcephalous, 4 yrs	25	40	30	85°	45.0	
" "	25	45	25	70°	40.0	
Marie Martel, imbecile	40	50	35	95°	55.0	
ADULTS.						
Viellard	55	65	--	----	72.5	
"L'homme adulte"	55	60	50	125°	72.2	
Charruas	50	50	45	120°	66.2	
Fieschi	55	70	35	125°	71.2	
CRIMINALS.*						
Balazo	45	51	--	----	60.2	Robber and murderer.
Madarazo	50	59	--	----	68.5	Thief. Worse than Balazo.
Kuss	45	47	--	----	64.2	Killed his son. Drunkard.
Perndinucz	45	44	--	----	63.5	" "
Sinka	60	68	--	----	73.2	Bank note forger.
Maglenov	50	53	--	----	67.0	Vengeful, mentality low.

* *Anat. Studien an Verbrecher-Gehirnen.* Benedikt. Wien, 1879.

CRIMINALS—Continued.	I.	II.	III.	IV.	V.	
Paunoviczo	50	49	--	----	66.0	Mentally weak; murd'r
Faczuna	50	58	--	----	68.2	Habitual thief.
Budimcic	45	43	--	----	63.2	Murderer; incapable of culture.
Rozsa	50	48	--	----	65.8	Robber by heredity; noted.
Pantalic	50	44	--	----	64.8	Hired murderer.
Mia	50	48	--	----	65.8	" "
QUADRUMANA.						
Chimpanzee	30	40	--	110	52.2	Mivart, "Man and Apes," p. 318.
"	30	40	--	----		
"	--	--	30	----		Huxley, "Man's Place in Nature." Frontispiece.
Orang-outang	30	30	--	115	47.5	Mivart, p. 318.
"	20	30	--	----		
"	--	--	30	----		Huxley, ibid.
Cynocephalus	25	--	40	105	46.2	
"	15	20	--	----		
Saimiri, adult	25	25	20	75	36.2	Mivart, p. 318.
QUADRUPEDS.						
Beaver	--	--	25	----	29.0	
Agouti	10	10	--	----	18.0	
Porcupine	15	15	--	----	23.0	
Paca	10	10	--	----	18.0	
Rabbit	10	10	--	----	18.0	
Dog	15	15	20	60°	27.5	
Wolf	15	15	15	60°	23.8	
Fox	20	20	20	63°	30.8	
Lion	20	20	30	60°	32.2	
Cat	20	20	30	50°	30.0	
Panther	17	25	30	65°	34.1	
Brown Bear	20	25	35	50°	32.2	
Brown Coati	20	25	35	50°	27.5	
Polecat	--	25	--	----	25.0	
Ferret	--	15	--	----	20.0	
Otter	16	20	30	40°	26.5	
Sheep	12	19	25	55°	27.7	
Ox	20	20	25	55°	30.0	
Horse	25	30	25	65°	36.2	
Stag	20	25	--	----	34.0	
Roebuck	18	25	--	----	33.0	
Boar	15	20	--	----	30.0	
Kangaroo	8	10	--	----	15.0	
Tonquin pig	10	15	20	45°	22.5	
Elephant	25	35	30	70°	40.0	
Embryo of Cow	10	10	15	10°	11.2	

I do not propose to laboriously twist the foregoing figures to suit any theoretical notions of how they should be interpreted. In the first place they are not accurate enough; then there are many things to be considered in connection with the figures, which require more consideration than the limits of this article will admit, or our present literature and knowledge justify. Assuming these results as somewhere near the truth, we would naturally be attracted by some discrepancies—the five months' fœtus in one case being less developed anterior

to the sulcus Rolando than the preceding fœtus of four and a half months. There is in this case nothing stranger than that one child may be born with more defective lungs than another. The capacity for cells and fibres may exist without their actual presence, so when estimating by measure and weight we must microscopically demonstrate the existence of the cells and their connections, or the estimate will be valueless. A rough way of correcting such measurements seemed to present itself in the angular differences with which each medulla oblongata joined the base of its brain. Invertebrates with determinable nervous cords and cephalic ganglia have all lying in the same plane, and in a general way as we ascend through the vertebrate series we find that either the spinal cord or the medulla or both tend to form less obtuse angles with the anterior base of the cerebrum. In some human brains this angle is quite 90°. The more posterior situation of the foramen magnum in lower quadrupeds and its migration forward in bimana and quadrumana must be in pursuance of a law, and it seemed most rational to refer it not alone to an attempt to equilibrate the cranial weight, for the massive jaws of the orang, chimpanzee and gorilla are not thus relatively compensated. The more upright the habitual position of the animal the greater would this straightening process proceed between spine and cranium, but the increase in mass and weight of the frontal lobe seems to determine the cerebral overlap of the cerebellum and the creation of less obliquity between the medulla axis and brain base. The much-admired brain of Fieschi, the would-be regicide, according to my hypothesis indicated great brain capacity by the posterior position of the paracentral gyrus; but the angle at which the medulla joined the brain, if we are to rely upon the excellent engraving of Leuret, implied that while the larger anterior mass existed it was not well stored with psycho-motor cells and tracts. This is the aspect these features presented to my mind. Under rigorous examination they may prove utterly worthless in this connection, but as bare facts read in this way they promise something at least to the investigator. Taking the position of the occipital outlet by itself, the little Chrysothrix would most resemble man, and this would warn us that broad classifications cannot

be made upon single anatomical facts, though this case is exceptional, the positions of the foramen magnum being most posterior in the lemurs and passing forward in the order Mycetes, Cynocephalus, Gorilla.

Gratiolet, Van der Kolk, Vrolik, Marshall, Rolleston, Mivart and Huxley record their verdicts in favor of the advanced position the orang should occupy in the scale of animals, owing to the resemblance between its brain and man's. But, if Mivart's engraving facing page 138, in his *Man and Apes*, faithfully represents the position of the sulcus Rolando, the brain of the orang according to the views here announced is certainly far behind the chimpanzee in indications of intellect as the animal itself is in sprightliness and ability to learn "tricks." Gratiolet represents the brains of the chimpanzee and orang so nearly alike as to suggest the probability of some error in the text in naming them. The mean of the measurements, by lowering Gratiolet's orang and elevating Mivart's, places the chimpanzee at the head of the apes as having the greatest area of cortical motor centres. This is a position to which the chimpanzee is entitled according to Spitzka's careful investigations, as will incidentally appear from the following, which I abstract from articles by that gentleman, on the peduncular tracts of the anthropoid apes, published in the JOURNAL OF NERVOUS AND MENTAL DISEASE:

"Meynert has shown that the relations of the peduncular tracts and basal ganglia to each other present well-marked differences in different animals, and that the superiority of the cerebral hemispheres in man is projected in a corresponding predominance of the pes pedunculi, the pons proper and the anterior pyramids."

"On examining the base of the chimpanzee's brain we are struck by the close resemblance to that of a child, especially is this resemblance marked in the peduncular tracts. The corpora mammillares are prominent and distinct from each other, being separated by a deep notch. The crura are almost cylindroid, they have a bold convex contour and are not irregularly bulged out by deeper ganglia as in lower animals. The pons is massive. The posterior outline is straight, and beneath it a coecal depression exists exactly as in the human subject.

It is noteworthy that the diminution which the medulla undergoes from before backwards is gradual and even, not sudden as in the baboon, Ateles and Cebus."

"Taking these parts as a whole, and comparing them with lower animals and man, we remark as points possessed in common by man and his anthropoid relatives the massive character of the pons, especially in its anterior third. A second feature is the bold contour of the pyramids at their point of exit from the pons. In monkeys even with as high a hemispheric development as Semnopithecus, Macacus and Ateles, the anterior half of the medulla oblongata narrows down very suddenly in joining the posterior half. The suddenness of this reduction in mass is, as Meynert has shown, most marked in lower brains, and is therefore still greater in the dog and rabbit than in the monkeys mentioned. *It is due to the presence in the anterior half, and absence in the posterior half, of a considerable amount of molecular nerve substance.*" (Italics mine.) "Comparing the base of the chimpanzee's peduncular tracts with those of a well-developed human brain, we observe that the pons is slightly less voluminous and the pyramids flatter between the olivary bodies. Particularly in its anterior half does the chimpanzee's pons resemble the same part of certain human brains—in depth especially."

"The crura cerebri of the orang are more convex and are separated by a deeper interpeduncular notch than in the other human-like apes. There is a great discrepancy in regard to the columns of Türck; none of my human specimens show less than .0369, the chimpanzee measures .0295 and .0300, the cebus monkey .0243, taking each entire area to equal 1.0000. It is noteworthy that the anterior pyramids are proportionately defective as compared with those of man. The fibres of the motor decussation occupy a much larger area in man, and as the pyramidal fibres which are still vertical in this altitude are also more voluminous, it results that with the same configuration of the grey substance the antero-posterior diameter of the human medulla is relatively greater. Taking the transverse diameter as a standard of 10, the antero-posterior is 9.2 in man, 8.8 in the anthropoid. The nucleus cuneatus is larger in the chimpanzee, while the nucleus graci-

lis is of the same dimensions and as scattered as in the latter. The further we pass from the decussation to the pons, the flatter does the chimpanzee's medulla become as contrasted with that of man; this approximation to the lower animals is, however, still more pronounced in the baboon and lower monkeys. The proportion of anterior pyramid in man is .1555 to the chimpanzee .1111. At the altitude of the middle olivary region the anterior pyramids vary from .1050 to .1750; in the same sections the chimpanzee has .0825, the cebus .0500. We have now arrived at that region, in which, according to Meynert, the proportions of the fibre masses subservient to the intelligence can be best ascertained as contrasted with the fibres and ganglia concerned in automatism. *It is well known that the pes or basis of the crura stands out more or less prominently in different individuals:* in some it is flat and broad, in others narrow and high, and in still others as broad as in the former and as high as in the latter. This third condition I have found especially associated with well convoluted hemispheres. I believe that many insanities may yet be traced to an inadequate projection of a well-developed hemisphere. If we were to represent the average development of the higher (hemispheric and cerebellar) tracts of the human being as 100, the chimpanzee would rank about 75, the baboon at 40, the cebus at 25, the dog at 7½."*

"In the rabbit the crura cerebri are small, and the pons, which is derived from them, is rudimentary; on both sides of the point where the anterior pyramids emerge, there is a band of transverse fibres known as the trapezium (Dean), which is about as deep as the pons itself. In the dog, the pons already preponderates over this trapezium, in the baboon but a small edge of the latter is visible, in the chimpanzee and the human being it is completely hidden from view. With the increase of the pes pedunculi and parallel increase of the pons proper and progressive concealment of the trapezium, the anterior pyramids gain in bulk, they become more columnar in character, and a body which has hitherto lain behind them is pushed to the outer side. This is the explanation for the presence of

* Spitzka—"Peduncular Tracts of Anthropoid Apes." This journal July, 1879.

a distinct olivary protuberance in man, and of its absence in the lower mammalia.

"We thus perceive that anatomical peculiarities apparently of the most independent and disconnected character, are, in reality, but an expression of one great harmony, and that the shape, volume, and relations of the basi-cerebral parts are but the expression of the ratio of prosencephalic preponderance. This preponderance increases to such a degree in the anthropoid apes and man that the cerebral hemispheres may well be likened to a great empire, on whose strength depends the proper subjection and prosperity of tributary states."* I have chosen the foregoing as the best differential measurements on record, most pertinent to the present subject. It is to be regretted that the friability of Dr. Spitzka's specimens prevented him from continuing his investigations above the basal ganglia. The point, however, is sufficiently demonstrated, that the connections of the psycho-motor area of the cortex even as low down as the pyramids, and with intervening ganglia interposing themselves, preponderate in a ratio agreeing with the views of this paper, and indicate that the cortical region under discussion, itself, is likewise proportionately developed.

Meynert stated that the diameter of a fasciculus depends upon the mass of grey matter with which it is connected. Spitzka modifies this, and claims that "with the increasing development of the cortex the aggregate mass of projecting associating and commissural fibres grows more rapidly than the cortex itself. A richly convoluted brain has relatively more white substance than a poorly convoluted one; the higher we ascend the thinner becomes the relative thickness of the grey substance as compared with the white lamina entering it, but the *absolute* thickness of the cortex increases, and not only this, *but it also becomes richer in cellular elements.* The outer layer of the cortex, which is very poor in nerve cells in all animals, is relatively thinner in man than in lower animals. As we ascend in the scale of intelligence this layer is displaced by the formation of cells in the layer just beneath.

* Spitzka—"Contributions to Encephalic Anatomy." This journal, July, 1878.

"The nerve cell of the cerebral cortex is a free nucleus in the menobranchus, bipolar in the amphiuma (Schmidt), has but few processes in the scaly reptiles, fewer in the rabbit than in the dog, in the dog than in the ape, and in the ape than in man. (Herbert Major states in his paper on the cortex of a cynocephalus baboon, that he could discover no other difference between the nerve pyramids of the human and simian cortex than the lesser richness in processes of the latter. We can confirm this observation for macacus and cebus; in the chimpanzee we could discover no difference, taking into account that the staining was imperfect.) The proteus, amphiuma, reptile, rabbit, dog, ape and man occupy with regard to the respective number of processes appended to the cortical cell the *same order which they occupy in the intellectual series.*"*

Betz † claims that on the surfaces of the hemispheres there are two fundamental regions which are nearly divided by the sulcus Rolando, anterior to that fissure the grey cortex is characterized by a predominance of large pyramidal cells over the globular cells. The orbital region is included in this division. Back of this furrow are all the sphenoidal and occipital lobes and the median portion to the anterior border of the quadrilateral lobule. There the granular cells preponderate and the large ones are relatively rare. "The anterior lobe," says Charcot, "may be called the department of the giant pyramidal cells or the motor cells *par excellence.* This department embraces the entire ascending frontal convolution, the superior extremity of the ascending parietal convolution, together with the paracentral lobule. *Betz has observed in the dog the same kind of cells at those points designated by Fritsch and Hitzig as motor centres, otherwise spoken of as the parts neighboring the crucial sulcus.* Interest is added by the fact that in the dog the giant pyramidal cells exist *nowhere else* but in the regions called psycho-motor." Histologically as well as physiologically the sulcus Rolando and crucial sulcus are analogous, the main point being that homologous psycho-motor centres, indicated by these giant cells, are further back in man than in lower animals.

* Spitzka.—"Architecture of the Brain." This journal, October, 1879.

† P. Betz of Kiew—*Anatomischer Nachweis zweier Gehirncentra.*

Such centres situated back of the Rolandic sulcus as elicit movements by stimulation are in most cases only apparently motor. In the case of the angular gyrus Ferrier * says, that inasmuch as its destruction causes no paralysis it must be regarded as a sensory centre from which the excitation has been conveyed forward to motor parts. The centre for movements of the eyes and head is the farthest forward, and in the animals where this centre was found at all it bordered posteriorly the region known as "inhibitory" (to be mentioned hereafter), and was situated farther forward in the descending scale of intelligence. Centres for movements of the arms and legs cluster in the vicinity of the summit of the Rolandic sulcus and paracentral gyrus at the point where the largest motor cells were found. These centres are also moved forward gradually in a descending series of animal life, and maintain a position posterior to what Leuret calls the crucial and Owen the frontal sulcus.

Dr. Herbert Major † carefully examined the minute structure of the chacma baboon's brain and noted general resemblance to the human brain, except that the large cells of the second layer of the frontal and parietal convolutions were not so frequent and the processes of these cells are less numerous than in the corresponding cells of the human brain. He believes that the more numerous and complex anastomoses have relation to the superior functional activity of man. Bevan Lewis ‡ details the results of his examination of the cortices of the human brain and those of a cat and sheep. He found the clusters of these motor cells to be more numerous in the human brain, the processes numbering from seven to fifteen to each cell, with a remarkable constancy in their occupancy of definite areas; the largest cells were found at the summit of the lobulus Rolandicus anterior. The cat's brain presented fewer cells with less numerous processes, the average number of the latter being eight, those of the sheep being still less numerous. The largest cells found in the anterior lobe of the human brain measure .126 millimetre; in the sigmoid gyrus of the cat .106; in the corresponding position in the sheep .065.

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* Op cit., p. 166.

† *Journal of Mental Science*, January, 1876.

‡ *Brain: A Journal of Neurology*, London, April, 1878.

Dr. H. D. Schmidt, of New Orleans* found the first traces of these cells to exist in the human fœtus at the third and fourth month, becoming very noticeable at the seventh month. M. Tarchanoff† at the Soc. de Biologie, June 28, 1878, stated that from his investigations and Soltmann's, the psycho-motor centres in rabbits and dogs do not appear till the tenth day, when the senses are developed. The actions of external agents are, therefore, indispensably necessary for the formation of these centres. In Guinea pigs and hedgehogs, born with full exercise of their senses, the cortices were better developed. The brain of the rabbit contains but few giant and no pyramidal cells. He found that the administration of phosphorus accelerated the development of the functions of the psycho-motor centres.

The activity of this anterior cerebral area was brought forward in a paper on Cerebral Thermometry, read by M. Paul Broca, before the Association Française pour l'avancement des Sciences, August 30, 1874.‡ Dr. Landon Carter Gray, of Brooklyn, N. Y., § devised an apparatus for making cerebral thermometric measurements, and fixes the normal temperatures of parts of the skull, from a great number of observations, as follows: The average temperature of the left frontal station is 94.36°, the right being 93.71°; left parietal station, 94.44°, right, 93.59°; left occipital, 92.66°, right, 91.94°. Dr. Gray observed a rise of temperature in the parietal motor area of from .50° to 2.50° in persons after mental exercise; in general the extreme frontal and occipital regions lowering in temperature, the left side being warmer than the right. Results varied, occasionally giving an elevation of temperature in the occipital, but there was always an increase noted in the region of the sulcus Rolando. Drs. Maragliano and Seppilli‖ reviewed the results of Broca and Gray, adding experiments of their own upon lunatics. They describe an elevation of 1° to 2° (Fahrenheit) in the motor area, with a smaller increase posterior to this during the period of agita-

* JOURNAL OF NERVOUS AND MENTAL DISEASE, July, 1877; p. 436.

† *Le Progrès Médical*, July 6. This journal, October, 1878.

‡ *Le Progrès Médical*, 1877.

§ JOURNAL OF NERVOUS AND MENTAL DISEASE, January, 1879.

‖ *Revista Sperimentale de Freniatria e di Medicina Legale.*

tion, over that noticed during the period of calm, in these cases.

There are certain craniological bearings naturally pertaining to this subject, but there are so many elements which combine to defeat any endeavor to connect cerebral and cranial prominences, that, except in a general way, I cannot see how we can be enabled to attach any importance to mere skull shape. Dr. Gustave Le Bon, at the Soc. de Méd. Pratiques,* gave the results of his cranial measurements, and demonstrated that there was no asymmetry of right and left sides. It is probable his measurements were confined to the outer surfaces. In cutting open the skull of a mongrel dog, the deep indentations in the bones made by the convolutions beneath, and the great thickness of the cranium suggested to my mind the idea that doubtless a relatively large brain may be imparted to offspring by one parent, and a thicker skull may be derived from the larger boned or less intelligent parent; so that through life the unfortunate individual thus endowed will possess a brain which in its growth is cramped and retarded by its osseous envelope, having actually to erode and absorb the bony tissue to make room for brain development. The dog mentioned was quite intelligent, but suffered from epileptic attacks, during one of which it was shot. The meninges were quite adherent to the skull, and no other than the defects mentioned existing, color was lent to the supposition that this terrier and mastiff mongrel had suffered from incongruity between brain and brain case. This blemish may readily occur in mulattoes or other progeny of mismated races or individuals. Herodotus† notes the thickness of the skulls of the Egyptians, and attributes it to the action of the sun upon their uncovered heads. Sœmmering‡ says the skull of a centenarian is two-fifths lighter than in middle age. This is, however, in keeping with the lessened weights of all bones in the aged. There is, nevertheless, a remarkable variation in skull thickness between crania generally, that has never

* *La France Médicale*, No. 28, 1878.—JOURNAL OF NERVOUS AND MENTAL DISEASE, Oct., 1879.

† *Thalia*, XII., quoted by Holden, *Osteology*, p. 110.

‡ Holden, loc. cit.

been satisfactorily accounted for. Schroeder Van der Kolk agrees with Gall that the skull thickness is modified by brain growth.* The skull of Asseline (the recently deceased member of the Mutual Autopsy Society of Paris) was so thin in places as to be translucent, according to the published accounts.

Ferrier† claims that "the reaction between the sensory and motor centres involves consciousness, and that in general the lower down the animal as regards the perfection and complexity of the nervous system, the less marked are the distinctions of cortical areas, and the more capable are the subordinate centres of performing the offices apparently devolving on the cortex in the higher animals."

Dr. W. H. Broadbent, of London, in a communication to the International Medical Congress, at Geneva, Switzerland, Sept. 9–15,‡ concludes that: "The cortical motor centres are the points of departure for the descending voluntary impulses, the starting places for ideo-motor actions. The cells of the motor zone (which resemble those of the anterior horns of the cord) are the apparatuses by which the dictates of the intelligence are formulated for expression or for transmission outwards."

Aside from diagnoses and autopsies, the only person on record who has ever made a practical use of the connection between cortical and peripheral apparatus, to educate the centres through their peripheral connections, was Dr. Edward Seguin, of New York, the author of *Idiocy and its Treatment.* In an article entitled the "Psycho-physiological Training of an Idiot Hand,"§ Dr. Seguin describes the satisfactory progress he made toward developing the mentality of an idiot child through a skillful course of manual instruction. The child was taught gradually to use the hitherto "lifeless" hands. The automatic centres were first exercised in this way, and eventually the child was induced to exert volition,

* *Functions of the Brain*, p. 294.

† "Atrophy of left hemisphere of the brain, with co-existent atrophy of right side of the body."

‡ *Gaz. des Hopitaux*, No. 30, 1877.—JOURNAL OF NERVOUS AND MENTAL DISEASE, Jan., 1878.

§ *Archives of Medicine*, October, 1879.

by instruction in certain motions to be repeated at the command of the trainer. In this way the highest centres were reached, and the Doctor experienced all the results he could have hoped for. The most astonishing part of this transaction is this passage in the monograph at its conclusion: "If the idiot whose case is represented to you has improved under the care of his good teacher, if hundreds of others improve in public institutions (under the care of women whose names are never pronounced with sufficient respect), *the sovereignty of the brain is at an end*, and the physiological doctrine of *decentralization* contains in germ a new doctrine and new methods of education." It would thus appear that while the Doctor was practically making use of a physiological fact, he was actually deducing from his methods and results most erroneous "decentralization" ideas. What was really accomplished in this case, was the exercise of hitherto dormant psycho-motor cells.

While the region in the vicinity of the sulcus Rolando, and more especially the portion immediately in front of it in the brains of the primates, is devoted to what may be strictly termed psycho-motor areas, Ferrier* demonstrated that the orbital extremity of the frontal lobes possessed characteristics not to be overlooked. He found that by cutting off these extremities, no paralysis ensued, but that the animals thus operated on, "while not actually deprived of intelligence, *had lost, to all appearance, the faculty of attentive and intelligent observation.*" This part of the brain Ferrier designates as the "inhibitory." Simple inspection of the relative thicknesses of these corresponding portions in the ape and man show that the latter has a vastly superior development of this part. The hollow appearance of the basilar part of the orbital extremity of the monkey's brain, and the flattened and higher appearance of the supra-orbital cerebral division obtaining in man, are to be seen at a glance. Necessarily the diminution of this space would occur *pari passu* with the anterior advancement of the psycho-motor centres in a descending scale of animal intelligence. Cruveilhier associates idiocy with defective development of the frontal lobes; and the follow-

* Ferrier, *Localization of Function in Diseases of the Brain.*

ing authors are cited by Ferrier* as recording instances where accidents or disease had destroyed the integrity of the anterior portion of the brain, with consequent mental deficiencies supervening: Harlow, Bouillaud, Trousseau, Selwyn, Pitres, Morrin, Padeau, Tavignot, Fayrer, Marot, Renault, Petit, Charcot, Andral, Bergeron, Hertz, Reed, Begbie, Cholmeley, Evans, Hewitt, Lepine, Bouilly, Baraduc, Davidson, and others. Treves cited one case of the kind where even bilateral destruction of the anterior frontal region caused neither motor nor sensory disturbance.

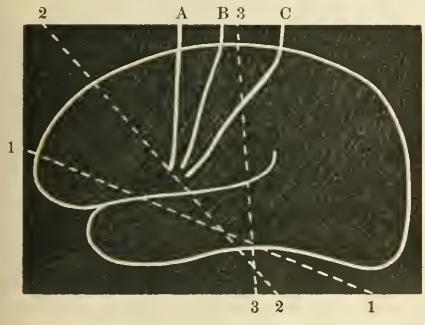
Carpenter,† in discussing the functions of the nervous system in general, says, that "when different organs are so far specialized as to be confined to distinct portions of the system, and each part, consequently, becomes possessed of a different structure, and is appropriated to a separate function, this repetition of parts in the nervous system no longer exists; its individual portions assume special and distinct offices; and they are brought into much closer relation to one another by means of the *commissures* or connecting fibres, which form a large part of the nervous system in the higher animals. It is evident that between the most simple and the most complex forms of this system there must be a number of intermediate gradations, each of them having a relation with the general form of the body, its structure and economy, and the specialization of its distinct functions. This will be found, on careful examination, to be the case; and yet, with a diversity of its parts as great as exists in the conformation of other organs, its essential character will be found to be the same throughout." This extreme anterior portion of the brain seems to be essentially for the inter-relation of the psycho-motor cells, and, while uniting them commissurally, is diminished necessarily in the same ratio of diminution of the cortical motor areas downward toward the invertebrates.

The few accompanying illustrative figures are adapted from Ferrier, Pozzi, Mivart, Benedikt, Gratiolet and Leuret. A much more intimate acquaintance with the habits of animals

* Ibid.

† *Principles of Comparative Physiology*, p. 642.

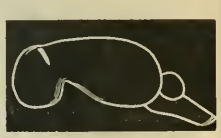
than at present exists, would be needed for a critical comparison between brain features and intelligence; and even upon the order of precedence of domesticated creatures, there are a variety of opinions. It seems to me that in estimating animal peculiarities we do not sufficiently consider that there are gradations of reasoning powers between individuals of each kind as well as between different men. In the different breeds of dogs will this Rolandic position vary especially.



This figure indicates by the position of the Rolandic sulcus A, low mental types. It is situated a little farther forward in the baboon, an animal which Mivart mentions as "low, brutal and degraded." It is also about the position of the sulcus at the sixth month of fœtal life and in idiots. B marks its place in the infant at birth and in imbecility, as represented by Pozzi's drawing of the brain of Marie Martel. The orang-outang's sulcus Rolando is slightly posterior to this, but the frontal lobe is quite pointed and less massive than that of human beings. This location for the sulcus also resembles that represented by Benedikt in the brain of Budimcic, the murderer, who was "incapable of culture." An attempt is made at C to locate the sulcus as usually placed in man. The bank note forger Sinka, and Fieschi, owned sulci occupying places situated more posteriorly.

The dotted lines projected through the medulla axis, mark boundaries for frontal areas. 1 is peculiar to human fœti under six months, microcephales and to lower animals. Even the chrysothrix, whose foramen magnum is situated centrally in the skull base, has, as may be seen in another cut, this obliquity of cerebro-medullary junction. 2 is characteristic of the fœtus after six months, and nearly represents that feature in Fieschi. 3 indicates the centrally balanced cerebrum of adult man.

In this figure the diminutive frontal region of the kangaroo is shown. An intelligent gentleman, a pharmacist, residing at Sandhurst, Australia, assured me that the ordinary sheep is the intellectual superior of the kangaroo. I mention this because Gratiolet notes the placid expression and peacefulness of this animal as an evidence that it should occupy the exalted place he accords it, near the ox and horse. Were such things as mildness indicative of mentality, the gentle idiot would rank before Blucher or Frederick William the First.

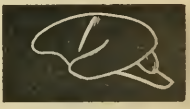


The horse, as here sketched, has comparatively a large frontal lobe. Some horses doubtless are as intelligent as elephants, at least they are as capable of being trained.

The adult saimiri (chrysothrix) or squirrel monkey is diminutive in size but resembles man closely in many anatomical features. The sharpness of its orbital lobe, the anterior situation of the sulcus Rolando, and the medullary angle are indicated in the figure.

Aristotle, Buffon and Gratiolet exalt the character of the elephant, the first claiming that "the elephant surpasses all animals in comprehension,"* while menagerie attendants state that this animal is very brutal, with overestimated reasoning powers and is to be mainly governed by violence and through fear. His frontal brain development places him between the horse and baboon in intelligence.

Microcephalic brain of a child four years of age. Lenret shows the cerebrum to be much distorted and very little of the brain is in front of the sulcus Rolando. The medulla joins the brain at an angle greater than is found to exist in the lowest anthropoid.



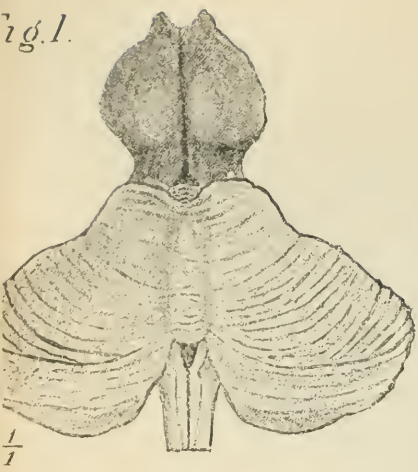
This last figure is intended to show that however closely the chimpanzee's brain may resemble man's, the medullary junction and the lesser anterior brain mass separates the two completely.

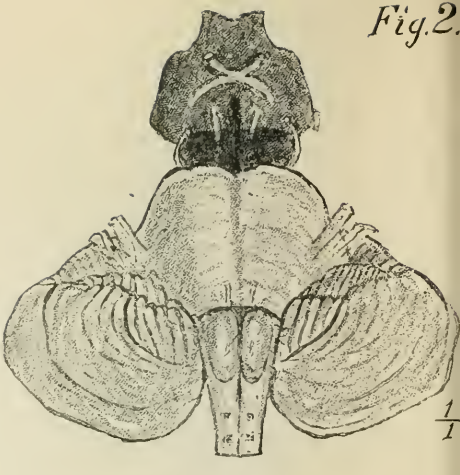
* Aristotle I., ix; ch. 72.

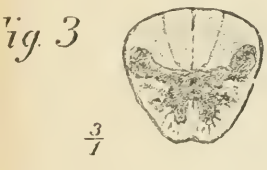
The estimated position of the proposed line projected through the medulla axis may afford a rough means of ascertaining the relative degrees of mentality indicated by fossil as well as by later crania. In this way it would appear that the megatherium was beneath the pterodactyl. But so great is the range of this matter of comparative intelligences and so varied are the views of authors that we can do no more than to refer to them in this connection. The later works on this subject that we have seen are by George J. Romanes,* and W. Lauder Lindsay.† The latter attempts to outline the subject of mind in the lower animals, and to illustrate their possession of the higher mental faculties as they occur in man.

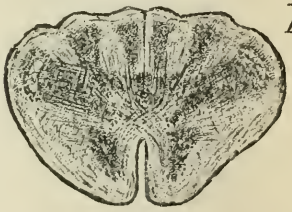
* Nineteenth Century, 1878. Dublin Lecture, British Association, Aug. 16, 1878.

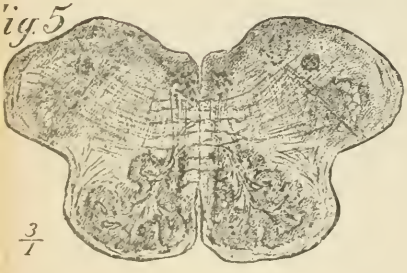
† *Mind in the Lower Animals in Health and Disease.* Appleton & Co.

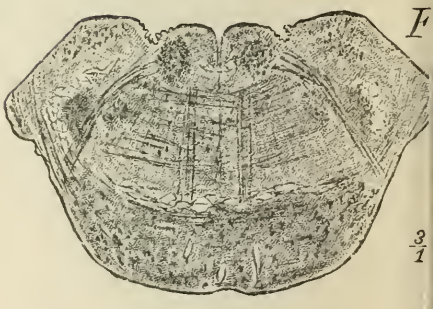


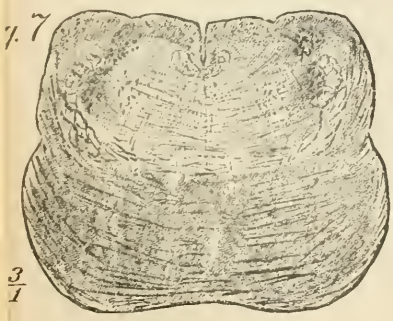


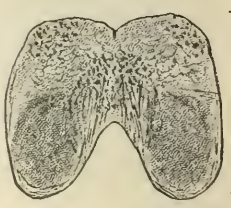












BRAIN OF MICROCEPHALIC INFANT.

THE SENSORY TRACT IN THE CENTRAL NERVOUS SYSTEM.*

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IT is the object of this essay to determine the course of the tracts which convey general sensations from the surface of the body to the cortex of the brain, and to ascertain in what region of the cortex the sensations are consciously perceived. By the term general sensations it is intended to include tactile sensations, the sensations of pain and temperature, and the muscular sense. In order to determine the course of the tracts it will be necessary to trace the anatomical connection between the skin and the cortex of the brain as far as possible by the various methods at command. In order to ascertain the region of the cortex in which sensations are perceived the physiological experiments which have a bearing upon the subject will be reviewed, and the pathological cases in which sensory symptoms can be connected with localized disease will be cited.

I.—The anatomical connection between the surface of the

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body and the cortex of the brain may be conveniently divided into four parts:

1. The peripheral nervous system, by which sensations reach the spinal cord.

2. The sensory tracts in the spinal cord.

3. The sensory tracts in the medulla, pons, and crura cerebri.

4. The sensory tracts in the brain.

1.—*The peripheral nervous system* requires but a brief mention since its anatomy is a subject remote from the present one. Sensory impressions are transmitted from the surface of the body to the spinal cord along mixed nerves made up of fibres which are partly sensory and partly motor. These nerves after passing between the vertebræ into the spinal canal divide into two divisions, one of which enters the spinal cord upon its antero-lateral surface, and the other of which enters it upon its postero-lateral surface. It was one of the early discoveries of nervous physiology, made independently by Magendie and Sir Charles Bell, that the anterior of these divisions contained all the motor fibres of the nerve, and that the posterior of these divisions contained all the sensory fibres of the nerve. It is, therefore, with the nerve-fibres entering the spinal cord by the posterior nerve-roots that this essay has to deal.

2.—*The sensory tracts in the spinal cord.*—At its point of entrance into the spinal cord each sensory nerve-trunk loses its sheath, and its separate fibres at once diverge in various directions. Two main divisions of the nerve are well marked. The first passes directly into the posterior gray horn. The second passes into the posterior lateral white column, and is the larger of the two.

(1) The nerve fibres entering the posterior gray horn at once diverge from one another. Some of these end in the small cells of the posterior horn. A part of these are lost in

the network of nerve fibres in the substantia gelatinosa. A part of these pass forward to the anterior gray horn of the same side. Others pass over to the gray matter of the other side through the posterior gray commissure. A considerable part pass forward and inward, and then turn upward in a bundle named by Kölliker the longitudinal bundle of the posterior horn, and by Clarke the ascending bundle of Deiters, and having ascended a longer or shorter distance again become horizontal and join gray cells in the posterior horn.

(2) The nerve fibres entering the posterior median white column divide into: (*a*) a small bundle which turns downward, and having passed from one to three centimetres in this direction, enters the posterior gray horn[1]; and (*b*) a large bundle which turns upward, and in its course approaches the antero-lateral angle of the posterior column. This latter bundle sends out its fibres in various directions as it passes upward. Many of these fibres turn outward and enter the posterior gray horn at various levels. The remainder pass upward and are lost. The termination of those entering the gray horn is various. Some end directly in the small cells of the gray horn. Some pass forward to the anterior gray horn of the same side. Some terminate in the groups of cells lying in the posterior lateral part of the anterior horn. Some are lost in the network of Gerlach, in the region of the cells of the column of Clarke, and are doubtless connected with these cells. Many of the fibres pass by way of the posterior gray commissure to the opposite side of the cord, where they end in the gray network, or in the cells of the anterior horn.[2] The various cells in which these fibres terminate send out two or more protoplasmic

[1] Schultze: *Arch. f Psychiatrie*, xiv., 2. Ueber secondären Degeneration im Rückenmarke.

[2] Flechsig: "Leitungsbahn im Gehirn u. Ruckenmark," 1877. Schwalbe: "Neurologie," 1881. Ross: "Diseases of Nervous System," Anat. introduction (1883).

prolongations and these in turn uniting make up a complex network throughout the gray substance of the cord.

Thus all the nerve fibres entering by one nerve-trunk terminate in the gray matter of the cord.[1] The level of their terminal cells is not that of their entrance, but each spinal nerve is connected with successive layers of the gray matter from a point three cm. below its entrance to a point at least eight cm. above and possibly higher.[2]

From the cells in the gray matter and from the network of fibres new nerve fibres arise which pass backward to the posterior median column, and form it. Some of these fibres come from the inner side of the posterior horns and from the network near the Clarke column of cells, and pass through the posterior lateral column to reach the posterior median column. Others come from the posterior commissure of the gray substance, and turning directly backward pass along the sides of the septum posterius and thus reach the median column.[2] This posterior median column is made up entirely of long fibres passing up to the medulla and conveying sensations from the lower extremities.[3] The posterior lateral column below the cervical region contains chiefly short fibres passing from the nerve-trunks to the gray matter as already described; and possibly short fibres between adjacent segments of the cord. In the cervical region it also contains long fibres which pass up to the medulla—and which probably convey sensations from the upper extremities.[3] The direct cerebellar columns, lying in the periphery of the lateral columns are made up of fibres which arise from the cells of the columns of Clarke—cells which are considered the terminal stations of some of the sensory fibres entering the posterior horn. These

[1] Hermann's "Handbuch. d. Physiol." Eckhard. P. 159.

[2] Flechsig: "Leitungsbahn im Gehirn und Ruckenmark," p. 311.

[3] Schultze: *Arch. f. Psychiatrie*, xiv., 2. Ueber secondären Degeneration. Flechsig: Plan des mensch. Gehirns.

columns were thought to convey sensory impulses by Meynert,[1] and the fact of their origin from sensory cells, proven by Flechsig,[2] is supposed to confirm this hypothesis, which is further strengthened by the fact that they degenerate upward (centripetally) after lesions in the cord.

It has been recently asserted that some of the sensory fibres in the cord pass upward in the portion of the lateral columns which lie between the gray matter of the cord and the direct cerebellar columns. (Pyramidenbahn and Seitenstrang reste.) This hypothesis is not proven, and the fact that in descending degeneration involving these columns after a lesion of the brain or upper cord, as well as in primary lateral sclerosis which affects these columns in their entire extent, all sensory symptoms are wanting, seems to oppose the hypothesis.

Anatomical study therefore indicates that in the spinal cord the sensory tracts lie in :

1. The gray matter of the cord ;
2. The posterior median and posterior lateral columns ;
3. The direct cerebellar columns.

Physiological experiment and pathological study have shown that sensory impressions pass immediately, after their entrance into the cord, to its opposite side, at least partially if not wholly.[3] The decussation of the fibres conveying sensations of pain and temperature along any nerve occurs at a lower level than that of the fibres conveying tactile sensations.[4] The fact that the sensations of pain and temperature may be lost while those of tactile sensations are unaffected,[5] and the fact that tactile sensation may be impaired while sensations of pain and temperature

[1] Meynert : *Arch. f. Psych.*, iv.

[2] Flechsig : " Leitungsbahn," p. 299.

[3] Hermann's " Handbuch d. Physiol.," p. 167.

[4] Köbner, *Arch. f. klin. Med.*, xix., 169, art. on Spinal Hemiplegia—forty-eight cases.

[5] Bernhardt, *Berlin. klin. Wochenschr.*, Jan. 24, 1884.

are normal,[1] indicates that these two classes of sensation are transmitted along different tracts in the spinal cord in man. In animals sensations of pain and temperature are transmitted upward through the gray matter of the cord; and tactile sensations are sent along the posterior white columns.[2] It is probable that the same is true in man, but this is not yet proven, as it is possible that in disease of the cord in man sensations may pass upward through the gray matter, which would normally go along the posterior columns.[3]

It may therefore be stated that the various functions of the various sensory tracts is not absolutely determined in man. It is known that a unilateral lesion of the cord in man produces a loss of muscular sense on the side of the lesion, and a loss of the sensations of touch, pain, and temperature on the side opposite to the lesion.[4] It is also known that sclerosis of the posterior lateral columns, which involves the fibres of the sensory nerves entering the cord through them, produces disturbances of the sensations of touch, pain, and temperature, as well as a marked disturbance of the muscular sense. Such a sclerosis is followed after a time by secondary scleroses in the posterior median and direct cerebellar columns, but it is as yet impossible to distinguish the symptoms due to each of these processes. After a transverse myelitis from any cause a secondary degeneration upward of the sensory tract occurs, the process advancing up to the medulla in the posterior median and direct cerebellar columns and for several centimetres up-

[1] Schüppel, *Arch. f. Psych.*, ix.

[2] Schiff, "Physiologie"; confirmed by M. Foster and Dalton.

[3] Wundt, "Physio-Psychol.," pp. 107–110; also Hammond, Case of Allochiria, N. Y. Neuro. Soc. Trans., Jan., 1883; and cases of allochiria in *Brain*, vol. iv., No. 2, vol. v., No. 3.

[4] Brown-Séquard, *Arch. de Physiol.*, 1868; Köbner, *Arch. f. klin. Med.*, xix., where forty-eight cases with autopsies are collected. Brown-Séquard considered that the muscular sense was transmitted through the motor columns of the cord, but more recent investigation has demonstrated the existence of sensory nerves from the muscles which enter the posterior columns, and are therefore in connection with the sensory tracts.

ward in the posterior lateral columns,[1] and when this is total all sensations are cut off from below. When the process is only partial, however, sensation is but slightly affected, if at all; though whether this lack of symptoms is due to the escape of some white fibres in the degenerated columns, or to a vicarious action of the gray matter, is undecided, although the former is probably the case.

None of the pathological processes in the spinal cord which affect the sensory tracts afford a basis for distinguishing between the functions of these various tracts. The only conclusions possible in the present state of knowledge are:

(1) In the spinal cord sensory impressions are transmitted through the posterior median and posterior lateral and direct cerebellar white columns, and through the gray matter.

(2) All sensory impressions except those of muscular sense decussate in the cord soon after their entrance to a great degree, if not wholly.

3.—*The sensory tracts in the medulla, pons, and crura cerebri.* On reaching the medulla the sensory tracts of the cord undergo changes of direction and division which are difficult to trace. It is impossible to rely solely upon any single method of determining their course. A number of methods, however, are at present used to trace the direction of a nerve-tract. These are as follows:

(1) By means of a series of thin continuous sections, sagittal, horizontal, and vertical, the continuity of fibres can be followed.

(2) As the medullary sheaths of the fibres of each tract develop in the fœtus at various times, their presence or absence in a definite area, in fœtuses of various ages, affords valuable information regarding the course of the tracts.[2]

[1] Schultze, *Arch. f. Psychiatrie*, xiv., 2, Über secondären Degeneration; and Homen, *Virchow's Arch.*, Sept., 1882.

[2] Known as Flechsig's method.

(3) If a definite tract be divided in a new-born animal, and it survives the operation, the parts connected with that tract fail to develop as the animal grows, and hence a *post-mortem* examination of the full-grown animal will reveal an atrophy in the course of the tract, formerly divided, whose course it is desired to trace.[1]

(4) A local focus of disease in any nerve-tract is followed in the course of a few weeks by a process of degeneration in that tract, which probably proceeds in the direction in which normal nerve impulses are sent. Hence, some time after a lesion, the tract leading from that lesion may be distinguished from all other tracts by the condition of degeneration.

(5) By collecting a large number of cases of local lesion limited to a small area of the nervous system, and ascertaining the symptoms common to all these cases, valuable positive information regarding the function of the area involved by the lesion is afforded. The evidence thus obtained may be tested by a second collection of cases of local lesion involving all other parts of the nervous system excepting the area concerned in the first set of cases, and observing in these the function which remains unaffected. If the facts afforded by the positive cases are substantiated by those obtained from the negative cases, they may be considered as established upon a firm basis.

It is by the use of all these methods, and by a selection of the common facts established by them, that we shall attempt to determine the course of the sensory tracts in the medulla, pons, and crus.

(1) The continuity of the sensory tracts of the cord, with certain parts of the medulla, is easily ascertained, and is not a matter of dispute. All authorities admit, that the posterior median column (the column of Goll) terminates

[1] Known as Gudden's method.

in the funiculus gracilis of the medulla, and its fibres enter the cells of the nucleus gracilis. The posterior lateral column (the column of Burdach) terminates in the funiculus cuneatus and its nucleus, which lies just external to the funiculus gracilis. The direct cerebellar column of the cord passes directly to the cerebellum, by the way of the corpus restiforme of the medulla, the column lying just external to the funiculus cuneatus. The gray matter of the cord undergoes a change of form and of location in becoming continuous with that of the medulla. Each posterior horn becomes greater in area at its extremity, and smaller in area at its junction with the central gray matter, and finally its extremity is separated from the central gray matter by a set of white fibres which come from the nucleus cuneatus and nucleus gracilis, and pass inward toward the median line. While this change of form is proceeding, the location of the posterior horn is also changed. It is displaced laterally by the constantly increasing size of the parts lying between it and the median fissure, and later by the gradual opening out of the central canal into the fourth ventricle by means of the separation of the sides of that fissure. Thus it comes to lie in the lateral part of the medulla, a little posterior to a horizontal line through its centre. The microscopic appearance also changes. Instead of a compact mass of small gray cells in the cord, lying in a fine network of fibres, the sensory gray of the medulla presents the appearance of scattered gray cells lying in a coarse network of fibres. These fibres are partly protoplasmic prolongations of cells, and partly distinct white fibres passing in all directions. The appearance of this area in the medulla has been described by the name which it bears—viz.: the formatio reticularis. In the lower part of the medulla, therefore, the sensory tracts on each side consist of three gray col-

umns—the funiculus gracilis, the funiculus cuneatus, and the formatio reticularis; and a single white tract—the direct cerebellar column. The latter passes out of the medulla by the inferior peduncle of the cerebellum, and terminates in the cortex and central gray matter of the vermiform lobe of the cerebellum. It does not enter into the description of the sensory tracts above this level, and will therefore be traced separately further on. The course of the sensory impulses from the three gray columns upward through the medulla and pons is still undetermined, since irreconcilable differences exist between the various anatomists who have studied the subject. The following views have been advanced by different authorities:

Meynert [1] traces fibres from each nucleus gracilis and nucleus cuneatus (1) to the olivary body of the same side, where fibres arise which cross to the opposite side and go to the cerebellum through the corpus restiforme; (2) through the sensory decussation to the opposite side of the medulla, where they turn upward in the interolivary tract, lying adjacent to the raphé and dorsad to the pyramidal tract. These fibres pass up through the pons, lying just dorsad to the pyramidal tract, and in the crus they divide, some ending in the substantia nigra, and some going to the external third of the longitudinal bundles of the pes of the crus, and thence to the posterior part of the internal capsule. He states that the outer part of the formatio reticularis, in which lie the sensory roots and cells of the V., and VIII., and IX. nerves, is homologous to and a continuation of the substantia gelatinosa of the posterior gray horns of the cord below, and is continuous above with the formatio reticularis lying external to the red nucleus of the tegmentum of the crus, whence fibres ascend to the posterior part of the internal capsule, and to the optic thalamus.

Flechsig [2] traces (1) fibres from the nucleus gracilis through the upper decussation to the opposite interolivary tract, in which they ascend and then pass to the lateral part of the lemniscus, in which they go upward between the corp. quad. and the red

[1] Stricker's "Handbook of Histology," Sydenham edition, vol ii., p. 525. *Arch. f. Psych.*, iv., p. 405.

[2] Flechsig: "Leitungsbahn im Gehirn und Ruckenmark," 1876. *Arch. f. Heilkunde*, xviii., p. 280, 1877. "Plan des menschlichen Gehirns," 1883, pp. 21, 22. *Arch. für Micro. Anat.*, xix., p. 60, 1881.

nucleus to the posterior third of the internal capsule. (2) Fibres from the nucleus cuneatus (*a*) to the olivary body of the same side, which is connected (α) with the lemniscus of the same side, and (β) with the cerebellum of the opposite side; (*b*) to the formatio reticularis of the same, and possibly to the opposite side, whence fibres pass both to the optic thalamus and to the cortex through the internal capsule. He considers the lemniscus and the formatio reticularis as the direct continuation of the posterior columns of the cord, and opposes the view which connects these columns with the cerebellum. With the formatio reticularis are also continuous the gray matter of the posterior horns of the cord and the white fibres lying adjacent to it (Seitenstrang reste).

Aeby [1] traces (1) the fibres from the nucleus gracilis through the upper decussation into the interolivary tract, thence through the lemniscus to the optic thalamus; (2) the fibres from the nucleus cuneatus to the olivary body of the same side, thence to the corpus restiforme of the opposite side, and in it to the cerebellum, whence they issue again in its superior peduncle, and decussating a second time below the red nucleus end in it or in the optic thalamus above.

Roller [2] considers the interolivary tract and its continuation upward in the lemniscus as the chief sensory tract of the medulla and pons, and traces to it fibres from both the nucleus gracilis and nucleus cuneatus of the opposite side, and fibres from the olivary body of the same side, with which both the nuclei of the same and of the opposite side are joined. The lemniscus does not decussate in the crus, but passes directly to the posterior part of the internal capsule, and to the optic thalamus.

Henle [3] describes a second decussation of the lemniscus in the crus in the roof of the aqueduct of Sylvius, a view with which the description of Wernicke is in accord. Henle does not attempt to trace each tract of the cord to the brain.[4] He states that the greater part of the posterior columns of the cord join the anterior column of the medulla and thus ascend, a position in which he stands alone, and which has never been confirmed.

Schwalbe [5] says that a connection of the nucleus gracilis and

[1] Aeby: "Schema des Faserverlaufes im Gehirn u. Ruckenm." Bern, 1883.

[2] Roller: "Die Schleife." *Arch. für Micro. Anat.*, xix., 2, p. 303.

[3] Henle: "Handbuch der Nervenlehre," 1879, p. 272.

[4] *L. c.*, p. 329.

[5] Schwalbe: "Lehrbuch der Neurologie," 1881, p. 633.

nucleus cuneatus, with the formatio reticularis and olivary body, though probable is not proved, and that their connection with the cerebellum is very improbable. He considers the formatio reticularis as the continuation of the seiten strangreste, and traces its fibres to the optic thalamus. He does not trace a sensory tract directly from the cord to the brain, but confines himself to a review of other anatomists, preferring to leave the question undecided.

WERNICKE [1] has given the subject most careful study, and come to the following conclusions. Since the sensory impulses cross the median line in the cord soon after their entrance, and since lesions of the cord or of the internal capsule produce hemianæsthesia of the opposite side, the sensory tract in one side of the cord must be continuous with the internal capsule of the same side. Therefore there is either no sensory decussation in the medulla or there is a double sensory decussation between the spinal cord and the internal capsule. If the first alternative is adopted the sensory tract must lie in the medulla in the external lateral part of the formatio reticularis. This conclusion is reached by a process of exclusion, the function and connection of all other parts being distinctly non-sensory, except the lemniscus, which, however, cannot be included as it decussates. But Wernicke has observed a case of tubercle of the pons,[2] situated just in the external lateral part of the formatio reticularis in which no anæsthesia of the parts below was produced. He therefore rejects this alternative, and adopts the second one, according to which a double decussation of the sensory tract occurs between the cord and capsule. This double decussation he traces as follows : [3]

A. Fibres from the nucleus gracilis cross in the upper or sensory decussation to the interolviary tract and lemniscus, ascend in it to the roof of the aqueduct of Sylvius beneath the corpora quadrigemina, where they recross (in the decussation described by Henle), and then pass up into the internal capsule, and to the optic thalamus.

B. 1. Fibres from the direct cerebellar tract of the cord pass along the inferior cerebellar peduncle to the vermiform lobe of the cerebellum where their first decussation occurs.

2. Fibres from the nucleus gracilis pass along the inferior cerebellar peduncle of the same side to the vermiform lobe where their first decussation occurs.

[1] Wernicke : " Lehrbuch der Gehirnkrankheiten." Bd I., 1881.

[2] *Arch. f. Psych.*, vii.

[3] I reverse the order in which he states the facts, in order to trace the tracts from below upward in the direction in which they transmit impulses.

3. Fibres from the nucleus cuneatus pass to the olivary body of the same side, thence by the fibræ arcuatæ to the inferior pednucle of the opposite side, thus reaching the dentate nucleus of the cerebellum.

The fibres then issue from the cerebellum by two paths : (1) by the middle peduncle, when they recross in the pons and turn upward in its anterior part to pass into the outer third of the pes of the crus, and thence reach the internal capsule ; (2) by the superior peduncle, when they recross in its decussation below the red nucleus, and, passing around or through this, reach the internal capsule.

The last authority to be mentioned is SPITZKA,[1] whose description of the sensory tract agrees in some points with that of Meynert and in some with that of Wernicke. He holds that the fibres from the nucleus gracilis pass through the sensory decussation of Meynert to the interolivary tract of the opposite side, and thence in the stratum intermedium (a term which Spitzka applies to the longitudinal fibres of the pons lying just posterior to the pyramidal tracts, and which is the same as Henle's Bundle vom Fuss zu Haube, and which is continuous with Meynert's stratum intermedium at the level of the substantia nigra of Sömmering) to the posterior part of the internal capsule, thus forming a direct sensory tract to the cerebrum.[2] He holds that the fibres from the nucleus cuneatus divide into a larger and a smaller set of fibres. The larger set decussates through the olivary bodies, and thence passes to the cerebellum by way of the restiform column. The smaller set does not decussate, but passes to the inner portion of the restiform column of the same side, forming the inner division of the inferior peduncle of the cerebellum, and terminating in the nucleus fastigii.

The direct cerebellar column joins the restiform column, and thus represents a direct uninterrupted communication between the sensory periphery and the hemispheres of the cerebellum. The restiform column of each side is therefore made up of (1) the direct cerebellar column of the same side ; (2) a part of the column of Burdach of the opposite side, whose fibres have passed through

[1] The Relations of the Cerebellum, *Alienist and Neurologist*, Jan., 1884. Case of Pons Lesion, *Amer. Jour. of Neurology*, Feb., 1884.

[2] Spitzka does not seem to notice what Wernicke pointed out, that, if such were the course of the sensory tract conveying tactile sensations, a lesion of the internal capsule would produce anæsthesia on the *same* side of the body as the lesion. For the sensory impulses along such a tract would have crossed once in the cord and a second time in the medulla, thus regaining the side they came from.

the olivary bodies ; (3) a part of the column of Burdach of the same side. The restiform column goes to the cortex of the cerebellar hemisphere, and a small portion of it to the nucleus dentatus of the cerebellum. From these parts new tracts issue which convey sensory impulses to the cerebrum. The first of these tracts is the middle peduncle of the cerebellum, in which fibres pass from the hemisphere of the cerebellum to the pons of the opposite side, end in the gray matter of the pons, and thence go up by new longitudinal fibres in the pons, which pass into the pes, and thus to the internal capsule. (This corresponds to Wernicke's first recrossing tract.) The second of the tracts passes from the nucleus dentatus of the cerebellum along the superior peduncle to the red nucleus of the opposite side, and thus into the internal capsule. (This corresponds to Wernicke's second recrossing tract.) Spitzka holds that the cerebellum is a special cerebral segment for all the sensory tracts, into which they are temporarily deflected on their way to the cerebrum. He therefore favors the view of Wernicke as to the existence of a double decussation between the cord and the capsule for all fibres excepting those of the column of Goll. Spitzka, therefore, traces two distinct sensory tracts from the cord to the brain. One of these goes directly through the interolivary tract, stratum intermedium, crus, and internal capsule to the cortex. The other is deflected to the cerebellum, and, having decussated twice between the lower level of the medulla and the level of the red nucleus, reaches the internal capsule and joins its fellow. The first of these corresponds to Meynert's tract. The second of these corresponds to Wernicke's tract.

It is evident that the different views here stated cannot be reconciled. It is also evident that the method of tracing fibres followed by these anatomists is inadequate to decide between their differences. The actual course of the sensory tracts must be traced by the aid of other methods.

(2) The method of Flechsig gives little satisfactory information as to the sensory tracts, as he himself admits, and the conclusions reached by him are based largely upon the results of pathological study. They have been already stated.

(3) The method of Gudden has been pursued by but one experimenter,[1] and but one of his experiments was successful. This, however, affords important imformation and is therefore stated in full.[2] Von Monakow of St. Pirminsburg divided one half of the spinal cord just below the medulla in a new-born rabbit. Six months afterward the animal was killed. The following parts above the place of division were found atrophied: the posterior median column with the nucleus gracilis except in its median half which had not been involved in the division; the posterior lateral column with the nucleus cuneatus; the direct cerebellar column and its tract in the corpus restiforme; the formatio reticularis in its external two thirds lying internal to the ascending root of the fifth nerve, from the medulla as far up as to the anterior corpus quadrigemnum; the lateral part of the lemniscus; the fibres connecting the nuclei gracilis et cuneatus with the olivary body (bogenfasern); the centre of Deiters or outer acoustic centre, on the side of division. On the side opposite the division the olivary body and the lemniscus were partly atrophied. The atrophy could not be followed up to the optic thalamus or internal capsule, hence v. Monakow concludes that no direct sensory tracts exist from the cord to the brain, but that all impulses are sent to gray centres in the medulla and pons, and thence are transmitted indirectly by new fibres upward. The paths of sensory impulses as determined by his experiment would be the formatio reticularis and interolivary tract in the medulla and the lemniscus and formatio reticularis in the pons and crura, and the corpus restiforme to the cerebellum. But as the atrophy in the corpus restiforme involved only the direct cerebellar columns all the way to the cerebellum, and affected its inner

[1] That is for the purpose of determining the course of the sensory tracts.

[2] Monakow: "Exper. Beitrag zur Kenntniss des corp. Restiform." *Arch. of Psych.*, xiv., 3, Nov., 1883.

third only as far as the Deiters centre which lies in the medulla, he considers the transmission to the cerebellum of sensory impulses, except by the direct cerebellar columns, a matter of much doubt. These results of his investigation agree with the statement of Flechsig in the most important particulars and oppose distinctly the statement of Wernicke as to a double decussation through sensory tracts passing to the cerebellum.

Closely allied to this method of Gudden and in some respects in correspondence with it is a method of investigating the course of sensory tracts in infants born with defective brains. If (as Flechsig teaches) the tracts between brain and spinal cord develop in the direction in which they convey impulses, it would be possible to distinguish between centripetal and centrifugal fibres definitely in the nervous system of an infant born with a nervous system deficient either as to brain or as to cord.

The subject of microcephalic brains is one to which hitherto little attention has been given, probably because of their rarity, and also because their importance for anatomical purposes has not been appreciated. I have been able to find but one well recorded case of a microcephalic brain.[1] I have been fortunate enough to secure such a brain, through the kindness of Dr. Northrop, Pathologist of the New York Foundling Asylum, and its examination has been the original motive for this essay.

The following is the description of this brain.[2] The spinal cord below the second cerv. vert. was unfortunately not removed.

[1] This is the case described by Rohon, and published in pamphlet form by him. My thanks are due to Dr. Spitzka for calling my attention to, and for lending me, this pamphlet which was sent to him by the author.

[2] It may be as well to state that this infant presented by the breech, was born alive, and lived one week. It took nourishment and cried like a normal infant, but made no voluntary movements and seemed incapable of perceiving sensations. It slept much of the time, waking only to nurse. It did not attract attention by any peculiar symptoms, and no further history of it was taken.

Microcephalic Infant who Lived Seven Days.

Autopsy.—Head : On opening the head by means of incisions along the sutures a large amount of serum was evacuated. The two parietal bones were removed together. It was then found that there was a marked deficiency in the development of the brain, there being no evidence of cerebral tissue in the anterior and middle cranial fossæ or above the tentorium cerebelli. The *dura mater* lined the cranial cavity but presented at several points an abnormal appearance. Anteriorly, in the median line where two layers should been reflected from the sides of the groove to form the falx cerebri but one layer was present springing from the left margin of the groove. The right layer was deficient for a space of two inches anteriorly, so that at this place the falx cerebri was deficient upon the right side, and a probe could be thrust forward into a cul-de-sac (the foramen cæcum) at its anterior extremity. The falx was normal in its formation and attachments posterior to the coronal suture but its margin hung free in the cranial cavity, there being no brain tissue on either side. The longitudinal sinus in the falx was closed anteriorly opposite the coronal suture so that it did not open into the cranial cavity through the deficiency above described. About three quarters of an inch to the left of the falx, and extending parallel with it, arising from the inner surface of the frontal bone, and from the left parietal bone, was an abnormal reduplication of the dura mater similar in appearance to the falx but less extensive. This layer of dura was attached posteriorly to the margin of the left petrous bone, anteriorly to the left posterior clinoid process of the sphenoid bone. It was therefore stretched across the left anterior and middle fossæ and divided the space into two parts, the outer of which was twice the size of the inner. Contained within the layers of this process of the dura and situated near its free margin about half way from the inner surface of the frontal bone to the apex of the petrous bone was a small mass of tissue oval in shape, one inch in antero-posterior and vertical diameters and half an inch in thickness, which had the gross and microscopic appearance of cerebral substance. It had no connection with the cerebral tissue at the base and must have developed independently. It was the only evidence of an attempt at the formation of cerebral lobes. As it could have developed from the prosencephalon only, and as no other tissue was present which had developed from that portion of the cerebral vesicle, it seemed probable that its presence indicated that the prosen-

cephalon was torn away from the thalamencephalon at a very early stage of fœtal life and had therefore failed to develop excepting to this rudimentary degree. On the right side a fold of dura mater extended across the middle cranial fossa. It arose from the posterior margin of the lesser wing of the sphenoid from the clinoid processes, and from the margin of the petrous portion of the temporal bone. It did not reach as a whole the inner surface of the squamous portion of the temporal bone but was held out toward, and partly attached to that bone by six fibrous bands which radiated from its free margin. There was no similar fold on the left side. Other parts of the dura, viz. : the parietal layers and the sinuses, were normal.

The mass of cerebral tissue present was surrounded by a thick *pia mater* containing many *blood-vessels*. A plexus of these vessels lay upon its upper surface. Upon its lower surface the distribution of the vessels was abnormal. The vertebral arteries joined to form a basilar artery, which lay upon the pons, but was not straight, but was thrown into numerous curves. This basilar artery divided into two arteries, each of which passed outward and forward, giving off numerous vessels to the cerebellum, and to the plexus already mentioned, and joined at their anterior terminations the internal carotid arteries. There were no anterior cerebral or anterior communicating arteries, and hence the circle of Willis was not complete.

The cerebral mass which was present is represented in its exact size in figures 1 and 2. The *upper surface* presented the appearance of a normal, but small, cerebellum and medulla, while anterior to the vermis cerebelli a mass of gray matter was seen not presenting an appearance similar to the corpora quadrigemina or optic thalamus, but consisting of a mass of irregular gray substance, continuous with a second mass, the latter having an appearance of two symmetrical oval bodies with smooth surface separated by a shallow fissure. This gray mass was very soft, and in the process of extraction it was injured upon the right side, and was detached from the pons upon both sides.

The cord being cut through, the medulla and cerebellum were lifted carefully, and the entire cerebral mass was removed from behind forward. All the cranial nerves from the xii. to the iii. were divided in turn, none being deficient. From the thick mesh of pia mater and connective tissue lying beneath the irregular gray mass anterior to the pons, the optic tracts, chiasm, and nerves were easily distinguishable, but the optic nerves were smaller than

in a normal brain of like age. There was no appearance of lobulation, and no fissures were seen in the gray mass from which the optic tracts issued to form the chiasm. The tracts could not be traced outward, but sank directly into the mass as represented in fig. 2. The *lower surface* of the brain presented a number of abnormal features. The *medulla* below the level of the olivary bodies was small, and the anterior pyramids were wholly wanting. The olivary bodies were abnormally large and lay adjacent to one another, being separated by a deep fissure. No fibræ arcuatæ were visible. The *pons* presented a normal appearance externally. The longitudinal fibres characteristic of the *crura cerebri* were wanting, and the level of the mass of gray matter, whose surface was rough, which extended forward from the anterior margin of the pons was much below that of the normal level of the crura. From this mass of gray matter the iii. nerves issued directly, and were larger in size than in a normal brain.

The *cerebellum* was somewhat smaller than normal, but appeared to be perfectly developed in all respects; the normal lobes being well defined by the normal fissures. It measured 7.0 by 3 by 1.6 cm. The gray mass lying anterior to the pons presented two anterior prolongations, which ceased abruptly at a a point 1 mm. from the mass.

The entire cerebral mass was put in Müller's fluid to harden, and at the end of six weeks was placed in alcohol.

It was evident from the parts of the cerebrum missing, that the prosencephalon had failed to develop, thus causing an absence of the entire cerebral hemispheres, the olfactory bulb and nerve, the corpus callosum, the corpora striata, the anterior commissure, and the fornix.

The thalamencephalon, or posterior half of the anterior primary vesicle, had developed to some extent, as was evident from the two masses of gray matter from which the optic tracts and nerves arose, but its development was not normal. For the purpose of accurate comparison the normal brain of a child one week old was obtained and hardened, and horizontal sections were then made with a Thoma microtome through the medulla, pons, and crura, both of the normal and of the microcephalic brain. The sections made were from 20 to 30 μ thick, those in the crura being necessarily thicker than those in the medulla and lower pons, on account of the brittle nature of the tissue. Every set of ten successive sections was given a number, and one or two of each set of these sections were stained and mounted. Weigert's method of

staining with acid fuchsin[1] was used, and also the method with ammoniac-carmine. From the mounted sections those were selected for drawing which demonstrated the course of the various tracts and the presence or absence of the various nuclei and objects of interest. It was considered needless to draw all the sections made. Those which are given are drawings of sections at intervals of about 1 cm. from the first cervical nerve upward.[2]

In the spinal cord at the level of the first cervical nerve (fig. 3) it will be noticed that the postero-lateral columns are absent and that in consequence the shape of the cord is abnormal. The anterior gray horns are small, but their cells present a normal appearance. The anterior fissure is deep at the normal position of the pyramidal decussation. The posterior columns, the posterior gray matter, and the caput cornu on both sides are normal.

In the medulla, at the level of the beginning of the nuclei gracilis and cuneatus, the first decussating fibres appear; at the same level, the ascending root of the fifth nerve become distinct. The contrast between the large number of decussating fibres in the normal, and the small number in the abnormal specimens, was marked. The angle at which these fibres decussate was greater (*i. e.*, more obtuse) than that at which the fibres of the pyramidal tracts in the normal specimen met, thus indicating that they are independent of any pyramidal fibres. In the next figure, fig. 4, the origin of these decussating fibres is seen to be from the nuclei gracilis and cuneatus. *The existence of this decussation, in a specimen in which pyramidal tracts are wholly absent, proves the independent existence of the sensory decussation.* It is noticeable that the number of decussating fibres is small, the decussation occupying a small area in all the specimens through this series, and lying deep within the medulla at the bottom of the anterior fissure, and just anterior to the central canal. At the centre of the decussation, where its fibres are most numerous, the area occupied is but one third of that occupied by the decussation at this point in the normal specimen. It is, therefore, evident that in a normal medulla the decussation of motor and sensory fibres takes place, at least in part, at about the same area, and that the pyramidal and sensory decussations do not lie entirely separate, the one below the other. The absence of pyramidal tracts and decussation influences the shape of the medulla to a marked degree,

[1] See Weigert's article, *Centralbl. f. med. Wissenschr.*, 1882, Nos. 40 and 42.

[2] Eighteen drawings with the sections accompanied the essay. Only those which are necessary are given here.

and also the arrangement of the gray matter; the anterior horns not being cut off from the central gray matter in the abnormal specimen until the sensory decussation is fully developed. There is little difference between the normal and abnormal specimen as far as the development of the nuclei gracilis and cuneatus, the nuclei cuniati accessores, the formatio reticularis, and the fifth nerve are concerned. In the sections just above fig. 4, the numerous fibres passing to the olivary body of the *same* side from both nuclei, were clearly seen. In fig. 5, the abnormally large size of the olivary body in the abnormal specimen is already evident, and a large number of fibres are seen issuing from the large olivary body and passing toward the raphé. Many of these can be followed across the raphé into the formatio reticularis of the opposite side, where they are lost as they turn toward the restiform body. The inner olivary nucleus is smaller in the abnormal specimen, and is seen to be circular; its cells are of the same size and character as those of the olivary body. The outer olivary nucleus is absent in the abnormal specimens.

The interolivary tract appears to be much more narrow, and has a shorter antero-posterior (ventro-dorsad) diameter than in the normal specimen; this is evident at all levels, but especially in section fig. 5. The small size of the interolivary tract in this brain supports the assertion of Flechsig. According to Flechsig, the normal interolivary tract consists of two parts. The smaller of these is the continuation upward of sensory fibres which have decussated in the sensory decussation. It increases in size from below upward, in the reverse ratio to that in which the nuclei gracilis and cuneatus decrease in size,—that is, as these nuclei send fibres upward to the opposite interolivary tract, they grow smaller and it grows larger. The larger of the two parts of the interolivary tract is a continuation of that part of the lemniscus which comes from the lenticular nucleus through its pillar (the Linsenkernschlinge). In this microcephalic brain there was no lenticular nucleus; the lemniscus was small, and this second or larger part of the interolivary tract was absent. There were present nuclei gracilis and cuneatus, and the smaller part of the interolivary tract was present. *It is, therefore, evident that a portion of the interolivary tract develops from below upward, and is in relation with the nuclei gracilis and cuneati.* And, further, that a portion of the interolivary tract develops from above downward, and being in relation with the nucleus lenticularis is absent when it is absent.

The raphé in the abnormal specimen is deficient in the number of its antero-posterior (ventro-dorsad) fibres. This is to be brought into connection with the deficiency of the pyramidal tracts, by means of which the central cortex is joined with the cranial nerve nuclei. It has been supposed that fibres leave the pyramidal tracts at various levels in the pons and medulla, and, turning backward, pass along the raphé to the cranial nerve nuclei. The absence of these fibres taken in connection with the absence of the pyramids proves this hypothesis to be well founded.

There is a marked difference between the abnormal specimen and a normal one in the number of decussating fibres in the anterior (ventrad) part of the raphé. This is doubtless due to the same cause as the deficiency of fibres between the pyramids and the cranial nerve nuclei, and may be considered as evidence that the fibres from the pyramids to the nuclei not only pass in the raphé, but also decussate in the raphé soon after entering it, and at a point nearer to the anterior (ventrad) than to the posterior (dorsad) surface of the pons and medulla.

It is to be noted that the shape, size, and appearance of the gray matter on the floor of the fourth ventricle, of the cranial nerve nuclei, and of the roots of the cranial nerves, of the ascending root of the fifth nerve, of the solitary bundle, and of the formatio reticularis, do not differ at all from those of a normal brain. Although to external appearance the medulla was apparently deficient in arciform fibres, the sections show that these are present in about normal numbers.

The deformity in the general shape of sections through the medulla is due to the absence of the pyramid, to the large size of the olivary bodies, and to the existence of a deep fissure between them.

In the pons (figs. 6 and 7) the most noticeable feature of the specimens is the total absence of the *longitudinal fibres* in the ventral half. This half of the pons is made up of transverse fibres coming from the cerebellar hemispheres and of gray nuclei which are scattered irregularly between the transverse fibres, and which cannot be separated into superficial and deep layers as in the normal specimen. The absence of the longitudinal fibres diminishes the size of the pons, and also its shape, the ventral half being narrower in ratio to the dorsal half than in the normal specimen.

The same comparative deficiency of fibres in the *raphé*, both ventro-dorsad and decussating, which was found in the medulla is evident in the pons, and confirms the supposition that these fibres

have a relation to the longitudinal fibres of the pons, and join these with the cranial nerve nuclei.

In a normal pons the *lemniscus* is a noticeable feature, being made up of the fibres from the interolivary tract, which change their relative position as they ascend, turning ventrad and laterad; so that from occupying a rectangular area adjacent to the raphé with its long diameter parallel to the raphé in the lower part of the pons, it comes to occupy an oval area adjacent to the pyramidal tracts with its long diameter at right angles to the raphé in the upper part of the pons. The position of the lemniscus in the abnormal specimen is normal, but its size is small, and the area occupied by its fibres is about one half that in the normal specimen. This corresponds to the deficiency of fibres in the interolivary tract which has been noticed. The fibres of the lemniscus which are deficient are those which lie most internally in the normal lemniscus, the portion which, according to Wernicke, can be traced to the linsenkernschlinge, a formation which is absent in this specimen. As the lemniscus is traced upward through the pons its fibres become more and more deflected away from the median line, so that at the upper level of the pons (fig. 7) they are seen to approach the lateral boundary of the dorsal half and to be curving upward (dorsad).

It is evident, therefore, that a continuous tract can be traced from the nuclei gracilis and cuneatus across the median line, through the interolivary tract into the lemniscus, and upward into the tegmentum of the crus cerebri, where it lies in the external lateral part of that body.

The *formatio reticularis* presents a normal appearance in the abnormal specimen throughout the pons.

The *posterior longitudinal bundle*, whose fibres in the medulla could not be accurately distinguished from those of the interolivary tract, is well seen in all the sections through the pons, and presents a normal appearance both as to size and as to situation.

The nuclei upon the floor of the fourth ventricle occur in their regular order and position and present nothing worthy of remark. The ascending root of the trigeminus increasing in size as it ascends through the lower half of the pons, meets the descending root at the junction of the upper and middle thirds, and being joined by the motor fibres from the motor nucleus the nerve issues from the lateral part of the pons.

The *ependyma* upon the floor of the fourth ventricle appears to be of abnormal thickness, and the cells of cylindrical epithelium

are large. The ependyma is thrown into folds along the floor, so as to give a crenated appearance to all the upper margins of the sections. It is also to be noticed that a distinctly marked post-*fissure* exists throughout the pons, which is abnormal. Numerous vacuoles are seen throughout the pons, and the blood-vessels are very numerous. When the crura cerebri are reached (fig. 8), the abnormal specimen is seen to differ widely from a normal one, owing to the facts that the roof of the aqueduct of Sylvius is wanting, and that *there are no corpora quadrigemina.* The gray matter lying beneath the floor of the fourth ventricle becomes very thick, and there appear groups of gray nuclei in the external dorsad portion of the specimens, to which nothing analogous is found in the normal specimen. The nuclei of the fourth and third nerves appear to be crowded inward toward the median line and to be displaced downward (ventrad), so that many of the cells lie ventrad of the posterior longitudinal bundle. The number of cells from which third nerve fibres arise, and the number of the third nerve fibres seem to be greater than normal. The third nerve issues from between the red nuclei. The fourth nerve fibres pass outward along the upper border of the red nuclei. The red nucleus is visible, and the decussation of the superior peduncle of the cerebellum, whose fibres are destined to end in this nucleus, is evident in the specimens just below this level. Before the brain was cut it was noticed that the superior peduncles of the cerebellum met and disappeared beneath the lemniscus at a more obtuse angle than normal, and this is evident from the sections, in which the superior peduncles in their full extent were seen to appear suddenly and to decussate at a very obtuse angle.

Many of the sections through the crura cerebri were imperfect. This portion of the brain in the specimen consisted almost entirely of gray matter, and was very difficult to handle, and badly hardened.

As already stated, the peduncles of the crura were absent, the tegmentum alone being present. This left the substantia nigra and red nucleus lying free upon the ventrad surface, with a deep fissure between the two halves of the tegmentum. Some of the gray substance thus lying free crumbled away when the specimen was handled, although great care was taken to prevent such crumbling. It is possible that the portion thus destroyed may have formed a substantia nigra, but in the section as drawn, no such collection of cells (which at this age would not be pig-

mented) was found. The *red nucleus*, however, is intact in several sections and presents about the normal size, shape, and appearance. It is limited ventrad, fig. 8, by a few fibres which pass around it, and which may be likened to the arciform fibres of the medulla, but from their direction cannot be considered as belonging to the superior peduncle of the cerebellum. In fig. 8, the fibres of the third nerve are seen to pass through the red nucleus. The extent and contour of the red nucleus, are well seen in fig. 8, which is made about at its centre. At a higher level a collection of cells of oval shape was seen lying between the red nucleus and the posterior longitudinal bundle. Its shape and appearance resembled that of the corpus Luysii, but its position was not that of that body, which is not found in the specimen. The absence of corpora quadrigemina is evident, but the thick layer of gray substance forming the dorsal border of the crus with its collection of gray cells does not resemble in respect of shape or microscopic appearance, in any degree, these deficient bodies.

It was impossible to obtain sections through the portions of the cerebral mass lying above this level, on account of the imperfect hardening of the specimen. Inspection of the cut surface with a lens showed no appearance of white fibres. There was simply a mass of gray substance which crumbled on the knife, surrounded by a layer of thick connective tissue. Optic tracts could not be found.

To sum up the results derived from the examination of this specimen it may be stated that the chief characteristics are (1) the total absence of pyramidal tracts in all parts of the nervous system, and the deficiency of a portion (about half) of the lemniscus, in the crura, pons, and medulla; (2) the presence of a portion of the lemniscus, and of the interolivary tract; (3) the presence of the formatio reticularis in its entire extent; (4) the presence of the gray nuclei on the floor of the fourth ventricle, with their nerve-roots and nerves; (5) the presence of the peduncles of the cerebellum, and of a normal cerebellum.

These facts are to be brought into relation with the absence in the cord of those tracts which are known to be motor, and the presence of those tracts which are known

to be sensory. It seems evident, therefore, that in this microcephalic brain all the motor tracts in the crura, pons, and medulla are wanting, and that all the sensory tracts are present. The specimen is, therefore, of great value in tracing the sensory tracts.

The sensory tracts in the cord were normal and led to the portions of the medulla which have been stated as sensory. These are the nuclei gracilis and cuneatus, formatio reticularis, and direct cerebellar tract. From the nuclei gracilis and cuneatus some fibres passed through the sensory decussation into the interolivary tract, whence they went upward, formed the outer portion of the lemniscus, and turning upward, passed external to the red nucleus toward the internal capsule. From the nuclei gracilis and cuneatus other fibres passed to the formatio reticularis, there joining fibres which came from the gray matter of the cord; and these together passed up through the medulla, pons, and crura without decussating. The direct cerebellar tract went to the vermiform lobe of the cerebellum—to end in its cortex.

The result of the examination of this specimen supports the conclusion of Flechsig, that tracts develop in the direction in which they convey impulses. Flechsig reports a case [1] which, in some respects, resembles this one. In his case a division had occurred between the brain above and the pons below the corpora quadrigemina; the pyramidal tracts in the pons are wholly wanting, and the lemniscus was diminished to one third of its natural size,[2] while no changes were found in the dorsal half of the pons. In the medulla, the olivary lobes were small, the interolivary tract was reduced to one half its normal extent, the place of the absent pyramids was taken up partly by arciform fibres and partly by "gelatinous substance." The upper decussation was normal. The lower decussation was absent and the fibres of the postero-lateral columns (*i. e.* pyramidenbahn) of the cord were absent.

[1] "Leitungsbahn im Gehirn u. Ruckenm.," p. 120.

[2] In the text the statement is that the lemniscus was reduced to two thirds of its normal size, but in the "Plan d. Mensch. Gehirns," p. 26, this is corrected so as to read one third, as stated above.

The sensory columns of the cord were normal in all respects. (See Taf. xvii., 1–12.)

In Rohon's case [1] there had developed a cephalic mass above the pons about as large as the cerebellum ; and from this a very few fibres issued, forming a thin crusta on each side, but ending in the substantia nigra. In the pons and medulla the same deficiency of longitudinal fibres and pyramidal tracts was found, as in Flechsig's case. But Rohon found a lower pyramidal decussation present. (The plate which he gives as a proof of this pyramidal decussation is a plate of the lower part of the sensory rather than of the motor decussation, as is seen by the fact that the nuclei gracilis and cuneatus can be seen in it, and the number of fibres decussating is much less than in a normal brain at this level.) He, therefore, concludes that in the process of development the pyramidal fibres develop from the brain downward to the point of decussation, and from the cord upward to the point of decussation, and these two parts developing independently unite to form a continuous tract. This conclusion is in direct opposition to the conclusion reached by Flechsig, that the pyramidal tracts develop from above downward along their entire extent.

Up to this time no case has been offered to decide the controversy. But the case here described is such a case, and it proves that the assertion of Flechsig was well founded. In this case the pyramids are wanting in pons and medulla, the pyramidal decussation is wanting, and the continuation of the pyramidal tracts in the spinal cord in its postero-lateral columns is absent. If the pyramidal tracts develop from above downward the case is easily explained. The hemispheres were absent, and the pyramidal tracts failed to develop. If the pyramidal tracts develop partly from below upward, why were they absent in this case, when all other parts of the cord which are known to develop from below upward were present ?

While it is impossible to dispute Rohon's assertion that the pyramidal decussation was present in his case, the diagrams which he offers fail to prove it ; and the case of Flechsig, taken in connection with the case here described, prove that Rohon's generalization is erroneous. *The pyramidal tracts develop from above downward in the direction in which they transmit impulses.*

In Rohon's case the lemniscus and interolivary tract are not of normal size, but are more fully developed than in Flechsig's

[1] Rohon : "Untersuchungen über den Bau eines microcephalen Hirnes." Wien, 1879.

case, or in my case. Rohon was able to trace lemniscus fibres to both corpora quadrigemina, which was impossible in the other cases. The other parts were normal in all three cases.

It may therefore be stated that in the brains of three microcephalic infants all the voluntary motor tracts had failed to develop. This failure had not extended to the gray matter of the floor of the fourth ventricle, to the posterior longitudinal bundle, to the formatio reticularis, to the outer half of the lemniscus, to the olivary bodies and interolivary tract, or to the cerebellar peduncles. In some or all of these parts therefore the sensory tracts must lie. It can be shown from pathological facts, that the sensory tracts do not lie in the gray matter of the fourth ventricle, nor in the posterior longitudinal bundle. The olivary bodies are in functional relation with the cerebellum, for they are anatomically joined to it by the inferior peduncles; atrophy of one cerebellar hemisphere is always associated with atrophy of the opposite olivary body; destruction of an olivary body produces cerebellar incoördination. It can be shown that the sensory tracts do not lie in the olivary bodies or cerebellar peduncles. Hence by exclusion it is evident that the sensory tracts must lie in the formatio reticularis, in the lemniscus, and interolivary tract.

It may be added here in order to complete the record of this case, that sections through the superior vermiform lobe of the cerebellum and through the cerebellar cortex showed their structure and nuclei to be normal.

(4) The method of tracing tracts by means of observing secondary degenerations in the medulla and pons has been of more use in determining the motor paths than the sensory. After lesions in the motor area of the brain, either in the cortex or in the internal capsule, the longitudinal fibres lying in the middle two quarters of the crus cerebri and in the anterior (ventral) half of the pons and medulla degenerate

downward.[1] After lesions in the crus or pons involving the parts lying posterior to these tracts a secondary degeneration downward is observed in the inner two thirds of the lemniscus, which can be followed through the interolivary tract and into the olivary body of the same side[2]; and a secondary degeneration upward is observed in the outer third of the lemniscus, which can be followed upward nearly to the optic thalamus, and into the internal capsule of the same side. This latter is the only tract in the pons in which an ascending degeneration has been traced, and its discovery confirms the assertions already stated of various anatomists, that in the lemniscus at least a portion of the sensory impulses pass upward to the brain.

(5) In the absence of further information to be derived from the methods of investigation already considered, it is necessary to consider the more carefully the facts afforded by a study of pathological cases. Lesions of limited extent in the medulla, pons, and crus are not infrequent, but cases available for the present purpose are very rare. This is due to several causes. A lesion of any considerable extent in these parts, especially if situated in the posterior (dorsal) half of the medulla or pons, usually causes sudden death by injuring the centres of the pneumogastric nerves. A lesion in the anterior half of the medulla or pons involves the motor fibres only, and gives no information regarding the exact course of the sensory tracts. The arterial supply of the anterior part of the medulla and pons is derived from the basilar artery, and disease of this vessel may give rise to symptoms chiefly referable to the tracts in the anterior half of the pons. It is from disease of this vessel that lesions of the pons are usually due, softening from

[1] Türck: "Wien. akad. Sitz. Bericht." *Math-nat. Cl.*, 1851, Bd. vi., S., 288. Charcot: "Localization des maladies cerebrales." Paris, 1881.
Brissaud: "Recherches sur la contracture permanente des Hemiplegiques." Paris, 1880.

[2] And in one case as far downward as through the sensory decussation to the nucleus gracilis. Spitzka: *Amer. Jour. of Neurology*, Feb., 1884.

embolism or thrombosis, and hemorrhage being the forms of lesion most frequent in the pons. For these reasons cases of disturbance of the sensory tracts from lesions in these parts are rare. From a lack of accurate knowledge of the microscopic anatomy of the parts many descriptions of lesions are so indefinite as to be of little use in determining the connection of symptoms with lesions. And lastly, the record of the symptoms in many cases is imperfect. Under these circumstances it is not surprising to find that Nothnagel, whose study of local brain lesions is the most complete and careful of any hitherto published,[1] concludes that the cases recorded up to the time of his writing (1879) were not sufficient in number, nor in such accord, as to warrant any statement more definite than the following: "The lesions of the pons producing anæsthesia are usually situated in the lateral portions in the vicinity of the floor of the fourth ventricle, and all kinds of sensations are equally affected." Since the publication of his work, however, a number of cases have been published in various countries and in different journals which, when collected and compared, afford valuable information on this subject. It is to the study of these cases that we now proceed.

It is well known that lesions limited to the anterior (ventrad) half of the pons, and affecting only the transverse and longitudinal bundles, do not produce sensory symptoms. Many cases are recorded which establish this fact. They need not be cited here.[2] It is admitted that disease which does not extend to or involve the parts lying dorsad of the deep transverse fibres of the pons does not give rise to disturbance of sensation. It follows that the sensory tracts

[1] Nothnagel: "Topische Diagnostik der Gehirnkrankheiten," Berlin, 1879.

[2] The following cases are referred to, however, as they illustrate this position: S. M. Burnett, Knapp's *Arch. of Ophthal.*, vi., 469; Janeway, *N. Y. Med. Jour.*, xxxi., 66; Crandall, *Phil. Times*, ix., 313; Pousson, *Progrès Médical*, x., 560; Ballet, *Progrès Médical*, viii., 657; Gautier, *Gaz. Hebdom.*, 1881, p. 701; *Brain*, vii. and viii.; Wernicke, iii., 415; Judell, *Berlin. klin. Wochen.*, 1872, No. 24.

do not lie in the pyramidal tracts, or in the transverse fibres, or in the gray nuclei of the pons lying between these parts—*i. e.*, in the parts ventrad of the lemniscus.

Lesions situated in and limited to the gray matter of the floor of the fourth ventricle, not destroying or compressing subjacent parts, have been recently studied by Weichselbaum,[1] De Jonge,[2] and Luys.[3] Twenty-three cases of such lesions have been collected by these authors. The symptoms in all these cases were referable to destruction of the cranial nerve nuclei. In none of them were there disturbances of sensation in the body. It follows that the sensory tracts do not lie in the gray matter of the floor of the fourth ventricle, a conclusion which is confirmed by the study of cases of bulbar paralysis, in which sensory symptoms do not occur unless the lesion extends to the formatio reticularis.

By exclusion therefore the conclusion is reached that the sensory tracts in the pons must lie between the deep transverse fibres and the gray matter of the fourth ventricle; that is, in the lemniscus or formatio reticularis.

In the following cases these tracts were involved in the pons or in the medulla, and disturbances of sensation were produced. In some of them both the parts mentioned were affected, and then all kinds of sensation were disturbed. In others but one of these parts was affected, and then some kinds of sensation escaped. The study of these cases therefore will establish not only the course of the sensory tracts already indicated by the anatomical and embryological investigations already described, but may indicate the course of different sensory tracts. The cases are first cited and then analyzed.

Case 1.—Softening in the pons and medulla—sensory symptoms.

[1] Weichselbaum: *Wien. med. Wochensch.*, 1881, No. 32.
[2] De Jonge: *Arch. f. Psych.*, xiii., p. 666.
[3] Luys: *L'Encephale*, 1883, No. 3.

Male, aged fifty, after having suffered for some time from vertigo, was suddenly seized with a feeling of fulness in the head, and a peculiar paræsthesia of the right half of the body, not including the face. This paræsthesia became less after a few days, but there remained a diminution of sensation and a marked ataxia in the right limbs. Eight months after the attack the patient was examined by Dr. Kahler, who found that in both right extremities there was a very marked ataxia upon any motion, which was not increased when the patient's eyes were closed, a loss of the power of perceiving the location of these limbs, and a loss of the sensation of pressure. He had a constant feeling in the right half of the body as if the muscles were contracted. There was no loss of muscular power, and no diminution of tactile sensibility. Other symptoms were nystagmus, paralysis of the left abducens, paralysis of the muscles which open the glottis on the right side. The patient was under observation for two years, during which time there was no change in the symptoms. Cause of death not stated.

Autopsy.—A brown discoloration was found upon the floor of the fourth ventricle in the caudad part of the pons, which was found to correspond to a focus of softening. This focus began in the middle of the left olivary body, and extended upward for 8 mm. through the dorsal segment of the pons in its centre, on both sides of the raphé, but more especially on the left side. The left olivary body and the left interolivary tract were the parts chiefly affected by the lesion. The pyramidal tracts in the pons and medulla, and the lateral parts of the pons and medulla presented a normal appearance.—O. Kahler: *Prager medisch. Wochensch.*, 1879, Nos. 2, 3, and 4.

In the discussion of this case Kahler cites two cases of Leyden's. One of these cases he quotes as follows:

"In a case of Leyden's, in which very marked ataxia of all four extremities was present, the autopsy showed the presence of three small foci of embolic softening lying in the middle of the substance of the pons, but not affecting the pyramidal tracts."[1]

The other case is as follows:

Case 2.—Softening of interolivary tracts in the medulla—general ataxia.

Male, aged sixty-two, was suddenly seized with vertigo and headache, but was able to walk home. From the onset of the

[1] This case of Leyden's is recorded in his "Klinik d. Ruckenmark—krankheiten," I.

attack he was unable to swallow, and three days afterward was brought to the hospital. He complained of headache and vertigo, of inability to swallow, and of inability to stand or walk, or to use his arms and hands on account of loss of power of co-ordination. Examination showed pupils equal ; no paralysis or anæsthesia of the face ; speech indistinct, especially in pronouncing *T* ; tongue protruded straight, but slowly, and tremor was marked ; total inability to swallow ; constant hiccough ; marked ataxia of both hands and both legs, so that he cannot feed himself, or stand, and walking is impossible, even with assistance. Sensibility seemed perfectly normal in all parts of the body, as was also the sense of pain. In the course of a few days an intense redness of the face was noticed. The symptoms remained stationary. He grew weaker, and became delirious, respiration became irregular, and in six days after the attack he died.

Autopsy.—All parts of the nervous system were normal excepting the medulla oblongata. In the medulla an area of softening was found extending vertically from the middle of the olivary bodies cephalad ½ cm. to their upper limit, and occupying the entire interolivary tract on both sides from the gray matter of the floor of the fourth ventricle to the pyramidal tracts, which latter tracts were slightly involved in the degeneration. The entire raphé was destroyed. The lesion did not reach the pons. The nerve fibres were swollen or atrophied or in a state of degeneration. The process was an acute myelomalacia.—Leyden : *Arch. f. Psych.* vii., pp. 57–61.

Case 3.—Softening of one lateral portion of the medulla—sensory symptoms.

Male, æt. fifty-six, on waking in the morning found that he was ill, was dizzy, and could not walk, having a tendency to fall to the left, but no paralysis. His left face felt cold, he could not talk plainly, and had difficulty in swallowing. When examined five days after it was found that he could walk only when assisted, and tended to fall to the left, though when seated all motions were good, without ataxia—the right arm trembling, however, slightly. No facial or hypoglossal paralysis, and ocular muscles normal ; swallowing difficult. Sensation was lost in the left side of the face and in the right half of the body and right limbs. The right limbs soon became livid. The patient always knew the position of his limbs. He died fourteen days after the attack, the pulse having been rapid, 130, from the first.

Autopsy.—An area of softening, due to thrombosis of the left vertebral artery, was found in the lateral dorsad caudad portion of the left half of the medulla. The left corpus restiforme, and adjacent part of the floor of the fourth ventricle and formatio reticularis were yellow and softened for 1 cm. in length. The area extended from the viii. to that of the xii. nerve, and involved the restiform body, the adjacent nucleus cuneatus, the formatio reticularis, the ascending v. root, the motor nucleus of the vagus and its fibres. The olivary body, the sensory nucleus of the vagus, and the hypoglossal were not involved.—H. Senator: *Arch f. Psych.* xi., p. 713.

Case 4.—Softening of one lateral portion of the pons—sensory symptoms.

Male, æt. 50, after suffering for several months from headache and vertigo, was suddenly seized with a sensation as if the entire right side of the body was swollen. When this passed off there was found to be a diminution of the tactile and muscular senses in the right side of the body (not including the face), diplopia, rotary nystagmus, and strabismus due to paralysis of the left sixth nerve, and marked ataxia in the right limbs. The senses of pain and temperature were not affected. Sense of location and of pressure much disturbed. A constant sensation of formication and distension was present in the entire right half except the face. The voice was loud and hoarse, and the patient could not whisper on account of paralysis of the right vocal cord. In the course of the following year a right-sided facial paralysis developed gradually, including the uvula. The disturbance of sensation increased, involving to some degree the senses of pain and temperature and the electro-cutaneous sensibility. The ataxia persisted till death, but true paralysis was not present. The speech became unintelligible as the facial paralysis increased, and the tongue became partly paralyzed. Before death the tongue could hardly be protruded and trembled constantly. He died three years after the onset of the symptoms.

Autopsy.—On the floor of the fourth ventricle beginning near the cephalad boundary and extending back to the striæ acusticæ was found an area of yellow-brown softening. On cutting the pons this was found to lie entirely in its dorsal third, and to the left of the raphé, and to extend from a point five mm. below the corp. quad. down to the upper limit of the olivary body in the medulla. The microscopic examination showed that the primary lesion was a softening due to thrombosis of the pons arteries, in the dorsad

division of the pons, near the raphé, and involved chiefly the formatio reticularis, the lemniscus, and the fibres of the left vi. The following parts were not involved : the pyramidal fibres, the entire transverse fibres of the pons, the middle peduncle of the cerebellum ; the entire nerve nuclei on the floor of the fourth ventricle, the right vi. fibres, and both vii. fibres. A secondary degeneration had developed downward, involving the interolivary tract and the left olivary body in its entire extent.—Kahler and Pick : *Vierteljahrsch. f. d. Prak. Heilk.*, 1879, Bd. 142, S. 96.

Case 5.—Hemorrhage in one lateral portion of the pons—sensory symptoms.

Male, fifty-eight, was suddenly seized with paresis of the left side of the body accompanied by total anæsthesia and loss of the skin reflexes, with total paralysis of the right facial, and abducens, and conjugate paralysis of the left internal rectus, with hyperæsthesia of the left half of the face. The tongue was paralyzed on the left side. Soon after the attack the temperature was lower on the left side of the body.

After one week the paresis had entirely passed away from the left side, but the anæsthesia remained, and was accompanied in the left hand by a loss of muscular sense which gave rise to marked ataxia. The left patellar tendon reflex was increased. The patient's chief complaint was of vertigo and tinnitus aurium. After five months the patient was examined again. There was then right facial paralysis with reaction of degeneration. There was conjugate deviation of the eyes to the left. There was no true paralysis of the extremities, but the ataxia made the left hand and arm useless, and it was slightly atrophied, but there was no contracture. There was partial anæsthesia of the entire left half of the body, greater in the arm than in the leg, and less in the face than in the body ; the sensations for temperature, pain, and pressure being entirely unperceived in the arm. The hyperæsthesia of the right face was no longer present, but the cornea and conjunctiva of the right eye were anæsthetic. Atrophy of the right half of the face, and difficulty of deglutition occurred during the last two months of life. All the symptoms persisted until death, which occurred nine months after the attack.

Autopsy.—Atheroma and miliary aneurisms were present in the larger brain arteries. A clot was found in the gray matter on the floor of the fourth ventricle, on the right side, in its upper half. The lesion involved chiefly the formatio reticularis, and the lemniscus. It had destroyed the right vi. and vii. nuclei and had

involved the post longitudinal bundle, thus probably producing the left internal rectus paralysis. It had reached and involved slightly the ascending root from the nucleus of the right v. and had also destroyed the descending root of the v. on the right side (which is thought by Meynert to go to the left v. nucleus). It had not reached the viii. centres. It did not affect the pyramids. Secondary degeneration was found to have taken place from the clot downward in the lemniscus, and this was traced to the inter-olivary zone and into the olivary body of the *same* side, this being much atrophied.—Meyer: *Archiv. für Psychiatrie*, xiii., p. 63.

Case 6.—Hemorrhage in one lateral portion of the pons; sensory symptoms.

Male, æt. 41, had an attack of vertigo followed by difficulty of motion in the right arm and leg with formication in them and in the left half of the face, and diplopia. The disturbance of sensasation remained for three months, while that of motion disappeared in a few hours. Nearly two years after the attack a second one, precisely similar, occurred, and he then entered Senator's division of the Augusta hospital. Examination showed analgesia in the left second branch of the trigeminus; neither eye could be turned to the left; paresis of the right arm and leg, which soon became almost total. Soon after paresis of the left facial and right hypoglossal developed and swallowing became difficult. The patient had a great diminution of the power of sensation of touch, pain, temperature, and the position of the paralyzed limbs, and the skin reflexes were here diminished, while the right patellar reflex was increased. These symptoms all increased in intensity, and it was also noticed that the right hand and forearm were warmer, damper from sweat, and more livid than the left hand and forearm. There were no urinary symptoms. His intelligence was undisturbed, and the special senses were normal. The anæsthesia and paralysis of the left half of the face and right limbs were nearly complete at the time of death, and the eyes were both directed to the right constantly—the pupils being normal. Seven weeks after his second attack he died.

Autopsy.—Atheroma and thrombosis of the left vertebral and posterior half of the basilar artery with softening of the pons and medulla were discovered. A hemorrhage was found on the floor of the fourth ventricle 1 mm. wide, 1 cm. long in the median line in the upper half. A second clot lay over the sixth nucleus. Section showed an extensive area of softening in the left half of the pons and medulla, involving chiefly its dorsad part near the gray matter

of the fourth ventricle, which appeared to be sunken in. This extended from the nucleus of the vi. to that of the xii. nerve, and to the lower end of the olivary body, being narrower at the ends than in the middle, and lying diagonally to the long axis of the pons, so that above, it was nearer the raphé and the dorsal surface than it was below. It therefore destroyed the formatio reticularis, all the nuclei in part from the vi. down to and including the hypoglossal, the lemniscus, and interolivary tract, the median part of the olivary body, the deepest fibres of the pyramidal tract, the ascending trigeminal root and the solitary bundle, and the median part of the restiform body. It touched the direct cerebellar column at the lowest limit. The right half of the pons and medulla were normal.

Remarks.—As the vi. nucleus was not involved it is necessary to suppose a centre for the conjugate movements of the eyes, lying outside of the vi. nucleus, which was destroyed by the lesion. The fibres to the right xii. were destroyed, while a portion of the left xii. nucleus remained, hence the deviation of the tongue to the right on voluntary motion.—H. Senator: *Arch. f. Psych.*, xiv., p. 2.

Case 7.—Hemorrhage into one lateral part of pons; sensory symptoms.

Male, æt. fifty-eight, was suddenly seized with faintness and vertigo, and then noticed that his right arm and leg were numb as if asleep, but were not paralyzed. The numbness continued for some months, during which time his vision was blurred. It then passed off, but he continued to use his hand in a clumsy way. Two years after the first attack he had an aggravation of his symptoms—the numbness increasing, and his tongue becoming paralyzed for a few hours. He was then examined by Dr. Spitzka, who found that the man's movements with his right leg and arm were jerky and clumsy, and that he felt insecure in walking in the dark. There was ataxia of the right arm, knee-tendon reflex, exaggerated on right side. Skin reflexes absent on right side. No trophic disturbances. Tongue deviates to right. Speech thick after talking some time. Tremor of lips is present. Electric reactions normal. Tactile sensibility impaired in right hand, and on dorsum of forearm; also in right foot. Sensation of pain is quite acute. Sensation of temperature quite impaired. He cannot judge differences of weight five times as great as those recognized on the left side, and cannot judge of the nature of surfaces with his right hand. The direction in which a cold rod is laid

upon his skin is not accurately judged on his right side in arm and leg, and on the body to a less degree—to within three inches of the median line. Marked loss of muscular sense in arm and leg. Sensation slightly impaired on right cheek and lips. After a year the numbness extended to the left foot, and three months later the symptoms had increased in intensity to a marked degree, and he began to have difficulty in swallowing. Six months later the trouble in speech had increased, and he stumbled in speaking. At this time he had two attacks of dizziness and fainting, and, in addition to former symptoms, contraction of right pupil was noticed. Four months after this he became somnolent and apathetic, yawned a great deal, breathed irregularly, and œdema of the right hand, with a decided right hemiparesis and paraplegic weakness of the lower extremities was present. He did not appreciate when his bladder was full. At this time he took to his bed. Soon after he noticed tingling of *both* lower, and a subjective sense of stiffness in both upper, extremities, more marked on the right side. One month later left ptosis developed, and the tongue now protruded to the left. A few days after this involuntary discharges began, his intellect for the first time began to wander, and he suffered much from a feeling of coldness, though the room was so hot that he was in a profuse perspiration, and his temperature was normal. Six days before his death his breathing became stertorous, and right ptosis with extreme myosis was found, the previous left ptosis having passed off. He died comatose at age of sixty-two.

Autopsy.—Dura normal. Opacities in the great falx. Cerebospinal fluid increased in quantity. Vessels calcified. In both hemispheres numerous capillary hemorrhages and small spots of softening were found, none more than 1 cm. in size. In the cerebellum several perivascular hemorrhages were found in the dentale nucleus and two miliary patches of softening, which were found to be recent, as were all the lesions except the one in the pons. In the pons an old hemorrhagic focus was found of irregular shape, lying wholly in the *left* half, and occupying the area of the lemniscus, being thus ventrad of the formatio reticularis, and dorsad of the longitudinal fibres of the pyramids.

The focus of disease consisted of a cavity with partly organized walls and intense contiguous tissue-changes. At the level of the motor nucleus of the v. the cavity was merely a slit, while the area of tissue-change around it occupied nearly the entire field of the lemniscus, failing to reach the raphé or the motor root of the

v. on either side. In lower sections the cavity became larger, and advanced ventrad involving the transverse fibres of the pons below the lemniscus. The contiguous tissue-change involved the raphé, and extended across the median line 2½ mm. The cavity extended caudad as far as to the facial-nerve nucleus, which, however, was not involved.

An area of *descending degeneration* was found, involving the lemniscus and the interolivary tract on the left side down to the sensory decussation, where it was followed through the decussation into the opposite side of the medulla, where it involved the nucleus gracilis to a considerable extent, and the nucleus cuneatus in a lesser degree. The olivary body on the side of the lesion was not involved in this degeneration, although the field around it on all sides was involved. The internal accessory olivary nucleus was not involved. An ascending degeneration was traced, involving the middle third of the lemniscus as high as the level of the corp. quadrigeminum post. It could not be traced higher, but it was impossible to identify that division of the lemniscus which passes into the thalamic region of the left side.—E. C. Spitzka: *Amer. Jour. of Neurology and Psychiatrie*, Nov., 1883. Published Feb., 1884.

Spitzka cites the following case of descending degeneration in connection with his case:

Case 8.—Among a number of cases of secondary degeneration in the pons medulla and cord studied by Homén (*Virchow's Archiv f. path. Anat.*, Bd. 88, S. 61–84), one is recorded in which a focus of softening in the left half of the pons was followed by degeneration downward, both of the pyramidal column and of the lemniscus. The latter was destroyed by the focus of disease at the level of the common nucleus of the abducens and facial nerves. Below this point the secondary degeneration of the lemniscus was traced as far as to the sensory decussation, lying in the lemniscus and interolivary tract. The case is accompanied by a very meagre history, which merely states that the patient was hemiplegic on the right side for three years. No reference is made to any sensory disturbance. It is, of course, impossible to draw any physiological conclusions from this case, but inasmuch as it supports the view of Flechsig that a portion at least of the lemniscus degenerates downward, it is cited here.

Case 9.—Tubercle of the pons; anæsthesia of the face alone. Male, æt. fifty-eight. Began to suffer in July from headache,

diplopia, and difficulty in opening the mouth. In August an examination showed the presence of left facial paralysis including all the branches of the nerve ; spasm of the left masseter ; no affection of the tongue ; ptosis of both eyes, especially of the left ; conjugate deviation of both eyes to the right, it being impossible to turn the right eye beyond the median line, or to move the left eye toward it. Pupils contracted. Sight impaired by old cataract. Smell, taste, and hearing normal ; possibly a slight degree of deafness in the left ear. Loss of sensation to all stimuli and numbness of the right side of the face and neck. No paralysis, ataxia, or anæsthesia in the body or limbs. No urinary symptoms. These symptoms, with headache, vertigo, and vomiting, persisted till death occurred in October.

Autopsy.—On the floor of the fourth ventricle in the middle of its left half a tumor 2 cm. wide and $1\frac{1}{2}$ cm. long was found, which had involved the lateral-dorsad part of the pons, but did not reach the transverse fibres. The tumor was a tubercle, and was not surrounded by any zone of softening. It involved the left common abducens-facialis nucleus, the left facial nucleus and genu, the motor root of the trigeminus, and the descending root of the trigeminus on the left side, which decussates, according to Meynert. At a lower level it involved the eighth and ninth centres on the left side. The lemniscus and the greater part of the formatio reticularis were unaffected.—Wernicke : *Arch. für Psychiatrie*, vii., p. 513.

Case 10.—Hemorrhage in one half of pons ; sensory symptoms.

Female, æt. forty. In June, 1881, she had a sudden attack of vertigo and loss of consciousness, which had been preceded by a continuous occipital headache for several days. On recovering consciousness she found the right half of her body totally paralyzed, and noticed that the left ear was deaf. She did not notice any loss of sensation. A few days after the sight became dim in the left eye, the cornea became inflamed and cloudy, and, finally, she lost the sight entirely in that eye. Some weeks after the attack her right limbs became rigid and remained so three months. She never had any embarrassment of respiration. In October she was seen by Dr. Miles. At that time the right leg could be moved slighly, and the right arm fairly well, but its motions were ataxic, and power of grasp was much less than in the left hand. There was bilateral facial paralysis involving all the branches of both nerves, and producing immobility of the face and defective

pronunciation of the labials. The tongue was protruded to the right with an irregular, uncertain motion. There was no muscular atrophy, and faradic reaction was normal in all the muscles. For five months after the attack there had been a tonic contraction of the muscles of mastication, so that the teeth were not separable more than a quarter of an inch. There was no trouble in swallowing. The nails were found to be growing faster on the paralyzed side, but were not ridged. There was incontinence of urine and fæces. Tactile sensibility was normal, except over a small area of the lower right face, and entire left half of the forehead, and the middle third of the flexor surface of the right forearm, which areas were also analgesic. Tactile sense of the tongue was good. Taste and smell intact. Total deafness of the left ear ; right ear normal. Speech was slow, jerky, and drawling, but there was no aphasia. There was no paralysis of the ocular muscles, and no ptosis. The left cornea was opaque from the presence of extensive pannus, and much hypertrophied.

In December, 1881, the spasm of the muscles of the left side of the jaw relaxed, and the right facial paralysis almost disappeared. By February, 1882, the left eye had atrophied and the cornea had sunk in. At times flashes of heat and redness, with tingling, occurred in the right half of the body and limbs, and the entire side was redder than the left. There was no polyuria. Patient was emotional, laughing and crying easily, but otherwise there was no loss of intellectual power. She grew weaker, bedsores developed, and she died May 21, 1882.

Autopsy (twenty-eight hours p. m.).—Dura normal. Pia opaque and thickened, but not adherent. Arteries atheromatous. On the basilar artery at the inferior margin of the pons, a fusiform aneurism was found of the size of a bean. The left vii. was seen to be smaller than the right vii. The hemispheres were slightly atrophied. The lateral ventricles were distended with serum, and the ependyma thickened. The brain was wet but normal. On the floor of the fourth ventricle, in its left half, just above and external to the eminentia teres, a small yellow irregular depression was seen. Transverse section at this point showed an old hemorrhagic focus of stellate form, extending from five mm. below the caudad border of the pons, five mm. internally to the floor of the ventricle cephalad, nearly to the superior border of the pons, growing smaller as it ascended. The centre of the lesion lay anterior (ventrad) to the genu nervi facialis of the left side. From this point two arms extended forward, nearly reaching the

superficial transverse fibres of the pons. Nowhere did it approach the median line. In the right half of the pons at a point slightly anterior (ventrad) to the vii. nucleus was another focus, also old, of the size of a No. 5 bird-shot. Microscopic examination showed the presence of miliary aneurisms, also a descending degeneration of the crossed pyramidal tract in the right half of the cord, and of the direct tract in the left half. The left v., vii., and viii. n. nuclei were involved.—F. A. Miles : *Archives of Medicine*, Aug., 1882.

The lesion involved both the formatio reticularis and the lemniscus in the pons, as can be seen in the drawing accompanying the case.

Case 11.—Hemorrhage in one lateral portion of the pons ; sensory symptoms.

Female, æt. forty-four, was suddenly seized with giddiness and faintness, but did not lose consciousness. Gradual loss of power supervened in the left arm and leg, with loss of speech and dimness of vision. The next day, on admission to King's College Hospital, there were found paralysis of the right facial, spasm of the left orbicularis palpebrarum, paralysis of the left hypoglossus, and of the left arm and leg, most marked in the extensor muscles ; anæsthesia (partial) and analgesia of the left arm and leg, but no anæsthesia of the face. The symptoms increased during the following two weeks, and paralysis of the right abducens and difficulty of deglutition developed. During the last six days there was constant twitching of the right extremities, and the bladder was paralyzed. She died eighteen days after the attack.

Autopsy.—A fusiform hemorrhage was found in the pons, extending along almost the whole length of the right side. It was 2 cm. long, and its anterior (cephalad) margin was 0.5 cm. caudad of the corp. quad. post., while its caudad margin was 1 cm. cephalad of the pyramid of the medulla. Its widest portion was situated at a depth of 1½ cm. from the ventrad surface of the pons. It did not cross the raphé, but opposite its posterior extremity, in the left half of the pons, was a small clot the size of a hemp-seed, which lay at a depth of 1 cm. from the anterior surface. The clot was wedge-shaped, and its apex projected slightly into the iv. ventricle.—F. Willcocks : from Clinic of Dr. Johnson, King's College Hospital Report in *Brit. Med. Jour.*, 1881, i., p. 272.

Case 12.—Tumor in one lateral half of pons ; sensory symptoms.

Female, twenty-eight, was suddenly seized with an acute pain in the right side of the head, and lost her consciousness. On recovering from the attack, she was found to have total paralysis of the left hand, paresis of the left leg, and anæsthesia in both these limbs ; also paralysis of the right motor v., right vii., right xii., and anæsthesia of the right half of the face. She was deaf in the right ear. Her speech was imperfect, and she had difficulty in swallowing. In this condition she lived fourteen months, and died of exhaustion.

Autopsy.—A semi-cartilaginous fibrous tumor was found in the dura and pia upon the right side of the pons and medulla oblongata. It extended from the point of exit of the v. backward for two inches, enclosing the right vertebral artery. The surface of the right crus cerebelli was softened, and so was the pons, upon which the tumor lay. It was incorporated with the substance of the right side of the medulla, and had produced softening throughout its tracts. The left side of the medulla was normal. The roots of all the right cranial nerves, from the v. to the xii., were compressed.—*Amer. Jour. Med. Sc.*, vol. xxviii., p. 106 (1841).

Case 13.—Softening of pons ; sensory symptoms.

Male, æt. seventy-four ; a complete left hemiplegia of the arm and leg, with impairment of sensation gradually developed, and remained for one year before death, the right side never being affected. The slighest abrasion on the left side produced marked ulceration. There was no incontinence of urine or fæces. He died suddenly.

Autopsy.—The meninges were congested, and there was some effusion in the ventricles and under the arachnoid. In the falx cerebri were found four small pieces of bone one fourth inch thick. On the floor of the lateral and fourth ventricles granular excrescences resembling boiled sago were found. In the pons was a wedge-shaped area of brown softening. It spread over the anterior surface of the pons in its entire width, and one half an inch antero-posteriorly. Its apex extended on the right side to a depth of three eighths inch, and involved the longitudinal fibres. On the left side it was superficial, and involved only the transverse fibres. —J. B. Tuttle : *Phil. Med. Times*, xii., p. 350.

Case 14.—Tumor of one half of pons ; sensory symptoms.

Female, æt. seventeen ; when admitted to the hospital was so stupid that no history could be obtained. On admission the following symptoms were found : complete paralysis and anæsthesia of the left half of the face ; complete paralysis and partial anæs-

thesia of the right arm ; paresis and impairment of sensation in the right leg, though she could walk ; loss of smell in left nostril (tested by ammonia, which indicates anæsthesia) ; no paralysis of the tongue ; loss of hearing in the left ear ; total paralysis of the left eyeball, without strabismus; conjunctiva and cornea acutely inflamed ; deglutition difficult, but appetite good ; no vomiting ; pulse rapid and weak ; temp. varied slightly from normal ; involuntary evacuations. Her mental faculties were so blunted that it was impossible to obtain reliable replies. She went into a condition of coma and died. (Duration not stated.)

Autopsy.—The pons was greatly distorted, and enlarged on the left side. Its surface was nodular, and its margins overlapped the medulla and crus. The left crus cerebri was also enlarged, and nodular. The left pyramid was compressed and indented, and the right pyramid pushed aside. The floor of the fourth ventricle was widened and bulged upward on the left side. The tumor was spherical in shape, occupied the left half of the pons, and had pushed the raphé to the right. No microscopic examination. No description of sections.—F. A. Miles : *Arch. of Medicine*, Oct., 1881.

Case 15.—Tumor of one half of the pons ; sensory symptoms.

Male, æt. eight. Nov. 1, 1877, he suddenly fell down, and on being helped up could not stand, and trembled greatly. Three days subsequently he had a similar attack, but this did not interfere with his going to school all the month. His teacher noticed that he was very clumsy, but did not think him stupid. For three weeks prior to Dec. 12th he suffered from darting pains through his head, occasional vomiting, and weakness in his left hand. When examined Dec. 12th there were found occipital headache, right facial paresis, head inclined to the left, ptosis of left eyelid, paresis of left hand, and an unsteady gait. In a week the symptoms had increased in degree, the left pupil was dilated, but the ptosis had disappeared in the left and appeared in the right eyelid. His skin was cool—pulse 80, regular—appetite good. There was no intellectual disturbance. Dec. 29th.—Vomiting is now associated with the paroxysmal headache, and at the same time his bowels move. His speech is indistinct, and he is now very garrulous, talking constantly. During January his mind became much weakened ; his special senses were not impaired ; strabismus of the right eye appeared, the ptosis remaining ; right half of face became anæsthetic ; he could no longer stand or walk, and his head seemed too heavy for the muscles which support it. The

optic discs were normal. The left hemiplegia became complete, and his right foot was kept in motion constantly ; the pulse became irregular and rapid, and on Feb. 10th he died of paralysis of the pneumogastric nerves.

Autopsy.—The right side of the pons was much larger than the left ; soft and white, in its entire extent. The change extended along the middle cerebellar peduncle into the right hemisphere of the cerebellum. The fifth nerve could not be traced through the mass. This portion of the pons consisted of round and oval cells with few nuclei in a granular stroma, with many vessels. The tumor was a soft glioma. No sections, and no accurate localization.—J. C. Mackenzie : *Cincinnati Lancet and Clinic*, iv., p. 150.

Case 16.—Abscess in one half of pons ; sensory symptoms.

Male, æt forty-four, while suffering from an abscess of the arm developed suddenly paralysis of the right facial, anæsthesia of the right trigeminus, paralysis of the left hypoglossus, and anæsthesia of the left side of the body. On the next day swallowing and speech became difficult, and spasms of the left arm and leg began and were followed by paresis. Two days after respiration became difficult and he died.

Autopsy.—(By Huguenin.) An abscess was found in the right side of the pons, involving the dorsad half almost in its entire extent without involving either the inferior or superior peduncles of the cerebellum. In the lower part of the pons the abscess had broken through the deep transverse fibres and had reached the longitudinal fibres of the anterior portion.—Bircher, *Schweitzer ärtzbl. Corresp. Bl.*, 1881, No. 4, quoted by Wernicke, *l. c.*, iii., p. 417.

Case 17.—Tumor of one half of the pons ; sensory symptoms.

Male, æt. thirty-two, syphilitic, was kicked in the head by a horse five years before the acute symptoms set in, but ever since that time had suffered from nocturnal headache, and occasional attacks of vertigo. Four weeks before his admission to the hospital he had a fall on the ice and hit his head. No ill effects of this fall manifested themselves until one week after it when he suddenly had an attack of vertigo and fell down. A few days after his sight became dim and he noticed a weakness of the right arm and leg. On admission it was found that his memory was defective so that history was uncertain. He was able to walk but his entire right side was paretic except the upper branch of the facial nerve. It was noticed that the right side of the forehead wrinkled more promptly than the left. Sensation was diminished

in the right limbs and in the left side of the face. Hearing, smell, and taste normal. Both eyes were constantly directed to the right and they could not be turned to the left of the median line. They were fixed and staring; pupils equal, and normal; in accommodation the axes converged slightly; the fundus was pale; the outline of the disc irregular, and the disc hyperæmic and opaque. The patient got worse gradually, and had to stay in bed on account of the vertigo. He had frequent attacks of epistaxis, and became anæmic and weak. On the day before death the limbs of both sides seemed equally paralyzed, and the mouth was drawn to the right side. There was decided loss of sensation in the right limbs and left side of the face. The eyes still deviated to the right and the pupils were small. He died of exhaustion. (Duration of illness not stated.)

Autopsy.—In the left squamous portion of the temporal bone a fracture of the skull—not depressed—was found, and here the dura was adherent, and on its inner surface was a small hard yellow nodule the size of a pea which had caused a slight depression in the left first temporal convolution at the junction of its middle and posterior thirds. A tumor one half inch in diameter was found in the body of the pons, causing a bulging of the floor of the fourth ventricle in its cephalad left part. The tumor was limited to the cephalad left quarter of the pons and did not cross the median line. It had pushed apart the anterior and posterior surfaces of the pons and had not disturbed their integrity(?). Microscopic examination showed it to be a gumma. There was descending neuritis of the optic nerves.—C. K. Mills, Jour. Ment. and Nerv. Dis., July, 1881.

Case 18.—Hemorrhage in one half of the pons; sensory symptoms.

Male, æt. twenty-four, suffered from headache for several days, and then from numbness and formication in the left arm. On Oct. 14th, was suddenly paralyzed in the left extremities and left side of the tongue and right side of the face, and articulation was impossible. The left extremities were anæsthetic. The paralysis subsided somewhat in the extremities before his death, which occurred on Nov. 24th.

Autopsy.—A clot was found in the right half of the pons, just beneath the floor of the fourth ventricle. It extended nearly to the corpora quadrigemina. The clot was hard, yellowish, and fibrinous, and the surrounding substance was normal. Very small hemorrhages were also found, one in the centrum ovale and one

in the left corpus striatum.—Mavot : quoted by Wernicke, *Lehrbuch d. Gehirnkr.*, II., p. 95.

Case 19.—Tumor of one lateral half of pons ; sensory symptoms.

Female, æt. forty, after suffering from headache, vertigo, and vomiting, with deafness and diplopia, developed gradually paralysis of the right abducens, of the left facial, of the left arm and leg, and total anæsthesia, with loss of muscular sense in the paralyzed extremities and in the left half of the trunk. Then followed attacks of pain shooting down the left arm and leg. The tongue protruded straight, and speech was perfect. The special senses were normal. Difficulty in chewing, with spasm of the masseters developed before death.

Autopsy.—In the substance of the right half of the pons a tubercular tumor was found, 2 cm. in diameter, which produced a bulging upward of the floor of the fourth ventricle. The tumor began at the cephalad border of the pons, and extended down to the inferior peduncle of the cerebellum, lying chiefly in the dorsad part of the pons near the floor of the fourth ventricle. The right middle peduncle of the cerebellum and the left half of the pons were normal. It was surrounded by a zone of softened tissue, which extended upward in the right crus cerebri nearly to the optic thalamus (ascending degeneration ?). In the lower extremity of the right posterior central convolution upon its surface a second tubercle was found of the size of a pea.—Mavot : *Bull de la Soc. Anat.*, Paris, mars, 1875, quoted by Nothnagel, *l. c.*, p. 121.

Case 20. Softening of one half of the pons ; sensory symptoms.

Male, æt. thirty-three, had for several days a feeling of numbness, cold, and weakness in the right arm and leg, and headache. Then paralysis in these limbs developed suddenly without loss of consciousness. Examination showed paralysis of the entire right side, with anæsthesia, the face being included. Duration of illness not stated.

Autopsy.—A thrombosis of the basilar artery had produced an area of softening in the pons, which involved its entire left cephalad dorsad half. Details of the lesion are wanting.—Nothnagel : *Topische diagnostic d. Gehirnk.*, p 112.

Case 21.—Sarcoma compressing the pons and crus ; ataxia.

Male, æt. nine, began to use his right hand in an awkward manner in May, 1874, and in the course of a few months had lost power almost completely in the right arm. He then began to

suffer from headache, nausea, vomiting, double vision followed by strabismus, due to paralysis of the left abducens He had occasional twitchings in the right hand, but no convulsions. During the next year the paralysis extended to the right leg, and there was a staggering gait. There developed ataxia and rigidity in the fingers of the paralyzed hand. Optic neuritis followed, and he suffered much from pain in the legs. His symptoms increased in severity until death, which occurred in April, 1880.

Autopsy.—A sarcoma was found upon the base of the brain, pressing upon the left crus cerebri and the pons. (Details wanting).—E. C. Seguin : "N. Y. Neurol. Soc. Rep.," JOUR. MENT. AND NERV. DIS., Jan., 1882.

CASE 22.—Sarcoma of one half of the pons ; ataxia and sensory symptoms.

Male, æt. thirty-eight, fell and hit his head on Jan. 13th, and since that time has suffered from headache, vertigo so severe that he fell at times, and occasional attacks of nausea, and pain and weakness in his right shoulder. Was admitted to the hospital Feb. 23d, when a paresis of the right arm with numbness, impairment of speech and of deglutition, and "a distortion of the features, most marked on the right side of the face" (left facial paralysis ?), were found. His gait was reeling. His memory was so defective that the history was obtained with difficulty. He was habitually constipated. There was no change in his condition up to June, when he had two apoplectic seizures, occuring at an interval of two weeks, and died.

Autopsy (twenty-four hours, p. m).—The lateral ventricles were distended with serum. A tumor, of the size of a hickory nut, was found situated in the pons, and involving chiefly the dorsad surface of the left lateral half. A second tumor of the same size was found in the extreme posterior projection of the right lobe of the cerebellum. Microscopic examination showed them to be round-cell sarcomata.—G. Hart : *St. Louis Med. and Surg. Jour.*, vol. xiii., p. 571.

CASE 23.—Glioma of the pons ; ataxia.

Male, æt. six and a half, in Nov., 1873, fell on the back of his head. Two days after this he had a headache, and two weeks after his gait became irregular and ataxic, so that he reeled in walking. He was restless at night, and had headache frequently, but no vomiting or constipation. In Jan., 1874, his speech became indistinct and jerky, and his memory began to fail. In April he had become very stupid, and his speech was slow and

unintelligible though he tried to talk much. There was marked ataxia of the head and all the limbs, but no paralysis or anæsthesia. There was exophthalmos, and all the motions of the eyes were performed slowly, but there was no disturbance of vision. Hearing was good. Later in the month he had involuntary evacuations of urine, and the left pupil was dilated. In May, vomiting, intermittent pulse, dimness of vision, and paresis of the facial muscles began, and an ophthalmoscopic examination showed atrophy of the left disc, and congestion of the right disc. On the 8th he became comatose, and on the 9th he died of apnœa.

Autopsy.—The pons was found to be enlarged in all directions, measuring two inches long, two and a half inches wide, and one and a half inches thick. In a depression along its centre ran the basilar artery, which was much stretched. The tubercula quadrigemina were pushed up and flattened; the cephalad anterior portion of the fourth ventricle was occupied by a rounded swelling, which was firm on the left side and elastic on the right side. The anterior pyramids at their entrance into the pons were elevated, but their point of entrance was deep and normal. Microscopic examination showed the existence of small, round, and polygonal cells, with granular matter in a stroma of thickened neuralgia, throughout the pons. (Glioma.) There was an atrophy of the optic nerves. Lateral ventricles distended by serum. —Gibney: *Amer. Jour. Med. Science*, July, 1875.

Case 24.—Abscess compressing the pons; sensory symptoms.

Male, æt. twenty-one. In Sept., 1869, began to suffer from headache, and, on the 10th, left facial paralysis developed and remained. He entered Massachusetts Hospital Oct. 27th, when left seventh and twelfth paresis was noticed, and it was found that, in walking, he was dizzy and inclined to fall to the left. Subsequently, paresis of the left arm and leg developed, and, one month after, involuntary motions of the hand and arm occurred, with stiffness on passive extension. One week later, inco-ordination was so marked in his legs that he could not stand. The sensation of the limbs was not affected, but he had pain in the left side of the face and on the inner side of the left arm, and the left cheek and eyeball were anæsthetic. There was a gradual loss of vision, first in the left and then in the right eye. He had been deaf in the left ear for years. There was occasional flushing of the face. During three days in December he lay in a stupor. In January, nausea and vomiting began, the head ache became intense, and, at last, dysphagia developed, and he died Feb. 18, 1870.

Autopsy.—Pia strongly injected at the base; four ounces of serum in lateral ventricles. Brain-substance firm with numerous puncta vasculosa. Left side of the cerebellum was prominent and half as large again as the other side, the medulla and pons being pushed to the right. The left fifth, sixth, seventh, and eighth nerves were covered in, and destroyed by, a morbid growth; the ninth, tenth, and eleventh were stretched over the abscess to be described; twelfth normal. Along the side of the medulla was a swelling—soft and fluctuating, which was found to be an abscess containing green pus; its cavity was as large as a walnut, was lined with a membrane covered with villi. It extended upward and inward under the medulla and lower edge of the pons, not affecting the cerebellar peduncles except by pressure, seeming to lie between the lower and middle peduncles and to press them apart. The lower peduncle was spread out over its surface; it extended down almost to the lower part of the left cerebellar lobe, near the median line, the corpus dentatum not being affected. On the outer side of this abscess was a second one, as large as a hickory nut, separated from the first by a layer of cerebellar tissue, and occupying the upper part of the upper portion of the left cerebellar lobe. The fourth ventricle was larger than normal, being stretched over the abscess. The morbid growth was a glioma consisting of cells in groups and fibrous tissue.—S. G. Webber, *Boston Surg. and Med. Jour.*, vol. lxxxii, p. 289.

Case 25.—Softening of the pons; hemiplegia, subsequently opposite facial palsy.

Male, suffered for four months from paresis of right arm and leg, with slight weakness of the right side of the face. He was then attacked with severe pain in his left jaw, which radiated from behind and below the left ear, over the side and front of the head and face, and, the next day, the left half of the face was found paralyzed. The pain continued for a month, and was associated with hyperæsthesia of the second branch of the fifth nerve. Reaction of degeneration not present. Two months after this attack, diabetes developed. All the symptoms persisted until nine months from the beginning of his illness, when he died suddenly. No mention is made of any disturbance of sensation, except in the second branch of the fifth nerve—on the left side.

Autopsy.—There was a depression of the convexity of the pons on the left side. Opposite to this, in the middle of the pons, was an irregular area of softening, situated in the pyramidal fibres, opposite and a little below the origin of the fifth nerve. Above, the

softening was in two foci, one of which was a cavity, the other being occupied by granular debris ; but, a little lower, these had blended into one of rather smaller size. The fibres of the fifth and seventh were both apparently undamaged, but the lesion in its lower part was close to the fibres of the seventh. In the middle of the pons, the left ascending sensory nucleus of the fifth presented two small foci of softening. The anterior pyramid of the left side was completely degenerated, and the degeneration was traced through the decussation to the opposite lateral column of the cord.—Gowers : *Brain*, pt. vii, p. 474.

CASE 26.—Tumor of the pons ; no sensory symptoms.

Male, æt. fifty-four, suffered for three months before his death from severe continuous headache, and two months before death noticed that his vision was disturbed by the fact that his eyes were constantly turned to the right. There was no diplopia. On examination, the head and eyes were found to be turned to the right, and the eyes, moved together, could not turn beyond the median line to the left, though the right eye alone could be turned for some distance to the left beyond the median line. Pupils equal and mobile. The position of the face was not due to paralysis or contracture of the muscles of the neck, and he could turn his head in any direction. *No paralysis or loss of sensation.* He had some dizziness, and staggered in walking. He died of pneumonia.

Autopsy.—No meningitis. No lesion in the hemispheres. A tumor was found in the pons at a level one cm. below (caudad) the apparent origin of the v. on the left side. It was so situated in front of (cephalad) the eminentia teres that it involved the course of the fibres of the left abducens, and by a little prolongation across the raphé toward the right side, interrupted the fibres of communication between the vi. and iii. centres. It did not involve the common nucleus of vi. and vii. It interrupted the posterior longitudinal bundle and the adjacent part of the raphé. No other lesion was found. The tumor was the size of a small nut. The position of the head was regarded not as compensatory for the position of the eyes, but as due to a severing of fibres joining the rotatory muscles of the head with their reflex centres. —Quioc : *Lyon Médicale*, 1881, July, Nos. 29, 30.

ANALYSIS OF THE SYMPTOMS.

The large number and varied character of the symptoms present in these cases make an analysis desirable ; and in

order to reach any conclusion from the study of the cases, as to the tracts conveying sensory impulses, such an analysis is necessary.

The Sensory Symptoms.

I. Disturbances of the tactile sense.

The tactile sense was disturbed to some degree in twenty-one cases. In cases 1, 2, 21, 22, 26 it was not affected. The distribution of the disturbance was as follows:

1. Anæsthesia of the right side of the face in cases 5, 7, 9, 10, 12, 15, 16, 20. Anæsthesia of the left side of the face in cases 3, 6, 10, 14, 17, 24, 25. Inflammation of the cornea coincident with the anæsthesia, occurred in cases 5, 10, 14.

In cases 3, 5, 6, 12, 14, 15, 16, 17, 24, 25, the anæsthesia of the face was on the same side as the lesion, and the lesion involved the ascending root of the trigeminal nerve in some part of its course in the medulla or pons, and did not involve the descending root. In cases 7, 9, and 20 the anæsthesia of the face was on the opposite side from the lesion. In cases 7 and 9 the lesion was situated so high in the pons (cephalad) as to involve the descending root of the trigeminus to some extent, but too high in the pons to affect the ascending root at all. The anæsthesia was slight in degree and was limited to the cheek and lip in case 7. It was well marked in case 9. In case 20 all details of the extent of the symptom and of the position of the lesion are wanting. In case 10 both sides of the face were anæsthetic, but the anæsthesia was limited to the upper branch of the trigeminus on the side of the lesion, and to the lower branch of the trigeminus on the side opposite to the lesion. In this case the lesion was so situated as to involve a small portion of the ascending root of the trigeminus and also a portion of the descending root of the trigeminus on the same side.

The conclusions to be reached from these facts are as follows:

(*a*) *Lesions affecting the ascending root of the trigeminus produce anæsthesia of the face upon the side of the lesion.*

(*b*) *Lesions affecting the descending root of the trigeminus produce anæsthesia of the face upon the side opposite the lesion.*

These conclusions confirm the statement of Meynert, that the descending root of the trigeminus decussates in the pons.

It may be noticed in passing that *a lesion, in order to produce any disturbance of sensibility in the face, must lie in the external lateral part of the formatio reticularis.* In the cases in which the face was not affected, the lesion lay elsewhere than in this portion. In the cases in which it was involved there was anæsthesia of the face.

2. Anæsthesia of the right limbs occurred in cases 3, 4, 6, 7, 14, 17, 20. Anæsthesia of the right arm alone occurred in cases 10, 22. Anæsthesia of the left limbs occurred in cases 5, 11, 12, 13, 16, 18, 19. In all these cases the anæsthesia of the limbs was upon the side opposite to the lesion, although the lesion was situated in all portions of the formatio reticularis from the lower limit of the medulla to the upper border of the pons. *It is therefore evident, first, that in the medulla and pons the sensory tract for each side of the body lies in the opposite half; and, secondly, that there is no decussation of the sensory tracts between the sensory decussation at the lower limit of the medulla and the upper border of the pons.* The facts are therefore opposed to the course of the sensory tracts described by Wernicke and Spitzka, in so far (1) as these tracts are supposed to undergo a second decussation between the sensory decussation and the internal capsule; and (2) as the sensory tracts are supposed to leave the medulla and to pass by way of the cerebellum around the pons. Lesions of the pons in any portion of its vertical extent (*i. e.*, between its cephalad and caudad limits) produce anæsthesia of the op-

posite half of the body. Therefore the sensory tracts must pass through the pons. Therefore they cannot pass through the cerebellar peduncles and the cerebellum unless we suppose that there are two independent sensory tracts, from each side to the cerebrum, a lesion in one of which suspends the function of the other also. It is very possible that some sensory impulses may pass to the cerebellum by the tracts described by Wernicke and Spitzka, and, setting up there a reflex action, be the means of exciting that organ to do its reflex work. But if so these are *not* the sensory impulses which pass to the higher cortical cerebral centres, or which are destined to awake in consciousness a perception of the sensation. The sensations which are perceived consciously are transmitted directly from the surface of the body through the spinal cord, medulla and pons into the internal capsule and thence to the cortical centres, and in their course undergo but one decussation. If that decussation is complete in the cord, the tract remains on the same side from the cord to the capsule. If that decussation does not occur in the cord it takes place in the sensory decussation at the lower part of the medulla.

In all these cases the lesion involved the formatio reticularis of the medulla or pons, and this was the only area of these parts which was affected in *every* case. By the term formatio reticularis I wish to include in the medulla (see fig. 5) the portion lying between the gray matter of the floor of the fourth ventricle and the pyramidal tracts in a dorso-ventrad direction, and between the interolivary tract and the ascending root of the trigeminal nerve in a lateral direction. In as much as there is no case on record in which the olivary body alone was destroyed, it is impossible to state whether it is to be included in the sensory area of the medulla. Its connection with the sensory parts of the cord has led Meynert to consider it a part of the

sensory tract. The question cannot be decided from pathological facts. In the pons the formatio reticularis (see figs. 6, 7) lies between the gray matter of the floor of the fourth ventricle and the lemniscus, in a dorso-ventrad direction, and between the raphé and the external border of the pons, in a lateral direction. There is one case on record (case 26) in which a lesion limited to the inner portion of the formatio reticularis near the raphé in the most cephalad quarter of the pons, and affecting the post. longitudinal bundle chiefly, produced *no* sensory symptoms. It is possible, therefore, that the sensory tracts for tactile sensation no where in medulla or pons approach the raphé, but the number of cases is too few to warrant any general positive statement.

That the sensory tract for the tactile sense lies in the formatio reticularis alone will be more conclusively demonstrated after a review of the other sensory symptoms.

When the cases in which anæsthesia of the face was associated with anæsthesia of the limbs are compared with the cases in which the face alone or the limbs alone were affected, and the situation of the lesion in these three classes of cases are compared, the following diagnostic points can be deduced:

1. *If in any case anæsthesia of one side of the face occurs (not due to neuritis of the trigeminus or to cerebral lesion), the lesion lies in the medulla or pons, in the outer third of the formatio reticularis. Its position in this part is to be determined by the other symptoms present; for, if it is situated high up (cephalad) in the pons, it will be on the side opposite to the anæsthesia, and if it is situated low down (caudad) in the pons or in the medulla, it will be on the same side as the anæsthesia.*

2. *If in any case anæsthesia of the limbs occurs (not due to cerebral lesion), the lesion lies in the medulla or pons, in the inner two thirds of the formatio reticularis, and upon the side opposite to the anæsthesia; or in the spinal cord.*

3. *If one side of the face and the limbs of the opposite side are anæsthetic, the lesion affects the entire lateral extent of the formatio reticularis, and lies in the medulla, or in the pons,* below *the point of union of the ascending and descending roots of the fifth nerve.*

4. *If the face and limbs of the same side are anæsthetic, the lesion lies in the brain at a point* higher than *the junction of the ascending and descending roots of the fifth nerve in the pons. Its position is then to be determined by other symptoms. It may involve the entire formatio reticularis in the upper pons, or crus cerebri; it may be situated in the posterior part of the internal capsule; it may lie in the centrum ovale destroying the radiation of sensory fibres from the internal capsule; it may be in the sensory area of the cortex in which all these traces terminate.*

See Diagram 1.

II. Disturbances of the sensation of pain.

The sense of pain was impaired in cases 4, 5, 6, 9, 10, 11, 19, 20. The subjective sensation of pain was present in cases 19, 21, 24. In a number of the cases no test of this sensation was made. The sensation of pain was found to be normal in cases 1, 2, 7. The distribution of the disturbance of the sensation of pain corresponded in all these cases with the distribution of the anæsthesia, and therefore the conclusions drawn from the disturbances of the tactile sense apply to those of the sense of pain. In the three cases in which the sense of pain was normal the tactile sense was also normal in two, and but slightly affected in the third. In all these three cases the lesion was chiefly in the interolivary tract and lemniscus, and only in the third was the formatio reticularis affected at all. No case of disturbance of the sensation of pain *alone*, accompanied by an autopsy, is on record.

Therefore, until further evidence is brought forward, it

must be accepted that *sensations of pain are transmitted through the formatio reticularis*, and have the same course as tactile sensations.

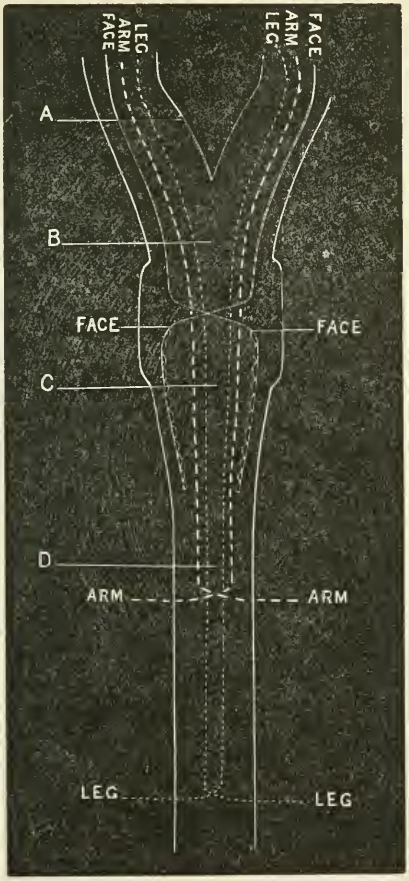


DIAGRAM I.

DIAGRAM OF TRACT CONVEYING TACTILE SENSATION FROM THE SURFACE TO THE INTERNAL CAPSULE.

A.—Lesion in Capsule producing Hemianæsthesia.
B.— " " Crus " "
C.— " " Pons (below upper third) or in Medulla producing Alternating Anæsthesia.
D.—Lesion in Cord producing Spinal Hemianæsthesia.

III. Disturbances of the sensation of temperature.

The sensation of temperature was impaired in cases 4, 5, 6, 7, in all of which cases the disturbance occurred in the

anæsthetic parts. In the remainder of the cases no tests for this sensation were applied. No conclusion can, therefore, be drawn as to the course of the tracts conveying this sensation, although the fact that *disturbances of the sensations of pain and temperature usually occur together* gives a certain probability to the hypothesis that these sensations follow the same tract in both cord, and medulla, and pons.

Subjective sensations of heat and cold were present in a number of cases: of cold, in cases 3, 7, 15, 20; of heat in 6, 10, 24. These subjective sensations had no relation to the anæsthetic parts, but were general, extending to the normal as well as to the affected limbs. In the cases in which the subjective sensation was that of heat, there was also a flushing of the parts in which the heat was felt. It seems probable, therefore, that in these cases the vaso-motor centre in the medulla, the existence of which, in animals, is undoubted, was involved. Foster[1] locates this centre in the medulla in a small area lying just above the calamus scriptorius, and it is a noticeable fact that in these three cases the lesion lay exactly in this region; while in three out of the four cases in which the sensation was one of cold, the same area must have been, to some extent at least, involved, and in two cases (cases 2 and 5) in which there was no subjective sensation, but in which there was other evidence of vaso-motor disturbance, the same area was affected. We have, therefore, eight cases in which *vaso-motor disturbance was associated with a lesion in the upper half of the medulla*, and thirteen cases in which this part was not affected, and in which no vaso-motor disturbance occurred. The *localization of the vaso-motor centre, therefore, which was reached by physiological research is confirmed by pathological observation.*

IV. Disturbances of the muscular sense and consequent ataxia.

[1] "A Text-Book of Physiology." M. Foster, 3d edition, page 218.

Ataxia was present in cases 1, 2, 4, 5, 6, 7, 10, 15, 17, 19, 21, 22, 23, 24.. In cases 1, 2, ataxia was not accompanied by other sensory symptoms. In cases 7, 10, 15, ataxia was marked while the sensory symptoms were slight, in two of these cases the anæsthesia being limited to the face. In the remainder both muscular and tactile senses were affected.

In the cases in which ataxia occurred without affection of tactile sensibility the lesion affected either the interolivary tract in the medulla, or its continuation, the lemniscus, in the pons.

In the cases in which affection of tactile sensibility occurred without ataxia, these parts were not involved in the lesion.

In the cases in which both ataxia and tactile anæsthesia occurred, these parts as well as the formatio reticularis were involved. *The conclusion is warranted that the muscular sense is transmitted along the sensory tracts which lie in the interolivary tract and lemniscus.* In the spinal cord the sensation of muscular sense ascends upon the same side upon which it enters, as we have already seen. But lesions of the interolivary tract and lemniscus produce ataxia in the limbs of the side *opposite to the* lesion. Therefore, *the sensations of muscular sense must decussate in the medulla. They do decussate in the sensory decussation of the medulla*, as is proven by the facts afforded by cases 4, 5, 7, 8, in which a descending degeneration was traced from a lesion of the lemniscus downward along the interolivary tract to the level of the sensory decussation, and in one case through the sensory decussation to the nuclei gracilis and cuneatus.[1] This course, therefore, corresponds to that of the pyramidal motor tracts.

[1] Kahler (*Praguer med. Woch.*, Jan., 1879) was the first to connect the symptom ataxia with a lesion of the interolivary tract. Meyer (*Arch für Psych.*, Feb., 1882) reported his case without alluding to Kahler. Senator (*Arch. für Psych.*, Oct., 1883) cited both these cases and added one of his own, and ascribed the ataxia to a lesion of the lemniscus as well as of the in-

The sensations of pressure and of the location of a limb are conveyed by the muscular sense and are included under it. In the cases in which they were tested and found wanting, cases 1, 4, 5, 6, 7, the disturbance was limited to the parts which were ataxic.

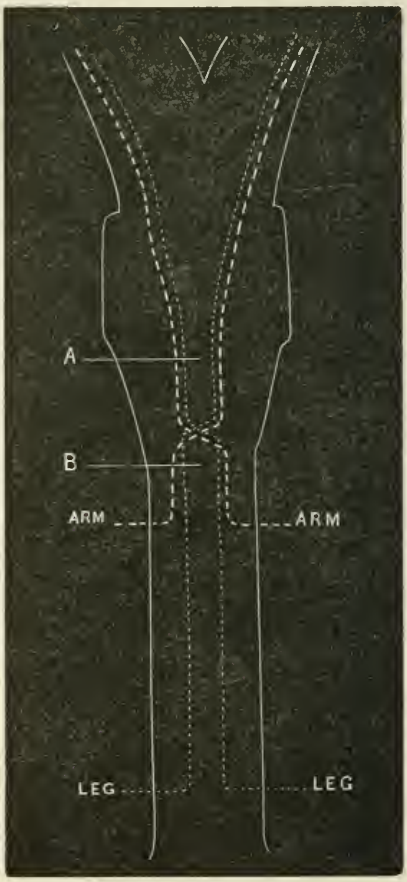


DIAGRAM II.

DIAGRAM OF TRACT CONVEYING MUSCULAR SENSE FROM THE LIMBS.

A.—Lesion above the decussation producing ataxia of opposite side.
B.—Lesion below the decussation producing ataxia of the same side.

terolivary tract. Spitzka. (*Amer Jour. Neurology*, Feb., 1884,) arrived at the same conclusion from his own case and quoted Meyer, but does not allude to Senator. Each of these observers, therefore, may justly claim the independent discovery of the same fact. All the cases hitherto published are here collected for the first time.

The results reached from the analysis of these cases are as follows:

1. The sensory tracts in the medulla are the formatio reticularis and the interolivary tract.

2. The sensory tracts in the pons are the formatio reticularis and the lemniscus.

3. The sensory impulses which have decussated in the spinal cord are conducted along the sensory tract through the medulla pons and crus to the internal capsule without recrossing the median line. These are the sensations of touch, pain, and temperature, and they are transmitted through the formatio reticularis.

4. Those sensory impulses which have not decussated in the spinal cord cross the median line in the sensory decussation of the medulla, and pass upward through the interolivary tract to the lemniscus, in which they ascend to the internal capsule. These are the sensations included under the term muscular sense.

5. If any sensory impulses pass to the cerebellum, they are only those whose result is to awaken reflex action, and they are not the sensory impulses whose reception in the cortical cells is capable of awakening a conscious perception of the sensation. The sensations consciously perceived do not pass through the cerebellum on their way to the cerebrum.

The course of the sensory tracts thus established by pathological cases is the same as that which has been determined by the anatomical and embryological researches of Flechsig, by the one experiment made in accordance with the method of Gudden, and by the investigation of the microcephalic brains in which the motor tracts were absent. It may therefore be accepted as the only one possible in the medulla and pons.

But if it be accepted that the muscular sense passes

along the interolivary tract and decussates in the medulla, while no other sensations so decussate, we may trace this tract downward into the cord, and thus determine the function of a portion of the cord. The interolivary tract is made up partly of fibres from the nucleus cuneatus and partly from fibres from the nucleus gracilis, and these are respectively the terminal stations of the columns of Burdach and of Goll. It is therefore probable that in these columns the muscular sense is transmitted, the column of Goll transmitting this sense from the legs, and the column of Burdach transmitting it from the arms. This hypothesis is substantiated by the fact that these are the columns which are diseased in locomotor ataxia, in which the most marked and constant symptom is a loss of the muscular sense. This does not exclude a transmission of touch also in these columns, since both columns are connected with the formatio reticularis, as well as with the interolivary tract. It makes it probable that Schiff's conclusions regarding animals are true as regards man, and that *in the spinal cord sensations of touch and of the muscular sense pass up in the posterior columns*, while sensations of temperature and pain pass up in other sensory tracts—viz., in the gray matter or in the direct cerebellar columns.

But it is improbable that sensations of pain and temperature pass in the direct cerebellar columns: first, because these columns only commence in the cord above the level of the first lumbar nerve at the level of the Clarke column of gray cells in the posterior median gray matter; and secondly, because these columns go to the cerebellum, while lesions of the cerebellum do not interfere with sensations of pain and heat or cold, while, on the other hand, such sensations are affected in diseases of the pons, through which the direct cerebellar columns do not pass. For these reasons it is probable that *sensations of temperature and pain pass up in the gray matter of the spinal cord.*

What, then, remains as a function for the direct cerebellar columns? I am not aware that any hypothesis has been offered, and where proofs are wanting, hypotheses are worth little. But anatomy and pathology combined seem to indicate a possible function for these columns, as follows:

1. The columns arise (Flechsig) from a set of cells (the Clarke column) whose extent is coextensive with the entrance into the cord of nerves which come from the thoracic and abdominal viscera, and which form a centripetal path for impulses originating within the great cavities of the body. 2. Diseases in the cord which interfere with the function of the direct cerebellar columns (viz.: transverse myelitis—myelitis of any kind) are attended by an irregular action of the organs within the great cavities, especially within the abdomen. The gastric crisis of locomotor ataxia and the habitual constipation of myelitis are examples in point. 3. Diseases of the cerebellum are known to give rise to disturbances of function of the viscera of the great cavities—especially of the abdominal viscera; indigestion, vomiting of a peculiar kind, obstinate constipation, polyurea, albuminuria, etc., being well-known symptoms of cerebellar disease.[1]

For these reasons I would advance the hypothesis that among other functions of the cerebellum that of a reflex centre for the proper regulation of the functions of vegetative life is one, and further that the *path of impulses between the vegetative organs and this reflex centre lies in the direct cerebellar columns of the spinal cord* as far as centripetal impulses are concerned.[2]

[1] It is a noticeable fact that these symptoms of cerebellar disease, *unlike the ataxia*, are not produced by lesions of the vermiform lobe *alone*, but occur when the cerebellar hemispheres are the seat of lesions, such as abscess and softening from embolism and thrombosis, which neither increase the intra-cranial pressure nor affect in any way directly the floor of the fourth ventricle and the pneumogastric centres there or in the flocculas.

[2] The cases here collected present many interesting features aside from those

SENSORY TRACTS IN THE CRURA CEREBRI.

The formatio reticularis and lemniscus continue upward through the tegmentum of the crus lying external to the red nucleus, and pass into the posterior part of the internal capsule. It is the opinion of Flechsig that the lateral lemniscus passes through the internal capsule into the corona radiata of the tegmentum and thus directly to the cortex. Roller, Forel, and Wernicke trace it in part at least to the laminæ medullares of the optic thalamus and make it end in this ganglion. Fibres from the formatio reticularis pass

concerning the sensory tract. The discussion of these symptoms, and of the light which they throw upon the normal functions of the medulla and pons, cannot be entered upon here, as these are subjects aside from the present purpose. The symptoms are, however, analyzed, and are reserved for consideration at another time.

1. Paralysis of the iii. nerve, right. Case 5.
   " " " left. Cases 9, 14, 15.
2. Paralysis of the vi. nerve, right. Cases 5, 11, 19, 26.
   " " " left. Cases 1, 4, 14, 15, 21.
3. Conjugate deviation of the eyes to the right. Cases 6, 9, 26.
   " " " " left. Cases 5, 17.
4. Ptosis. Cases 7, 9, 15.
5. Nystagmus. Cases 1, 4.
6. Diplopia. Cases 4, 6, 9, 19, 21.
7. Myosis. Case 15.
8. Mydriasis. Cases 7, 9.
9. Dimness of vision. Cases 7, 11, 17, 21, 23, 24.
10. Inflamation of the cornea. Cases 5, 10, 14.
11. Paralysis of vii. nerve, right. Cases 4, 5, 11, 12, 15, 17, 18, 20, 23.
    " " " " left. Cases 6, 9, 10, 14, 17, 19, 22,23, 24, 25.
12. Trismus. Cases 9, 10, 17.
13. Deafness in the right ear in cases 10, 12 ; in the left ear in cases 9, 14, 24.
14. Tinnitus aurium in case 5.
15. Difficulty in swallowing in cases 2, 3, 5, 6, 7, 11, 12, 14, 16, 22, 24.
16. Difficulty in speaking, not aphasia, in cases 2, 3, 4, 7, 10, 11, 12, 15, 16, 18, 22, 23.
17. Paralysis of the XII. nerve, right, in cases 2, 4, 6, 7, 10, 12, 20.
    " " " " left in cases 2, 5, 6, 11, 16, 18, 24, 25.
18. Paralysis of arm and leg, right, in cases 6, 10, 14, 15, 17, 20, 21, 22, 23.
    " " " left, in cases 5, 11, 14, 16, 18, 19, 24.
19. Increased knee-tendon reflex, right, in cases 6, 7.
    " " " left, in case 5.
20. Absence of the skin reflexes, right, in cases 6, 7.
    " " " left, in case 5.

Situation of the Lesion.

On the right side in cases 2, 5, 11, 12, 13, 15, 16, 18, 19, 23.
" left " " 1, 2, 3, 4, 6, 7, 8, 9, 10, 14, 17, 20, 21, 22, 24, 25.
On both sides in case 2.
In the medulla alone in cases 1, 2, 3, 6, 9, 24.
In the pons alone in cases 10, 11, 13, 14, 15, 16, 17, 19, 20, 21, 22, 25, 26.
In both medulla and pons in cases 4, 5, 7, 8, 12.

to the substantia nigra of the crus and end in it. Others pass to the laminæ medullares of the optic thalamus and end in it. Others still unite with fibres from the red nucleus, and leave the tegmentum in a large bundle which passes through the internal capsule in its posterior part and radiates toward the cortex (the Haubenstralung of Flechsig). No attempt has been made to assign functions to the various bundles of fibres issuing from the tegmentum.

Cases of lesion of the crus are rare, and those on record are chiefly lesions of the pes and not of the tegmentum. Nothnagel was able to cite but nine cases, and his conclusion as to the sensory tracts is merely negative. They do not lie in the inner two thirds of the pes, as lesions there give rise to motor symptoms only.

Two cases are on record in which the red nucleus has been diseased, and in neither of these was sensation affected.[1] The red nucleus is in anatomical connection with the opposite superior cerebellar peduncle, as is proven by the fact that atrophy of one cerebellar hemisphere is accompanied by atrophy of the opposite red nucleus. Lesions of the corpora quadrigemina usually involve the red nucleus, and in these there is no disturbance of general sensation.[2] Such lesions may produce incoördination which indicates a functional relation between the red nucleus and the cerebellum. There is, therefore, no reason to assign sensory functions to the red nucleus, or to suppose that the sensory tracts pass in the inner half of the tegmentum.

It is not probable that the sensory tracts pass in the outer third of the pes, for this part consists of fibres which connect the cerebrum and cerebellum, and is absent when either the cerebrum or cerebellum is absent.[3]

[1] Kahler: *Arch. f. Psych.*, x. Kahler and Pick: *Zeitsch. f. Heilk.*, ii., p. 305.

[2] See Wernicke: "Lehrbuch d. Gehirn.," vols. ii. and iii., sections on Corp. Quad.

[3] Compare Flechsig's case of deficient cerebellum with my case of deficient cerebrum, in both of which this part was absent.

It is therefore probable that in the crura the sensory tracts lie in the outer half of the tegmentum, in which part lie the formatio reticularis and lemniscus, which have been shown to be the sensory tracts in the pons. There are no pathological facts to prove or to oppose this hypothesis. It is reached by a method of exclusion, but until further facts are offered may be accepted.

SENSORY TRACTS IN THE BRAIN.

I.—*Anatomical.*

The formatio reticularis terminates at the upper level of the crura, and from this point fibres pass into the posterior third of the internal capsule. Into this part of the capsule the lemniscus also passes. These tracts here become indistinguishable, but are known to lie in the internal capsule, as they did in the pons, posterior to the motor tract. It is well-known that lesions of the posterior part of the internal capsule produce anæsthesia of the opposite side of the body; a pathological fact which confirms the course of the sensory tract through this part.

At the upper level of the internal capsule its fibres radiate toward the cortex. The point of radiation of the fibres which form the sensory tract is known as the "carrefour sensitif." This is just opposite the middle of the optic thalamus and at its upper level. Here, too, lesions produce hemianæsthesia.

Above the "carrefour sensitif" the nerve fibres from the capsule become so mingled with radiating fibres from the optic thalamus that in an adult brain they cannot be distinguished. Flechsig has found, however, that the former obtain their medullary sheaths at an earlier date than the latter in fœtal life. He was thus able to trace the sensory tract. He affirms that it approaches the posterior and inner border of the motor tract, and in the corona radiata passes to the cortex of the parietal lobe where it ends in

the convolutions lying posterior to the fissure of Rolando, and anterior to the occipital lobe of the brain.

From a review of the facts just stated it is evident that a direct anatomical connection can be traced from the surface of the body to the parietal cortex of the brain, by means of the sensory nerves, the sensory nerve-tracts in the cord, medulla, and pons, and in the interior of the brain itself.

It is admitted that no one nerve fibre can be followed from a point on the surface to a point in the brain. In fact it seems to be a law of nervous transmission that impulses are always modified in their course by passing into and then issuing from nerve cells. In the course of sensory impulses the cells in the gray matter of the cord, and probably those in the gray matter of the medulla, crus, and optic thalamus are traversed by some, if not by all of them. But however such cells may modify the impulse they do not interrupt it, and their presence does not affect the fact of a connection between the surface of the body and the cortex of the brain. The most considerable collection of cells in this tract is the optic thalamus, and for many years it has been thought to take a large part in the reception and transmission of sensory impressions. Recent investigation of cases of thalamus disease by Nothnagel[1] and Wernicke[2] has thrown some doubt upon its sensory function, as far as the sensations of touch, temperature, and pain are concerned. And it is still an open question whether the sensory symptoms occurring in its diseases are due to an implication of sensory centres, or to an affection of the sensory tracts passing along its periphery in the internal capsule. Until further facts are offered, however, the conclusions of Flechsig must be accepted, since these are the only ones well established and of value.

Anatomical study, pursued by the various methods of in-

[1] Nothnagel: "Topische diagnostic d. Gehirnk."

[2] Wernicke: *l. c.*, vol. ii. and iii.

vestigation at present known, leads, therefore, to the conclusion that the tracts conveying sensations of touch, pain, temperature, and the muscular sense, pass to the parietal region of the cortex of the hemispheres.

It is not known whether *all* the sensations from one side of the body pass to the opposite hemisphere of the brain. The pathological facts here cited would indicate that they do. It must be remembered, however, that for many years, and until a very recent date, it was supposed that a lesion of one hemisphere produced a paralysis upon the opposite side of the body *alone*. It is now admitted that, in as much as each hemisphere is connected with both sides of the body by motor tracts—the larger of the motor tracts decussating, and the smaller not decussating in the medulla,—a lesion of one hemisphere produces paralysis upon the opposite side of the body, and a certain amount of weakness upon the same side of the body. It has recently been established that a lesion of one hemisphere in the visual area produces, not blindness in the opposite eye as was formerly supposed, but a certain degree of blindness in both eyes, that in the opposite eye being greater in extent than that in the eye of the same side.[1] The olfactory fibres pass from each bulb to both hemispheres. The argument from analogy would therefore indicate that, as regards other sensations, each half of the brain is in relation with both sides of the body, its connection with the opposite side being more extensive than that with the same side. The probability therefore is, that *all* the sensations from one side of the body do *not* pass to the parietal cortex of the opposite side; but that while the majority so pass, a portion go up to the cortex of the same side from which they come. This probability is somewhat strengthend by the fact that

[1] See Mauthner: "Vorträge aus des Gesammtgebiet der Augenheilkunde." "Gehirn und Auge," Wien, 1883. Also Wernicke: *l. c.*, vol. i., p. 69. Also Starr: *Amer. Jour. Med. Science*, Jan., 1884.

complete hemianæsthesia from cortical disease is far less common than complete hemiplegia, a fact which would find an easy explanation if it could be shown that both sides of the body were connected by sensory tracts with each half of the brain. A more careful and accurate examination of future cases may furnish facts to decide this question. At present it can only be stated that a connection has been established between the sensory tracts and the parietal regions of the brain.

II.—*Physiological.*

The researches of physiologists in determining the functions of the brain have been productive of the most successful and brilliant results ever achieved in that department of science. From the beginning of investigation of this subject by Fritsch and Hitzig in 1870 to the latest experiments of Munk in the present year, a continuous succession of interesting and valuable facts have been discovered. This is not the place to trace the history of these discoveries, or to give in detail the methods pursued. The results which bear upon the subject in hand—the location of the sensory areas in the cortex—must be considered briefly.

Ferrier was the first to attempt to determine the sensory area of the cortex. Recognizing the fact that in animals no reliable information of sensory disturbance due to *irritation* of the cortex could be obtained, he resorted to the method of extirpating various areas of the cortex with a view of producing anæsthesia. As the result of several experiments, one of which is given in detail,[1] he concluded that the tactile centre was to be found in the hippocampus major and the uncinate convolution, in the monkey. As he admits that other regions, especially the occipital cortex and part of the corona radiata, were injured in the course of the experiment, the result cannot be accepted as con-

[1] Ferrier : "Functions of the Brain," p. 179.

clusive. That, in the monkeys operated on, tactile sense was destroyed, may be admitted, though it is not stated whether the loss of tactile sense was permanent or only temporary. But since, in destroying this sense, several regions of the brain were destroyed and the internal capsule was injured, it seems somewhat arbitrary to limit the function to one of the regions destroyed. That the tactile centre in man lies in the hippocampal region cannot be admitted; for, first, we have already seen that the sensory fibres do not pass to this region; and second, we shall see, in the pathological section, that lesions of this region do not produce anæsthesia.

The conclusion of Ferrier therefore stands without anatomical or pathological support.

It is probably from a repetition of his earlier experiments that Ferrier has been recently led to change his opinion. In his last announcement (Nov., 1883, Oration before the Med. and Chirurg. Society, London, rep. in *Brit. Med. Jour.*, Nov. 30, 1883) he says: "I have all along held, and hold, both on experimental and on clinical grounds, that the centres of common sensation, including muscular sensibility, are anatomically distinct from those of motion, and are situated in the subcortical region." He thus retracts the statement that sensory centres are situated in the hippocampal region and refuses to locate them on the cortex at all.

The experiments of Munk are more numerous, more carefully conducted, and more complete in observation and description, and his conclusions conform to both anatomical and pathological facts. His method is to extirpate limited areas of the cortex in various regions, and after the animal has entirely recovered from the temporary effects of the operation, to determine what functions are impaired. The animals used were dogs and monkeys. Munk has found

that the area of common sensation (Fühlsphäre)—including sensation of pressure, location of a limb, muscular sense, and touch, lies in the central region, including the anterior and posterior central convolutions, and in the adjacent portions of the cortex. He divides this area into special regions, for the hind leg, fore leg, head, eye, and ear muscles, neck and body. These regions coincide with, but are somewhat more extensive than, the corresponding motor centres for these parts. Destruction of any one of these regions, therefore, produces paralysis and anæsthesia in the parts with which it is connected. The paralysis Munk ascribes to the loss of the memory of the combination of impulses necessary to produce the desired motion (innervationsgefuhl and bewegungsvorstellungen). The anæsthesia persists for ten weeks after the extirpation of a single centre, after which time the animal recovers, the adjacent region taking up the function of the one destroyed. If the entire area, however, is extirpated, the anæsthesia is permanent and complete. To produce complete anæsthesia the entire parietal cortex and a portion of the frontal cortex, must be destroyed. The sensory portion of the cortex is therefore assigned by Munk to the parietal area and central region of the brain.[1] This conclusion is in harmony with the anatomical fact already stated, that this portion receives the sensory tracts.

The experiments of Goltz,[2] though interpreted by him as contradictory to the theory of localization, really substantiate indirectly the conclusion of Munk as opposed to that of Ferrier.

Goltz extirpated the greater part of the external convex surface of the hemispheres, leaving the portions at the base of the brain intact. He thus destroyed Munk's Fühlsphäre

[1] Munk's conclusions are to be gathered from his reports to the Physiol. Soc, of Berlin, to be found in *Pfluger's Arch. f. Physiologie.*

[2] Goltz: "Verrichtungen des Gehirns."

but did not injure the hippocampal region—Ferrier's area of touch. According to his description of the animals thus experimented upon, there was at first a decided loss of sensation to touch, pain, and the muscular sense, though he claims that this was not complete, and that after a time the animal recovered. While his experimental results are interpreted by him as contradicting the results of other experiments in favor of localization, it is evident that, as regards this special subject of the localization of the sensation of touch, his results are in accord with those of Munk and are opposed to those of Ferrier.[1]

The most recent experiments to determine the sensory centres of the cortex are those of Tripier, of Montpellier, France, reported in the *Revue Mensuelle de Médecine*, 1880–1881. Like other experimenters, Tripier has extirpated various areas of the cortex, and observed the results. He reaches the same conclusion as Munk, and affirms the existence of sensory centres on the so-called motor area. He finds that the sensory area for a definite limb coincides with, but is more extensive than, the motor area for the same limb. He thus arrives at a similar arrangement of sensory areas to that already described by Munk.

At the same time that Tripier was working in France, Moeli in Berlin was investigating the same subject. His results are published in *Virchow's Arch.*, Bd. 76, and coincide with those of Munk and Tripier.

Thus three experimenters, working independently, arrived at the same time at a similar conclusion, viz.: that the motor and sensory areas of the brain for any one limb coincide.

But physiological experiment, however precise, affords information concerning the functions of the brain in animals only. It is solely by means of the study of clinical cases

[1] This is not the place to discuss the merits of Goltz' objections to localization. The reader is referred to the *Journal of Physiology*, Dec., 1883, for a complete review and careful criticism of the respective results of Goltz and Ferrier.

that results can be reached in the case of man. The final appeal must, therefore, be made to pathology.

III.—*Pathological.*

Are there any cases on record in which the occurrence of sensory symptoms during life must be ascribed to a limited cortical lesion discovered at an autopsy and accurately localized? When Nothnagel wrote his great work "Topische diagnostic der Gehirnkrankheiten," in 1879, he was obliged to say: "Disturbances of sensation have as yet no bearing upon the diagnosis of cortical lesions." The stimulus given to the observation of brain lesions by Nothnagel's work was productive of many careful records of cases, and in 1880 Exner was able to find twenty two cases in the journals, in which sensory disturbance had been associated with cortical disease. In all of these cases he found the lesion to be within or very near to the central or motor region.[1] No general collection of cases of cortical lesion has been made since that of Exner; the cases collected by Charcot (*Rev. de Méd.*, 1883) being studied solely from the point of view of motor symptoms, and no mention being made of the condition of sensation in the majority of the cases. Exner did not have access to American journals when making his collection. I have, therefore, examined the American journals for the past twenty years, and the chief European journals published between Jan., 1880, and Jan., 1884, and have found a large number of cases in which sensory symptoms have been noticed, and in which a *post-mortem* examination has shown a lesion in the cortex of the brain. It is to the study of these cases that we at once proceed, the cases being first cited and then analyzed.

In the cases chosen there was but *one lesion of limited extent*, situated *in the cortex* of the brain, and *not affecting the*

[1] In sixteen of Exner's cases the central convolutions were actually involved. In the others the parietal convolutions near the post-central conv. were affected.

basal gauglia. These are the only cases from which conclusions can be legitimately drawn, and therefore all others are ruled out from consideration, although a large number were found in which sensory symptoms were associated with extensive or multiple lesions of the cortex.[1]

ANALYSIS OF THE CASES.

In all the cases here collected sensory symptoms were present. In all these cases an autopsy showed a diseased condition, limited to the cortex of the hemisphere of the opposite side, in the anterior or posterior central convolutions, or in the convolutions of the parietal lobules,[2] all other parts of the brain being normal.

The sensory symptoms were of different kinds, which may be classified into (1) symptoms of irritation of the cortex, including hyperæsthesia, hyperalgesia, paræsthesiæ, and subjective pain, and (2) symptoms of destruction of the cortex, including anæsthesia, analgesia, and ataxia. In a number of the cases, the first class of symptoms were succeeded by the second class in the course of the disease.

1. Sensory symptoms indicating irritation of the cortex.

These occurred in fourteen cases. In some the irritation was temporary, occurring just before or just after an epileptic convulsion. In these cases the condition was one of cortical epilepsy, and the diagnosis was confirmed by the autopsy, the local lesion being found in that part of the motor area corresponding to the muscles which first became convulsed. It is therefore evident that the sensory aura of cortical epilepsy may be brought into connection with cortical disease in the sensory-motor area of the brain.

In some of the cases the irritation was permanent, being due to disturbances of circulation in the cortex set up by a

[1] The American cases in this collection are cited by me in full in the *Amer. Jour. of the Med. Sci.* for July, 1884.

[2] For convenience and to avoid repetition, these convolutions are included in the term "*sensory-motor area*."

Case.	Sex.	Age.	Symptoms.	Duration.	Lesion.	Situation.	Reported by.
1	M.	67	Anæsthesia l. face and arm.	4 mos.	Softening from thrombosis.	A. C., P. C.; m. 1-3.	Petrina: *Zeit. f. Heil.*, ii., 388.
2	M.	30	" r. " "	3 mos.	Hemorrhage.	Sylvian Reg. A. C. l. 1-3.	" " ii., 388.
3	M.	33	Analgesia r. " "	3 wks.	Tubercle.	3 F. A. C. l. 1-3.	" " ii., 388.
4	F.	39	Paræsthesia r. " "	4 mos.	Gumma.	P. C.; l. m. 1-3.	Sands: *Med. News*, April, 1883.
5	F.	20	Analgesia r. " and body.	3 wks.	Softening.	A. C.; l. 1-3 3 F.	Petrina, *l. c.*
6	M.	28	Paræsthesia of arm.	2 yrs.	Softening.	P. C. m. 1-3; Ang. Gy.	Dieshfeld: *Practitioner*, May,'75.
7	M.	28	" "	2 yrs.	Tubercle.	A. C., P. C.; m. 1-3.	Edinger: *Arch. Psych.*, x., 93.
8	F.	18	Hyperæsthesia of r. arm and leg.	5 mos.	Sarcoma.	Sup. Par. Lob.	Monakow: " " xi., 626.
9	M.	28	Anæsthesia of r. arm.	10 days.	Abscess.	A. C., P. C.; m. 1-3.	Bender: *Deut. med. Woch.*, No. 50, 1882.
10	M.	27	" l. "	1 mo.	Tubercle.	A. C., P. C.	Bramwell: *Edin. Jour.*, xxiv.,145.
11	F.	56	" l. "	4 yrs.	Cyst softening.	Inf. Par. Lob. P. C.; m. 1-3.	Carter: *Med. Times and Gaz.*, ii., 399, 1880.
12	F.	60	" r. "	1½ yrs.	Old clot.	3 F.; A.C., l. 1-3, Is. Reil.	Wood: *Phil. Times*, v., 470.
13	M.	34	" r. "	3 mos.	Abscess.	Inf. Par. Lob. P. C.; m. 1-3.	Cock: *Amer. Jour.*, Oct., 1852.
14	M.	19	" l. "	1½ mos.	Softening.	A. C.; m. 1-3.	Noyes: " July, 1882.
15	M.	50	Paræsthesia of l. arm and leg.	4 mos.	Sarcoma.	A. C., P. C.; m. u. 1-3.	Peabody: *Arch. Med.*, April, '82.
16	F.	27	" l. " "	2 days.	Softening from thrombosis.	A. C., P. C.; m. u. 1-3	Bumstead: " Oct., '79.
17	M.	53	" and ataxia of l. arm and leg.	7 mos.	Tubercle.	Syl. Reg. Isl. Reil.	Seaman: *Phil. News*, Jan., 1883.
18	M.	54	Anæsthesia of r. arm and leg.	6 mos.	Abscess.	A. C., P. C ; m. u. 1-3.	Page: *Med. & Surg. Rep.*, xxi., 29.
19	M.	35	Paræsthesia of l. arm and leg.	2 yrs.	Gumma.	A. C., P. C.; m. u. 1-3.	Morton: *Chic. Jour. & Exam.*, xlvi., 21.
20	F.	38	Hyperæsthesia of l. " "	Not stated.	Gumma.	A. C., P. C.; u. 1-3.	Mills: *Arch. Med.*, Aug. 1882.
21	M.	57	Hyperæsthesia and anæsthesia of l. arm and leg.	"Some months."	Tubercle.	Sup. Par. Lob.	Petrina, *l. c.*
22	F.	2¾	Anæsthesia r. arm and leg.	2 days.	Tub. meningitis.	A. C., P. C.; Par. Lob.	Cerf: *Arch. klin. Med.*, xxxi., 431.
23	F.	2½	" r. " "	23 days.	Abscess.	Sup. Par. Lob.	Smith: Jour. Ment. and Nerv. Dis., July, 1880.
24	M.	66	" r. " "	18 mos.	Softening.	A. C., P. C.; m.l. 1-3 Inf. Par. Lob.	Mills: *Phil. Times*, ix., 246.
25	M.	—	" r. " "	28 days.	Abscess.	P. C. mid. 1-3 Inf. Par. Lob.	Liddell: *Amer. Jour. Med. Sc.*, July, 1883.
26	M.	36	" and ataxia l. arm and leg.	2 mos.	Softening.	P. C. and Inf. Par. Lob.	Carson: *Practitioner*, xv., 217.
27	F.	30	" l. arm and leg.	10 mos.	Carcinoma.	P. C. m. 1-3; Inf. Par. Lob.	Mills: *Arch. Med.*, Aug., 1881.
28	F.	53	" l. " "	7 mos.	Glioma.	A. C., P. C.; mid. 1-3.	Wood: *Amer. Jour. Med. Sc.*, April, 1864.
29	M.	—	L. hemianæsthesia.	1 yr.	Sarcoma.	A. C., P. C.; u. 1-3 Sup. Par. Lob.	Seguin: *Trans. Neuro. Soc.*, 1877.
30	F.	27	L. "	2 mos.	Sarcoma.	A. C., P. C.; m. u. 1-3.	Bell: *Amer. Jour.*, July, 1870.
31	M.	30	L. "	6 mos.	Tumor.	A. C., P. C.; m. u. 1-3.	Janeway: *Med. Record*, ix., 651.
32	M.	33	R. "	1¼ mos.	Abscess.	P. C., Inf. Par. Lob.	*Hospital Gazette*, vi., 552.
33	M.	50	R. "	3 mos.	Softening from thrombosis.	A. C. l. 1-3; Is. Reil.	Petrina, *l. c.*
34	M.	67	R. "	22 days.	Softening.	A. C., P. C. l. 1-3; Is. Reil.	Tripier: *Rev. Mens.*, 1880, p. 138.
35	F.	57	R. "	20 days.	Softening.	A. C., mid. 1-3.	" " 1880, p. 135.
36	F.	23	R. "	1 mo.	Softening from emb.	A. C., P.C. m.l. 1-3; Inf. Par. Lob.	Dejerine: *Prog. Méd.*, viii., 781.
37	F.	42	R. hyperalgesia.	3 days.	Glioma.	A. C., P. C., l. 1-3.	Cheesman: *Arch. Med.*, Aug., '81.
38	M.	66	R. hemianæsthesia.	4 mos.	Softening.	A. C., P. C. l. 1-3; Par. Lob.	Mills: *Med. Bulletin*, i., 13.
39	F.	51	R. "	2 yrs.	Clot.	Is. Reil.; Inf. Par. Lob.	Richardson: *Richmond Med. Jour.*, iii., 426.
40	M.	72	R. "	20 yrs.	Hemorrhagic cyst.	A. C., P. C.; Is. Reil.	Starr: *Amer. Jour.*, July, 1884.
41	M.	42	R. "	Not stated.	Clot.	Inf. Par. Lob.	Tripier, *l. c.*

lesion (embolism, thrombosis, hemorrhage), or to pressure upon the cortex by a tumor or clot. It was in these cases that the lesion, after for a time irritating the cortex, produced its disintegration, and the symptoms of irritation were followed by those of destruction.

2. Symptoms of destruction of the cortex.

These occurred in thirty-two cases. In all of these cases the loss of sensation was permanent, but in none of them was it complete. This fact adds probability to the hypothesis already advanced, that *each* side of the body is connected by sensory tracts with *both* sides of the brain, destruction of the sensory area of one half of the brain producing great impairment, but no absolute loss of sensation, in the opposite side of the body. This hypothesis also offers an explanation for some of the numerous cases which are on record, in which a careless examination of the condition of sensation failed to detect any anæsthesia, but in which a disease of the sensory-motor area of the brain was discovered after death. There are many such cases to be found in medical literature, and Ferrier has cited them in opposition to the views of Exner, who, as already stated, was the first to connect sensory symptoms with lesions of the motor area. Such cases are, however, of little value, since in many of them no mention is made of the condition of sensation, and in many the tests applied to detect the various kinds of disturbance of sensation were not accurately applied. Like the cases cited by Brown-Séquard in opposition to all the facts of localization, these cases do not bear a strict examination.

The forty-one cases here collected, together with the twenty-two cases collected by Exner, afford sufficient ground for the conclusion that lesions affecting the cortex of the brain in the central and parietal convolutions, may give rise to sensory symptoms.

It is well known that definite regions of the central convolutions of each hemisphere govern definite portions of the muscular system. The motor area for the opposite half of the face and tongue lies in the lower third of the central convolutions. The motor area for the opposite arm lies in the middle third of the central convolutions. The motor area for the opposite leg lies in the upper third of the central convolutions, including the paracentral lobule.[1]

It is interesting to find that a similar distribution of the sensory areas may be affirmed. In the cases here collected, the lesion lay in the lower third of the sensory motor area when the face was affected by sensory disturbances; the lesion lay in the middle third of this area when the arm was affected; the lesion lay in the upper third of this area when the leg was affected. When sensory symptoms occurred in both face and arm, the lower and middle areas, or their junction, were found diseased. When sensory symptoms occurred in both arm and leg, the middle and upper areas, or their junction, were found diseased. And it is a noticeable fact that in no case were face and leg affected together without implication of the arm, a fact which affords a clear indication that their areas are separated by that of the arm.

It is therefore justifiable to conclude that:

1. In the cortex of the brain sensations of touch, pain, temperature, and the muscular sense are perceived.

2. These perceptions occur in the gray matter of the anterior and posterior central convolutions and of the parietal convolutions; sensations from one side of the body being perceived in the opposite half of the brain in a more intense degree than in the same half of the brain.

[1] See Charcot: "Localization des Maladies Cérébrales," Paris, 1876; and collections of cases in the *Rev. de Méd*, 1877, 1879, and 1883.
Nothnagel, "Topische diagnostic der Gehirnkrankeiten," 1879.
Exner, "Untersuchungen ub. d. Local. d. Functionen in d. Grosshirnrinde," 1880.
Wernicke, *l. c.* Ferrier, "Localization of Functions in the Brain," 1878.
Starr, *Amer. Jour. Med. Sci.*, April and July, 1884.

3. The various sensory areas for various parts of the body lie about and coincide to some extent with the various motor areas for similar parts—the area for the face, arm, and leg, lying in the lower, middle, and upper thirds of the sensory-motor region respectively.

4. While the motor area is confined to the central convolutions, the sensory area includes to some extent the convolutions of the parietal lobe which lie adjacent and posterior to them. It is therefore more extensive than the motor area.

These conclusions are further strengthened by a review of the cases of lesion of other areas of the cortex. Such cases cannot be cited here.[1] It may, however, be affirmed that in cases of disease limited to the frontal, temporal, sphenoidal, or occipital regions of the brain disturbance of sensation is not observed.[2]

GENERAL CONCLUSIONS.

A study of the anatomy of the central nervous system by the various methods at present used, an examination of embryological facts both in normal infants and in cases of abnormal development, a review of the results of physiological experiment and a collection of pathological cases of small lesion limited to definite tracts and to definite areas of the cortex of the brain, have led to the following conclusions:

A.—The surface of the body is connected with a definite region of the surface of the brain by distinct tracts which convey sensory impressions. These impressions enter the spinal cord by the posterior nerve-roots, and then ascend in different tracts.

(*a*) The impressions destined to awaken the sensation of

[1] See authorities cited in last note.

[2] This is also the result reached from a study of the American cases which I have collected and published in the *Amer. Jour. of Med. Sci.*, April and July, 1884.

touch pass at once to the opposite half of the spinal cord, to a great extent if not entirely, and ascend in the posterior white columns, the impressions from the legs passing in the posterior median, and those from the arms in the posterior lateral columns. On reaching the medulla these impressions pass to the formatio reticularis of the same side on which they were in the cord, and ascending in this tract through the pons and crus, reach the internal capsule, where they are conducted by a tract lying in the inner half of its posterior third to the corona radiata, whence they diverge to the cortex of the central and parietal regions, the impressions from the leg being perceived in the upper third of these regions, and those from the arm being perceived in the middle third of these regions.

(*b*) The impressions destined to awaken the sensations of pain and temperature also cross the median line immediately after entering the spinal cord, and pass up in the gray matter of the cord, probably in its posterior inner part. On reaching the medulla they enter the formatio reticularis, and from this point their course is identical with that of the impressions of touch already traced.

(*c*) The impressions destined to awaken the sensation of the location and of the motion of a limb ascend in the spinal cord, in the posterior white columns of the same side upon which they enter; the muscular sense from the legs passing up in the posterior median, and that from the arms in the posterior lateral columns. On reaching the medulla, these impressions pass to the opposite side through the sensory decussation, then ascend in the interolivary tract, to the pons, where they enter the lemniscus, and gradually turn outward from the median line as they pass up through the pons. In the crus, these impressions are conducted by the lateral lemniscus, which lies in the outer third of the tegmentum, and which passes directly into the internal capsule,

there being situated in the middle part of the posterior third, external and in close approximation to the sensory tract of touch already described. From this point, the course of these kinds of impressions cannot be distinguished from one another, and their termination is in the cortex of the central and parietal regions, the muscular sense of the leg being perceived in the upper third, and that of the arm in the middle third, of these regions.

B.—Sensory impressions from the face enter the pons by the sensory root of the trigeminus, and pass downward to sensory cells which lie in the lateral portion of the formatio reticularis, and which are arranged in a column extending from the junction of the upper and middle third of the pons, to the lower limit of the medulla. The upper portion of this column receives the fibres from the upper branch of the nerve ; the middle portion from the middle branch, and the lower portion from the lower branch. The course of sensory impulses, from these sensory cells to the brain, is in the lateral part of the formatio reticularis of the same side upon which they enter, up to the junction of the upper and middle thirds of the pons, where they cross the median line and join the sensory impressions from the body in the formatio reticularis. In the crus cerebri, these impressions pass in the outer half of the tegmentum, and, thence entering the posterior third of the internal capsule, pass on to the corona radiata. In the divergence of fibres in the corona, these impressions pass along the lowest radiation, and thus reach the lower third of the central and parietal regions, where they are perceived. It is, as yet, impossible to distinguish between the course of tactile, painful, and muscular impressions from the face to the cortex of the brain.

C.—Sensory impressions from the great cavities of the body enter the spinal cord by the posterior nerve-roots, and are probably received by sensory cells which lie in the inner

and posterior part of the gray matter of the spinal cord—the Clarke column of cells. From these cells, these impressions pass outward to a white column lying in the lateral periphery of the cord—the direct cerebellar column—in which they pass upward to the medulla, and on through the restiform body to the cerebellum, to terminate either in the nucleus dentatus or in the cortex, or in both. The existence of a decussation of these impulses is undetermined, but, if it occurs, it must take place soon after their entrance, as each direct cerebellar column passes to that hemisphere of the cerebellum of the same side upon which it lies. The impressions thus transmitted to the cerebellum are not those of touch, pain, temperature, or the muscular sense, and are probably impressions connected with the functions of vegetative life and destined to awaken reflex actions.

If the facts here presented prove of value in aiding the diagnosis of local foci of disease in the central nervous system, the object of the author will be attained.

NEW YORK, No. 29 East 62d St.

A Resumé of Lectures upon the Structure and Physiology of the Central Nervous System, with a Localization of its Lesions.

DELIVERED BY

DR. C. EUGENE RIGGS

BEFORE THE MEDICAL CLASS OF THE UNIVERSITY OF MINNESOTA.

1889-90.

PREFATORY NOTE.

This resume' fulfils a promise made to my class of last year that I would endeavor to give them in a tangible and concise form the results of the latest research into the anatomy and physiology of the central nervous system. In so doing it will be observed that I have added to already familiar facts, much material of recent publication.

The Review of the Map of the Human Brain and the Localization of the Functions of the Segments of the Spinal Cord I have been able to add through the courtesy of Dr. Starr. They will prove invaluable aids to the student of nervous anatomy.

The Spinal Cord.

V.

THE ANATOMY AND PHYSIOLOGY OF THE SPINAL CORD, WITH LOCALIZATION OF ITS LESIONS.

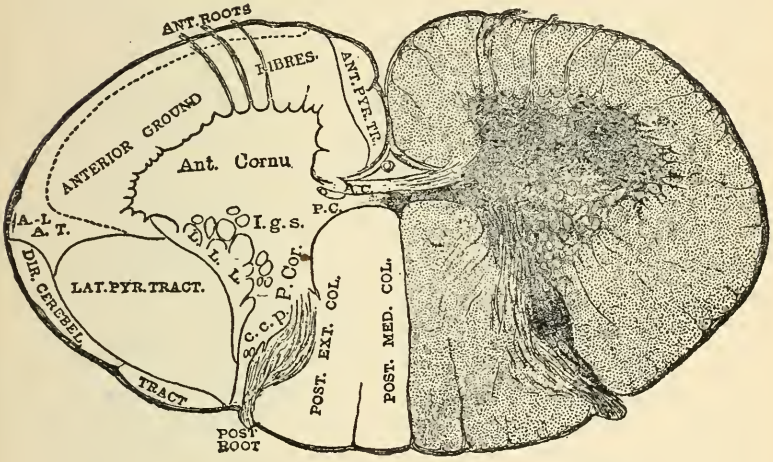


DIAGRAM I.

Section of the Spinal Cord in Cervical Region. A. C., anterior commissure; P. C., posterior commissure; I. g. s. intermediate gray substance; P. Cor., posterior cornu; c. c. p., caput cornu posterioris; L. L. L., lateral limiting layer; A.—L. A. T., antero-lateral ascending tract, which extends along the periphery of the cord. (After Gowers.)

COLUMNS OF SPINAL CORD.	THEIR FUNCTIONS.
Crossed Pyramidal Tract,	Voluntary Motion.
Direct Pyramidal Tract,	Voluntary Motion.
Col. Goll,—Post. median Col.,	Muscle and Tactile Sense.
Col. Burdach,—Post. ext. Col.,	Unknown. "Habitat of Tabes."
Direct Cerebellar Tract,	Muscle Sense from Trunk. (Flechsig.)
Ant. Lat. Ascending Tract,	Temperature and Pain.
Ant. Ground Fibres,	Commissural Fibres.
Lat. Limiting Layer,	Commissural Fibres.

For a different view of these functions, see "Secondary Degenerations of The Spinal Cord."—(TOOTH.)

The MOTOR TRACT—direct and crossed,—arises in the motor area of the cerebral cortex, descends through the internal capsule, crus, pons and anterior pyramid, into the cord; 80 per cent. of the fibres decussate in the medulla, forming the crossed pyramidal tract of

the opposite side, and extend to the end of the cord. The remaining fibres form the direct pyramidal tract of the same side, usually terminating about the mid-dorsal region; these fibres cross (?) in the anterior commissure and pass to the anterior cornu of the opposite side.

Sensory Tract. *a.* The fibres conveying impressions destined to awaken muscle sense ascend in the column of Goll, decussate in the medulla (sensory decussation of Meynert) and pass to the opposite side, whence coursing through the lemniscus, or interolivary tract, to the pons, there entering the fillet, they pass through the crus by way of the upper fillet and at once enter into the middle third of the posterior division of the internal capsule, external and in close approximation to the sensory tract of touch, temperature and pain, and terminate in the cortex of the central and parietal regions.

a. According to Flechsig (quoted by Gowers) the direct cerebellar tract, which, arising from the lower dorsal region, leaves the lateral column at the level of the first cervical nerve and passes up in the restiform body to the cerebellum, conveys muscle sense from the lower part of trunk.

b. The fibres conveying impressions destined to awaken tactile sense decussate and pass to the opposite side of cord, ascend the column of Goll and in the medulla course in the formatio reticularis of the same side of which they were in the cord, thence ascending in this side through the pons and crus, reach the internal capsule, lying in this tract in the inner half of the middle third of the posterior division. They terminate in the cortex of the central and parietal regions.

c. The fibres conveying impressions destined to awaken sensations of temperature and pain cross the median line immediately after entering the cord, pass up in the antero-lateral ascending tract, and in the medulla enter the formatio reticularis; from this point their course is identical with those of tactile sense.

THE CONNECTIVE TISSUE AND NEUROGLIA OF THE SPINAL CORD.

Connective, or supporting tissue is found throughout the entire cord; its character has been a subject of much discussion and has

given rise to great diversity of opinion. "It not only presents all the characters exhibited by the connective-tissue of other organs, but even passes over into direct continuity with indubitable connective-tissue. Certainly it is a connective-tissue in which the intercellular substance is reduced to a minimum. In most places it takes the form of a close network of fine fibres which can be followed to connective-tissue cells. * * * This (the connective-tissue) is the stroma in which the nervous elements and their vessels are disposed. Sometimes disease so destroys these latter structures that only the connective-tissue skeleton remains."

The neuroglia "Is a substance which appears peculiarly suitable for filling out the spaces which are not occupied by the other elements, taking its place in the constitution of the general mass with out offering any resistance to the free circulation of the nutrient juices. This is the function of the neuroglia which, in the form of an excessively fine granular mass, constitutes the matrix of the gray substance; this said, its description is exhausted. The neuroglia must be regarded as a peculiar kind of intercellular substance, for the cells from which it takes origin are not found in the adult organism. The free nuclei often pointed out as belonging to the neuroglia are probably nothing more than leucocytes escaped from blood vessels. The fine granules of the neuroglia apply themselves to the processes of both nerve cells and connective-tissue, and sometimes are seen attached to isolated elements. They were called by Boll interfibrillar granules. Their chemical nature, as well as their morphological constitution, precludes us from regarding them as part of the connective-tissue. They must be looked upon as elements peculiar to the nervous system. The total amount of the neuroglia is very small. Most of what was hitherto regarded as ground-substance has been proved to be a network of nerve fibres, medullated and non-medullated." Obersteiner.

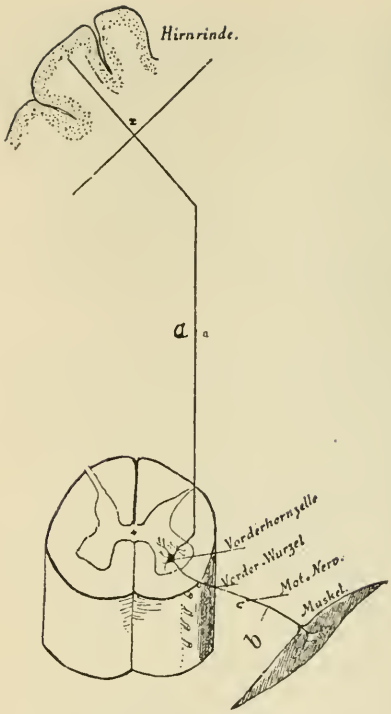


DIAGRAM II.

a. Cerebro-Spinal Segment. b. Spino-Muscular Segment. (After Edinger.)

The accompanying diagram shows that the motor path is composed of two segments.

a. Cerebro-spinal or Pyramidal.

b. Spino-muscular.

The fibres of the latter are far more numerous than the former. The cerebro-spinal fibers, when they pass into the intermediate gray substance, divide into a fine net-work or fibrillæ called the net-work of Gerlach, which unites with the ramifying processes of the ganglionic nerve cells * in the anterior horns. It is very probable that each pyramidal fibre through this fibrillary net-work is connected with a number of multipolar cells. The conduction in the cord is mostly on the opposite side to the hemisphere of the brain in which the impulses originated, to a less extent on the same side, (direct pyramidal tract).

Loss of motor power will occur from any interruption of the motor tract. Lesion of the pyramidal fibres causes loss of motion and increase of the reflexes. Damage of the spino-muscular segment causes loss of motion, of reflex action and atrophy of the muscles.

* "The nerve cells are the real nerve centres. To the fibres belongs only the task of conducting the stimuli transferred to them.

Many other functions of the nervous system besides simple conduction belong to the cells. The cells are the stations; the fibres the railroads which connect the stations together. * * * Every fibre may be regarded as a conducting path either connecting two nerve cells or a nerve cell and a peripheral end-organ, be it motor or sensory. An end-organ may be looked upon indeed as the terminal station of the road which extends outward from the nerve network." Obersteiner.

Flexor spasm is due to over-action of the centers for cutaneous reflex action. Extensor spasm is caused chiefly by over-action of the muscle-reflex centres, although it may be caused by cutaneous impressions. Contracture means a persistent shortening of a muscle depending on active contraction of the fibres and not on tissue change. Incoördination of movement is a symptom, motor in character, but sensory in origin. (Gowers.)

Disease of the white matter, especially the motor path, (not involving the ganglionic cells of the anterior cornua) does not affect the nutrition of the neuro-muscular system; hence in tabes dorsalis, lateral sclerosis, disseminated and traumatic sclerosis the normal muscular formula is not altered, although in their later stages there might occur quantitative changes. It is therefore evident that *electrical investigation is by no means a ready, much less a scientific method* of distinguishing between organic and functional disease.

VISCERAL NERVES.

a. A cervico-cranial outflow in connection with the vagus and spinal accessory nerve—(cervical splanchnics).

b. A thoracic outflow—(abdominal splanchnics).

c. A sacral outflow—(pelvic splanchnics).

These fibres (about 1-10 the size of motor fibers) arise in the brain and course along with the pyramidal fibres, pass down with them through the pons and enter the anterior pyramids, (from higher centres in the brain to lower ones in the cord) just as spinal nerves. They are found in both the pyramidal and the direct cerebellar tracts; from which they cross to the posterior vesicular column of Clarke, their chief destination in the cord; thence they pass out with the anterior nerve roots, forming after their exit the white ramus communicans, then to the ganglia of the sympathetic nerves, undergo interruption in their cells, become non-medullated and are distributed to the vessels and viscera. The afferent fibres return to the cord by the posterior roots and in it ascend to the brain by the column of Goll and antero-lateral ascending tract.—(*Tooth*).

X.

SENSORY SYMPTOMS.

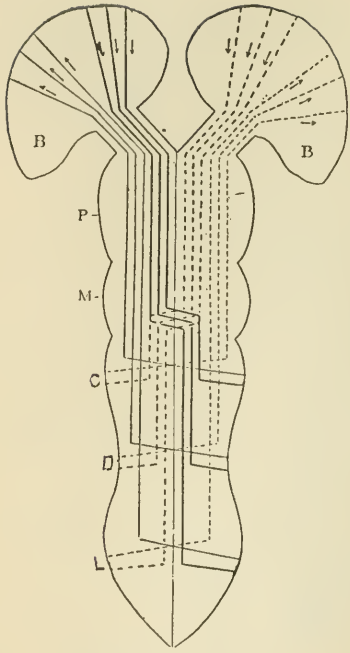


DIAGRAM III.

Diagramatic representation of the course of the Nerves of Common Sensibility and Motion. (After Bramwell.)

B. Brain. P. Pons. M. Medulla. C. Cervical Enlargement. D. Dorsal Region. L. Lumbar Enlargement.

Sensations of touch, temperature and pain decussate in the cord, muscle sense in the medulla. Sensibility may be intact when motor paralysis is absolute. The division of the sensory path into upper a id lower segments is conceivable as analogous to that of the motor path. Total lesion of the posterior roots causes loss of reflex action as well as loss of sensation. Interruption of the sensory conduction tracts higher up in the cord leaves reflex action unaffected, sensibility to temperature is rarely affected without that to pain. Consciousness may be influenced directly from the muscles, as the pain of cramp proves. Pain referred to the spine is usually due to disease of the meninges or bones, and seldom to organic disease of the cord, for in the latter it is generally referred to the neighborhood of the spine, to the loins or to the sacrum, rarely to the cord itself; its diagnostic importance is materially lessened by its frequent occurrence in gastric, abdominal and neuralgic affections.

Excentric or radiating pains are caused by irritation of the posterior nerve roots in their passage through the intervertebral foramina, through the membranes or through the posterior columns of the cord; this accounts for the absolute rigidity of the spine observed in patients suffering from chronic inflammation of the meninges. Irritation of the sensory conducting tracts at a higher level usually causes pains similar but less acute; these latter closely resemble rheumatism, like it being influenced by atmospheric changes, and because of this similarity great care in diagnosis should be observed.

Girdle pain (the zone of hyperæsthesia,) is due to irritation of the sensory nerve fibres of the healthy region above, and adjacent to, a transverse or unilateral lesion of the cord. Paræsthesia occurs alike in functional and organic disease and indicates neither the locality of the disorder nor the character of the disease.

Extreme pain in an anæsthetic area (Anæsthesia Dolorosa) is due to irritation of a sensory nerve trunk, posterior nerve root or postero-external column; the pain being referred by the perceptive cerebral center to its accustomed peripheral distribution (the anæsthetic area in question). Spasms and contractures in spinal meningitis may be due to irritation of the posterior or anterior nerve roots.

Reflex Action.—Loss of the reflexes may be due to interruption anywhere in the course of the reflex arc. Disease of a motor center or its nerves causes atrophy of the muscular area and loss of reflex action. Disease in the sensory center of a reflex arc may impair the deep but not the superficial reflexes or vice versa. Loss of all reflex action may casually follow acute lesion of the cord due to irritative inhibition, while permanent loss of cutaneous reflexes may ensue in brain disease, e. g. hemiplegia.

Disease of the pyramidal fibres or their trophic centers in the cerebral cortex, gives rise to excessive action of the deep reflexes, while lesion of the cord cutting off the inhibitory centers from above in like manner causes an excess of the cutaneous reflexes.

Loss of Visceral Control.—*Simple incontinence* is the condition in which the urine dribbles from the bladder as fast as secreted, owing to the destruction of the vesical center in the lumbar enlargement. *Intermittent incontinence* is due to interruption of the voluntary path, in consequence of which the sphincter acts automatically whenever sufficient urine has accumulated in the bladder to excite the detrusor muscle to contraction. *Overflow incontinence* occurs when, voluntary control being lost, a certain degree of distension of the bladder is attained, the pressure being sufficient, the sphincter relaxing, to expel its contents; such relaxation is constantly occurring under the high pressure which is continually renewed consequent upon the flow from the kidneys. This condition in tabes frequently causes death, and should be carefully guarded against. While the action of the bladder is the more complex, the same explanation may be applied to the sphincter ani.—(Gowers, Bramwell.)

XII.

TOTAL TRANSVERSE LESIONS OF THE SPINAL CORD.

A total transverse lesion is limited in vertical extent and separates the brain from all parts below the lesion, hence cutting off all motor and sensory impulses from below the damaged segment.

a. When a lesion involves the lower part of the lumbar enlargement, the sensory and motor supply to the skin and muscles of the leg and foot is lost, (4th lumbar to 3rd sacral); the inner side of the leg escapes because it is supplied by the anterior crural nerve from the lumbar plexus,—(comes from upper part of the lumbar enlargement). The lowest nerves supply the anus and perineum.

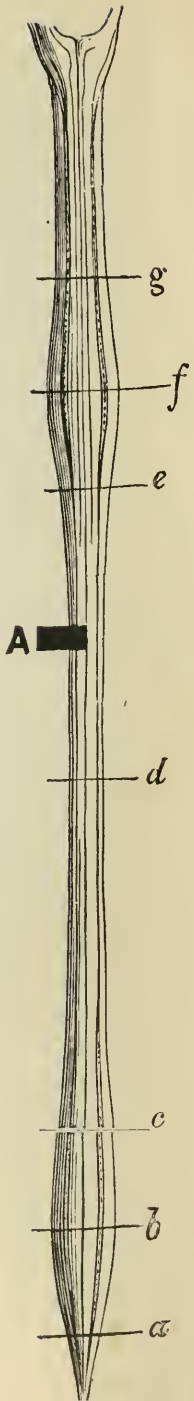
b. A lesion in the middle of the lumbar enlargement causes paralysis of the flexors of the knee and the hip muscles, (glutei, quadratis and gemelli) and loss of sensation in the skin of the lower part of the gluteal region; it also causes an involvement of the vesical and anal centres.

c. The skin and muscles in the front and outer part of the thigh are affected when the lesion is in the upper part of the lumbar enlargement, whence comes the anterior crural and obturator nerves, the latter of which supplies the adductors. The skin on the upper and outer side of the thigh and the part adjacent to the scrotum and in the groin loses sensibility only when the disease damages the highest part of the lumbar enlargement, from which the 2d and 3d lumbar nerves arise; then the flexors of the hip are paralyzed.

d. A transverse lesion in the dorsal region gives rise to the following symptoms:

A. Paraplegia.—R. D. in the muscles of the segment where the lesion is situated.

B. The loss of common and special sensibil-



C. Increased reflexes.

D. Retention of fæces and urine,—intermittent incontinence.

E. Electrical reactions normal below lesion.

F. Vaso-motor paralysis.

G. Hyperæsthesia at upper part of lesion.

H. Trophic changes in sensory area of diseased segment. Priapism may occur when lesion is in upper dorsal region.

K. Ascending and descending degenerations.

e. A lesion of the lowest part of the cervical enlargement (first dorsal nerve) causes the intrinsic muscles of the hand first to be affected, commencing with the little finger.

f. Involvement of the middle of the cervical enlargement (fifth, sixth and seventh cervical nerves) affects the shoulder muscles and the seratus magnus, with a general loss of power and sensation. Above the level of the sixth pair the trapezius and sterno-cleido-mastoid muscles suffer; they derive their innervation from the spinal accessory.

g. A lesion between the fourth and fifth cervical causes paralysis of the diaphragm and the lower part of the neck becomes anæsthetic.

UNILATERAL LESIONS OF THE SPINAL CORD—BROWN-SEQUARD'S PARALYSIS.

A. A Spinal Hemiplegia is where both an arm and a leg are affected. A Spinal Hemi-Paraplegia is where a leg or an arm alone is affected. There is usually some loss of motion on the opposite side of the body in unilateral lesions owing to a slight involvement of the adjoining half segment, or to disease of the direct pyramidal tract, the lesion being on the same side. The sensory areas of the diseased segment become anaesthetic and a zone of hyperæsthesia occurs at the upper border of the lesion.

The symptoms on the side of the lesion are:

A. Motor palsy.

B. Hyperæsthesia.

C. Loss of muscle sense.

D. Reflex action increased, lessened at first by inhibition.

E. Temperature first increased by one to three degrees Far., then sinks below normal.

F. The bladder and rectum at first impaired, and always somewhat weakened.

The symptoms on the side opposite the lesion are:

A′. Anæsthesia.

B′. Muscle sense normal.

C′. No palsy.

D′. Reflex action normal.

E′. Temperature normal.

(Gowers, Strümpell & Bramwell.)

LOCALIZATION OF THE FUNCTIONS OF THE SEGMENTS OF THE SPINAL CORD.*

SEGMENT.	MUSCLES.	REFLEX.	SENSATION.
2d and 3d Cervical.	Sterno Mastoid. Trapezius. Scaleni and neck. Diaphragm.	Hypochondrium (?) Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Back of head to vertex. Neck.
4th Cerv.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator Longus. Rhomboid. Supra & Infra Spinatus.	Pupil 4-7 cerv. Dilatation of the pupil produced by irritation of neck.	Neck. Upper shoulder. Outer arm.
5th Cerv.	Deltoid. Biceps. Coraco-Brach. Brachialis Anticus. Supinator Longus. Supinator Brevis. Deep muscles of shoulder blade. Rhomboid. Teres Minor. Pectoralis (clavicular part). Serratus Magnus.	Scapular. 5th Cerv. to 1st Dors. Irritation of skin over the scapula produces contraction of the scapular muscles. Supinator Longus. Tapping its tendon in wrist produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm. Anterior upper two-thirds of arm.
6th Cerv.	Biceps. Brachialis Anticus. Pectoralis (clav. part). Serratus Magnus. Triceps. Extensors of wrist and fingers. Pronators.	Triceps. 5th to 6th Cerv. Tapping elbow tendon produces extension of forearm. Posterior wrist. 6th to 8th Cerv. Tapping tendons causes extension of hand.	Outer side of arm and forearm. Inside and front of forearm.
7th Cerv.	Triceps (long head). Extensors of wrist and fingers Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis (costal part). Latissimus Dorsi. Teres Major.	Anterior Wrist. 7th-8th Cerv. Tapping anterior tendon causes flexion of wrist. Palmar. 7th Cerv.—1st Dors. Stroking palm causes closure of fingers.	Inner and back of arm and forearm. Radial distribution in the hand.

8th Cerv.	Flexors of wrist and fingers. Intrinsic muscles of hand.		Forearm and hand; median and ulnar areas.
1st Dors.	Extensors of thumb. Intrinsic hand muscles. Thenar and Hypothenar eminences.		Ulnar distribution to hand.
2d and 12th Dors.	Muscles of back and abdomen. Erectores Spinae.	Epigastric. 4th–7th Dors. Tickl'g mammary region causes retraction of the epigastrium. Abdominal. 7th–11th Dors. Stroking side of abdomen causes retraction of belly.	Skin of chest and abdomen, in bands running around and downward corresponding to spinal nerves. Upper gluteal region.
1st Lumb.	Ilio-psoas. Sartorius.	Cremasteric. 1st–3d Lumb. Stroking inner thigh causes retraction of scrotum.	Skin over groin and front of Scrotum.
2d Lumb.	Ilio-psoas. Sartorius. Flexors of knee (Remak). Quadriceps femoris.	Patella tendon. Striking tendon causes extension of leg.	Outer side of thigh.
3d Lumb.	Quadriceps femoris. Inner rotators of thigh. Abductors of thigh.	Bladder centre. 2d–4th Lumb.	Front of thigh.
4th Lumb.	Abductors of thigh. Adductors of thigh. Flexors of knee (Ferrier). Tibialis Anticus. Peroneus Long.	Rectal centre. 4th L. to 2d Sac. Gluteal. 4th–5th Lumb. Stroking buttock causes dimpling in fold of buttock.	Inner side of thigh and leg to ankle. Inner side of foot.
5th Lumb.	Outward Rotators of thigh. Flexors of knee (Ferrier). Flexors of ankle. Peronei. Extensors of toes.	Achilles tendon. Over extension causes rapid flexion of ankle, called ankle clonos.	Lower gluteal region back of thigh. Leg and foot outer part.
1st and 2d Sacr.	Flexors of ankle. Long flexor of toes. Intrinsic muscles of foot.	Plantar. Tickling sole of foot causes flexion of toes and retraction of leg.	Leg and foot except inner side. Perineum and back of Scrotum. Anus.

*This valuable table of Dr. M. Allen Starr's is inserted by his permission.

The Brain,

INCLUDING

Dr. Starr's Review of Flechsig's

Plan of the Human Brain.

THE PONS, THE MEDULLA AND THE CRANIAL NERVES, WITH THEIR RELATION TO EACH OTHER.

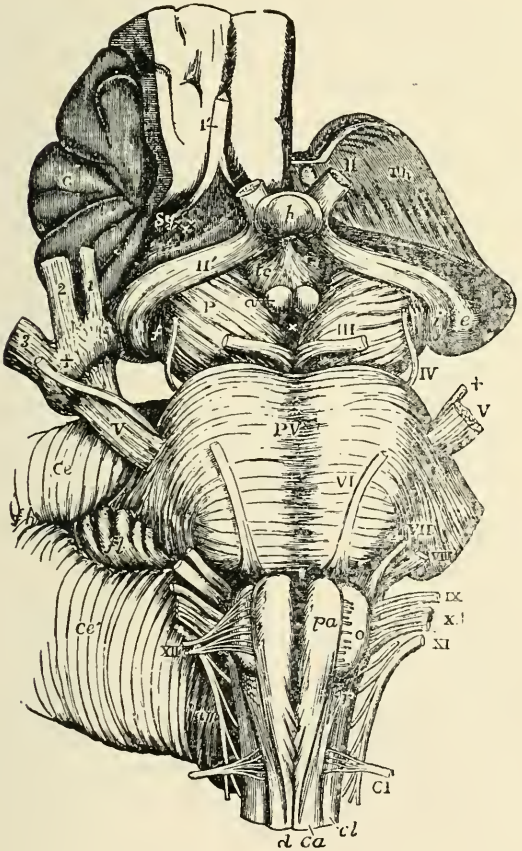


DIAGRAM V.

THE BASE OF THE BRAIN. (Allan Thompson.)

I to XII, the cranial nerves; Th., optic thalamus; h, putuitary body; tc., tuber cinerium; a., corpora albicantia; P., pes pedunculi; i., interior and exterior geniculate body; PV., pons varolii; pa., anterior pyramid of medulla; o., olive; d., decussation of anterior pyramids; ca., anterior column of spinal cord; cl., lateral column of spinal cord: Ce., cerebellum; fl., flocculus of cerebellum.

The medulla and pons are clearly shown by the above figure, to connect the cord below with the brain and its crura above. Behind them is seen the cerebellum; in them are found the nuclei of the cranial nerves from the fifth to the twelfth inclusive; the XII, XI, X. IX and VIII arise (superficial origin) from the medulla, while the V, VI and VII spring from the pons. The function and distribution of these nerves are as follows:

XII. The Hypoglossal Nerve is motor in function and supplies all the extrinsic muscles of the tongue, also certain others in connection with the hyoid bone. Whatever sensibility it possesses is derived from its free communication with other nerves.

XI. The Spinal Accessory Nerve consists of two portions, the spinal and accessory. The latter joins the pneumogastric nerve; the remainder is distributed to the sterno-cleido-mastoideus and trapezius muscles, and is motor in function.

X. The Pneumogastric or Vagus Nerve has a very extensive distribution, supplying the pharynx, larynx, trachea, lungs, heart, œsophagus and stomach. It communicates freely with cranial, spinal and sympathatetic nerves and hence its functions are of a very mixed character, partly motor, partly sensory and partly of a nature allied to those of the sympathetic system.

IX. The Glosso-Pharyngeal Nerve is principally distributed to the mucus membrane lining the upper part of the pharynx, the Eustachian tube, the arches of the palate, the tonsils, and to the sides of the posterior part of the upper surface of the tongue; it also gives filaments through its tympanic branch to some parts of the middle ear. It is a nerve of the special sense of taste and of ordinary sensation to the parts which it supplies, and is the chief centripetal nerve engaged in the act of deglutition.

VIII. The Auditory Nerve is the nerve of the special sense of hearing and is distributed to the internal ear.

VII. The Facial Nerve is purely a nerve of motion, and supplies the muscles of the face. Through the large superficial petrosal it is distributed to some of the muscles of the soft palate and to the intrinsic muscles of the tongue through the chorda tympani. Its branches ramify freely with the sensory fibres of the fifth before their termination in the facial muscles.

VI. The Sixth Nerve (Abducens Oculi) is the motor nerve of the external rectus muscle of the eyeball.

V. The Fifth Nerve (Trifacial or Trigeminal). Pierret compares all the intercranial nerves to a single spinal nerve of which the sensory portion of the fifth nerve may be considered the sensory root and its motor portion, together with all the motor nerves,—the oculo motor, pathetic, sixth, facial and hypoglossal may be regarded as the motor or anterior root. The trigeminal is the chief nerve of common and muscular sensibility to the face; it gives motor power to the muscles of mastication, and one of its branches contains filaments appropriated to the special sense of taste. The two upper portions of this nerve are purely sensory, while the lower one is very complex, containing not only motor and sensory filaments, but those of special sense (gustatory) as well.

IV. The Fourth Nerve (Pathetic or Trochlear) is the least of the cranial nerves and ends in the superior oblique muscle of the eyeball; it communicates with the sympathetic and often with the opthalmic division of the fifth, and according to Bidder, sends a filament to the dura mater.

III. The Third Nerve (Motor Oculi) is entirely motor in function; it sends branches to five of the seven muscles of the orbit, also one to the opthalmic ganglion, thereby supplying motor power to the iris.

It will be observed that the motor nerves of the pons and medulla are the motor branch of the trigeminal, the abducens, the motor portion of the glosso-pharyngeal and of the vagus and the hyperglossal, while the sensory nerves of the same area are the trigeminal, the auditory and the glosso-pharyngeal.

In the medulla and pons are found important nerve tracts, viz: The formatio reticularis, the lemniscus, the fillet and the pes pedunculi, along which are conveyed the sensory, motor, vaso-motor and trophic impulses from the brain to the cord and from the cord to the brain necessary for the maintenance of life. In the medulla are complex centers for the various organic functions. These are: a, The respiratory and convulsive centres; b, vaso-motor centre; c, cardio-inhibitory centre; d, diabetic centre; e, centre for deglutition; f, the centre controling the œsophagus and the stomach; g, the centre for the secretion of saliva, sweat and urine; h, the centre for co-ordination (?).

The pons varolii is intimately connected with co-ordination. Stimulation or section of it causes pain with forced or disorderly movements, followed by sensory, motor, and vaso-motor paralysis.

(*Foster, Landois and Stirling, Starr, Buzzard and Flower.*)

THE MEDULLA OBLONGATA.

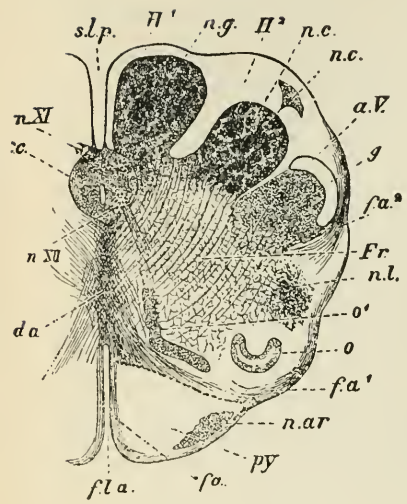


DIAGRAM VI.

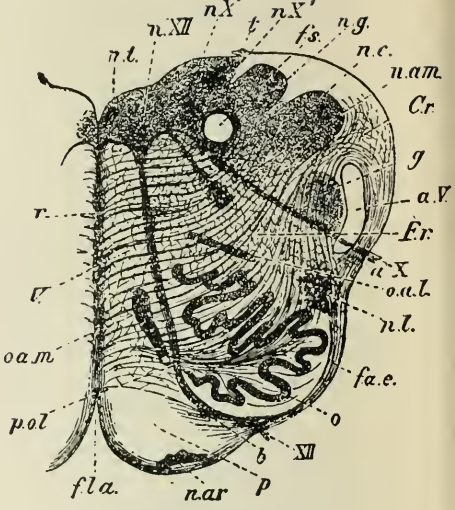


DIAGRAM VII.

Diagram 6. Section of the medulla oblongata at the upper decussation of the pyramids, f.l.a., anterior, s.l.p., posterior median fissure; nXI, nucleus of the accessorius vagi; nXII, nucleus of the hypoglossal; da, the so-called superior or anterior decussation of the pyramids; py, anterior pyramid; n. ar, nucleus arciformis; O, median parolivary body; o′, beginning of the nucleus of the olivary body, nl, nucleus of the lateral column; Fr, formatio reticularis; g, substantia gelatinosa, with (aV) the ascending root of the trigeminus; nc, nucleus of the funiculus cuneatus; nc′, external nucleus of the funiculus cuneatus; ng, nucleus of the funiculus gracilis (or clava); H^1, funiculus gracilis; H^2, funiculus cuneatus; c c, central canal; fa, fa^1, fa^2; external arciform fibres. ×4.

Diagram 7. Section of the medulla oblongata through the olivary body. nXII, nucleus of the hypoglossal; nX, nX′, more or less cellular parts of the nucleus of the vagus; XII, hypoglossal nerve; X, vagus; n. am, nucleus ambiguus; nl, nucleus lateralis; o, olivary nucleus; o.a.l., external, and o.a.m, internal parolivary body; fs, the round bundle or funiculus solitarius; Cr, restiform body; p, anterior pyramid, surrounded by arciform fibres; f. a. e., p. o. l. fibres proceeding from the olive to the raphe (pedunculus olivae); r, raphe. ×4.—(*After Landois and Sterling.*)

The medulla is, in shape an irregular, truncated cone; the difference in form between it and the cord is due to an altered position of its parts, viz.: the crossed pyramidal tract, the direct cerebellar tract, the termination of the posterior columns in the slender and cunate nucleus, etc., with the addition of nervous matter not found in the spinal cord. The spinal cord is divided by the anterior and posterior roots into three columns or areas, viz.: anterior, lateral and posterior. Quain affirms that both morphologically and in a measure physiologically, the spinal accessory, pneumogastric and glosso-pharyngeal nerves are the upper prolongation of the sensory nerve roots of the spinal cord. (These nerves, along with the ascending root of the fifth, are said, by Meynert, to belong to the lateral mixed system because they possess motor as well as sensory nuclei.) The hypoglossal nerve may, in like manner, be considered as the upward prolongation of the anterior roots of the spinal cord; by analogy, therefore, we may locate three columns in the medulla—the anterior, lateral and posterior areas of Flechsig. A glance at the above diagram will show the spinal accessory nerve traversing the substance of the medulla to reach its nucleus in the gray matter near the central canal, and in so doing it marks off an oval area on each side, posteriorly—the posterior area of the medulla; and it will be observed that it corresponds to the posterior columns of the spinal cord, viz.: the columns of Goll and Burdach. You can readily trace the hypoglossal nerve as it courses its way through the medulla to its nucleus situated in the gray matter near the antero-lateral side of the central canal, in the closed portion of the bulb; that portion of its substance lying between the hypoglossal nerve and the antero-median fissure is wedge shaped in form and is called the anterior area of the medulla, that portion lying between the hypoglossal and the spinal accessory nerve is called the lateral area of the medulla and upon its upper surface is situated the olivary body.

The Posterior Area of the Medulla.—The column of Goll ends in the nucleus of gracilis, that of Burdach in the nucleus cuneatus. The funiculi of gracilis and of Burdach are longitudinal prominences given to the median and postero-lateral columns by the aggregation in them of nerve cells from the base of the posterior cornu, forming the above named nuclei of these columns. Although quite small at the point of origin, they are yet of comparatively

good size where the central canal opens into the fourth ventricle. Between the postero-lateral column and the postero-lateral groove is seen the longitudinal prominence of Schwalbe—the funiculus of Rolando; the tubercle of Rolando which causes this projection on the surface of the medulla is produced by the increased size of the gelatinous substance of the posterior cornu of the cord as it becomes the nucles of the fifth nerve which from this point courses upward through the medulla into the pons varolii; here its sensory fibres enter and terminate at different levels in the pons and medulla.

The Lateral Area of the Medulla is the continuation upward of the lateral column of the cord with the exception of its crossed pyramidal and direct cerebellar tracts; the function* of the olive shaped prominence on the surface of this area is unknown.—(*Starr*, *Quain*, *Gowers*, *Landois and Sterling.*)

Anterior Area of the Medulla. The pyramid of this area, as is beautifully shown in Diagram VIII, is formed by the crossed pyramidal tract of the opposite side passing through its own anterior cornu, crossing over the anterior median fissure, and uniting with the direct pyramidal tract of the same side. The upper prolongation of the balance of the anterior column is dorsad to the pyramid and hence is concealed from view. By the passage of the crossed pyramidal tract through the anterior cornu, the caput cornu (see C. a.) is completely separated from the rest of the gray matter and is ultimately pushed over into the lateral area of the medulla by the interpolation of the olivary body and the formation of the anterior pyramid; the greater part of its substance is broken up into a reticulated or basket-like network, the formatio reticularis (see f. r.) a small nucleus being left in the lateral area which is called the nucleus lateralis. (n. l., fig. 7.)

*Dr. Bruce, in the *Reports from the Royal College of Physicians, Edinburgh, 1890*, asserts that the connection of the interolivary body, by means of an acoustico-olivary tract with the auditory nerve, as shown by himself, serves to throw some light on the special function assigned to that body in maintaining the equilibrium of muscular balance.

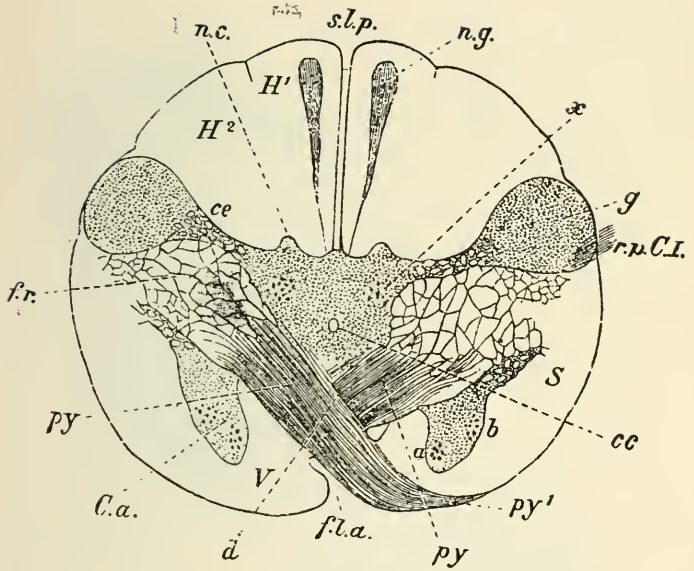


DIAGRAM VIII.

Section of the Decussation of the Pyramids, f.l.a., anterior median fissure, displaced laterally by the fibres decussating at d; V, anterior column; C.a., anterior cornu, with its nerve cells, a. b.; cc, central canal; S, lateral column; fr, formatio reticularis; ce, neck, and g, head of the posterior horn; r. p. C. I., posterior root of the first cervical nerve; n. c., first indication of the nucleus of the funiculus cuneatus; n.g., nucleus (clava) of the funiculus gracilis; H^1, funiculus gracilis; H^2, funiculus cuneatus; s. l. p., posterior median fissure; x groups of ganglionic cells in the base of the posterior cornu. ×6.

Tooth suggests that certain fine fibres from the antero-lateral ascending tract of the monkey end in this nucleus, this being the case it is reasonable to infer that a similar termination may occur in man. The base of the cornu remains as a small portion of gray substance close to the antero-lateral aspect of the central canal. While that portion of the medulla lying between the two olivary bodies may be designated as the interolivary space, it should be borne in mind that this is not the interolivary tract. Fibres pass to either restiform body from the olive of its own side, but it receives a greater number of fibres from that of the opposite side. Although decussation takes place between the two olivary bodies in the median raphe, no fibres pass from one olive to the other.

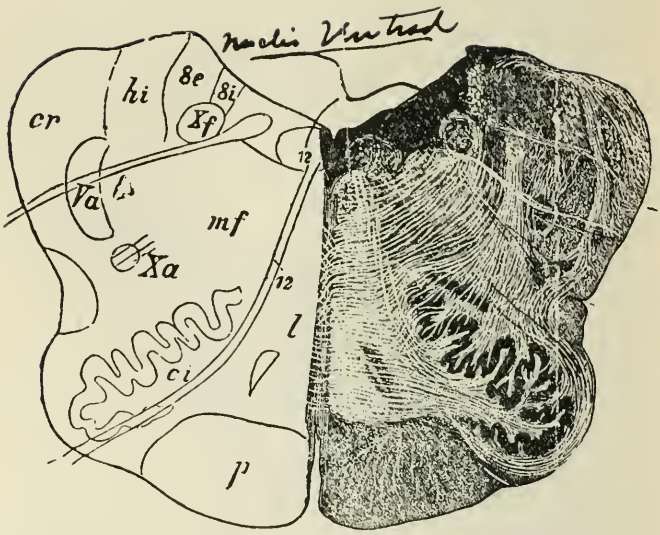


DIAGRAM IX.

Section through the medulla. (Wernicke ×4, quoted by Starr) p, pyramid; l, lemniscus or interolivary tract, ci., fibres from the olive which cross the raphe and pass outward and backward to enter the inferior cerebellar peduncle, cr.; mf., formatio reticularis, in which Xa, the deep nucleus of the vagus (nucleus ambiguus) lies; Va, ascending root of the trigeminal nerve; 12, hypoglossal nucleus and root; Xf, respiratory bundle around which, in the floor of the fourth ventriele, the vagus nucleus lies, and below which the vagus root issues; 8e and 8i, auditory nuclei; hi., nucleus cuneatus, from which the fibres pass outward and enter cr.

The Formatio Reticularis occupies the whole of the anterior and lateral areas of the medulla dorsal to the pyramids and olives; it is divided on each side into two parts by the hypoglossal nerve; the inner part, near the median raphe, contains the continuation of the anterior column of the spinal cord and the lemniscus,—the interolivary tract. The outer part lies between the interolivary tract and the ascending root of the trigeminus nerve, in a lateral direction; in the anterior area of the medulla it contains no nerve cells and hence is called the formatio reticularis alba to distinguish it from the lateral area which contains gray matter and is named formatio reticularis grisea. This latter tract transmits sensations of

touch, temperature and pain through the medulla, and is called the sensory tract. It is made up, according to Dr. Starr, of "Nerve fibres passing in three directions; a, transversely, the commissural fibres of the cranial nerve nuclei; b, ventrad, the fibres of the cranial nerve roots and arciform fibres; c, longitudinally, the sensory tract. The longitudinal fibres can be traced from the gray matter and various columns of the spinal cord, through the formatio reticularis to its upper level in the tegmentum of the crus, whence they issue to enter the posterior part of the internal capsule. * * * *
In the outer third of this part is found a column of peculiar structure, resembling the substantia gelatinosa of the posterior horn of the spinal cord,"—the ascending root of the fifth nerve as before noted.

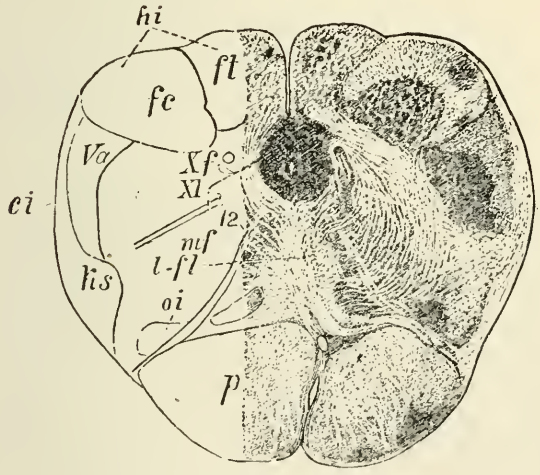


DIAGRAM X.

Section through medulla, Level of sensory decussation (Wernicke ×4, quoted by Starr) ft, nucleus gracilis; fc, nucleus cuneatus, from which fibres curve around the central gray matter, and then decussate and turn upward (cephalad) in the interolivary tract, l. ft; Va, ascending root of the trigeminal nerve; ci, inferior peduncle of the cerebellum, of which ks, direct cerebellar column, of the cord, is a part; Xf, respiratory bundle; XI, medullary nucleus of spinal accessory nerve and its root; 12, hypoglossal nucleus and root; mf, formatio reticularis; oi, olive.

The Tract of Muscular Sense courses in the formatio reticularis alba and is called the lemniscus or interolivary tract. "It conveys sensations upward and hence is traced in that direction. It arises from the nuclei gracilis and cuneatus on the dorsal surface of the medulla, in which the columns of Goll and Burdach end; it then crosses the median line (see Diagram X, ft and fc) decussating with its fellow of the opposite side in the upper decussation of the medulla, (sensory decussation of Meynert, piniform decussation of Spitzka.) The fibres after decussating turn upward, enter the interolivary tract, and pass onward through the medulla beyond the upper level of the olives, into the pons. Here this tract broadens out, becomes almost ribbon-like in its appearance and hence has been called the fillet. In the pons the fillet lies behind the deep transverse bundles of the pons and just in front of the formatio reticularis (see fig. XI.). As it passes upward it receives additional fibres from the superior olive (So, above diagram), but these again leave it at the level of the posterior corpora quadrigemina, while the main part of the fillet passes onward in the outer part of the tegmentum of the crus cerebri and thence into the posterior part of the internal capsule, whence it radiates to the parietal cortex of the brain."

Quain traces the tracts of the cord into the medulla as follows; the funiculi gracilis, cuneatus and Rolando arise from the posterior column of the spinal cord where it ends in the medulla; the two former end in the nuclei gracilis and cuneatus, while the latter terminates in the tubercle of Rolando. The crossed pyramidal tract leaves the lateral column of the cord to unite with the direct pyramidal tract of the opposite side, thus forming the anterior pyramid; while the direct cerebellar tract, about the middle of the medulla, passes obliquely backward to join the restiform body; the remainder of the lateral column forms the longitudinal fibres of the reticularis grisea. The direct pyramidal tract of the anterior column of the cord unites with the crossed pyramidal of the opposite side, which has decussated in the anterior commissure of the medulla, to form the pyramid of the same side, while the balance of the fibres pass dorsad to the pyramid and form the longitudinal fibres of the reticularis alba: they also give origin to The Posterior Longitudinal Bundle.

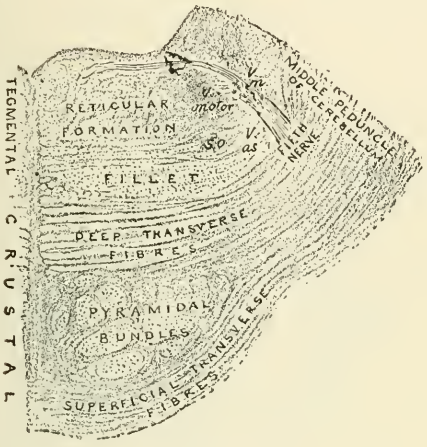


DIAGRAM XI.

Diagram of one-half of a section through the middle of the Pons. So, superior olivary body; Vas, ascending root; Vm, middle nucleus; V motor, motor nucleus of fifth nerve.—(*Gowers.*)

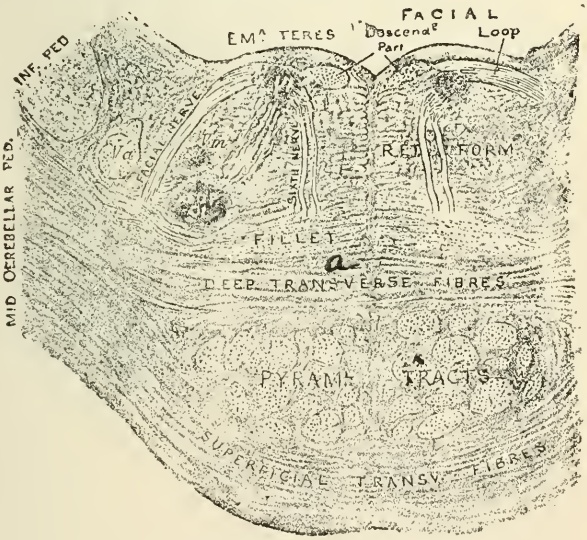


DIAGRAM XII.

Transverse section of the Pons at the level of the nuclei of the sixth (VI) nerve. Ret. Form. Reticular Formation; Vm, motor nucleus of the fifth nerve. Va, its ascending root seen in section. Sup. ol. Superior olivary body. A, Corpus Tropezoides, (modified from Gowers.)

The pons is simply the continuation upward of the medulla oblongata and contains the upper half of the fourth ventricle. Diagram V shows how "The bridge of Varolius" connects the lateral hemispheres of the cerebellum with each other on the ventral surface of the pons. Diagram XI shows two kinds of transverse fibres, superficial and deep, and between them is clearly seen the upward prolongation of the longitudinal fibres from the medulla. These commissural fibres contain between them large masses of gray matter. The Corpus Trapezoides is formed of deep transverse fibres (see Diagram XI, a.) in the lower half of the pons, passing from the upper olive of one side to cerebellum of other side and is also connected with the lemniscus. Between the deep transverse fibres are found large masses of gray matter—the nuclei pontis. It is in the gray matter of this basal region that the longitudinal fibres from the frontal, the occipital and parietal lobes of both cerebral hemispheres terminate and from it that transverse fibres arise and pass laterally to the cerebellum. "The majority of the fibres from one-half of the pons pass to the opposite hemisphere of the cerebellum and thus cross the median line where they interlace with those of the other side. The gray masses of the pons are thus interposed in a continuous tract between the cerebrum and the cerebellum. Each cerebral hemisphere being connected with both cerebellar hemispheres, but to a far greater extent to the opposite hemisphere than to the one of the same side."—(*Starr.*)

Aside from keeping up a communication between the cerebrum and the cerebellum, the function of these tracts is unknown. The dorsal portion of the pons consists principally of the formatio reticularis, the fillet and the gray matter of the medulla. As in the medulla, so likewise here, there is an interpolation of gray matter—the superior olivary body. (See Diagram XI, So.) From the pons there arise the seventh, fifth (partially), and sixth nerves.

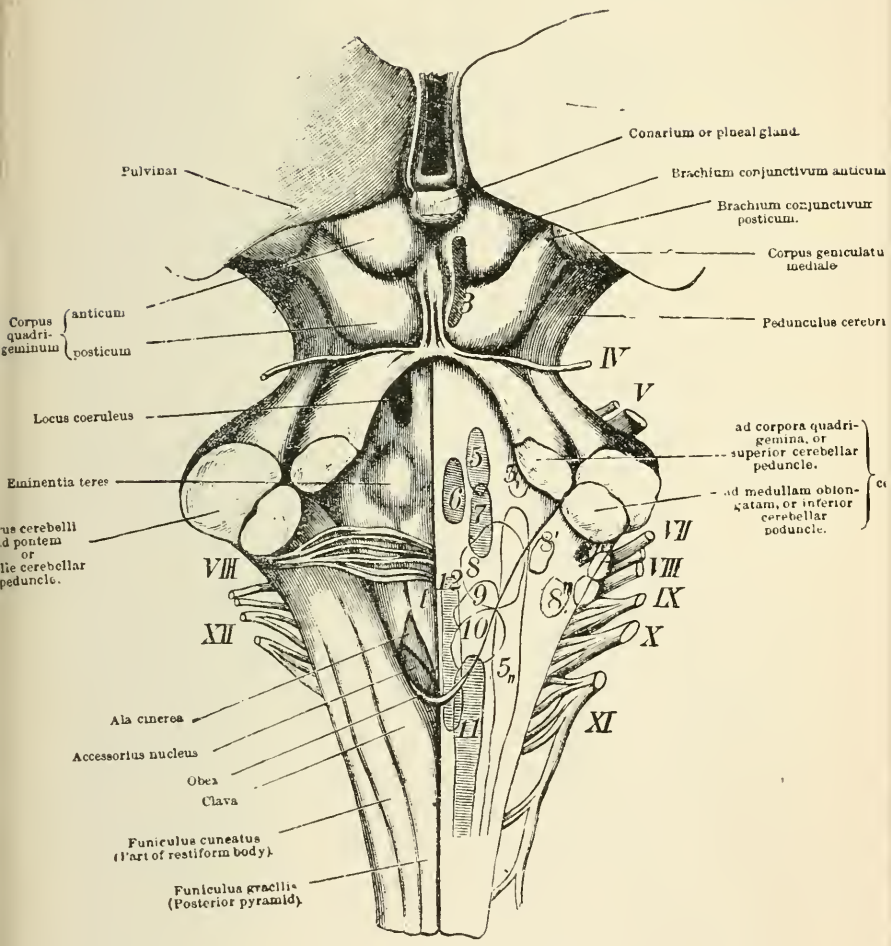


DIAGRAM XIII.

Medulla oblongata with the corpora quadrigemina. The numbers IV-XII indicate the superficial origin of the cranial nerves, while those (3-12) indicate their deep origin, i. e., the position of their central nuclei; t, funiculus teres. (*Modified from Landois and Stirling.*)

12. The Fasciculus Teres. It is from this longitudinal mass of large multipolar nerve cells (which is derived from the base of the anterior cornu, and which extends cephalad from the decussation of the anterior pyramids to the calamus scriptorius of the fourth ventricle) that the hypoglossal nerve arises. (See fig. XIII, 12.) Its fibres pass out obliquely through the inner part of the reticular formation and emerge from the medulla between the anterior pyramid and the olivary body.

11. Spinal-Accessory. 10. Eminentia Cinerea-Vagus. 9. Ala Cinerea-Glosso-Pharyngeal. Diagram VI (n. XI) shows a group of nerve cells dorsad to the hypoglossal nucleus, it is the beginning of the nucleus of the spinal-accessory nerve. The lower fibres of this nerve, which supply the muscles of the neck, arise from the anterior cornu of the upper cervical cord, not from the accessory nucleus. The fibres from the latter nucleus arise from the medulla and innervate the larynx, and hence have a similar origin to that of the vagus. After the central canal opens out into the fourth ventricle, the accessory nucleus lies laterad to that of the hypoglossal as seen in the above diagram. From the upper part of this column of gray matter (ala cinerea, eminentia cinerea) arise the vagus and glosso-pharyngeal nerves. These nerves emerge from the medulla between the olive and restiform body; in their course through the bulb they pass through the longitudinal fibres of the ascending root of the fifth nerve. The vagus and glosso-pharyngeal nuclei are composed of small and large nerve cells, the latter are deeply situated and are probably connected with the motor fibres of the upper part of this column. Fibres pass to these nerves from the respiratory bundle of Krause, also from the raphe. According to Meynert, quoted by Gowers, the pneumogastric sends fibres to the cerebellum. In this way he accounts for the disturbance of its gastric functions when there is involvement of equilibration, as in the vomiting of vertigo.

To the outer side of the nuclei of the vagus and glosso-pharyngeal nerves, (see fig. VII, fs) is a round, longitudinal bundle ascending cephalad to the highest part of the glosso-pharyngeal nucleus, and descending, caudad to to the middle of the cervical enlargement, and possibly lower. It is known as the Slender Column of Clark, the Respiratory Bundle of Krause, the Trinural Fasciculus of Spitzka, and the Funiculus Solitarius. Some fibres from the vagus, glosso-pharyngeal, and the spinal accessory nerves pass into it, hence it

has been called their ascending root. (Meynert.) It is supposed to be involved in the process of respiration, since if it is divided on both sides, respiration is entirely suspended.

8. The Tuberculum Acousticum. Underneath this tubercle of Schwalbe lies the chief auditory nucleus,—the inner auditory nucleus. It descends caudad in the medulla as far as the middle of the vagal nucleus and ascends cephalad (passing underneath the stria acoustica) into the pons, attaining its largest size at the junction of the pons and the medulla, where it almost touches the middle line; it diminishes rapidly in size as it extends upward into the pons and is here shifted to the side of the ventricle by the appearance between it and the median sulcus, of the nucleus of the sixth nerve.

8′. Dieter's Nucleus. The external auditory nucleus lies just on the outside of the inner auditory nucleus just described; while it does not descend caudad as far in the medulla as the main nucleus, it does reach as far cephalad in the pons, and superiorly it rather increases than diminishes in size. It is broken up by the fibres of the superficial root as they pass through it on their way to the inner auditory nucleus.

8″. The Accessory or the Anterior Nucleus of the auditory nerve is represented in the upper part of the medulla by a mass of nerve cells lying in the angle formed by the junction of the two roots; the main part of this nucleus ascends cephalad into the pons and lies in front of the restiform body on the outer side of the deep root.

It will be observed that we have only referred to three nuclei of origin for this nerve instead of four as are usually described. Laura and Gowers call in question one of these three, viz.: Dieter's nucleus, leaving simply the inner and anterior auditory nuclei as its source.

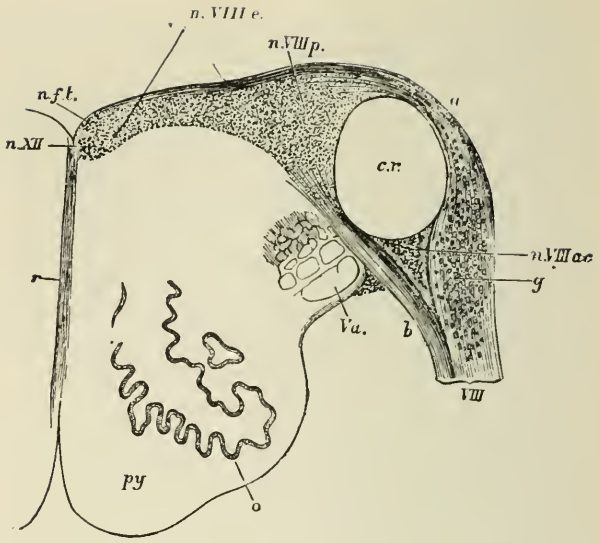


DIAGRAM XIV.

Transverse section of the upper part of the medulla oblongata. py, pyramid; o., olivary nucleus; V. a., ascending root of the fifth nerve; VIII, inferior (posterior) root of the auditory nerve, formed of two parts, a. and b., which enclose the restiform body, cr.; n. VIII. p.; Dieters nucleus of the auditory nerve; n. VIII. ac., accessory or anterior nucleus; g., ganglion cells in the root; n. f. t. nucleus of the funiculus teres; n. XII, nucleus of the hypoglossal; r. raphe; n. VIII e., inner auditory nucleus. (Modified from Schawlbe.)

The auditory nerve at the junction of the pons and the medulla, as shown in the above diagram, is joined to the latter by two roots, one of these, the superficial, (posterior) root arises, as already indicated from the inner auditory nucleus, (see diagram XIV, n. VIII e.), and winds around the restiform body (cr) to unite with the deep root (b) on its ventral surface, while the deep (anterior root penetrates the substance of the bulb and terminates in Dieter's nucleus (see above diagram, n. VIII. p.); some of the fibres of the superficial root (a) are continuous with the stria acoustica, even passing over to the opposite side; others join the restiform body coursing in the same direction as its fibres, while still others, according to Meynert, join the commissural fibres of the pons and pass to the cerebellum by the way of the opposite restiform body.

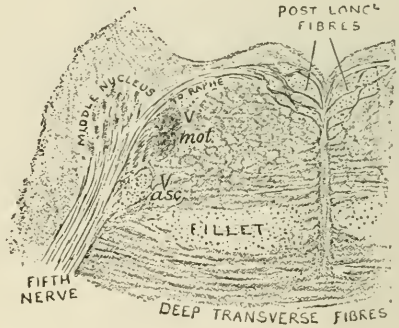
The deep root (b) not only pierces the medulla and ends chiefly in Dieter's nucleus, but from it also fibres go to the main auditory nucleus, to the restiform body of the same side and through it to the middle lobe of the cerebellum; "These latter fibres come from the semicircular canal and subserve, probably, not auditory sensations, but impressions from the canal, determined by the position and movement of the body," (Gowers); and finally from it fibres pass to the anterior nucleus. Spitzka describes the auditory path to the hemispheres as follows: "From the cochlea, by way of the posterior division (superficial root) of the auditory nerve through the corpus trapezoides to the opposite superior olive, thence to the lateral lemniscus, to the posterior corpora quadrigemina, to the internal geniculate body, and thence through the corona radiata to the first temporal convolution."

7. The Nucleus of the Facial is deeply situated in the formatio reticularis dorsad to the superior olive; to reach this nucleus its fibres pass in the outer part of the reticular formation in their approach to the nucleus of the abducens, around which they make an acute bend, (the knee of the facial) and turn downward, the majority of them passing on the mesial side of the sixth nucleus, (some pass through the nucleus itself) on their way to the deep-seated nucleus above described. "We do not know whether the connection between the lips and tongue is subserved by fibres that pass between the two nuclei (hypoglossal and facial) or whether, as is possible, the nerve fibres from the lips actually arise from the hypoglossal nucleus; many of the fibres of the facial nerve, turning downward at the loop, have a longitudinal course in the inner part of the reticular formation and may readily reach the hypoglossal nucleus." —(*Gowers,*)

6. Eminentia Teres.—Diagram XII (em. teres) shows us the nucleus of the sixth nerve, lying underneath the most prominent part of this eminence. Ferrier regards this nucleus as practically an upward continuation of the motor column which gives rise to the hypoglossal nerve in the medulla. Duval and Laborde believe that fibres pass upward to join the nuclei of the third and fourth cranial nerves of the opposite side. The posterior longitudinal bundle probably furnishes this avenue of communication, its function being to associate all the motor nuclei with each other, thus maintaining

a constant relation between the motor column of the gray matter of the fourth ventricle and its upward prolongation in the pons varolii.

5. The Fifth Nerve emerges from the pons through the inner part of the cerebellum, (see Fig. 5. V.+.); this figure shows that it consists of two roots of unequal size,(+.V.) the smaller being motor and the larger sensory. The deep origin of the trigeminus is more extensive than that of any other cranial nerve; cephalad it arises from a group of large nerve cells that surround the aqueduct of Sylvius, while caudad it originates from as low a point in the medulla as the tubercle of Rolando and by some competent observers it is supposed to extend as far down as the second or third cervical segement; thus its deep origin reaches from the anterior quadrigeminal tubercle to the third cervical segment. The force of Pierret's statement that the sensory root of this nerve represents the sensory roots of all the motor cranial nerves from the hypoglossal to the third, can now be fully appreciated.



Origin of fifth nerves *V mot*, motor nucleus *V asc.*, ascending root.

DIAGRAM XV.

Origin of Fifth Nerves—V. mot., motor nucleus; Vasc., ascending root.—(*Gowers*,)

While many of the fibres of the sensory root end in the middle nucleus (see above diagram, middle nucleus), which is situated beneath the outer edge of the floor of the fourth ventricle, the majority of the fibres turn downward and descend into the pons and medulla forming its ascending root (diagram XV, V asc.); they terminate

in the column of gelatinous substance found throughout this region, which as previously stated marks the upward prolongation of the tubercle of Rolando.

The Medial Root is composed of fibres of the fifth nerve which pass through the longitudinal bundles, cross the raphe and go to the opposite side of the pons; (see diagram XV, to raphe) fibres also go to the cerebellum.

The Motor Root ends in a cluster of nerve cells that lies on the inner side of the inner sensory nucleus. (Above diagram, V. mot.)

The Descending Root which arises from the large nerve cells situated in the gray matter of the aqueduct near the superior quadrigeminal body, is regarded by Meynert as sensory, by Henle and Forel as motor and by Gowers as yet undetermined.

We are now prepared to make the following summary of the deep origin of the cranial nerves. The vagus, the glosso-pharyngeal arise in the medulla; the spinal accessory arises both from the upper cervical cord and the medulla; the auditory arises from both the medulla and the pons; the facial and the sixth arise solely from the pons, (the facial does not spring from the nucleus ambiguous); and finally that the fifth nerve arises from the upper cervical region (?), the medulla, the pons varolii and the crura —(*Bastian, Quain, Gowers, Ferrier, Stewart, Webber, Starr, Landois and Sterling.*)

For the purpose of conciseness and clearness, the following summary may be helpful:

A. Boundary of the formatio reticularis in the medulla and pons.

a. That the formatio reticularis (formatio reticularis grisea) is that portion of the medulla lying between the gray matter of the floor of the fourth ventricle and the pyramidal tracts, in a dorso-ventrad direction, and between the interolivary tract (formatio reticularis alba) and the ascending root of the trigeminus nerve.

b. The formatio reticularis is that portion of the pons that lies between the gray matter of the floor of the fourth ventricle and the fillet, in a dorso-ventrad direction, and between the raphe and the external border of the pons in a lateral direction.

c Anatomically speaking, the term formatio reticularis includes under it both the white and the gray reticular formation, but it is better, as some neurologists have done, to restrict its meaning solely to the latter—formatio reticularis grisea. Precedent has given us

the authority to speak of the whole in terms of one of its parts, as in the cas above where we refer to interolivary tract instead of the formatio reticularis alba.

B. The tracts of common and special sensibility (muscle sense) in the medulla and pons.

a′. The sensory tracts in the medulla are the Interolivary Tract and the Formatio Reticularis.

b′. The sensory tracts in the pons are the Fillet and the Formatio Reticularis.

c′. That the sensations of touch, pain and temperature (common sensibility) which have decussated in the spinal cord, are transmitted through the formatio reticularis in both the medulla and pons.

d′. That the sensations of muscle sense which have not decussated in the cord, but cross the median line in the sensory decussation of the medulla, are transmitted by the interolivary tract in the medulla and the fillet in the pons; the sensations of pressure and location are conveyed by the same fibres and are included under the same terms as muscle sense.

e′, "If any sensory impulses pass to the cerebellum, they are only those whose result is to awaken reflex action, and they are not the sensory impulses whose reception in the cortical cells is capable of awakening a conscious perception of the sensation. The sensations consciously perceived do not pass through the cerebellum n their way to the cerebrum."—(*Starr.*)

In the preceding discussion the terms lemniscus and interolivary tract are used interchangably to indicate the path of muscle sense in the medulla, while the term fillet is used to designate the corresponding tract in the pons.

LESIONS OF THE MEDULLA AND PONS.

Diagram V shows the surface attachment and relative position of the cranial nerves and by a careful study of it one can readily see the various groups of palsies occurring in disease of the base of the brain. The third pair of nerves, it will be observed, arises nearest the middle line, and because of this, a lesion may involve both of them more readily than any other pair. From diagram V we also see that the hypoglossal nerves, although closely related to each other, are separated by the anterior pyramids; it is seldom that both are

affected by lesions outside the medulla. The two sixth nerves are seen to lie near together, issuing from the medulla at the lower border of the pons; they are often affected by disease because of their long course close to each other through the most prominent part of the pons: distant pressure, also, readily affects them. At its surface attachment the fifth nerve is seen to be isolated and alone; the one nearest to it is the sixth, hence the latter is the one most frequently associated with it in paralysis; the third and fourth pair also lie near it. It will be seen that the vagus, spinal-accessory and glossopharyngeal nerves lie in close contact as do also the facial and auditory. Because of the close proximity of the hypoglossal to the spinal-accessory in its outward passage, palsy of the tongue sometimes occurs with that of the vocal chord of the same side.

Disease of the medulla and pons gives rise to two classes of lesions, namely, system and indiscriminate (focal) lesions; the former always attack the conducting (motor and sensory) paths and are called primary in order to distinguish them from Wallerian degeneration of the same tracts, which is of course secondary. Lesions situated cephalad to the bulb and pons cause descending degeneration of the motor tract; while those caudad to them cause ascending degeneration of the sensory path. The above is an excellent illustration of the general law, enunciated by Waller, that degeneration in nerve fibres takes place in the direction of their nerve impulses. Bulbar paralysis is the best type of a system lesion of the motor tract.

The symptoms following indiscriminate or focal lesions depend not only upon the nerve fibres and nuclei involved, but also upon the extent of the tracts (motor and sensory) invaded, hence it follows that these lesions cause symptoms not only grave in character but of exceeding complexity as well. Focal disease of the medulla is said to cause diabetes, polyuria, albuminuria, salivation, etc.

LESIONS OF THE SENSORY AND MOTOR CONDUCTING PATHS IN THE MEDULLA AND PONS.

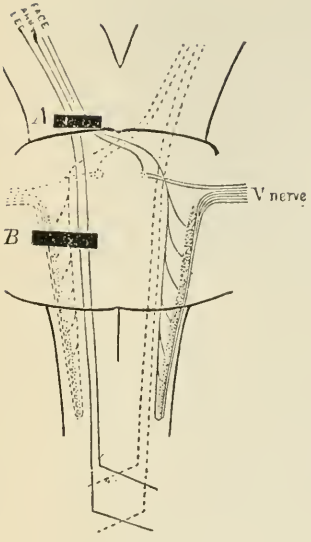


Diagram 16.

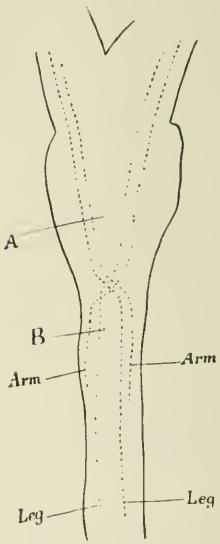


Diagram 17.

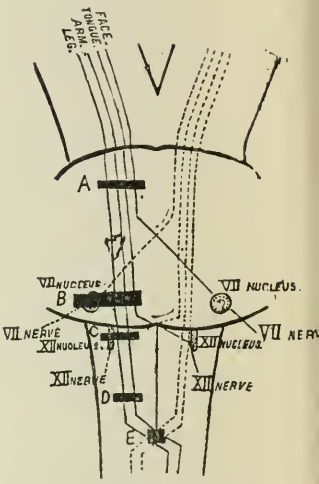


Diagram 18.

DIAGRAM XVI.

The course of the sensory tract in pons and medulla. A, lesion in tegmentum of crus cerebri, or upper quarter of pons, producing hemianaesthesia of the opposite side; B, lesion in formatio reticularis of pons or medulla, producing alternating anaesthesia. (*Starr.*)

LIAGRAM XVII.

The course of tract conveying muscular sense from the limbs. A, lesion above the decussation, producing ataxia of the opposite side; B, lesion below the decussation, producing ataxia of the same side. (*Starr.*)

DIAGRAM XVIII.

The motor tract in pons and medulla. Lesion at A causes hemiplegia of opposite side to lesion; lesion at B causes alternating paralysis; lesion at C causes paralysis of tongue on side of lesion and of extremities of opposite side; lesion at D causes paralysis of extremities of opposite side; lesion at E causes paralysis of extremities of both sides.—(*Starr.*)

SENSORY TRACT.

a. If in any case anaesthesia of one side of the face occurs, not due to neuritis of the trigeminus or a cerebral lesion, the lesion lies in the medulla or pons in the outer third of the formatio reticularis; its position in this part is to be determined by the other symptoms present, for if it is situated high up, cephalad, in the pons, it will be on the side opposite to the anaesthesia, and if it is situated low down, caudad, in the pons or medulla, it will be on the same side as the anaesthesia.

b. If in any case anaesthesia of the limbs occurs, not due to cerebral lesion, the lesion lies in the medulla or pons in the inner two-thirds of the formatio reticularis and upon the side opposite to the anaesthesia or in the spinal cord.

c. If one side of the face and the limbs of the opposite side are anaesthetic, the lesion affects the entire lateral extent of the formatio reticularis and lies in the medulla, or in the pons below the point of union of the ascending and descending roots of the fifth nerve.

d. If the face and limbs of the same side are anaesthetic, the lesion lies in the brain higher than the junction of the ascending and descending roots of the fifth nerve; in the pons its position is then to be determined by other symptoms; it may involve the entire formatio reticularis in the upper pons or crus cerebri, it may be situated in the posterior part of the internal capsule, it may lie in the centrum semi-ovale, destroying the radiation of the sensory fibres from the internal capsule, it may be in the sensory area of the cortex in which all these tracts terminate.

Muscle Sense Tract.—Sensations of muscle sense being transmitted by this tract, (the interolivary tract or lemniscus in the medulla, the fillet in the pons) a lesion of it causes ataxia in the limbs of the opposite side; a unilateral ataxia may indicate a lesion in the course of the fillet.

Motor Tract.—Lesion of the motor tract in the upper third of the pons (Fig. XVIII, A.) causes hemiplegia of the side opposite the lesion; a lesion in the medulla or lower third of the pons (B) causes alternating paralysis, i. e. paralysis of the face on the side of the lesion with paralysis of the arm and leg on the opposite side.

In the first case (hemiplegia) the upper branch of the facial nerve is not affected, the patient can close his eye on the paralysed side and the facial muscles react to Faradism. In the second case, (alternating paralysis) all branches of the facial nerve are involved, the eye remains open and the reaction of degeneration develops soon after the lesion occurs; in this case it is usual to find a paralysis of the abducens nerve on the same side as the facial palsy and the eyeball will be turned inward. The motor fibres to the tongue take a similar course to those of the face but leave the motor tract at a point in the lower third of the pons; having reached the hypoglossal nucleus in the medulla, they end, but the hypoglossal nerve passes outward, lying in close proximity to the motor tract in the medulla as shown in the diagram.

A lesion in the lower third of the pons or in the medulla (C) may thus cause paralysis of one side of the tongue and of the opposite arm and leg.

In the medulla the motor tracts pass in the anterior pyramids and before entering the spinal cord undergo partial decussation; a lesion in one pyramid will cause paralysis of the opposite arm and leg but as the two pyramids lie side by side, lesions of the medulla, by affecting both at once, may produce paralysis of both arms and legs and this is especially the case if the lesion lies so near the spinal cord (E) as to involve the decussation. The various forms of paralysis produced when both sides of the pons are involved at once can be ascertained by reference to diagram XVIII. In all these affections of the motor tract a condition of spastic rigidity may follow the paralysis and an increase of tendon reflexes is usually observed; in a few cases of pons lesions however the tendon reflexes have been lost. The electric condition of the paralysed muscles in the limbs is normal from the outset and no rapid atrophy occurs.

Lesions of the Centers lying in the pons and medulla. Lesions which irritate the tracts from the cortical centers to the motor nerve nuclei in the pons and medulla or lesions in their vicinity which cause irritation, may produce spasm of the muscles supplied by them, thus trismus with grinding of the teeth, nystagmus, conjugate lateral deviation of the eyes, spasm of the face, spasm of the throat and larynx and spasm of the tongue, may occur from lesions of an irritating nature. Such are small hæmorrhages, small areas of softening or small tumors in the pons or medulla. Such lesions

must lie in or near the floor of the fourth ventricle or in the course of the nerve roots to produce these spasms.

Lesions of the Motor and Sensory Nerves.—The Motor Nerves Lesions which interrupt the tracts from the cortical centres to the motor nerve nuclei, which destroy those nuclei or which interfere with the conduction of nerve impulses along the nerve root and nerve trunk will produce paralysis. The rarest form of paralysis is that of the muscles of mastication; while that of the external rectus muscle or the tongue or the facial muscles is very common; occasionally, however, paralysis of the vagus or of the muscles of deglutition is seen. If the lesion be situated in the nerve nuclei, rapid atrophy occurs in the paralysed muscles and is accompanied by the reaction of degeneration; if, however, the lesion invades the central tracts, there is no pronounced atrophy of the muscles neither is their normal muscular formula altered. Lesion of a nerve root or trunk is accompanied by the same symptoms as that of its nucleus, hence other symptoms are necessary to establish a diagnosis of a lesion in the nucleus.

When paralysis of the muscles supplied by one cranial nerve alone occurs, the probability is in favor of a peripheral nerve lesion, viz.: a meningetis or tumor on the base of the brain, and a central lesion can only be thought probable when an implication of other muscles in the region of other nuclei ensues. A very extreme contraction of the pupils is said to be seen in cases of sudden lesion of the pons. Paralysis of the motor portion of the glosso-pharngeal with dysphagia, is a not infrequent symptom of pons disease.

Paralysis of the motor and vaso-motor mechanism, governed by the vagus nerve is a constant symptom of disease in the lower part of the pons and medulla.

Arrest of respiration and heart action from lesion of this nucleus is the cause of sudden death which so frequently attends sudden lesions in the medulla and pons. When, however, the lesion is of a slowly progressive nature, (as tumor or sclerosis about a hæmorrhage) symptoms of irritation may occur from gradual invasion of the pneumogastric centres. Projectile vomiting; slow, very rapid, intermittant or irregular pulse, dyspnoea, loss of voice, polyuria and glycosura are symptoms which have been ascribed to such lesions. The hypoglossal nerves, owing to their large nuclei, and their passage through the middle of the medulla, are frequently involved in

medullary disease. Paralysis of the tongue occurs more frequently than spasm or twitching from their involvement, and when both sides are paralysed, talking, chewing and swallowing are interfered with.

An electric examination of the intrinsic muscles of the tongue showing the reaction of degeneration, proves the lesion to be medullary and not cerebral.

b. The Sensory Nerves. Lesions which affect the trigeminal nerve produce anaesthesia of the face; trophic disturbance of the eyeball has not yet been observed in cases of lesion of the fifth root or nucleus. In disease of the pons and medulla the auditory nerve occasionally suffers. Two sets of symptoms are then produced, one referable to the implication of the fibres conveying auditory impressions, the other due to the irritation or destruction of fibres concerned in equilibration. Deafness has been observed in pons disease on the side of the lesion, and is probably due to destruction of the auditory fibres passing inward to the inner auditory nucleus on the floor of the fourth ventricle, or to an implication of that nucleus. Lesions in any part of the auditory tract should produce deafness in one ear; disturbance of equilibrium may occur from lesions in the lateral portion of the pons if such lesions interrupt the part of auditory nerve conveying such sensations to the cerebellum. Vertigo, which as a frequent symptom of pons lesion, and rotatory movements of the body toward one side, which is an occasional symptom of pons disease when the lesion involves the lateral portion of the pons and the inferior and middle cerebellar peduncles, are probably due to an involvement of this portion of the auditory nerve in the lesion. The rotatory movement is probably due to a voluntary attempt to correct a subjective sense of motion; the patient really suffers from vertigo and thinks that he is turning or falling toward one side; he therefore tries to prevent his falling by turning toward the other side.

In two cases in which the nucleus of the sensory part of the glosso-pharyngeal nerve has been involved, a loss of taste has occurred.

Vaso-motor Centres. Lesions in the upper half of the medulla produce marked vaso-motor symptoms; the vaso-motor centre is bi-lateral and each centre controls the circulation of the same side of

the body. Among the symptoms of disease in its area are vaso-motor paralysis, with flushing of the surface and sensation of heat and abnormal sweating. When these symptoms are limited to one lateral half of the body and are associated with other symptoms of bulbar disease, they are valuable as the signs of the situation of the lesion.

Sudden lesions of the pons are not infrequently attended by general convulsions followed by coma; the convulsions are probably caused by irritation of the motor tracts passing through the pons.

There are certain general symptoms, such as headache, vertigo, disturbance of vision, etc., that can be more properly ascribed to variation in the inter-cranial circulation than to any definite local disease.

Lesions of the medulla oblongata are frequently bi-lateral in character and there early appears prominent motor symptoms in the mouth, throat and larynx; all the extremities may be paralyzed to a greater or less degree, there may also be disturbed heart action.

There may be, says Dr. Bastian, "a great general similarity between the symptoms occasioned by disease in the pons and disease in the bulb. Certain symptoms may be common to both, such as difficulties of a marked character in articulation and deglutition, associated with bilateral paralysis, more or less complete in degree, together with loss of emotional control; on the other hand, there are often distinguishing signs pointing to the existence of a lesion in one locality rather than in the other. Some of the principal differential signs are these:—

A. Combination of signs pointing to a lesion in the pons.

a. The co-existence of marked difficulty in deglutition and in articulation with an extremely well marked paralysis of the facial muscles, either on the opposite or on the same side as the paralysis of limbs.

b. No loss of phonation or paralysis of the palate; preservation of its reflex and of that of the pharynx.

c. Evidence of paralysis of the fifth or of the sixth nerves. The existence of well marked sensory defects in the paralysed limbs or half of the body.

d. Strabismus, external or internal, from paralysis or irritation of the sixth nerve.

B. Combination of signs pointing to a lesion in the bulb.

a′. The co-existence of extreme difficulty in articulation and in deglutition, with paresis involving the lower facial muscles only, (especially the orbicularis oris), and generally of a bilateral type.

b′. Often, loss or great impairment of phonation, with paralysis of the palate together with loss of its reflex excitability, and also of that of the pharynx.

c′. Evidence of defects of hearing of centric type.

d′. Absence of well marked sensory defects (?) in the paralysed limbs.

e′. Marked interference with the action of the heart or disturbance of the respiratory rythm."—*Gowers, Seguin, Starr.*

Note.—I am greatly indebted in the preceding discussion of the lesions of the medulla and pons to Dr. M. Allen Starr, from whose writings, by his special permission, I have drawn freely without verbal change.

THE CRURA CEREBRI AND ITS LESIONS.

By crura cerebri we mean not only the two thick strands of longitudinal fibres, (the cerebral peduncles), which emerge from the pons on their way to their destination in the cerebrum, but under that term we include also the substantia nigra, the tegmentum, (cap) along with its grey matter, nerve cells, nerve fibres, reticular formation, fillet, posterior longitudinal bundle and red nucleus; also the Sylvian aqueduct with its surrounding gray matter, in which lies certain cranial nerve nuclei, and the lamina quadrigemina which forms its posterior walls.

The aqueduct of Sylvius is a narrow passage, a little more than half an inch long, in which the fourth ventricle ends and which abruptly, in turn, opens into the third ventricle. Diagram XIX shows the iter to be surrounded by a thick layer of gray matter (centrales hohlengrau), which is prolonged from the gray matter of the fourth ventricle. In addition to the nerve cells which are scattered through the gray matter of the aqueduct, are certain well defined columns of nerve cells with which are connected the roots of the fifth, fourth and third nerves. By reference to the above diagram the third is seen to arise from a column of large nerve cells, (Nucl. N. oculomot.), lying in the ventrad portion of the gray matter of the aqueduct on either side of the median raphe. The nucleus of the oculo-motorius is situated in the region of the superior quadrigeminal body and reaches cephalad as far as the posterior commissure and extends caudad along by the nucleus of the fourth nerve. Ventrad to the nucleus of the third is seen the posterior longitudinal bundle (Hint. Langsbundel).

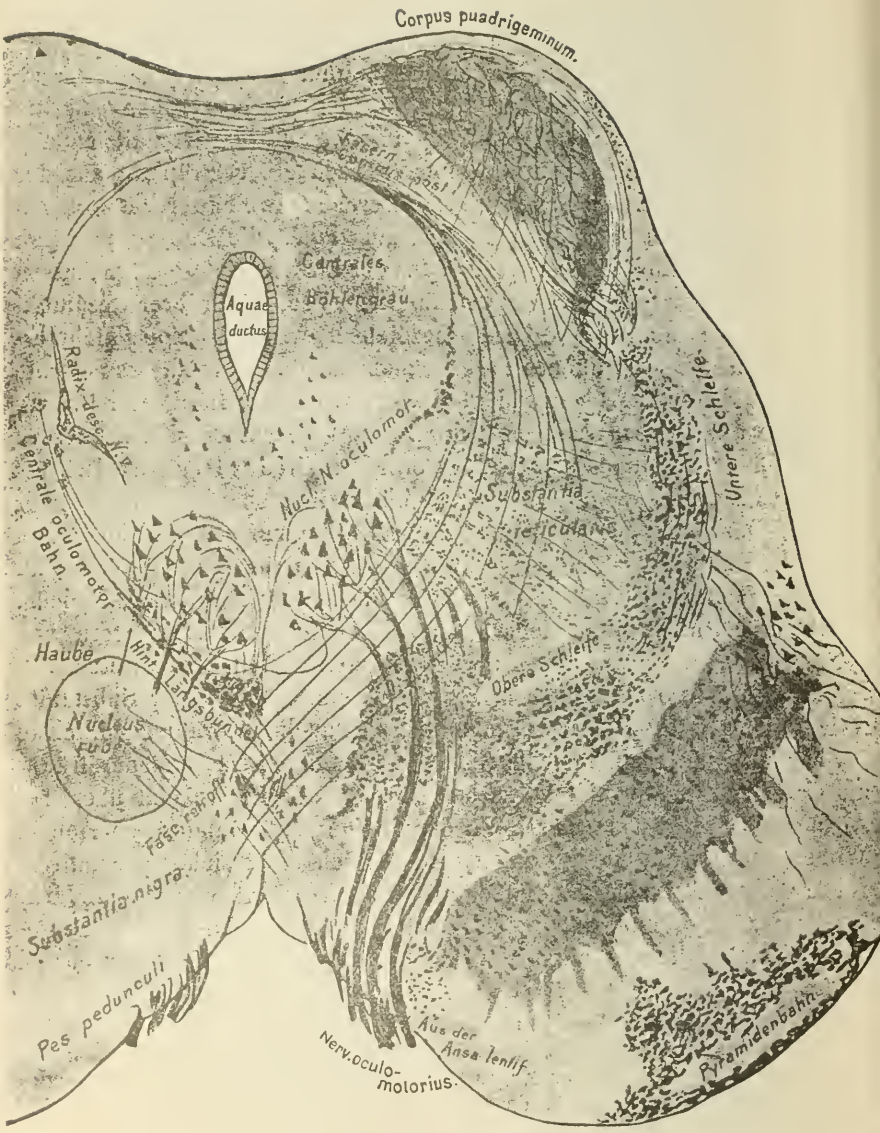

Corpus quadrigeminum.
Centrales Höhlengrau
Aquae ductus
Radix descend. N. V.
Centrale oculomotor. Bahn.
Haube
Nucleus ruber
Fasc. retroflex.
Substantia nigra.
Pes pedunculi
Nerv. oculo-motorius.
Aus der Ansa lenticul.
Pyramidenbahn.
Untere Schleife
Obere Schleife
Substantia reticularis
Nucl. N. oculomot.

DIAGRAM XIX

Centrales hohlengrau,—gray matter of the aqueduct; Fasern d Commis post.,—fibres of the posterior commissure; Untere Schleife,—lower fillet; Obere schleife,—upper fillet; Centrale oculo-motor Bahn,—the central oculo-motor tract; Hint langs bundel,—posterior longitudinal bundle; Haube,—the tegmentum or cap; aus der ansa lentif,—from the ansa lenticularis; Pyramidenbahn,—pyramidal tract. (After Edinger.)

Most of the fibers of the central tract (See diag. XIX, Centrale Oculo-motorbahn,) which, arising in the cortex, pass down by way of the internal capsule into the crus, are seen to end in the oculo-motor nucleus of the same side; some however are observed to pass to the nucleus of the opposite side. From this nucleus the nerve fibres pass forward through the tegmentum and its red nucleus to emerge from the inner surface of the crus cerebri at the oculo-motor groove just cephalad to the pons. The fourth nerve arises from a nucleus situated just between the superior and inferior quadrigeminal bodies, caudad to that of the third nerve; its cells are larger than those of the nucleus of the latter nerve and they are probably a continuation upward of the anterior horn of the spinal cord. From these cells the fibers of the fourth nerve pass obliquely downward and backward in one or more bundles, coursing along in the wall of the aqueduct to reach its posterior extremity; from this point the fibres curve inward and cross over the aqueduct in the valve of Vieussens to the opposite side, emerging from the velum close to the middle line; some observers do not regard the decussation as absolute, but affirm that part of the fibres are derived from the nucleus of the same side. It must not be forgotton here that the fourth and third nuclei are connected with that of the sixth by the posterior longitudinal bundle, so that all the nerves to the ocular muscles are thus correlated at their centres. Again referring to the gray matter of the aqueduct in Diagram XIX, we observe the highest centre of origin of the fifth nerve (Radix desc. N. V.). It is a group of large vesicular nerve cells situated laterad to the aqueduct; its function, as before stated is unknown.

The formatio reticularis (This net like formation which is rich in nerve cells, does not show the distinctive features of gray substance because of the predominance of white fibres and is known as substantia reticularis. Obersteiner) is much smaller in size in the crus than in pons or medulla; at its upper level (the level of

the superior quad. body) distinct bundles of fibres pass out into the brain. Chief among them is the sensory tract which at this point enters the posterior part of the internal capsule, lying in the inner half of its posterior third, thence by the way of the corona radiata it passes to the cortex of the central and parietal regions, and conveys sensations of touch, temperature and pain. Fibres also pass to the anterior corpora quadrigemina, the optic thalamus and to the central gray matter of the third ventricle.

There is still much difference of opinion concerning the character and ultimate termination of the fibres of the fillet; the subject is one of much complexity and there is yet a vast amount to learn concerning the origin and destination of its fibres. The nomenclature of the fillet is entirely lacking in uniformity and it has been suggested by a recent author that the difficulties which beset a comprehension of this vexed subject are due as much to a confusion of names as to a complexity of structure. According to Roller most sensory nuclei are connected with this tract and this assertion aside from the fact that it originates in the posterior columns would mark it as a sensory path, but since it degenerates both cephalad and caudad we may infer that its function is not entirely sensory in character. It forms the outer and ventrad boundary of the tegmentum and is made up of two sets of fibres, upper (obere schleife) and lower (untere schleife) fillet; the upper fillet is the larger and contains fibres from the interolivary tract which have probably been derived from the olivary body and from the nuclei gracilis and cuneatus; these fibers pass upward external to the red nucleus of the tegmentum, turn outward and pass through the internal capsule to end in the parietal cortex. The lower fillet comes from the superior olive and from the corpus trapezoides, and terminates in the posterior corpora quadrigemina. (Starr.)

One of the important tracts in the tegmentum is the superior peduncle of the cerebellum. It arises chiefly from the dentated nucleus of the cerebellum; fibres also pass to it from the superior and middle portion of the cerebellum and from the nucleus of the auditory nerve(?): passing upward from the cerebellum, it disappears beneath the quadrigeminal body, decussates with its fellow of the opposite side, and apparently terminates in the red nucleus of the tegmentum,—a tract of gray matter with large pigmented cells.

While a majority of the fibres do end in the cells of the red nucleus, a certain number, lying dorsad, separate into two bundles, one of which enters the internal capsule and the other passes to the optic thalamus.

The substantia nigra is a mass of gray matter (see above diagram) which separates the crusta from the tegmentum; it derives its name from its appearance which is caused by having scattered through its substance irregular nerve cells very darkly pigmented. From one-third to one-half of each cell is filled up with pigment which does not appear until extra-uterine life. According to Obersteiner the pigment in these spindle shaped cells of the substantia nigra is never present in lower animal life and its cells are distinguished from those of the locus cœruleus by the greater diameter and the round vesicular form of the latter. It extends from the upper border of the pons to the posterior border of the corpora albicantia. Glancing at the diagram it will be seen that on the ventrad surface of the substantia nigra there are irregular projections of the gray matter between which are bundles of white fibres, the stratum intermedium of Meynert; their origin and destination are different from those of the other fibres of the crusta. The fibres of the posterior commissure turn downward and pass under the corpora quadrigemina and enter the formatio reticularis where they are lost.

The corpora quadrigemina are masses of gray matter intermingled with fibres, that form the posterior wall of the crura cerebri (see diagram, corp. quad.) From the "brachium" of the anterior tubercle fibres pass to the external geniculate body and to the optic radiation by way of the posterior portion of the internal capsule. In like manner fibres pass by way of the "brachium" of the posterior quadrigeminal body to the internal geniculate body and are thus "in connection" according to Von Gudden, quoted by Gowers, "with the commissural fibres of the optic tract, which at the optic chiasma, turn back along the opposite tract to the opposite internal geniculate body and corpora quadrigemina." Fibres pass to the quadrigeminal bodies from the reticular formation and the fillet(?). Meynert believes that fibres pass to the nuclei of the third nerve from the gray matter of these tubercles.

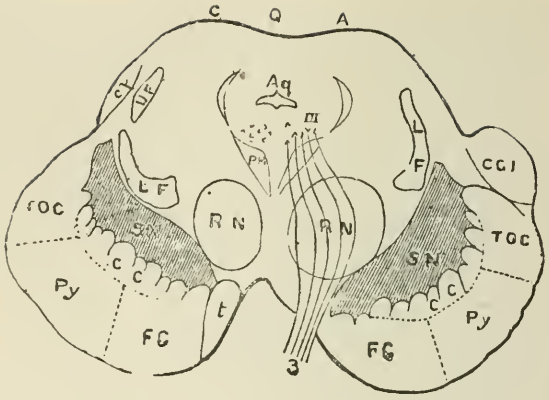


DIAGRAM XX.

Section of the Crura cerebri (Modified from Wernicke by Gowers) L F, U F, upper and lower fillet; C Q A, Anterior corp. quad.; Aq, aqueduct; III, Nucleus of the third nerve, (3); P. H., posterior horizontal fibres; c p, brachium of the post. corp. quadrigemina; R N, red nucleus; S N, substantia nigra; C G I, internal geniculate body; T O C, temporo-occipital cerebellar fibres; Py, pyramidal fibres; F C, fronto-cerebellar fibres; C C, caudate-cerebellar fibres; (stratum intermedium of Meynert); inner fibres of crusta to tegmentum.

Of the fibres that form the crusta less than half belong to the pyramidal tract. Glancing at the above diagram the relations of the white fibres of the crusta are readily seen. They are observed to lie partly on the inner and outer sides of the pyramidal fibres and also above, i. e. between the latter and the substantia nigra. Of these various sets of white fibres of the cerebral peduncle those alone on the medial side, (F C,) pass up to the internal capsule and are distributed to the pre-frontal lobe (Gowers); they degenerate downward and end in the gray matter of the pons, and from the nerve cells of this gray matter in which they end the fibres of the middle cerebellar peduncles arise and pass to the lateral and posterior regions of the cerebellar hemisphere, the larger number going to the opposite cerebellar body, the less to that of the same side. The small bundle of fibres (t) differs in course from the rest, for instead

of terminating in the gray matter of the pons it passes backward, joins the fibres of the fillet and is there lost. The fibres (T O C) that lie outside the pyramidal tract do not enter the internal capsule but pass to the cortex of the occipital and temporal lobes. This bundle of fibres also ends in the gray matter of the pons similarly to that on the inner side of the pyramidal tract, and according to Fleschig is chiefly connected with the upper surface of the cerebellar hemisphere near the middle lobe; it degenerates downwards.

The lenticular loop (see above diagram, Aus der Ansa lentif) is formed from fibres that come from the caudate nucleus passing through the lamina medulares of the lenticular nucleus and is joined by fibres from the outer body of the nucleus, thence the two sets unite in one and pass down the crus and enter the substantia nigra, forming the stratum intermedium of Meynert and finally end in the column of Turk. By reference to diagram XX it will be seen that this last bundle of crustal fibres (C C) is situated between the pyramidal fibres and the substantia nigra, and is known to degenerate downward.

Lesions of the Crura Cerebri and the Quadrigeminal Bodies. A lesion of the tegmentum of the crus would necessarily cause involvement of the sensory paths that pass through it, giving rise to anaesthesia. Incoördination would occur with paralysis of the third nerve. By reference to diag. XIX the third nerve is seen to pass through the red nucleus. A lesion affecting the crusta in which the pyramidal tract courses would cause hemiplegia of the opposite side. Since the third nerve issues from the groove on the inner side of the crusta (Sulcus-oculomotorii) a lesion at this point would involve the nerve on the side of the lesion, hence, there would be paralysis of the third nerve on the side of the lesion, with hemiplegia of the opposite side of the body, a characteristic lesion of the crustal region. So little is definitely known about the function of the quadrigeminal tubercles that lesions of these bodies are as yet imperfectly recognized. Hæmorrhage into their substance is a rare accident, tumors developing in them are a matter of record; and softening of both infrequently happens. A lesion involving the anterior pair is said to cause both blindness and loss of pupil reflex, also strabismus and nystagmus.

Disease of the posterior pair gives rise to disturbance of co-ordination, and as both anterior and posterior quadrigeminal bodies are usually involved at the same time, a combination of the above symptoms may be expected. *Gowers, Starr, Bastian, Quain, Flechsig, Obersteiner.*

THE INTERNAL CAPSULE AND ITS LESIONS.

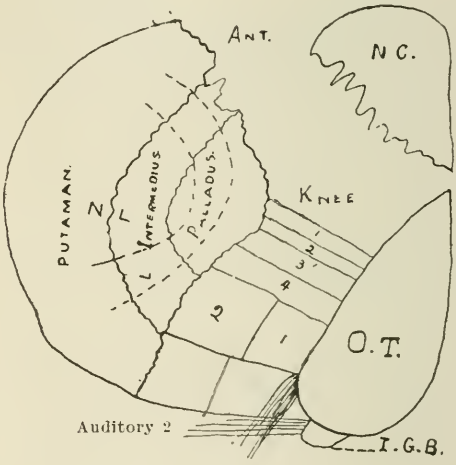


DIAGRAM XXI.

Ant.—Fibers from frontal to inner ⅓ crus and pons nucleus.
" " " " " optic thalamus.
" " " N. C. to N. L., thence Lenticular Loop.
Knee—Fibers to 3d frontal convolution—(speech tract).
Post.—Ant. (⅓) Fibers of motor tract in this order:
(1) face, (2) tongue, (3) arm, (4) leg and trunk; hence, monoplegia from capsular lesion is possible.
Post.—Middle (⅓) Fibers of the sensory tract.
(1) Fibers of touch, temp., pain, Formatio Reticularis.
(2) Fibers of muscular sense from Lemniscus.
Post.—Post. (⅓) (1) Fibers of visual tract.
(2) Fibers of auditory tract.
Fibers go to O. T. from Occipital, Parietal and Temporal lobes.
—After Dr. Starr.

The internal capsule is a layer of white substance formed by the fibres from the crura cerebri passing between the gray masses of the basal ganglia (the 'tween brain and the fore brain) on their way to the cerebral cortex. Glancing at the above diagram, we see it is divided into an anterior division, a knee and a posterior division. The latter is still further divided into three parts, the anterior (⅓), middle (⅓), and posterior (⅓). Fibres (the frontal pontine tract) pass through the anterior division of the internal capsule to the frontal lobes and the nucleus caudatus. The pyramidal tracts terminate in the Rolandic area, namely the central convolutions, the paracentral lobule and the anterior part of the parietal lobes, while in the middle and posterior thirds of the posterior division of the capsule the various sensory tracts mingle together forming the "carrefour sensitif". The hindermost fibers of the capsule (auditory, 2, visual, 1,) are distributed to the temporal and occipital (optic radiations of Gratiolet) lobes. The ultimate termination of the various sensory fibres is practically the same as that of the motor fibres,—the cortical, motor and sensory areas, to a great degree coinciding. The various strands of fibers just described as being found in the internal capsule are compressed, as it were, in a narrow space between the gray masses of the optic thalamus and the corpus striatum, but as they emerge in the centrum semi-ovale they spread away from each other in all directions to the cerebral cortex, forming the corona radiata Reilii. This stratified arrangement as portrayed in the above diagram, is in perfect harmony with their known function which has experimentally been demonstrated, of late by Beevor and Horsley and previously by Franck and Pitres. "It has been satisfactorily established that the cones of medullary fibres corresponding to the respective cortical centres are functionally differentiated (the stratified arrangement as seen in diagram No. XXI is according to their function, Fleschig has observed that the fibre tracts traversing the internal capsule are inconstant in their relation to its knee) like the cortical centres themselves, and maintain their individuality and are echeloned in definite and regular order in the internal capsule." *

The significant fact in the latest investigation in this line is that

* The Croonian Lectures on Cerebral Localisation, by David Ferrier. M. D., British Medical Journal, June 7th, 1890.

these functionally stratified fibres of the internal capsule are simply message carriers to and from the cerebral cortex and that the cortical cerebral centres to which they are ultimately distributed are likewise functionally differentiated; also that the latter (the cortical centres) store up and transform like other nerve centres, stimuli which they have received into their own energy. (Ferrier). That the above theory of the function of the fibres of the internal capsule does not lack scientific certitude is shown by the statement of Ferrier that "after the removal of the cortex, the medullary fibres lose their excitability, like motor nerves, separated from the anterior cornu of the spinal cord, so that after a lapse of four days no reactions can be produced by the strongest stimulation." It may be well to recall the fact that the pyramidal tract is "a long, unbroken fibre route between the cortex of the great brain and the cells of origin of the motor nerves." By reference to diagram II, we see that A represents the cerebro-spinal segment, B the spino-muscular segment. Between the two divisions of the nerve fibre one nerve cell (at least one) of the anterior horn will be observed, or it may be its homologue, a motor cell of the medulla oblongata. Spitzka has made the very remarkable discovery that not only the dolphin, which has no hind limbs, possesses only a rudimentary pyramidal tract, but that the same condition of things is also found in the elephant and armadillo. Obersteiner calls attention to the fact that the pyramidal fibres become medullated in the centrum semi-ovale and that the myelination proceeds in a caudad direction, requiring several weeks to reach the lumbar cord.

LESIONS OF THE INTERNAL CAPSULE.

Lesions of the anterior division of the internal capsule are rare and if they do not involve its knee, there are no known symptoms by which they can be recognized; for it will be remembered that the function of the tracts in the anterior division of the capsule is still undetermined. The most common form of hemiplegia is the result of a lesion involving the knee and the anterior third of the posterior division of the capsule; the face, tongue, arm, leg and trunk being the parts affected; if the lesion be situated on the left side transient speech defect will be present (see diagram XXI). If the lesion be of sufficient magnitude to destroy the medullary cones

of this region, they, being cut off from their trophic centres in the cerebrel cortex, will degenerate caudad and late rigidity will of necessity follow: convulsions, with deviations of the head and eyes are usually present in these cases. Monoplegias not infrequently occur, the hæmorrhage being small and involving any single one of the motor tracts. If the lesion invades the middle and posterior thirds of the posterior division of the capsule, hemianaesthesia with impairment of the special senses will occur, (see above diagram 2-muscle sense fibres, 1-touch, temperature and pain fibres; 2-auditory, 1-visual). If the destruction of the "carrefour sensitif,"—the "sensory crossway" of Charcot—be only partial, the hemianaesthesia will be incomplete, and according to Gowers, during the remainder of life pains will often be felt in the affected limbs. Lesions of the optic thalamus and corpus striatum give rise to no known spmptoms unless they interfere with the integrity of the capsular fibres. Athetosis, post paralytic chorea and clonic spasms that so frequently occur in the wake of lesions developing in this region are due, not to the involvement of the thalamus or striate body but to the irritatio ı these lesions cause of the pyramidal tract in its course through the internal capsule. Should a hæmorrhage occuring in either the corpus striatum or optic thalamus be sufficiently profuse to break through into the lateral ventricles a fatal result would speedily follow. The anterior cerebral artery gives a branch to the caudate nucleus and the internal posterior optic artery supplies the inner aspect of the thalamus: a rupture of either one of these vessels is especially liable to cause extravasation of blood into the ventricles. An embolism or thrombus of the Sylvian artery (the middle cerebral) would cause softening of the internal capsule and the basal ganglia with the exception of those areas which are supplied by the above named arteries; the symptoms following such a lesion would be hemianaesthesia, with dullness and disturbance of the mental faculties for a time; if the lesion be on the left side aphasia will be present due to the involvement of the cortical system of the Sylvian. The basal arteries of the Sylvian are of great importance because "they supply the caudate nucleus, except its head, the lenticular nucleus, the internal capsule and part of the optic thalamus". They are divided by Duret into two groups, (1) internal, (2) external; the latter is again divided into two sets, an anterior set called

the lenticulo-striate arteries and a posterior set, the lenticulo-optic arteries. It is only in the anterior division (the lenticulo-striate arteries) that we are now interested. One vessel of this set is generally much larger in size than the others and is especially liable to rupture. (an accident which is likely to happen to both divisions of the external set). This larger vessel courses on the outer side of the lenticular nucleus and has been most appropriately called by Charcot "The artery of cerebral hemorrhage"; the extravasation of blood is either in the outer layers of the lenticular nucleus (the putamen) or just outside of the lenticular body; this region is the most frequent site of cerebral hæmorrhage. The curved lines seen on the surface of the lenticular body in the above diagram indicate the direction, from without inward, in which pressure is exerted by hæmorrhage as just described.

In hemiopic headaches, a variety of hemicrania, the hindermost fibres of the posterior division are involved, the patient complains of a blur before the eyes which is evidently due to hemiopia of a transitory character; he will generally rub his eyes and complain of a feeling similar to that of being snow blind; associated with the ocular symptoms are subjective sensations of light, viz. a cloudy film with bright edges or a flickering or dazzling light before the eyes which persists for a few moments, then disappears to occur again and again. Perhaps the most alarming phase of the disease to the patient is the hemianaesthesia, the hemiplegia and the aphasia which may occur in the course of the attack; hemianaesthesia of the hand is of common occurrence, that of the leg much less so; the hemiplegic condition may, for the time be absolute, the patient not being able to use either his arm or his leg; the aphasic state usually lasts from fifteen to thirty minutes but has been known to persist for as much as fifteen hours. (Mauthner). These symptoms are usually followed by an intense headache. On the contrary however there may be no pain but simply a mental stupor; after a prolonged sleep all these disagreeable symptoms pass away and the patient enjoys his usual health, at least until a recurrence of the attack. One cannot fail of being impressed with the wonderful resemblance between hemiopic headaches and epilepsy; these striking phenomena are supposed to be due to a transient anaemia occurring in the "sensory

crossway" of Charcot. (*Gowers, Quain, Starr, Bastian, Ferrier, Obersteiner.*)

THE FIBRE SYSTEMS OF THE BRAIN.

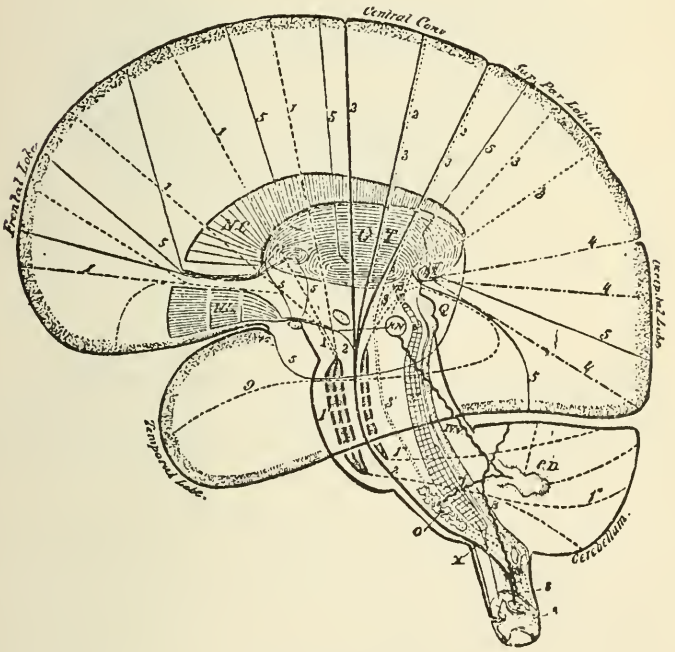


DIAGRAM XXII.

Projection Fibres in the Centrum Ovale and their Terminations. (Modified after Flechsig. Quoted from Starr.) 1, Tract from frontal lobe to pons nnclei, thence to cerebellum; 2, motor tract from central convolutions through capsule, crus, pons and medulla to the cord through the decussation; 3, sensory tract from parietal lobe through capsule, tegmentum and medulla to posterior columns of cord; 3′, by way of the lemniscus: 3″, by way of formatio reticularis; 4, visual tract from occipital lobe to optic thalamus; 5, optic thalamus radiation to all the lobes; 9, auditory tract from temporal lobe to optic thalamus; O. T., optic thalamus; N. L , lenticular nucleus; N. C., caudate nucleus; R. N., red nucleus of tegmentum; O, olive; C. D., corpus dentatum; 7, tract between O. and C. D.; Q. Corp. quadrigem.

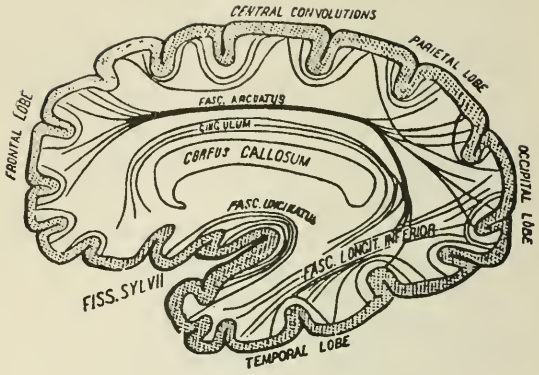


DIAGRAM XXIII.

Association Fibres in the centrum ovale. (Modified after Edinger. Quoted from Starr.) Fasc. arcuatus=fronto-occipital and fronto-temporal tracts; Fasc. uncinatus=temporo-frontal tract; Fasc. longit. inferior=occipito-temporal tract.

The centrum semi-ovale is composed entirely of nerve fibres; these may be divided into three intra-cerebral tracts, viz. the projection, commissural and association fibre-systems.

The Projection Fibre System "includes those fibres which join a definite area of the cortex with parts of the nervous system lying below." (Starr). One end of the projection fibre terminates in the cerebral cortex and the other either in "the nervous mechanism in the basal ganglia, the brain axis or the spinal cord." The cortex is, according to Meynert, devoted to conscious processes and under the term projection system he includes all the conducting paths (fibre routes) whose function it is to keep up a communication between the cerebral cortex and the outer world. "Through the fibres of this system sense pictures are projected on the perceptive cortex and further; not only are movements of ones own body the source of sensations of movement which are represented in the brain in the same way as phenomena of the outer world, but the cortex also by means of the motor tracts reflects

outwards again the states of stimulation, information with regard to which is transferred to it by means of sensory nerves. *

The majority of the projection fibres (diagram XXII, 5) end in the optic thalamus. "Two large bundles are however separable from the mass, one of these passes inward and forward from the occipital lobe and joins the pulvinar of the thalamus and the external geniculate body, (diagram XXII, 4) this is now known as the visual tract and conveys impulses received from one-half of each retina by the optic thalamus to the like named occipital lobe, * * * *. A second bundle passes from the temporal lobe inward to the thalamus and internal geniculate body, lying just beneath the first at its point of entrance into the thalamus, (Diagram XXII, 9). This conveys impulses of sound from both ears to each temporal lobe, * * *. These are the only bundles of the thalmic radiation whose functions are determined." †

The frontal pontine tract (Diagram XXII, 1) arises from the three convolutions of the frontal lobe and caudate nucleus (Obersteiner) and passes down through the anterior division of the internal capsule as far as the gray nuclei of the ventral half of the pons in which it terminates. The pons nuclei are also joined by "other fibres from both hemispheres of the cerebellum which enter the pons at its lateral surfaces in the middle peduncles. Thus it is evident that a connection exists between each frontal lobe and both cerebellar hemispheres, the crossed connection being greater than the direct one. In Flechsig's case of deficient cerebellum the pons nuclei and the fibres to them from the frontal lobe were found. In my case of deficient cerebrum the pons nuclei and the fibres to them from the cerebellum were found. Therefore each half of this tract, if it is a continuous tract, may develope independently of the other. Of the function of this tract we know nothing and of the functions of the frontal lobes and cerebellar hemispheres we know very little." †

* Meynert's scheme of the nervous system as described by Obersteiner, in his "CENTRAL NERVOUS ORGANS," page 168.

† THE INTRA-CEREBRAL TRACT. by M. ALLAN STARR, M. D. *Medical Record*, Feb. 13th, 1886.

The pyramidal tract (Diagram XXII, 2) arises from the posterior part of the third frontal convolution, the two central convolutions and from the paracentral lobule. Its fibres converge from the corona radiata to the middle portion of the upper surface of the internal capsule, (Diagram. XXI, knee, 1, 2, 3, 4,—the anterior third of the posterior division), the relative order of the fibres of this great motor tract with their functions has already been fully described in the discussion of the internal capsule. The projection fibres of the "sensory crossway" of Charcot yet remain to be described. (Diagram XXII, 3). They arise from the parietal lobe and from the central convolutions and pursue a course downward through the middle and posterior thirds of the internal capsule, (Diagram XXI, 2, 1,) into the formatio reticularis (the path for touch, temperature and pain) and fillet (the path for muscle sense) of the tegmentum of the crus and thence pursuing the course previously described to their final destination. They mingle with special sense fibres that pass to the optic thalamus (Diagram XXII, auditory 2, visual 1,).

The Commisural Fibre System by means of the corpus callosum and anterior commissure effects a connection between the two hemispheres of the brain, definite areas on the cortex of the one being thus connected with similar regions on the cortex of the other.

"In man at any rate, it looks as if each individual area on the general surface of the great brain was, without exception, united to its corresponding contra-lateral-area." (Obersteiner). The corpus callosum contains the commissural fibres for the frontal, parietal and occipital lobes of both hemispheres, thus bringing every lobe of each hemisphere into complete harmony of action with that of its fellow of the opposite side. The anterior commissure in like manner correlates and harmonizes both temporal lobes and is the medium of communication between the cortex and the olfactory lobes. The function of the commissural fibres is evidently, therefore, to bring into relation the two hemispheres and to render their activities symmetrical. We can test only the commissural fibres joining the motor convolutions of both hemispheres; if there is a failure to

perform corresponding bilateral motions in face, hands or feet, it would be inferred that there was a defect in function of the commissural fibres of these areas.

The Association Fibre System connects and brings into relation with each other the different areas of the same hemisphere. There are two groups of association fibres; the first is composed of short fibres which connect neighboring cortical areas; the second is made up of longer tracts of fibres which unite parts of the cerebral cortex situated some distance from each other. Diagram XXIII shows us the frontal and occipital lobes and the frontal and temporal lobes connected together by the fasc. arcuatus which is an excellent illustration of the second group of association fibres. By carefully studying the above diagram we see that each convolution is joined to the two adjacent convolutions by fibres which pass around the separating fissures, an instance of the first group of association fibres, also that fibres pass from each convolution to the convolution next but one and that hence each convolution has a probable connection with every other. (Starr). These fibres bring into functional connection distant parts of the brain and so provide the mechanism for unity of action. Dr. Starr suggests that their function is the association of concrete memories; if so lesion of the centrum semi-ovale involving the integrity of these parts would give rise to various forms of sensory, or amnesic, aphasia. Motor aphasia however can only be caused by a lesion of the posterior part of the third frontal convolution, (on the left side in right handed and on the right side in left handed persons) or by a lesion of the motor speech tract from Broca's centre to the motor nuclei of the pons and medulla. *

* Pathology of Sensory Aphasia, by M. Allan Starr, M. D. Brain, July, 1889.

CEREBRAL LOCALISATION.

The older physiologists believe with Flourens that "Functionally all parts of the cerebrum were alike and therefore any one part could act vicariously for any other;" the clinical and pathological investigations of Hughlings-Jackson suggesting the existence on each side of the fissure of Rolando of special centres for the movements of the leg, arm and face, which were experimentally demonstrated by Ferrier to have an actual existence in the cortex of the lower animals: also the prior investigations of Fritsch and Hitsig showing that the irritation of certain parts in the brain of animals "caused muscular contraction in well defined portions of the opposite side of the body, thus overthrowing the old idea that the gray cortical substance could not be irritated" have established beyond doubt the fact that differentiation of function exists in different parts of the animal brain. "These results were soon confirmed by numerous observations in cerebral pathology in man and to-day our information about the motor functions of the cerebral cortex forms the best known portion of the doctrine of cerebral localisation. * * * It is true that we are only just beginning to know something about the subject; there are numerous contradictory views asserted and numerous questions unanswered. * * * Much in it will surely be altered in the course of time; but still this doctrine of special localisation of the various cerebral functions marks out in general outline the only foundation upon which we can hope to erect a system of cerebral pathology and diagnosis."*

* Text-book of Medicine, page 672, Strumpell.

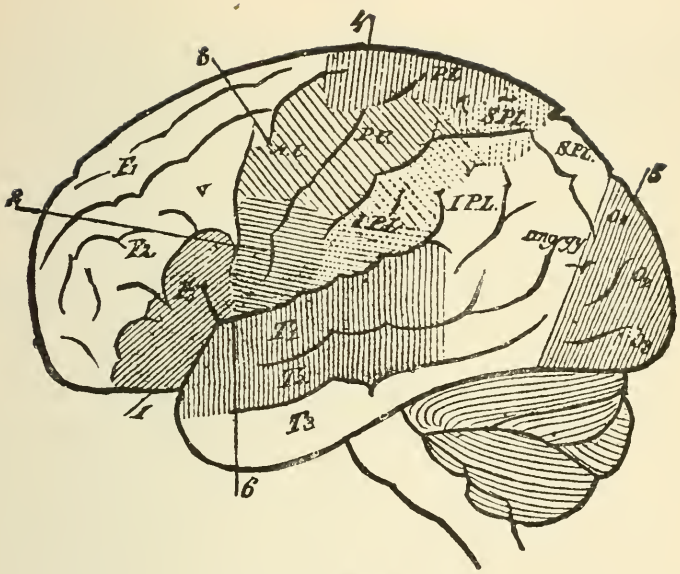


DIAGRAM XXIV.

Convolutions of the Brain (After Ecker, quoted by Starr). F., frontal; T., temporal; O., occipital; ang. gy., angular gyrus; A. C., anterior central; P. C., posterior central; P. L., paracentral lobule; S. P. L., superior parietal lobule; I. P. L., inferior parietal lobule; 1, area of speech, motor; 2, sensori-motor area of face; 3, sensori-motor area of arm; 4, sensori-motor area of leg; 5, visual area; 6, auditory area, area of speech, sensory.

The motor area includes not only the two ascending convolutions, (anterior and posterior central, or ascending frontal and ascending parietal) on either side of the fissure of Rolando but also the paracentral lobule on their medial aspect. Gowers believes that in addition it comprises part at least of the superior parietal lobule and the root of the first or highest frontal convolution (Diagram XXIV. A. C., P. C., S. P. L. and root of F_1). "The centre for the movement of the angles of the mouth lies opposite the fissure between the middle and lower frontal convolutions; the lips and tongue are apparently represented together in the lowest part of the ascending frontal and perhaps in the adjacent root of the third frontal." Gowers. The middle third of both central convolutions presides over the movements of the arm, (Diagram XXIV. 3). The upper

portions of both central convolutions and the paracentral lobule contain the motor centres which govern the movements of the leg and body. (Diagram XXIV. 4); it is not definitely known how far these centres extend backward upon the superior parietal lobule or forward on the ascending frontal, not further perhaps than the upper frontal sulcus. "Individual centres and cortex-fields are not to be considered as sharply outlined and definitely marked off from neighboring regions; the so-called centres are rather the spots of maximal relation to functions which fade away into neighboring areas. Hence it follows that the cortex-fields to a certain extent overlap one another." Obersteiner.

"Extirpation of the marginal convolution (in the monkey) causes paralysis of those movements which remain more or less unaffected after the destruction of the centres on the convex aspect of the hemispheres; namely, movements of the trunk, those of the hip muscles as well as some of those of the leg; in order however that these movements should be entirely paralyzed it is necessary that the marginal convolution should be destroyed in both hemispheres, as it would seem that the trunk movements are so bilaterally coördinated in the marginal convolution that the removal of one only is not sufficient to cause any very marked effect; when both are removed however the most absolute paralysis of the trunk muscles is induced. * * * Besides the movements of the trunk, (marginal gyrus in front of the paracentral lobule, Schäfer and Horsley) there are others which are also bilaterally represented in each cerebral hemisphere. This holds good in respect to the upper facial region as well as those of the larynx. Hence unilateral extirpation of the centres of these movements causes no, or scarcely any, perceptible impairment, and it is necessary that the centres should be destroyed on both sides in order that paralysis should result. It has been shown by Krause in dogs, and by Horsley and Semon * in monkeys, that unilateral extirpation of the laryngeal centres does not appreciably impair the adduction of the vocal cords; whereas phonation becomes volitionally impossible when the centres are destroyed in both hemispheres.

* "They find a motor centre in each cerebral hemisphere situated in the monkey just posterior to the lower end of the preventral sulcus at the base of the third convolution, and in the carnivora in the precrucial and neigh-

It would appear from the researches of Franck and Pitres, Exner, Leraschew, Sherrington that such movements as are not primarily bilaterally represented in each cerebral hemisphere are secondarily associated in accordance with an hypothesis originally advanced by Broadbent, by commissural fibres connecting the bulbar and spinal nuclei with each other. Though moderate stimulation of the cortical centres of the limbs gives rise to movements as a rule on the opposite side, yet it not infrequently happens, if the stimulation be increased, that movements occur in the limbs on both sides. These are, however more pronounced on the opposite than on the same side.

In the monkey as well as in man, it is not unusual to find descending degeneration in both lateral columns as the result of unilateral cortical lesions. According to the recent researches of Sherrington if the cortical lesions affect only the centres for the limbs bilateral degeneration does not occur, to any extent at least; but this is very pronounced if the lesion affect the marginal convolution. The degeneration is confined to the pyramidal tracts on the same side as far as the decussation of the pyramids, but becomes bilateral in the spinal cord. In the case however of the more obviously bilateral laryngeal centre, degeneration is well marked in the pyramids of both sides. These facts as well as clinical observations in man show that even in the case of the limb each hemisphere represents both sides of the body, mainly the opposite but to some extent also the same side. It was first pointed out by Brown-Sequard and his observations have been confirmed by Pitres and Friedlander that lesions which induce hemiplegia of the opposite side also cause some diminution in the energy of the movements of the limbs on the same side. This is a result which we should expect if each hemisphere were in relation with both sides of the body. * * *

boring gyrus * * * that stimulation of this point produces complete bilateral adduction of the vocal cords * * * and finally, that stimulation of the more peripheral parts of the area evokes 'less and less perfect adductions as they proceed from the focus outwards and when the extreme margin of the area is excited, only what is known as the "cadaveric position" is assumed by the vocal cords.'" On the Central Motor Innervation of the Larynx. By Dr. H. Krause, Berlin. The British Medical Journal, Jan. 18th, 1890.

It has now been established beyond all question that cortical lesions of the motor zone in man, if they are such as actually to destroy and not merely to push aside, the gray matter of the respective centres, invariably cause paralysis of volitional motion in the related parts. Such results invariably followed not only destructive lesions of disease but also surgical excisions of the cortical centres. Not only does general hemiplegia result from lesions of the whole of the Rolandic area but limited lesions cause limited paralyses or monoplegia of the face, arm and leg in precise correspondence with the results of experiments on monkeys."*

A cortical lesion may affect one or more contiguous areas of the motor region, rarely all of them. A circumscribed destroying lesion in the motor area of the cortex produces paralysis of one side of the face or of one arm or of one leg, and we designate such a paralysis as monoplegia of the face, arm or leg. In like manner an irritating lesion of the same region, if circumscribed, will give rise to monospasm of the face, arm or lower extremities; hence monoplegia and monospasm are the principal symptoms of disease of the Rolandic area. Knowing the relation that the motor zone bears to the varied movements of the body, we can locate during life with much certainty the exact point in the brain surface where the lesion is situated. It is more common to observe a combined paralysis of two portions of the body than a single one as the result of a cortical lesion; the most usual form being a simultaneous palsy of the arm and face, less frequently of the arm and leg. A glance at digram XXIV. is sufficient to show us that no single lesion could produce a paralysis of the leg and face at the same time, while the arm centre escapes uninjured. "As a matter of fact" says Strumpell "no such combination has ever been observed." Irritating lesions, as stated, may produce monospasm which after the manner of monoplegia above described may affect a single arm or the arm and face, etc., or the tonic and clonic spasms resulting from such a lesion may spread over the entire half of the body, ofttimes the convulsions become general. Such spasmodic seizures are because of their likeness to genuine epilepsy called cortical epilepsy, partial epilepsy or Jackson-

* The Croonian Lectures on Cerebral Localisation. By David Ferrier, M. D. The British Medical Journal, July 12th, 1890.

ian epilepsy. "Numerous cases of disease have taught us that these circumscribed epileptiform attacks occur almost exclusively in affections of the motor cortex. They furnish information as to the precise locality of the lesion; for spasms in the distribution of the facial nerve imply that mainly the lower third of the central convolutions is affected; of the arm, the middle third; and of the leg the upper portions of the same. At the same time the spasm and the paralysis vary greatly in their relations to each other. Often, for example, when there is haemorrhage into the central convolutions, violent, unilateral convulsions come on simultaneously with the paralysis. In the case of tumors and other lesions which develope slowly, partial epileptiform spasms will often appear quite a long while before there are any symptoms of paralysis. Finally, it is not rare for epileptiform attacks to occur repeatedly in regions that are already paralytic. Either of the occurrences described in the two preceding sentences are particularly strong evidence that the cortex cerebri is diseased. Besides the pronounced epileptic attacks, disease of the motor region of the cortex may give rise to less violent symptoms of motor irritation, like occasional twitching, rythmical twitching and tonic contraction." *

The success achieved by cerebral surgery has only become possible through the known facts of cerebral localisation as already detailed, while it in turn has served to verify in man the experimental data derived from the lower animals. Dr. Lucas-Championniere, of Paris, has recently called attention to one especial feature of this subject. A patient who had suffered from a spontaneous haemorrhage was left "with paresis of the right lower limb, marked contraction of the right hand and epileptiform attacks, which last as time went on increased more in frequency and intensity." Dr. Lucas-Championniere located the haemorrhagic clot in the ascending frontal convolution and trephined with the view "of liberating the compressed and irritated cerebral structure." An encysted clot was found "the cyst freely opened and the cavity washed out with antiseptic fluid." The patient made a good recovery and walked much better than before the operation; the contraction of the right arm passed away twenty-four hours after the removal of the hae-

* Text-book of Medicine, Strumpell, page 675.

morrhagic deposit. Two months later he suffered from an epileptic attack, no more however occurring up to the report of the case to the Academie de Medicine four months afterward.*

" The limits of the region receiving impulses which awaken the perception of touch, temperature and pain are not fully determined. It is known however that the motor and sensory regions coincide,— the sensori-motor area. While it is probable that the sensory region extends beyond the motor and includes the parietal lobules, which lie posterior to the motor area, (diagram XXIV. I. P. L.,S. P. L,) lesions affecting the posterior central convolution give rise to combined motor and sensory symptoms, the sensory areas lying in the same order as the motor areas, face, arm, and leg in the lower, middle and upper thirds respectively. Lesions in the motor area anterior to the fissure of Rolando usually produce paralysis without anaesthesia. Lesions in the parietal lobules may produce anaesthesia but do not cause paralysis. Each sensory area is in functional relation with the opposite limb to a much greater degree than with the limb of the same side. Mono-anaesthesia may therefore occur from cortical lesions, the loss of sensation is rarely total, as it is probable that the decussation of sensory impulses is rarely complete.

The degree of impairment of sensation is only to be ascertained by comparison of the affected limb with the other three. If the sensory area is not destroyed but is only irritated, subjective sensations in the limb whose area is affected occur and such mono-paraesthesiæ are valuable indications of cortical lesions when disease in other parts is excluded. Monospasm and monoplegia, mono-paraesthesia or mono-anaesthesia are therefore the chief symptoms of cortical disease in the sensori-motor area. The two former indicate an affection lying anterior to the parietal lobules. The two latter may occur when these also are involved."†

* British Medical Journal, May 17, 1890.

† Reference Handbook of Medical Sciences, Diagnosis of Local Lesions in the Brain. M. Allen Starr, M. D.

Ferrier does not think that the sensory centres fuse with the motor in the cerebral cortex, but that on the contrary they are located in the falciform * lobe.

"It is not unlikely that, besides representing sensibility of the opposite side of the body generally, certain parts of the falciform lobe may represent more particularly the sensibility of special regions, but though I occasionally notice in my experiments on the hypocampal region.—like Horseley and Schäfer in their experiments in the gyrus fornicatus,—that it seemed as if one region had been more affected than another, yet this was at times not apparent, and in general the anaesthesia has affected the whole of the opposite side, arm, leg and trunk. Evidence is therefore so far not conclusive of the existence of any altogether specialized centres in this general area. It is probable however that a certain degree of localisation may be established through the associating fibres which undoubtedly connect this region with the motor centres of the cortex. It has not yet been found possible to produce total persistent loss of all forms of tactile and common sensation on the opposite side after destructive lesions of the falciform lobe but this may be due to the fact that this lobe has never been absolutely destroyed throughout its whole extent. It is probable however that common sensibility may, to some extent at least, be bilaterally represented, so that a certain amount of compensation may be affected by the falciform lobe of the other hemisphere. The paths of connection between the falciform lobe and the sensory division of the internal capsule have not yet been traced by any anatomist, but in view of the proofs which have been above given of the relation between the falciform lobe and tactile and common sensibility, it is obvious that the hypothesis of Fleschsig as to the distribution of the sensory tract in the parietal lobule requires amendment.

No scheme of the cortical distribution of the sensory tracts can be admitted as correct which does not connect them with the cortex of the callosal and hypocampal convolutions. * * * From various considerations above advanced I conclude that the motor centres of

* "Broca describes the gyrus fornicatus,—and its prolongation the anterior part of the uncinate gyrus (Quain)—with the addition of the olfactory tract as a special lobe, lobus limbicus. Similarly Schwalbe on genetic grounds, institutes his lobus falciformus, which comprises the gyrus fornicatus, septum pellucidum, and fascia dentata." Obersteiner.

the cortex are not the centres of tactile or general sensibility, nor are they the centres of the muscular sense, whether we regard this to depend upon centripetal impressions, conscious or unconscious, or on a sense of innervation, but that they are motor in precisely the same sense as other motor centres; and, though functionally and organically connected, are anatomically differentiated from the centres of sensation general, as well as special." *

If a lesion involve the posterior part of the third frontal convolution on the left side in right handed, and on the right side in left handed persons, motor or ataxic aphasia will result. (diagram XXIV. 1) "In this area are located the memories of the combination of motor acts necessary to the pronunciation of words, memories which have been acquired by practice. If these memories are blotted out the ability to initiate the impulse required to produce a given sound is lost and speechlessness is the result. When this convolution alone is affected the patient can understand what is said to him and may be able to write but cannot talk." *

According to Gowers, Landouzy and Grasset have located the centre for the movement of the upper eyelid in the inferior parietal lobule.

The cortical centre of the sense of hearing is situated in the first and second temporal convolutions (diagram XXIV. 6, T_1, T_2). "In favor of this localisation we have, apart from the results of experimental investigations, the appearances presented in cases of word deafness, in which disease lesions of this region especially on the left side are almost always found. In the brain of deafmutes a very perceptible atrophy of the upper temporal convolution may be present, although the peripheral stem of the auditory nerve is intact." Obersteiner. A lesion of this region causes loss of hearing in the opposite ear which is not lasting however because compensation is possible by the corresponding centre of the opposite side.

"Each auditory nerve must be structurally connected with both hemispheres although only the connection with the opposite hemisphere is habitually in functional action." Gowers. Destruction of these convolutions causes deafness while the irritation of them gives rise to auditory hallucinations.

* The Croonian Lectures on Cerebral Localisation. By David Ferrier, M. D. The Britith Medical Journal, July 5th and 12th, 1890.

The cortical visual centres are located by Ferrier and Yeo in the the occipital lobe and the angular gyrus. "I contend," says Dr. Ferrier, "that the only hypothesis which seems to harmonise with all the facts is that the angular gyri are more particularly the centres for clear vision; each mainly for the eye of the opposite side. * * *

The probability is that any apparent relation between lesions of the cuneus and the occurrence of hemiopia is due to the special proclivity of this region to affections by morbid vascular conditions and to coincident implication of the optic radiations of the occipito-temporal regions." *

Seguin and Nothnagel believe that the optic radiations centre chiefly in the cuneus,—the triangular lobe which lies between the parieto-occipital and calcarine fissures. Exner regards the gyrus occipitalis primus as "The most concentrated and active portion of the cortical field of vision." Wilbrand regards the apex of the occipital lobe as the principal visual centre. "We do not know" says Gowers "the exact position of the visual centres in the occipital lobe. Hemianopia has resulted from disease of the apex of the lobe, the outer surface and the medial surface, but in some cases of partial lesion the white fibres of the optic tract may have been involved. Complete hemianopia has been most frequently produced by disease of the apex of the lobe and especially of the cuneus."

"Despite" says Obersteiner "the diversity of opinion, one will probably not go far wrong in placing the cortical visual area in the visual lobes, and of these lobes the most probable seat of vision is the CUNEUS, at the same time the fact must not be lost sight of that each visual centre is connected in a partially crossed manner with both eyes. * * * Whether or not the motor centres for the extrinsic eye-muscles lie in this region, extending somewhat over to the neighboring parts of the parietal lobe must be left undecided."

* "The occipito-angular region (that is the occipital lobe and angular gyrus) is the visual area of the cortex. Complete destruction of this area in the one hemisphere causes permanent hemianopia to the opposite side by paralysis of the corresponding halves of both retinae; while bilateral destruction causes complete and enduring blindness of both eyes. Apart from the loss of vision there are no other motor or sensory defects. The sensibility of the eyeball is intact, and the ocular movements are absolutely unimpaired. There is no impairment of the sensibility or motor power of the limbs. The other special senses are unaffected. If the destruction of the occipito-angular region is incomplete, unilaterally or bilaterally, the resulting hemianopia in the one case is not enduring, nor is the blindness permanent in the other." The Croonian Lectures on Cerebral Localisation. By David Ferrier, M. D. The British Medical Journal, June 14, 1890.

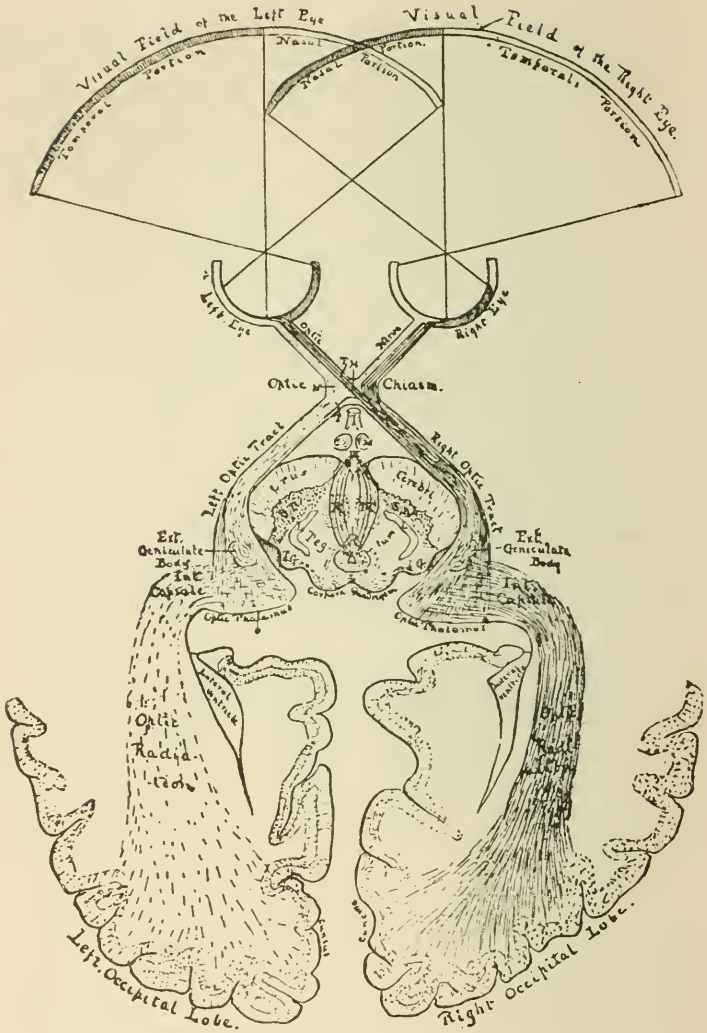


DIAGRAM XXV.

Diagram to Show the Course of the Visual Impulses from the Eyes to the Brain. I. I., infundibulum; C. M., corpora mammilaria, 1 1 1, exit of the third nerve; S. N., substantia nigra of crus; L., lemniscus of tegmentum; I. G., internal geniculate body; R. N., red

nucleus of tegmentum; N., lesion in chiasm causing unilateral nasal hemianopsia; T., lesion in chiasm causing unilateral temporal hemianopsia; H., lesion in chiasm causing bilateral temporal hemianopsia. The shading indicates the fibres, lesions of which at any place between the optic chiasm and the right occipital lobe will produce bilateral homonymous left hemianopsia. The portions of the visual field affected by such lesions are also shaded. The eyes are supposed to be directed in parallel lines and not to converge to a point. Quoted from Starr.

Each optic nerve unites with its fellow of the opposite side in front of the tuber cinerum and forms the optic chiasm. (Diagram XXV.)

According to Obersteiner the number of fibres in each nerve averages 438,000. The chief bulk of the chiasm is composed of three kinds of fibres. "1st, fibres from the lateral halves of the retina which occupy the lateral borders of the chiasm and go to the optic tract of the same side. 2d, fibres from the mesial side of the retina, which cross in the chiasm to the tract of the opposite side. 3d, fibres which occupy the back of the commissure and extend from one tract to the other—Gudden's commissure."

In the horse and the rabbit the decussation of the fibres of the optic nerve is absolute, no part of the visual field is common to both eyes, while in dogs, cats, monkeys and man the decussation is partial, the eyes to a greater or less degree being directed in parallel lines. A larger percentage of the fibres of the optic nerve decussate than pass to the occipital lobe of the same side. Starr.

"Each optic tract after leaving the chiasm passes around the crus cerebri, lying directly upon the fibres which pass through the foot of the crus (pes pedunculi), and ends on the level of the tegmentum of the crus in the external geniculate body, in the pulvinar of the optic thalamus, i. e. the eminence forming its posterior surface, and in the corpora quadrigemina anteriora, which latter it reaches by the brachium conjunctivum. The last named fibres of the optic tract have probably nothing to do with conscious vision, and may therefore be excluded from consideration in studying hemianopsia.

They form the sensory part of a reflex arc, whose motor part is made up of the motor nerves to the eyeballs. The functions of this reflex mechanism are to direct the motions of the eyeballs, and to regulate the process of accommodation and the size of the pupil. The primary visual centres are therefore the external geniculate body and optic thalamus. The fibres of the optic tract end in the cells of these ganglia, and from these cells new fibres arise which collect in a large tract and issue from the posterior external angle of the optic thalamus into the posterior third of the internal capsule. The visual tract turns upward and backward in the internal capsule, radiates into the centrum semi-ovale of the occipital lobe, and passing around the outer border of the posterior horn of the lateral ventricle, (see above diagram) terminates in the convolutions of the occipital cortex including the cuneus. At no point in this course is any decussation found. The only decussation of fibres between the eye and the cortex is in the optic chiasm." *

By reference to the proceeding diagram it will be seen that each occipital lobe is in direct anatomical connection through the optic tract, chiasm and nerves with the like named half of each retina. Owing to the refractive power of the lense objects on either half of the visual field are thrown upon the opposite half of the retina.

Loss of sight restricted to one-half of the visual field is called hemianopsia. When this condition is present in the outer half of the field of vision it is termed temporal hemianopsia, when present in the inner half, nasal hemianopsia. If the like named halves of both eyes are affected the condition resulting is that of homonymous or lateral hemianopsia, if the unlike named halves of both eyes are involved heteronymous hemianopsia occurs.

Ferrier regards "The partial, quadrant or sector-like defects in the upper or lower halves of the visual field as merely a form of incomplete hemianopsia. * * * The pathology of these sector-like defects is a matter of conjecture, they have not been conclusively brought into relation with lesions of any particular portion of the cortex, and the probality is that they are dependent rather upon partial lesions of the optic radiation than of the cortical centres

* Reference Handbook of the Medical Sciences. Hemianopsia. M. Allen Starr, M. D.

themselves." As opposed to Ferrier's views Munk, Schäfer and Gowers believe that the upper, lower, outer and inner portions of the retina are especially projected upon the corresponding portions of the occipital lobe. A lesion sometimes causes crossed amblyopia (a dimness or loss of vision in the opposite eye,) not hemianopsia; Gowers accounts for this by supposing that "on the outer surface in front of the occipital lobe there is a higher visual centre in which the half fields are combined, and the whole opposite field is represented." Ferrier has found by experiments on animals that lesions of the angular gyrus (ang. gy., diagram XXIV.) produce a similar condition. Pathological evidence in man seems to confirm the hypothesis of Gowers and at the same time establishes identity of function between the angular gyri of men and the lower animals.

A lesion may involve the optic tract simply or the cortical centre in which it terminates. "A distinguishing test between tract and central hemiopia has been suggested by Wilbrand and advocated by Wernicke and Seguin which consists in determining whether a pencil of light thrown upon the blind side of the retina induces contraction of the pupil or not. As the optic tract is the path of the fibres which excite pupillary contraction through the oculo-motor centres as well as those which excite visual sensations in the cortex lesions of the optic tract will cause not only hemiopia but also paralysis of the reflex reaction of the pupils to light; whereas lesion of the cortical centres will cause hemiopia, but leave intact the pupillary reaction." *

A lesion on the outer side of the optic chiasm (diagram XXV. N) will give rise to unilateral nasal hemianopsia while if a lesion be situated at T in above diagram either before or after the decussation of the optic nerve in the chiasm unilateral temporal hemianopsia will occur.

Bilateral hemianopsia is more frequently seen than the unilateral variety. Should a lesion destroy the direct optic tract on both sides bilateral nasal hemianopsia will be produced. A lesion destroying the decussating fibres, passing through the inner portion of the optic chiasm, will cause bilateral temporal hemianopsia (dia-

* Croonian Lectures on Cerebral Localisation. By David Ferrier, M. D. The British Medical Journal, June 21, 1890.

gram XXV, optic chiasm.) Tumors, syphilitic and tubercular growths, tabes etc., are the most common lesions of the chiasm. Bilateral homonymous hemianopsia will occur when a destructive lesion is situated at any point in the optic tract itself or in the cortical visual centres in which this tract terminates. Should the tract be invaded as it courses around the crus, not only will hemianopsia occur but there will most probably be involvement of the motor tract and the third nerve as well, (diagram XXV.); thus homonymous hemianopsia, hemiplegia of the opposite side and oculo-motor paralysis of the same side will of necessity occur. Or if a lesion invade the external geniculate body and optic thalamus, and be at all extensive the sensory paths in the tegmentum will be destroyed and hemianaesthesia, hemiataxia and hemianopsia will occur. A lesion involving the basal ganglia or the optic tract in its passage through the posterior third of the posterior division of the internal capsule will generally include some, and, if very extensive, all of the sensory and motor tracts of the capsule, thus producing hemianopsia, hemiplegia and hemianaesthesia of the opposite side. " If it is the left hemisphere, however, in which the disease is present a condition of word blindness is not infrequently associated with it; and in all the cases of word blindness hitherto reported, hemianopsia has been found; this is probably due to the destruction of the association fibres between the occipital and temporal lobes which lie side by side with the visual tract."* Starr.

* Consult Reference Handbook of the Medical Sciences, Hemianopsia. By M. Allen Starr, M. D.

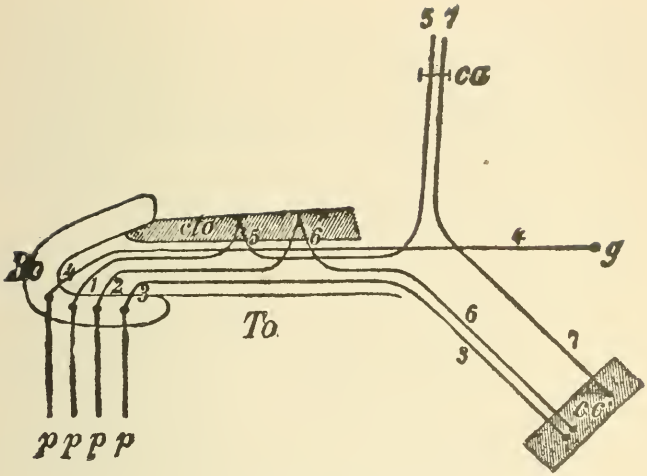


DIAGRAM XXVI.

Scheme of the central apparatus of smell.—Bo, bulbus olfactorius; To, tractus olfactorius; p, Schneiderian membrane; ecto-cortex of the olfactory tract; cc, cortex cerebri; g, central brain ganglia; ca, commissura anterior; 5, olfactory portion of the anterior commissure; 7, hemispheral (temporal) portion of the same. After Obersteiner.

The anatomical relation of the central apparatus of the sense of smell furnishes more reliable data as to its cortical centre than that derived from physiological experiment upon the lower animals.

In man and the monkey the olfactory organs are but imperfectly developed. Broca divides the mammalia into two classes, the osmatic, or those animals in which the sense of smell is well developed, and the anosmatic, or those in which it is poorly developed or entirely absent. "The peripheral nerves of smell originate in the pigmented regio-olfactoria of the Schneiderian membrane; they are non-medullated and pass through the perforations of the cribiform plates into the interior of the skull-case where they attach themselves to a greyish-yellow rounded body of small size in Man, the Bulbus Olfactorius, (Diagram XXVI. p p p p & Bo,) * * * The olfactory bulb lies on the orbital surface of the frontal lobe at the front of the sulcus olfactorius. It is free on all sides with the exception of its attachments to the olfactory nerve, and a strong

stalk or peduncle, wrongly called the olfactory nerve, which runs backwards to join with the rest of the brain,—the tractus olfactorius, (Diagram XXVI. To.) * * * The olfactory nerves (p p p p) find their first interruption in the olfactory bulb, (Bo), the bulb is comparable to the nuclei of origin of most other nerves, or in some sense to the ganglion cell-layer of the retina, perhaps also the spinal ganglia; but in no sense to the cortex." *

The olfactory tract (tractus olfactorius) is in shape triangular; the basal surface of this triangle is composed of medullated nerves, next to this is a layer mostly of connective tissue, while the rest of the tract (the apex) consists of nerve matter derived from a modified cortex, (Diagram XXVI. cot.) The anterior commissure (ca, Diagram XXVI.) simply provides for those portions of the cerebral cortex which are not connected with each other by the corpus callosum; the function of both these commissures being "to unite together identical points on the two hemispheres." The anterior commissure thus unites together the temporal perhaps occipital, lobes and the cortex of the olfactory tract. It is divided into the olfactory and temporal portions; the former connects the olfactory tracts and bulbs with each other, the latter unites the hippocampal lobules. Ferrier.

Ganser believes that the anterior commissure contains no decussating fibres of the olfactory tracts, only commissural. "If," says Ferrier, "the olfactory tract is related to the opposite hemisphere the path is not through the anterior commissure." Luciani thinks that each cortical olfactory centre is in relation with both nostrils but especially that of the same side. "It is however difficult with our present knowledge to trace anatomical connection between the nostril and the opposite side of the brain. * * * The matter is therefore one which requires further investigation." Ferrier. Clinical experience however confirms the opinion of Luciani, "since there are cases in which organic disease of one hemisphere has caused loss of the sense of smell in addition to that of the other special senses on the opposite side." Gowers. Obersteiner in his scheme of the central olfactory apparatus (Diagram XXVI. To.) distinguishes four kinds of fibres.

* Central Nervous Organs, pgs. 265-272. Obersteiner.

"(1) those from the bulb to the cortex of the tract, (Diagram XXVI. 1, 2.)

(2) Those from the bulb which run in the tract, without coming into connection with its cortex, backwards towards other portions of the cortex (3) or through non-cortical ganglia (4).

(3) Fibres arising from the cortex of the tract, and extending via anterior commissure to the cortex of the opposite side (5).

(4) Fibres from the cortex of the tract which run to other parts of the cortex or elsewhere in the brain (6). It can not be stated whether the very strong root of the olfactorius which passes to the nucleus amygdaleus and cornu Ammonis consists of fibres of class 2 or class 3. * * * If it is asked—To what portions of the cortex of the great brain do the olfactory nerves stand in direct relation?—The cortex of the olfactory tract itself should be first mentioned. To this can be added, in all probability, the nucleus amygdaleus, the anterior end of the cortex of the gyrus hippocampi as well as perhaps the frontal end of the gyrus cinguli." *

"A consideration of the almost exclusive part played by sensations of smell in the daily life of most carniverous animals would prepare us to expect that a very large proportion of the cortex of their brains must be devoted to their reception. If the brains of a large number of different carnivores, such as is exhibited in the fine collection in the Hunterian museum, are compared together it is obvious without need for measurement that the temporal lobe is very much larger in the dog, wolf, jackal and other animals which track their prey with the nose, than in other mammals.

Felines detect the proximity of their victims in the forests and jungles rather by listening for broken twigs and crackling leaves and sticks than by sniffing along their trails; in carnivores of this habit, which might be distinguished as "springing" animals, the temporal lobe projects forwards to a less extent than it does in the running hunters. A comparison however of the aquatic otter with its terrestrial congeners is most instructive. The otter trusts to its sensitive whiskers for guidance amongst the snags and stones in the pools of brown water which the salmon frequent. Its sense of smell is extremely deficient, and corresponding with this its tem-

* Central Nervous Organs, page 275. Obersteiner.

poral lobe is reduced to very small proportions. Herbivorous animals rely upon their sense of sight for safety. As far as possible they feed in open ground, keeping watchful guard. Doubtless they quickly discover any taint in the air when their enemies depart so far from their usual practice as to hunt from the windward, but the use which they make of the nose to escape the enemy is not comparable in intensity or specialisation to the following of a trail which crosses and recrosses countless other lines of scent. On the other hand, they make selection of favorite herbs and avoid poisonous ones with the aid of smell so that the difference between the two classes of vegetable and animal feeders is one of degrees rather than of kind, whilst carnivores are "osmatic," par excellence herbivores cannot be justifiably termed "anosmatic." * * * A study of the comparative anatomy of the brain throws much light upon questions of cortical localization, and will probably be the ultimate tribunal to which all experimental evidence will be submitted."*

The localization of the cortical centres for taste, according to Gowers, is unknown. "As to the sense of taste," says Dr. Ferrier, "I have not succeeded in differentiating any special region related to this faculty, but that it is in close relation with the olfactory centre is probable from the facts described. It was noted in connection with electrical irritation of the lower extremities of the temporo-sphenoidal convolutions in the monkey, and of the same region in the brain of the cat, that movements of the lips, tongue, cheek-pouches and jaws were occasionally induced phenomena which might be regarded as indications of the excitation of gustatory sensation. This interpretation received support from the above described results of destructive lesions; and we have therefore reasonable grounds for concluding that the gustatory centres are situated at the lower extremity of the temporo-sphenoidal lobes in close relation with those of smell. This would enable us to explain the occasional occurrence in man of anosmia and ageusia as the result of severe blows on the head, especially the vertex. A blow in this region causes counter-stroke of the base of the brain, particularly in the region of the olfactory centres. No doubt many of the cases of

*Translator's note, Obersteiner's Central Nervous Organs, pages 276-277.

so-called loss of taste and smell are merely cases of loss of smell only, the impairment of taste extending only to the perception of flavors, which as is well known, are compounds of taste and smell together. But there are cases in which both smell and taste proper are impaired or abolished by cranial injuries, and it is permissible to suppose that this may be caused by concussion and contusion of the lower extremities of the temporo-sphenoidal lobes, where the olfactory centres certainly and the gustatory centres in all probability are situated." *

There is still much uncertainty as to the function of "that region of the brain which lies in advance of the Rolandic area and is marked off by the precentral sulcus." Attention has already been called to the frontal pontine tract which arises from the three convolutions of the frontal lobe and, according to Obersteiner, from the nucleus caudatus, (Diagram XXII. 111) and passes through the anterior division of the internal capsule to the pons nuclei and thence to the cerebellum; as previously stated its function is unknown. Electrical irritation of the frontal centres both prefrontal and postfrontal "causes opening of the eyes, dilatation of the pupils and conjugate deviation of the head and eyes to the opposite side."

Complete destruction of the frontal region produces "not only conjugate deviation of the head and eyes but also a temporary paralysis of those movements which are excited by electrical stimulation, namely elevation of the eyelids and dilatation of the pupils." Ferrier.

"There is a quite generally accepted view that the cortex of the frontal portion of the brain is to be regarded as 'the seat of the higher psychical functions.' Some few cases are on record where extensive bilateral lesions of these parts had for their only symptoms mental disturbances. In general paralysis also, and in other forms of dementia it is very probable that the atrophy is greatest in the anterior part of the cerebrum. Nevertheless, we cannot

* Functions of the Brain. Ferrier, page 321.

emphasize too much the fact that we have no certain knowledge about the minute relations of the psychical functions to the different sections of the brain."* Striimpell.

* It is interesting to note in this connection that in the case quoted by Dr. J. H. McBride (The Journal of Nervous and Mental Disease, Aug., 1890) involvement of this region produced "dilatation of the pupils and a staring expression that was always very noticable." Dr. McBride also states that the memory of the patient began to fail about a year from the beginning of the trouble, and during the last months of his life there was great mental impairment.

REVIEW

—OF—

FLECHSIG'S PLAN OF THE HUMAN BRAIN.

By M. ALLEN STARR, M. D.

Plandes Menschlichen Gehirns. Auf Grund eigener Untersuchungen entworfen von Dr. Paul Flechsig. Leipzig, July, 1883. —A Map of the Human Brain.

With the object of presenting in an accessible form the result of his recent researches in the anatomy of the brain, which have been published from time to time in various journals, Flechsig has issued this pamphlet, containing a concise and complete statement of his discoveries, and a diagram * of the principal nerve tracts. Inasmuch as it is now conceded that the method of Flechsig surpasses all others in the accuracy of its results, his statement is worthy of attention. The conclusions presented by him are based partly upon facts gathered from the study of the development of the nerve-tracts in the embryo, and partly upon those afforded by the investigation of secondary degeneration after limited lesions, and partly upon the results of the examination of a brain in which the cerebellum was entirely absent.

* Diagram XXII. page 43. This diagram should be carefully consulted in the study of this review.

They are as follows:

I. There exist direct nerve-tracts between the cortex of the cerebrum and the motor and sensory nerves.

1. The direct motor tract arises from the cortex of the anterior and posterior central convolutions, of the paracentral lobule, and of the parietal lobe as far back as the præcuneus. Its anterior limit of origin is the sulcus præcentralis. Its posterior limit is not fully determined. The greater part of this direct motor tract is formed by the "pyramidal tract," which joins the cortex with the motor nerves of the body and which forms the pyramid of the medulla. Lying anterior to this tract in the centrum ovale and internal capsule, and internal to it in the crus cerebri, is the smaller part of the direct motor tract which connects the lower division of the anterior central convolution with the nuclei of the facial and hypoglossal nerves in the medulla. As this smaller part does not reach the pyramid of the medulla, it is not included in the "pyramidal tract." This direct motor tract has no connection with the basal ganglia nor with the gray matter of the pons. In the internal capsule it occupies the middle third; in the crus, the median portion of the outer half; in the medulla each tract divides into a large crossed pyramidal tract which decussates in the lower decussation, and lies in the posterior part of the lateral column of the cord, and a small direct pyramidal tract which lies in the anterior median column of the cord. In the cord the individual fibres leave the tracts at various levels and pass into the gray anterior horn of the side on which they lie, there entering the large multipolar ganglion cells. The majority of the pyramidal fibres end in the two enlargements (cervical and lumbar) and thus are in physiological relation with the nerves of the extremities.

2. The direct sensory tracts are less accurately determined.

The *olfactory* tract, * on leaving the olfactory bulb, divides into

a. A branch to the base of the frontal lobe, to the gyrus fornicatus (?);† the inner root.

b. A branch to the cortex of the sphenoidal lobe, to the gyrus uncinatus; the outer root.

c. A branch which passes backward through the lamina perforata anterior (to the caudate nucleus) (?); the middle root.

The *optic* tract, made up of fibres from both optic nerves, after leaving the chiasm and passing around the crus, sends (*a*) the larger number of its fibres into the corpus geniculatum externum; from which fibres in turn radiate outward through the posterior part of the internal capsule to pass to the occipital lobe. (*b*) ‡ The smaller number of its fibres pass to the anterior pair of the corpora quadrigemina. These fibres join the cells of the gray surface layer (Rindenschicht), and then turn back and pass over the corpus geniculatum externum to join the radiating fibres to the occipital lobe. The termination of these radiating optic fibres in the cortex is in the occipital lobe and especially in the cuneus, but with them are associated many fibres from the optic thalamus.

* "Owing to the original formation of the olfactory bulb and tract as a diverticulum from the anterior cerebral vesicle, the cavity of which has become almost obliterated, the remnants of its original connection with the mesial, external, upper, and lower aspects of the cerebral hemisphere are still spoken of as roots of the olfactory tract. Though in man and the monkey all the said roots, except the external one, are practically obliterated, yet in other animals, in whom the sense of smell is largely developed, four roots are usually described, namely, an external root passing to the hippocampal lobule; a superior and middle root, connecting the tract respectively with the grey matter of the base of the frontal lobe and the trigonum olfactorium, or gray matter of the anterior perforated space; and an inner root, which appears to fuse with the anterior extremity of the callosal gyrus. The connection of the olfactory tract, by means of its inner and outer root, with the anterior and posterior extremities of the falciform lobe, has been compared by Broca to a tennis racquet, of which the circumference is formed by the falciform lobe and the handle by the olfactory tract and bulb." Ferrier.

† The question marks are inserted by Flechsig to indicate that the statement is hypothetical.

‡ "The smaller number of fibres (b) of the optic tract pass to the corpora quadrigemina (anterior), cross over and become the sensory root of the reflex arc of the third nerve for accomodation and pupil reflex as well as direct the motions of the eye. They have nothing to do with conscious vision." Starr.

The *auditory* tract* and the *gustatory* tract have not yet been followed.

The tract conveying sensations from the skin is not fully traced, and it is questionable whether it is a direct tract. Clinical facts indicate the following course as the most probable in the brain.

The tract conveying general sensations from one-half of the body passes through the tegmentum of the pons and crura of the opposite side (Grosshirnschenkelhaube) into the posterior part of the internal capsule, which lies between the pyramidal tracts and the corpus geniculatum externum; thence radiating, its fibres pass to the cortex of the cerebrum lying between the sulcus præcentralis and the occipital lobe, *i. e.*, to that part of the brain which is situated beneath the parietal bone.

II. There exists a system of nerve tracts in connection with the optic thalamus.

1. From the external surface of the optic thalamus nerve fibres pass outward in all directions, forming the corona radiata† and joining the thalamus with all parts of the cortex. The cortex of the frontal lobe is joined with the thalamus by means of fibres which pass through the anterior part of the internal capsule, and go to the anterior outer nucleus of the thalamus, and to the stratum zonale. The parietal cortex in its entire extent is joined with the thalamus by fibres which pass to the outer and inner nuclei and to the stratum zonale. The cortex of the temporo-occipital lobe sends a large bundle of fibres to the pulvinar (the outer posterior third) of the thalamus, and to the stratum zonale. The cortex of the Sylvian region is joined with the outer and inner nuclei of the thalamus and with its stratum zonale. The cortex of the hippocampal region is joined to the anterior outer nucleus of the thalamus by means of the fornix, which passing from the hippocampus, along the roof of the lateral ventricle, and then downward to the corpus mamillare, there turns upon itself and ascends to enter the lower surface of the thalamus. The lenticular nucleus sends fibres through the internal capsule to the basal portion of the thalamus, but whether these end there or pass on to the tegmentum of the crus, is undecided.

*Refer to Spitzka's description of the auditory tract, page 19, and see diagram XXII. 9.

†The corona radiata is formed by the convergence of all the fibers from the cerebral cortex to the internal capsule.

2. A second series of tract connects the thalamus with the crus cerebri and its tegmentum.

(1.) Vicq d'Azyr's bundle of fibres passes downward from the anterior outer nucleus of the thalamus to the corpus mamillare, there turns upon itself and some of its fibres go between the red nucleus of the tegmentum and the substantia nigra of the crus into the formatio reticularis of the pons, while the remainder join the fornix (as already described).

(2.) Meynert's bundle is made up of fibres coming from the stratum zonale of the thalamus, from the ganglion habenulæ, and from the gray matter lining the third ventricle, which unite and pass through or near the red nucleus, are then connected with the ganglion interpedunculare of Gudden* (?), and go downward toward the formatio reticularis of the pons, though they cannot be traced into it.

(3.) From the laminæ medullares† of the thalamus, especially from the middle third of the lamina medullaris externa, bundles of fibres pass down to the red nucleus of the tegmentum. It is questionable whether these come from the gray matter of the thalamus, or from the internal capsule; and whether they end in the red nucleus or pass on to the cerebellum by its superior peduncle. The latter hypotheses have much in their favor. The assertion of Meynert that they pass into the formatio reticularis of the pons is denied. The assertion of Wernicke that they form a part of the lemniscus is also denied. Their connection with the cerebellum is deemed probable, as they were absent in the brain in which the cerebellum was wanting.

(4.) It is uncertain whether the fibres from the optic tract which enter the thalamus come from the optic nerve or from the commissura inferior of Gudden.‡ It is possible that they do not end in the thalamus, but pass through it to the corpora quadrigemina.

III. There exists a system of nerve fibres in connection with the gray nuclei of the ventral half of the pons Varolii.

*The ganglion interpedunculare of Gudden is a small mass of gray matter where the crusta meet.

†The laminæ medullares are the two strips of white fibers through the gray matter of the optic thalamus.

‡Contrast this view of Flechsig's with the description of termination of the fibres of the optic tract given by Dr. Starr, pages 59 and 60.

1. A tract from the cortex of the frontal lobe* forms the anterior division of the internal capsule and passes downward through the inner two-fifths of the crus cerebri to the gray nuclei in the antero-median substance of the pons. From these nuclei new fibres arise which pass laterally through the middle peduncle of the cerebellum to go to the lateral and posterior portions of the hemispheres of the cerebellum. All these fibres develop their medullary sheaths at the same time, and all of them were absent in the brain which lacked the cerebellum. It is, therefore, probable that by means of these fibres the cerebrum and cerebellum are brought into functional relation—the nuclei of the pons being interpolated in their course.

2. A tract from the postero-median gray nuclei of the pons ascends through the external fifth of the crus cerebri, forms the basal part of the internal capsules, passes along the base of the lenticular nucleus, and into the corona radiata of the temporo-occipital lobes. In the occipital lobe its fibres turn backward and are lost. In the temporal lobe they pass forward and can be traced to its apex. The nuclei of the pons from which this tract arises are joined, by deep lying fibres in the middle peduncle, with the superior part of the cerebellum lying near the median line. The fact that in congenital absence of the cerebellum these tracts were wanting, leads to the supposition that they, too, are connecting tracts between the cerebrum and cerebellum. This tract is not to be regarded as an auditory tract because it does not obtain its medullary sheath until three or four months after birth, long after the time at which an infant can first hear. The same reason prevents the supposition that it conveys sensation from the skin, although it is in this tract that Meynert and Gratiolet located the tract of general sensation. Clinical cases give no facts to determine the function of these tracts.

3. A tract connects the pons nuclei with the corpora striata. From the nucleus caudatus fibres pass into the laminæ medullares of the nucleus lenticularis, where they are joined by fibres from the outer body of this nucleus, and thence the two sets in one bundle pass downward into the crus and enter the substantia nigra of

*By reference to diagram XXII. l. l. l., this tract from the frontal lobe to the cerebellum can be easily traced, see the frontal pontine tract, page 45.

Sommering, forming the stratum intermedium of Meynert.* Here a part of them are lost. The remainder pass onward, lying dorsad of the deepest transverse fibres of the pons, join the lemniscus in the middle of the pons and probably end in the nuclei of the pons (?) from which in turn new fibres pass to the cerebellum. This tract degenerates downward after lesions in the crus. Its function is unknown.

IV. There exist a number of important tracts in the tegmentum of the crura cerebri beneath the corpora quadrigemina.

1. The largest of these is the superior peduncle of the cerebellum (Bindearm). This tract arises chiefly from the nucleus dentatus of the cerebellum, which is in turn connected with the cortex of the cerebellum, and with the opposite olivary body by way of the corpus restiforme of the medulla. It arises partly from the cortex of the superior and middle portions of the cerebellum, and possibly from the nucleus of the auditory nerve. Passing upward from the cerebellum the superior peduncle disappears beneath the corpora quadrigemina, decussates and apparently ends in the red nucleus of the tegmentum of the opposite side. While the majority of its fibres do end in this nucleus, a certain number lying dorsad of it pass onward and then divide into two bundles.† The first of these turns forward and outward, and forms a part of the pillar of the lenticular nucleus ("Linsenkernschlinge"). The second turns upward and backward, passes through the internal capsule and forms a large part of the corona of the tegmentum ("Haubenstrahlung").

2. The lemniscus forming the outer and ventrad boundary of the tegmentum is made up of two sets of fibres, one of which, including two-thirds of the tract, degenerates downward; the other, including the remainder, degenerates upward. The large part of the lemniscus is made up of fibres which have come from the interolivary tract, and which have a probable connection with the olivary body, and with the nuclei gracilis and cuneatus. These pass upward in the tegmentum external to the red nucleus, turn outward,

*The stratum intermedium of Meynert ends in the column of Turk, see page 37.

†The pillar of the lenticular nucleus is used as a synonym for lenticular loop. See the description of the termination of the superior peduncle of the cerebellum given on pages 34-35.

go through the Luy's body and the internal capsule, and end partly in the lenticular nucleus and partly in its pillar.* The smaller part of the lemniscus is made up of fibres which have come through the upper or sensory decussation from the posterior columns of the spinal cord, which have passed external to the greater part of the lemniscus in the pons, and which, at the level of the corpora quadrigemina posterior, turn upward and backward, and going through the posterior part of the internal capsule, form a part of the corona of the tegmentum. It is possible that through this tract sensory impulses pass directly from the posterior columns of the cord to the "carrefour sensitif." In the upper part of its course in the tegmentum, the lemniscus has numerous fibres associated with it which lie on its median side, and pass from the crusta to the tegmentum, and are lost in the raphé.

3. The posterior longitudinal bundle lies beneath the corpora quadrigemina in the tegmentum. It can be traced from the anterior columns of the cord (Vorderstranggrundbündel) through the medulla and pons, lying just beneath the gray floor of the fourth ventricle near the median line. It passes beneath the corpora quadrigemina, then becomes small, and its fibres are lost in the gray matter of the third ventricle. Its individual fibres are of short course, and their function is to connect the various cranial nerve nuclei with one another. Between the nuclei of the III., IV., and VI. nerves it is of large size, since the fibres which join these nuclei, and convey impulses which produce conjugate movements of the eyes, are many.

4. Longitudinal fibres in the formatio reticularis. These fibres come partly from the lateral column of the cord (Seitenstrangreste), partly from the posterior columns of the cord, and partly from the corpus restiforme of the medulla. Many of them end in the gray cells which are scattered through the formatio reticularis, and from which new fibres arise. The area of the formatio

*"These fibres (the larger lemniscus) pass upward in the tegmentum external to the red nucleus of the tegmentum turn outward and end in the parietal (and central) cortex after passing through the internal capsule," and do not terminate as above described in the lenticular nucleus and its pillar. "The smaller part of the lemniscus comes from the superior olive and corpus trepazoides and goes to the posterior corpora quadrigemina," Starr.

reticularis diminishes from below upward, and from its upper extremity, at the level of the upper corpora quadrigemina, four distinct bundles of fibres branch out to pass on into the brain. These are as follows:

(*a*) The bundles of Vicq d'Azyr and Meynert, which pass to the optic thalamus (already described).

(*b*) Numerous fibres, to which are joined some of the fibres in Meynert's bundle and some in the posterior longitudinal bundle, which go to the central gray of the third ventricle.

(*c*) Many fibres which pass to the corpora quadrigemina anterior and:

(*d*) Fibres to the posterior corpora quadrigemina which either end in these bodies or join their brachia, and then pass onward into the corona radiata. The former (*c*) cannot be distinguished from fibres in the lemniscus, which pass to the corpora qnadrigemina.

The connection of the tegmentum with the higher parts of the brain is formed by means of two large bundles, *each* of which is made up of fibres from *all* the various tracts in the tegmentum. One of these bundles is the pillar of the lenticular nucleus (Linsenkernschlinge), the other is the corona of the tegmentum (Haubenstrahlung). The first passes to the lenticular nucleus, the second to the cortex of the parietal lobe. Inasmuch as both bundles contain sensory fibres, it is probable that one centripetal impulse passes to both parts, awakening complex reflexes in the basal ganglion and conscious perception in the cortex.

V. The tracts in the cord can be traced into the medulla. 1. The anterior median and postero-lateral columns of the cord have already been traced to the pyramids. 2. The posterior median and posterior lateral columns end in the nucleus gracilis and nucleus cuneatus respectively; the posterior median column being made up wholly of sensory fibres from the legs. From the nucleus gracilis fibres arise which decussate in the upper or sensory decussation, and pass partly into the interolivary tract, and partly into the olivary body. From the nucleus cuneatus fibres arise, some of which join those from the nucleus gracilis, and some go directly into the formatio reticularis of the same and of the opposite side. The continuation of the sensory tracts in the cord is therefore to be

found in both the interolivary tract and in the formatio reticularis of the medulla. 3. The direct cerebellar column of the cord passes into the corpus restiforme of the medulla, and thence to the vermiform lobe of the cerebellum, where it decussates and ends in the cortex. To this column are joined in the corpus restiforme fibres which pass from the formatio reticularis, and from the olivary body of the opposite side to the nucleus dentatus of the cerebellum. It is possible that some of these fibres also go to the cortex of the cerebellum.

While the anatomical statements of Flechsig differ in some particulars from those of Wernicke, Forel, and Spitzka, these authors agree in regard to the course of the greater number of the tracts here described. The method of descripion of brain anatomy pursued by Flechsig is far more clear than the old method of describing successive sections at various levels through the brain. To follow a definite tract in its course and to learn its relations to other tracts and to the basal ganglia, is not only to secure a distinct picture of the structure of the brain, but also to aid one materially in any attempt to localize a disease in that tract. The results of Flechsig may be accepted as the outcome of careful research by means of an original and trustworthy method of investigation. His presentation of the subject of brain anatomy in this concise but complete little work will prove of immense service to those who are laboring upon this complex subject.

The notes which I have appended to this Review of the Map of the Human Brain have been added for the purpose of explanation and of harmonizing it with more modern research.

I am indebted to the kind courtesy of William Wood & Co., of New York, for the use of cuts and the privilege of making extracts from Dr. Starr's articles in their excellent work, Reference Handbook of the Medical Sciences.

The source,—Landois and Stirling—of diagram VIII. was accidentally omitted.

Multiple Neuritis and its Relation to Certain Peripheral Neuroses.

THE MIDDLETON-GOLDSMITH LECTURES FOR 1887.

BY

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MULTIPLE NEURITIS AND ITS RELATION TO CERTAIN PERIPHERAL NEUROSES.

THE MIDDLETON-GOLDSMITH LECTURES,

Delivered under the direction of the New York Pathological Society, January 25 *and* 28, 1887.

Lecture I.

Mr. President, Ladies and Gentlemen of the Pathological Society:

In inaugurating the course of lectures which bears the name of the founder, it is becoming that some expression of gratitude should be made to Dr. Middleton Goldsmith. This can hardly be associated with any form of personal encomium, since that would be as distasteful to him as it is needless before this Society, which owes its existence in part to his exertion. It is proper, however, to emphasize our appreciation of the motives which have led to the establishment of this lectureship. Every one knows that in the rapid progress of medical science, in the great and needful division of labor which that progress has made imperative, there is offered to each individual worker a vast mass of facts which cannot be easily grasped or even readily comprehended. To classify these facts, to weigh their importance, to draw logical conclusions from them, and to present these conclusions in a clear and accessible form is a work of no little magnitude, but of absolute necessity. To accomplish this specialists are at work in every department, and it may be well to emphasize the fact that it is the necessity for their labor, rather than any temporary fashion, that has led to that specialization in medical science which is so often harshly criticised. But special studies lose half their value if they are not made generally available. And it is just here that a lectureship such as this has its use. It enables the results of individual labor to

reach beyond the specialist, to be placed before the general practitioner in a simple but comprehensive way. There is, therefore, no means by which an individual can make his influence more widely and permanently felt than by the endowment of such a foundation. For when his personal activity has of necessity ceased with the natural termination of an honorable career he may be sure that, yearly, facts and results are proclaimed in his name of immeasurable service to his successors and of benefit to the profession.

And now, before proceeding to the subject which is to occupy our attention for a time, let me express my obligation for the honor which this Society has conferred in inviting me to inaugurate this course of lectures. It is an honor to which I have no claim, and the duty incurred is one which I feel incapable of performing satisfactorily. I must beg your indulgence in the performance of the task you have laid upon me, and if I succeed in eliciting your interest I know that it will be because the subject we are to study together has a novelty which may compensate for the defects of its presentation.

The discovery of a new disease is never made suddenly. It is a gradual process, and certain stages in the progress toward its complete recognition may be observed. There is first the period of clinical observation, when isolated cases of an unfamiliar and mysterious affection are recorded as curiosities. To this succeeds the period of diagnosis, when, by a comparison of the now numerous cases, a clinical picture of the disease is gradually filled out. In this stage there is much to impede the progress of discovery. For not content with an analysis of symptoms, and a grouping of cases, the majority of observers offer theoretical explanations of the nature of the new affection, and an element of speculation enters which often obscures the facts. There is, however, a real progress in this period, for it is characterized by inductive reasoning from fixed data, and as a result reliable conclusions are reached which make a diagnosis possible. The third period is that of pathological discovery, when the morbid changes lying at the basis of the disease are accurately ascertained. In this stage erroneous theo-

ries are eliminated, true explanations for various symptoms become self-evident, and the exact nature of the affection is determined. The disease has now a status of its own. And at first this might seem to be the final stage in the progress of discovery. But it is not. There remains a period of etiological classification, when conditions, formerly supposed to be dissimilar, are found to have a common basis, when the pathological changes are ascertained to be the same although the clinical pictures have varied, and when classification of the various forms is rendered possible and a definition of the disease is reached.

Such a gradual advance toward general recognition is well illustrated by the history of multiple neuritis, which is to engage our attention at the present time.

I think we may claim that one of the first cases presented was by an American physician, Dr. James Jackson, of Boston, in 1822.[1] In a paper "On a Peculiar Disease Resulting from the use of Ardent Spirits," which he named arthrodynia, he gives a most graphic picture of what we now know to be one form of multiple neuritis. He says, "This disease comes on gradually. It commences with pain in the lower limbs, but especially in the feet, and afterward extends to the hands and arms. The hands may be affected first in some instances, and in all cases in an advanced state the pain is more severe in the feet and hands than in the upper part of the limbs. The pain is excruciating, but varies in degree at different times. It is accompanied by a distressing feeling of numbness. After the disease has continued a short time, there take place some contractions of the fingers, and toes, and inability to use these parts freely. At length the hands and feet become nearly useless, the flexor muscles manifesting, as in other diseases, greater power than the extensors. The whole body diminishes in size, unless it be the abdomen, but the face does not exhibit the appearance of emaciation common to many visceral diseases. The diminution is especially observable in the feet and hands; and at some time the skin of these parts acquires a

[1] New England Journal of Medicine and Surgery, vol. xi., p. 351.

peculiar appearance. The same appearance is noticed in a slighter degree in the skin of other parts. This appearance consists in a great smoothness and shining with a sort of fineness of the skin. The integuments look as if tight and stretched, without rugæ or wrinkles, somewhat as when the subjacent parts are swollen ; but the skin is not discolored. Yet in this disease there is not any effusion under the skin, and the character which this assumes arises from some change in the organ itself. The most characteristic symptoms are manifested in the limbs, but the pain is not limited to these—and other symptoms are exhibited in other parts. The pain sometimes shoots suddenly up one or both legs, and in one case it frequently passed up the back and then forward to the pit of the stomach. The functions of the stomach are always impaired. The mind is weakened. Sleep is prevented by pain. I believe that this disease is always fatal when the use of spirituous liquors is not abandoned before the powers of the digestive organs are greatly impaired."

It is hardly possible, even at the present day, to add to this description, which portrays in strikingly vivid language the main features of one form of multiple neuritis.

The next observations of importance in establishing a clinical picture were made by Magnus Huss, who, in his work upon chronic alcoholism, in 1852, gave a very complete description of alcoholic nervous symptoms, dividing the cases into paralytic, anæsthetic, convulsive, epileptic, and hyperæsthetic forms. But not content with the clinical side, he advanced to pathological hypothesis, and ascribed all the various symptoms to lesions in the central nervous system. His description was amplified and completed by Lancereaux, in 1864, in an article upon alcoholism in the "Dictionnaire Encyclopédique des Sciences Médicales." But the pathology of the disease was still a matter of speculation.

In 1855 the great work of Duchenne was published,[1] and in it a number of cases are recorded which we now recognize as multiple neuritis. In these cases there were sensory disturbances, consisting of pain, numbness,

[1] Electrization localisée.

and loss of sensation; motor disturbances, consisting of paralysis, with atrophy, especially marked in the distal parts of the extremities, and attended by a loss of faradic contractility in the paralyzed muscles; and cyanosis, coolness, and increased sweating in the affected limbs. Duchenne grouped these cases together under the title "Paralysie Générale Spinale Subaigüe Ascendante," because he thought a gradually advancing lesion in the spinal cord, from below upward, would explain the symptoms. It is true that he found no macroscopic change in the cord in the only case in which he made an autopsy. But when, under the leadership of the French school, from 1860 to 1865, the microscopic appearances in nervous lesions began to be studied, the hypothesis of Duchenne at once appeared to be verified, for it was found that such symptoms as numbness, pain, and anæsthesia were associated with lesions of the posterior columns of the spinal cord. And it was also proven that atrophic paralysis was caused by a degeneration of the ganglion-cells of the anterior gray horns of the cord, not necessarily visible to the naked eye. It seemed an easy step to the conclusion that when these symptoms occurred together the entire spinal cord was the seat of disease, and that wherever they occurred a spinal lesion was progressing. The pathology of this form of paralysis appeared to be definitely ascertained, and for many years the fallacy of such a conclusion was not detected. All atrophic paralysis was invariably referred to spinal lesions, because spinal lesions may cause atrophic paralysis.

But facts rarely accommodate themselves permanently to theories, and after a time a mass of very unwieldy facts began to accumulate. Cases of atrophic paralysis without spinal lesion were observed, and these threw doubt upon the theoretical pathology. The period of true pathological observation had begun, and gradually went on to completion. In 1864 Dumenil reported the following case, which deserves to be cited, as it is the first in which an autopsy established the existence of a wide-spread disease in the peripheral nerves as a cause of sensory, motor, and trophic symptoms.

Observation I.—A tailor, aged sixty-one, after suffering from prickling in the toes for two weeks, was suddenly taken with weakness in the left arm and right leg, and, a few days later, by the same paresis in the left leg. Within five days he could not stand or walk. The paralyzed feet hung flaccid, and were totally paralyzed; the thighs could be moved freely. Anæsthesia was found on the right sole and calf, and on the left foot and outer side of the leg. In the muscles of the hands and forearms a considerable atrophy, with paralysis, developed. The faradic contractility was abolished in the paralyzed muscles. He complained of a painful numbness in the paralyzed limbs up to the knees, and involving the hands. No improvement; death in four and a half months.

Autopsy: Spinal cord and nerve-roots were normal. The finer nerve-branches in the legs and hands were degenerated, only a small number of nerve-fibres being found. Single fibres showed no continuous myelin sheath; but this was segmented and granular. There was an increase of connective tissue, and many fat-cells in the nerves.[1]

This case being of an anomalous character excited very little attention. Two years later, however, Dumenil reported another, and published an elaborate article on peripheral paralysis, in which he said: "My own observations have convinced me firmly that many paralyses of obscure origin are caused by true spontaneous neuritis."[2] The second case, which with the first led to this conclusion, has been cited by Leyden as a case of neuritis, but it does not conform to any of the types of multiple neuritis, and is open to some doubt from the results of the autopsy, since numerous foci of disease were found in the spinal cord. We therefore pass it by.

Singular as it may seem, an interval of ten years elapsed before another case of similar nature, accompanied by a record of *post-mortem* examination, appeared.

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[1] Dumenil: Gaz. Heb., 1864, p. 203, and Gaz. Heb., 1866, No. 4.

[2] Ceque j'ai observé me donne la conviction intime que bien des paralysies de cause obscure ont leur point de départ dans les véritables névrites spontanées, Gaz. Heb., 1866, No. 4.

Then Eichhorst, of Berlin, reported the following interesting history:

Observation II.—A female, aged sixty-six, after suffering daily for two weeks from a chill, fever, and sweat, attended by malaise, anorexia, and constipation, noticed an œdematous swelling of both feet and legs, and complained of pain in the abdomen. A week later, on admission to the hospital, these symptoms continued. The urinary examination was negative. Three days after admission she suddenly felt a severe boring pain in the left leg, shooting into the toes, and at the same time a profuse sweat broke out over the calf and back of the foot. A few hours later a total paralysis was found in the muscles supplied by the peroneal nerve, with a marked anæsthesia. The electric reaction, at first preserved, was found two days later to be gone. After six days, during which she had no further chills, a paralysis of the anterior tibial nerve developed, and soon after of the posterior tibial nerve also. One week later entire paraplegia of the legs, with anæsthesia, severe pains, continual sweating, increasing œdema, and loss of the tendon reflexes had ensued. There followed a paralysis of the left, and soon after of the right, radial nerve. Moderate fever continued, and albuminuria appeared. Three weeks after her admission sudden blindness developed, the ophthalmoscopic appearances being at the time normal; the patient then lay in bed with eyes closed, unable to move a limb. The extremities perspired constantly, and were tender, any pressure on nerve-trunks being very painful. In the face there was no trouble, and the senses were normal except that of sight. No trouble in swallowing. Nothing abnormal about the viscera during the entire disease. No irregularity of pulse or respiration. Death occurred on the forty-fourth day of the disease.

Autopsy: Spinal cord absolutely normal. The nerve-trunks in the bicipital grooves appeared intensely red to the eye, the perineurium being discolored and the endoneurium blood-red. The same appearance was noticed in the large nerve-trunks of the arm in their course, as well as in the left tibial nerve. The microscopic

examination showed a remarkable distention and tortuosity of the blood-vessels of the perineurium; the vessel-walls were thickened, their nuclei increased. In the vicinity of the vessels a large number of lymphoid cells were found, which everywhere followed the vessels and infiltrated the connective tissue. There were also numerous fatty cells. The connective-tissue fibrillæ of the perineurium were thickened, shining, and swollen; their nuclei were increased and partly infiltrated with fat granules. Similar changes were seen in the endoneurium, viz., numerous extravasations of blood, which separated the nerve-fibres and compressed them. The nerve-fibres showed marked degeneration, especially those lying next the endoneurium, consisting of disintegration of the myelin sheath, and a distention and spindle-shaped swelling of the individual nerve-fibres. The nuclei of the sheath of Schwann were not increased in number, but the protoplasm about them was coarsely granular and opaque. The cells of the endoneurium were everywhere wanting, being replaced by fatty granular cells, even between the uninjured fibres.[1]

This is the first case to be found in which the microscopic appearances are described with sufficient detail to be satisfactory. It is to be noticed that here the lesion was an acute inflammation, and was, apparently, a diffuse one, both interstitial tissue and nerve-fibrils being involved in the process. That it was primarily an interstitial inflammation, and that the affection of the nerve-fibrils was secondary, due to pressure of the products of exudation, is clearly seen in the fact that those fibres were more seriously affected which lay near vessels, while the deeper fibres in large bundles were not at all degenerated. The appearance of the fibres was such as occurs in any degeneration from pressure. There is no reason, therefore, to believe that the process began diffusely.

As to the symptoms in this case, it is to be remarked that they were ushered in by an acute febrile movement with chills, and that severe pain was an early and promi-

[1] Eichhorst: Virchow's Archiv, Bd. 69, p. 265. 1876.

nent symptom; also that œdema and sweating were present; and that the optic nerves were involved.

The clinical picture in the following case of Joffroy, published three years later, was somewhat different, as was also the pathological condition.

Observation III.—A washerwoman, aged thirty-three, in the last stage of phthisis, was admitted to the hospital on March 5th. In February she had noticed a rapidly increasing weakness of her legs, and at the time of admission she could not walk or raise her feet from the bed. She could flex the knees but not extend them. There was no contracture, and the muscles were relaxed and flabby. Sensations to pain, temperature, and pressure impressions were normal, but the muscular sense was lost, and reflexes were diminished. She showed such a degree of mental weakness that tactile sense could not be tested. She had no shooting pains, no loss of control over bladder and rectum, no bedsores. Two weeks later the arms became involved in the paralysis, and atrophy, inco-ordination, and loss of muscular sense, with fibrillary motions, developed within a few days. There was great diminution of faradic excitability in all the paralyzed muscles. At the end of ten days the arms were entirely powerless, but retained their sensibility. On April 7th she died.

Autopsy: A chronic meningitis of the brain explained the mental symptoms. The spinal cord was normal. The nerves appeared normal; but microscopic examination showed very marked degeneration in all the nerve-trunks, but especially in the sciatic, radial, and ulnar nerves. There was a segmentation of the myelin sheath, which at places was reduced to a finely granular mass. Many sheaths of Schwann were filled with this mass, others were empty. The nuclei of the sheaths of Schwann were increased in number. All the spinal nerve-roots were normal. The changes in the nerves were followed down into the fine terminal branches in the thenar muscles. The muscles were atrophied and showed fatty degeneration.[1]

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[1] A. Joffroy: Arch. de Phys. norm. et path., 1879, pp. 172–198.

Here, in contrast with the preceding case, it is to be noted that the lesion was not attended by congestion of the nerves, or by any exudation of lymphoid cells, or by marked interstitial changes. The affection was a true parenchymatous inflammation, with degeneration of the myelin sheath and axis-cylinder. As a result, the macroscopic appearance of the nerves was not such as to attract attention, and it required a microscopic examination to demonstrate the changes present. Joffroy, who reports this case as one of general spontaneous neuritis, finds the lesion identical with that observed in cases of localized neuritis occurring from cold, from lead palsy, or as a sequel of the infectious diseases. In regard to the symptoms, also, the case contrasts strongly with the preceding one. The patient had phthisis. The disease advanced more slowly. Pain was absent, and the sensory symptoms were by no means prominent, the muscular sense being the only one affected.

In 1880, the following cases were observed by Leyden, in the Charité Hospital of Berlin, in both of which changes were found in the peripheral nerves. They are cited because they not only enlarge our clinical picture, but confirm the pathological conditions already described.

Observation IV.—A sailor, aged twenty-five, was suddenly seized with severe tearing pains in all four extremities, especially near elbows and knees, which were swollen, white, indurated, and very tender. The pains shot down the limbs, and were attended by formication and by diminished sensation in fingers and toes. High fever accompanied the onset. In a few days, as the fever subsided, a paretic condition with muscular atrophy was noticed in the forearms, and to a less extent in legs and feet. A gradual improvement took place in the course of a few months in the lower extremities, but the paralysis and atrophy in the arms increased in degree, and the appearance of the hands resembled that seen in lead palsy. There was a reaction of degeneration in the muscles paralyzed. A year after the onset he died of chronic nephritis.

Autopsy : A sclerotic atrophy of both radial nerves was found. The nerve-sheath was thickened. The

muscles were considerably atrophied. The anterior spinal nerve-roots, as well as the cells in the anterior horns, and the entire spinal cord, were normal. In the nerves of the lower extremities no changes were visible.[1]

Observation V.—The second case was that of a merchant, aged thirty-one, who noticed about Christmas, 1878, a feeling of formication in the toes and sole, of first the left, and then the right foot. This continued more or less severe, without any motor disturbance, until July 10th, when he found his legs heavy, and in two days this had so increased that he could not walk. At the same time a constant formication in the soles of the feet was felt. Within a few days similar symptoms appeared in the arms and hands. On admission to the hospital on July 17th, he complained of weakness of the legs and loss of sensation in the hands, also burning pain in the left hand and inner side of the left knee. The motion of the legs was powerless and uncertain, and there was paresis of the extensors of the hands and fingers. Sensation was much impaired in the periphery of the extremities; the tendon reflexes were abolished, the muscles flabby. The electrical examination showed a loss of faradic contractility, and a great diminution of galvanic contractility in the affected muscles. The liver was tender, and there was a slight jaundice; pulse 70; no fever. The paralysis increased slowly in extent and severity, and was accompanied by paræsthesia, pains, and diminished sensibility. The muscles atrophied, and showed the reaction of degeneration. After six weeks slight fever began, with rapid pulse and dyspnœa. The pains affected the upper part of the thorax, and the spinal column became sensitive as low as the eighth dorsal vertebræ. During the last days small rapid pulse, severe dyspnœa, purely costal respiration, delirium, somnolence, and exhaustion were noticed, and on September 3d he died.

Autopsy: The spinal cord was normal, the gray and white substance intact, the large ganglion-cells in normal form and number, though a few seemed to present a slightly swollen, glassy appearance which Leyden consid-

[1] Leyden: Charité Annalen, 1880.

ered the first indications of trophic disturbance. The nerve-trunks, especially the radials and peronei, presented the appearance of a high degree of fatty degeneration and atrophy. The myelin sheath was either wholly wanting or greatly divided and split up, and in a state of fatty degeneration; the axis-cylinder seemed varicose from deposits of fat in it. A marked infiltration of lymphoid cells, especially between the endoneurium and nerve-fibres, was noticed, more marked along the course of the small arterioles. These changes were present in a greater degree in the peripheral branches of the nerves, and seemed less in the nerves near the cord.[1]

It will be noticed that here the lesion corresponded quite closely to that in the case of Eichhorst.

Impressed by the striking features of these two cases, and by their similarity, both in clinical symptoms and pathological changes, to the cases of Dumenil, Joffroy, and Eichhorst already cited, Leyden undertook a review of atrophic paralysis.[2] He showed the confusion which had come from blindly referring all such cases to a single lesion in the spinal cord. He proved that previous authors had erroneously grouped together several diseases distinct from one another, both in their course and in their pathology. One of these is poliomyelitis anterior, acute and chronic, occurring in children and adults; a disease of the anterior gray horns, with local foci of inflammatory degeneration; a disease characterized by motor and trophic symptoms of peculiar distribution and typical course. A second of these is progressive muscular atrophy, with its corresponding bulbar affection, glosso-labio-laryngeal paralysis; a disease of the anterior gray horns and of the motor cranial nerve-nuclei, of a chronic degenerative, non-inflammatory kind; a disease characterized by trophic and motor symptoms of different distribution, nature, and progress from the first disease considered. A third disease is pseudo-hypertrophic paralysis, in which a fatty deposit in the muscle conceals the real atrophy in progress; a

[1] Leyden: Zeitsch. f. Klin. Med., 1880.
[2] Leyden: Zeitschrift für Klin. Med., I. Ueber Poliomyelitis und Neuritis.

disease of childhood, but not due to any spinal affection; a disease of the muscular tissue itself. And lastly, he drew the picture of multiple neuritis, a disease due to degenerative processes in the nerves, independent of spinal lesion; a disease characterized by atrophic paralysis, associated with marked sensory symptoms, and with tenderness of the nerve, by a typical course, and, usually, by a favorable termination. By this article Leyden established the status of multiple neuritis as a distinct disease. Its symptoms were analyzed. Its lesion was described. The pathological stage in the progress of its discovery was complete.

When the characteristic features of a new disease have once been clearly pointed out, it is remarkable to observe how rapidly cases of it begin to be recognized. In the two or three years which followed the appearance of Leyden's article, numerous cases of multiple neuritis were reported in the journals. And many physicians, reviewing their records, recognized, in cases previously obscure or imperfectly diagnosticated, typical pictures of the new disease. It may be well to consider a few of these cases in order to complete our clinical knowledge of the affection.

Observation VI.—A female, aged thirty, of intemperate habits, but otherwise in good health, after suffering from formication, coldness, and pains in her feet and legs for some months, noticed an œdema of both legs. This increased rapidly after a few days, and the swollen limbs became painful to touch or pressure, and were the seat of severe lancinating pains, which were worse at night. Within a month the same symptoms appeared in the arms and hands, and a marked hyperæsthesia developed in all the extremities, as well as a rapidly progressing paralysis; so that, on admission to the hospital, six weeks after the appearance of the œdema, it was impossible for her to lift her limbs from the bed, or to extend her hands and fingers. The movements in the distal portions of all the extremities were much more impaired than those near the trunk, and in the paralyzed extensor muscles the faradic excitability was almost abolished. Heart and kidneys normal. She had some fever, and was

delirious at night. The symptoms increased rapidly; the paralysis became total; respiration became difficult; the heart rapid, and a week after admission to the hospital she died.

Autopsy: Tubercles in the lungs. Brain and cord were normal. Nerve-roots were normal. In the nerves of the extremities marked degeneration was found, especially in the radial and tibial nerves. By the side of a small number of empty sheaths were found fibres, whose myelin was segmented and in drops, separated by empty spaces. The axis-cylinders were indistinct. There was no increase in the nuclei of the sheath of Schwann.

In discussing the case Dr. Lancereaux made the diagnosis of alcoholic paralysis, assigning the lesion to the nerves, and differentiating it from a myelitis or a meningitis. In so doing he criticised the view of Wilks and Lockhardt Clarke, who still considered alcoholic paralysis as a central disease. He cited another case, very similar, of a female aged thirty-three, in which the symptoms were pains, hyperæsthesia, tenderness, paralysis, with atrophy in the extremities, but in which œdema was not so marked and came on quite late in the course of the case. The same lesions were found. To these he added two cases in which the patients manifested the same symptoms, but had never drank. They were both, however, sellers of varnish, and lived day and night in an atmosphere permeated by alcoholic vapor, from which he concludes that chronic alcoholic poisoning can occur by absorption through the lungs—a valuable observation, but hitherto not confirmed.[1]

The following case was reported by Grainger Stewart, together with two other cases which resulted in recovery.

Observation VII.—A male, aged thirty-one, noticed during August, 1880, a weakness of the legs, and during the following month a pain of a prickling character in the legs and feet. These increased in intensity, and in October a similar feeling came on in the fingers and hands, accompanied by loss of power and stiffness.

[1] E. Lancereaux: De la Paralysie alcoolique, Gaz. Heb. de Méd., 1881, p. 120.

When seen, in November, he had tingling pain in both legs from the knee to the back of the foot, with numbness and feeling of cold in the toes and plantar surfaces, so also in the hands, to a less extent. No girdle pain or formication. Sensibility to touch was diminished in the legs and hands. Transmission of impressions was delayed. Sensibility to heat, tickling, and pain were all diminished, as was also the muscular sense in the feet. There was no nystagmus, although he complained of things dancing before his eyes. Sight was normal. There was no incontinence of urine or fæces. The skin reflexes were absent in the soles, but normal in the abdomen and groins. The knee-jerk was absent. Voluntary motion was greatly impaired in legs and arms, and attempts to use the muscles caused pain. Electric irritability of the muscles and sensibility of the skin was much diminished. There were no vaso-motor or trophic changes. His mental condition became changed during his stay, his memory was impaired, and he seemed drowsy. A month after his first examination he died of pneumonia.

Autopsy by Dr. D. J. Hamilton: The median, ulnar, and tibial nerves showed great changes. With a low power of the microscope the bundles of fibres appeared to be affected by fatty degeneration. With a high power it was found that the axis-cylinders were swollen so as to form a number of fusiform bodies in the course of the nerve-tubes. These at parts were divided into a number of round homogeneous colloid bodies. When set free these bodies underwent a fatty degeneration, forming compound granular corpuscles. In some fibres the axis-cylinder was totally destroyed, nothing but a quantity of fibrous tissue remaining. The cords of the brachial plexus and the sciatic nerves were normal. Slight evidence of secondary sclerosis in the spinal cord was found in the columns of Goll and in the direct cerebellar columns. Its origin could not be explained.[1]

It must not be supposed, from the fact that all the cases so far cited were fatal, that death is always the result in multiple neuritis. This is very far from the truth, and

[1] Grainger Stewart: Edinburgh Medical Journal, April, 1881.

probably, if the mortality had been greater, the pathology of the disease would not so long have eluded search. The fatal cases have been brought together in order that the pathological appearances observed might be noted and compared, and might become somewhat familiar by repetition. It is time to enter upon the more careful study of their pathology, and so, for a time, let us leave the clinical features of the disease.

It may not be out of place, before proceeding to discuss the changes occurring in inflammations of the nerves, to review the normal anatomy of a nerve.

The Histology of a Nerve-Fibre.—When a nerve-trunk is dissected, the connective-tissue sheath or perineurium enclosing its fibres torn away, and the individual fibres set free by teasing from the finer connective-tissue strands or endoneurium which bind them together, it is possible to distinguish certain parts, by means of appropriate methods of staining.

There is *first* the axis-cylinder, Fig. 1 (*a*). This is made up of a number of primitive fibrils arranged longitudinally and continuous throughout the length of the nerve. The fibrils are cemented together by a substance which appears finely granular. Each cylinder is supposed to represent a prolongation of a single nerve-cell; but whether the individual fibrils of which it is made up come from the same cell or from different cells, or from the fibrillary network in the central organ surrounding the cell, is a matter of hypothesis.[1]

There is, *secondly*, the myelin sheath surrounding the axis-cylinder, Fig. 1 (*b*). This is not a continuous tube, but consists of a series of little tubes, or interannular segments, placed end to end. The point of junction of two ad-

[1] Whatever their origin, it is certain that they pass together in the axis-cylinder to the periphery, and that there the axis-cylinder as such terminates, while the individual fibrils branch out in various directions, and, joining with other fibrils from other cylinders, form a fine plexus within the organ to which the nerve as a whole has gone. It has been thought possible to trace individual fibrils of the plexus into individual epithelial cells. Such is their termination in various internal organs and in the skin. This is not, however, the only manner of termination of the axis-cylinder, for individual nerves can be traced directly to terminal organs, such as the terminal plates upon the muscle, and the terminal bulbs and corpuscles in the skin; in which cases no division or branching of the fibrillary constituents of the axis-cylinder has been discovered. The termination in each of the organs of special sense is still different. The axis-cylinder can best be seen by the acid fuchsin stain.

jacent segments is indicated by a constriction in the contour of the nerve-fibre, and if the myelin be stained with osmic acid it is seen to be deficient at these constrictions. If, by any means, the nerve-fibre be broken, the myelin in any segment will run out and collect in drops, showing that it is a semi-fluid substance, of fatty nature.[1]

There is, *thirdly*, a connective-tissue membrane surrounding the myelin sheath, the sheath of Schwann, Fig. 1 (*c*). This, like the last, is made up of segments cemented to one another at the point of constriction of the fibre. This constriction is known as the ring or node of Ranvier, Fig. 1 (*d*), and at its situation the sheath of Schwann is the only covering of the axis-cylinder. On the inner side of this sheath, and half-way between two nodes, is found a nucleus, Fig. 1 (*f*). The sheath and node are made evident by staining with nitrate of silver, while the nucleus is seen best by staining with carmine or acid fuchsin. The mutual relation of the sheath of Schwann and the myelin sheath is best understood by the study of their development, segment by segment. Ranvier[2] likens the production of any one segment of these sheaths to the production of a fat cell from a nucleated cell. Every nucleated cell is surrounded by a layer of protoplasm. As the fat forms it collects within the layer of protoplasm in the vicinity of the nucleus, in the form of drops which finally unite into a mass. This mass is surrounded by a very thin layer of protoplasm and by the cell-membrane. On one side of it is seen the nucleus flattened out

FIG. 1.—A Normal Nerve-fibre. *a*, Axis-cylinder; *b*, medullary sheath; *c*, sheath of Schwann; *d*, node of Ranvier; *e*, incisure of Schmidt; *f*, nucleus of the sheath of Schwann

[1] The myelin sheath is not a necessary constituent of all nerve-fibres, for the majority of the nerves of the sympathetic system are devoid of such sheaths. Nor does the myelin sheath of the nerves of the cerebro-spinal system extend from end to end of the axis-cylinder. For the axis-cylinder first receives its sheath at some little distance from the cell from which it issues, and at its termination, where it breaks up into branching fibrils, the myelin envelope ceases. Throughout the course of the nerve, however, the myelin sheath is present in the cerebro-spinal nerves, forming a protecting envelope, and probably acting as an insulating substance as well.

[2] Ranvier: Leçons sur l'Histologie du Système Nerveux, tome i., p. 115.

against the inner side of the membrane in the layer of protoplasm. Now, if each segment of the sheaths corresponds to the fat cell, and the myelin to the fat, the structure of the segment is clear. The membrane of the fat cell is represented by the sheath of Schwann. At the point of constriction of the nerve-fibre this membrane is cemented to that of the adjoining segment. Beneath the membrane, and flattened against its side, is the nucleus of the segment, lying in a layer of protoplasm in the middle of the segment. This layer of protoplasm forms a lining everywhere to the sheath of Schwann. With that sheath it is reflected around the axis-cylinder. Within the lining, in a state of semi-fluidity, is contained the myelin, which will run out if the limiting membrane is broken. By appropriate staining fine lines can be seen passing between the outer and inner layers of protoplasm, through the myelin sheath, the so-called incisions of Schmidt, Fig. 1 (*e*). These have been considered little trabeculæ of protoplasm within the segment in the midst of which the myelin lies. Recent staining methods seem to indicate that they belong to the sheath of Schwann and are connective tissue. The layer of protoplasm lying against the axis-cylinder is the layer which was formerly described as the sheath of Mauthner. Some authorities consider that a layer of connective tissue similar to the sheath of Schwann surrounds the axis-cylinder, but this is still uncertain. Thus the nerve-fibre consists of a central conducting strand, surrounded and insulated by a series of tube-like segments of fluid contained within a membrane, which are joined to one another, forming a double protecting sheath.

Individual fibres are associated in bundles held together by fine connective-tissue cells whose nuclei can be seen in a carmine or fuchsin stained preparation, lying always adjacent to, but outside of, the sheath of Schwann.[1] This

[1] These connective-tissue cells which lie between individual fibres have been recently the subject of study by Adamkiewicz (Adamkiewicz, Ueber Ruckenmarkschwindsucht, Sitzungsbericht der Wiener Acad., Bd. 91, Abth., II.) and Rosenheim (Rosenheim, Arch. für Psych., xvii., p. 830). The former regarded them as nerve-corpuscles, but the latter has proven that they are true connective-tissue cells. They are best seen in specimens stained with a double staining of anilin gentian violet and methyl blue, though they can be seen distinctly in fuchsin stained preparations, as has recently been demonstrated by Dr. Van

has been called the endoneurium, while the connective-tissue sheath surrounding the entire bundle is named the perineurium. Capillary vessels with free anastomoses run within the nerve, their walls lying adjacent to the individual fibres, and thus affording a perfect nutrition.[1] Lymph-spaces also have been demonstrated within the nerve-sheath, but not among the fibres.[2] That the interfibrillary spaces of the endoneurium, however, open into these peri-fascicular lymph-spaces is probable, from the fact that they do so in other organs. While it is evident that the nutrition of the axis-cylinder is derived from the circulatory fluids, it is probable that it is only at the nodes of Ranvier that the absorption takes place, since elsewhere the myelin sheath interferes with osmosis. Thus, if the nerve be put in nitrate of silver, it is only opposite the nodes that the axis-cylinder becomes stained.

The Pathology of Multiple Neuritis.—In studying the pathological processes which occur in multiple neuritis, it is necessary to keep these various elements of the normal nerve in mind, since each element is subject to changes. The exact character of these changes is best understood by observing the results of nerve-degeneration artificially produced in animals. And a study of this will not be out of place here, for, as we shall see, the changes occurring in multiple neuritis correspond quite exactly to those produced by experimental degeneration.

In considering the pathology of degenerative neuritis we enter at once upon a mass of controversial statements. It would seem to be a simple matter to establish, by observations upon nerves which had been experimentally compressed or severed, the changes which ensue in nerve injuries. But, as a matter of fact, there is, perhaps, no field of experimental pathological inquiry in which the results have differed more widely. In the first place, it is probable that the rapidity and even the char-

Giesen, of the Laboratory of the College of Physicians and Surgeons. They appear on nerves after the age of five, and increase in number as the individual grows older, so that in elderly persons they are very numerous. They are round or spindle-shaped, and frequently appear to have little thorn-like processes. In addition to these connective tissue cells there are fine fibrils of connective tissue, and bands of tissue lying between the bundles of nerve-fibres.

[1] Ranvier, l.c., p. 253. [2] Ranvier, l.c., p. 258.

acter of the changes differ in different animals. Secondly, various methods of investigation, of hardening, dissecting, and staining the nerves, seem to have resulted in the production of different appearances. And, lastly, it is by no means certain that a uniform pathological process goes on after experimental lesions.

After a lesion of a nerve-trunk, a process of degeneration sets in at the point of injury, and involves a small portion of the central end and the entire peripheral part of the nerve, from the seat of injury onward. This process may be more or less complete, and may, or may not, be followed by a second process of regeneration in the diseased nerve. It is necessary to distinguish between the degenerative and regenerative processes; and, inasmuch as it is affirmed that they may proceed simultaneously in various parts of the same fibre,[1] it is not strange that the confounding of the two should have increased the confusion in the statements.

In early times it seems to have been a question whether the destruction of a nerve was ever recovered from, and while the majority of authorities rightly contended that this was possible, there were those who denied it vehemently. Steinneck,[2] in 1838, seems to have determined this finally in the affirmative, using both the physiological and anatomical data at his disposal to good advantage. The exact processes of degeneration were first carefully studied by Nasse[3] and Waller,[4] and their results having attracted attention to the phenomena in question, a host of investigators have followed their lead. The most careful examinations of the entire series of changes which go on in degeneration and regeneration have been made by Ranvier,[5] Philipeau and Vulpian,[6] and Dejerine,[7] in France; by E. Neumann,[8] S. Mayer,[9] and Wolberg,[10] in Germany; by Hanken[11] in

[1] E. Neumann: Ueber De- und Regeneration der Nerven, Arch. f. mikros. Anat., xviii. [2] Schmidt's Jahrbuch., Bd. 26, S. 102, 1840.
[3] Müller's Archiv, 1839. [4] Comptes rendus, 1852, vol. 34, p. 675.
[5] Leçons sur l'Histologie du Système Nerveux.
[6] Gaz. Méd. de Paris, 1859, Comptes rendus à la Soc. de Biol.
[7] Arch. de Physiol., 1875, p. 567.
[8] Arch. f. mikros. Anat., xviii., 302, 1880. [9] Zeitsch. f. Heilkunde, ii., 1881.
[10] Deut. Zeitsch. f. Chirurgie, xviii. u. xix., 1883.
[11] Internat. Monatsch. f. Anat. und Histologie, Bd. iii., 1886; Fortschritte der Medicin, 1886, December.

Holland ; by Schiff[1] and Tizzoni,[2] in Italy, and by Weir Mitchell[3] in America.

The majority of writers upon nervous diseases and upon general pathology seem to have followed Ranvier closely, without any mention of the fact that other authorities differ from his conclusions. It will be necessary here to present the various views which are held.

The Process of Degeneration.—When a nerve is compressed by a ligature or forceps without sufficient force to rupture the sheath of Schwann, the myelin is driven away from the point of pressure in both directions, and the axis cylinder is disintegrated and mingled with it. It might be supposed that the nodes of Ranvier would prevent such a driving back of the myelin, but they seem to offer but feeble resistance, so that the entire fibre on either side of the compressed spot is bulged out for some little distance, the sheath of Schwann between the distended portions being left empty or containing only a little granular débris. In a short time changes of a degenerative character are observed on both sides of the point of compression. Those on the central side are limited to the immediate neighborhood of the injured spot, and, according to Ranvier, do not affect the nerve for a distance greater than a centimetre from the point of compression.[4]

On the peripheral side of the point of pressure the de-

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[1] Comptes rendus, 1854, vol xxxviii., p. 448, and Zeitsch. f. Wissen. Zool., 1856, vii., p. 145.

[2] Centralbl. für. Med. Wissen., 1878, Nr. 13.

[3] Injuries of Nerves, 1872.

[4] Some recent investigations of Friedlander and Krause throw doubt upon this statement. (Fortschritte der Medicin, December 1, 1886.) They have discovered a considerable degree of atrophy in the nerve-trunks in the stumps of amputated extremities, and they traced this atrophy upward through the nerves, and into the posterior nerve-roots exclusively. They conclude, therefore, that the fibres affected are entirely sensory fibres. The axis-cylinder is only slightly affected by the atrophy, but the myelin sheath is wholly destroyed. Inasmuch as the degree of atrophy in a nerve-trunk was about the same whether the amputation had been made near the body or near the end of the limb, they conclude that only certain of the sensory fibres degenerate upward. And as the tactile corpuscles and terminal bulbs are only found in the most distal parts of the extremities, it is suggested that only those sensory fibres degenerate which arise from these so-called tactile bodies. If this is proven to be true by further observation, the tactile bodies will assume the rôle of trophic centres for the sensory nerves. And in this case anything which interrupts the connection between the tactile bodies and the posterior spinal ganglion will produce a centripetal degeneration in the nerve. Hence in neuritis from section or pressure, as well as in multiple neuritis, such secondary degeneration is to be expected and should be looked for.

generation is extensive, involving the entire nerve down to its finest terminations. The process is held by some to be a gradually advancing one along the fibre from the point of pressure outward, while others believe that it begins at once in the entire length of the nerve. The first change noticed is a breaking up of the myelin sheath into segments, and then into smaller masses and drops (Fig. 2, *a*, *b*), which finally undergo further disintegration, either by a fatty or albuminoid degeneration, or by a process of saponification, until a finely granular mass alone remains. Tizzoni states that this process is partly due to the activity of migratory white blood-globules, a view which Ranvier supports, and which Neumann and Mayer deny. If, as seems probable, the connective-tissue cells of Rosenheim take an active part in all processes of nerve inflammation, it is possible that these are the bodies which Tizzoni mistook for leucocytes. Ranvier holds that the segmentation of the myelin is due to the increase of the protoplasm about the nucleus of the segment, and that it is this protoplasm which replaces the myelin. Neumann holds that the granular mass resulting from the disintegration is not protoplasmic, but is a débris capable of acting as the basis for processes of regeneration after undergoing a chemical change. All authorities admit that the granular mass may be gradually absorbed, leaving the sheath of Schwann collapsed and empty, or containing only granules of débris or nuclei (Fig. 2, *c*, *d*).

As the myelin undergoes these changes the axis-cylinder usually becomes involved. Some authors, it is true, believe that it remains intact, and that, although deprived of its function, it is capable of resuming that function at any time when regeneration of the myelin sheath has taken place. Such authorities as Erb, Charcot, Weir Mitchell, and Wolberg have given their approval of this view. Ranvier believes that the protoplasm of the nucleus attacks and destroys the axis-cylinder as well as the myelin. Neumann holds that it is split up into segments like the myelin, becomes mingled with it, and undergoes the same process of chemical change, or absorption; and Mayer agrees with this view (Fig. 3, *a*, *b*, *c*).

The sheath of Schwann also takes part in the process

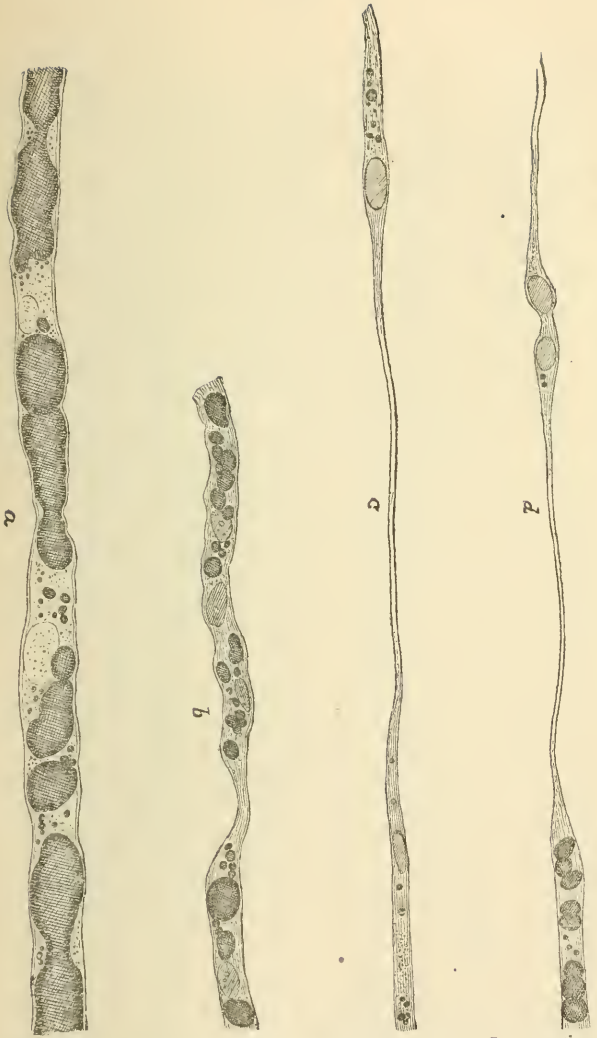

FIG. 2.—The Process of Degeneration. (After Ranvier.) *a*, Segmentation of myelin and axis-cylinder, with increase of protoplasm ; *b*, disintegration of myelin into drops, increase of nuclei, partial absorption of débris; *c*, complete absorption of the disintegrated mass, leaving the Schwann sheath empty, or containing only débris and nuclei ; *d*, the same, nuclei alone remaining.

of degeneration. When that process has fairly begun numerous nuclei are observed lining this sheath in each interannular segment. They may have come by a process of segmentation of the original nucleus of the segment, as Ranvier holds. But Neumann and Mayer have shown that they appear as early at the extremities of the segment as they do in the vicinity of the nucleus, and they therefore conclude that they are a free formation originating in the granular or protoplasmic mass. Tizzoni thinks them emigrated corpuscles. The most recent view of all is that of Rosenheim, that they come from the connective-tissue cells along the sheath which divide and multiply, and show powers of emigration as soon as the process of degeneration begins. When the granular mass is absorbed these nuclei remain scattered along the sheath of Schwann, and it has been suggested that, when in an empty sheath a new axis-cylinder appears, it owes its existence to these nuclei, which arrange themselves in a line and develop into the new fibre (Wolberg). If no regeneration occurs they disappear gradually, and then the only relic of the former nerve-fibre is the empty, collapsed sheath of Schwann, which remains as a connective-tissue strand.

The increase of nuclei and connective-tissue fibrils in the endoneurium and perineurium which accompanies the process of degeneration, aids in the transformation of the nerve into a band of connective tissue.

The degeneration which affects the nerve is continued to the terminal plates upon the muscle, and these are changed into a mass of granules and finally absorbed, a connective-tissue plate being left.[1] Whether any changes occur in the sensory terminal organs, such as the tactile corpuscles or terminal bulbs, has never been ascertained. Those who believe that the individual axis-cylinder fibrils terminate in the epithelium of the skin may cite the trophic changes which often occur on the surface as evidence that this covering of the body shares in the nerve-changes.

Authorities seem to agree that the same results follow

[1] Gessler: Die Motorishe Endplatte und ihre Bedeutung für die Periphere-Lähmung. Leipzig. 1885.

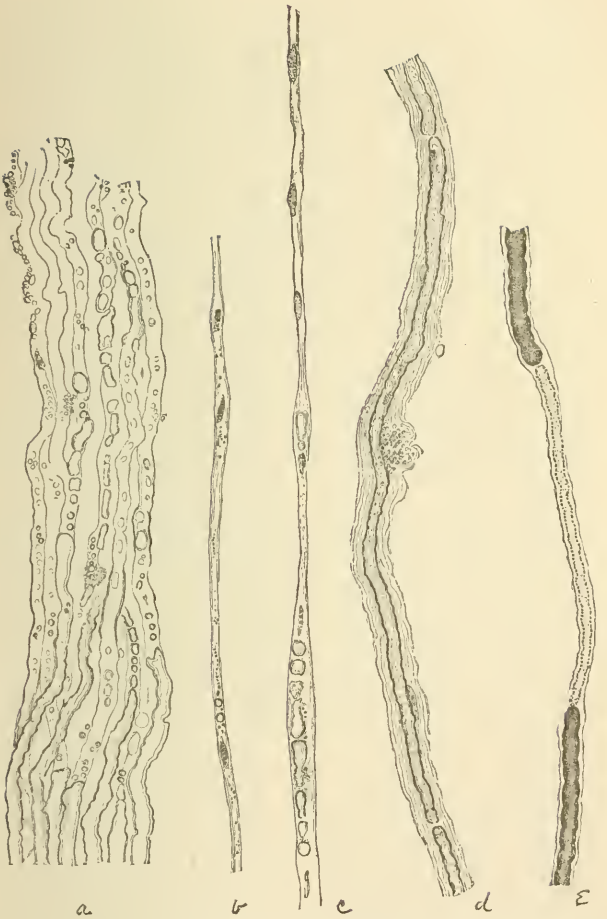

FIG. 3.—The Process of Degeneration and Regeneration going on in Normal Nerves. (After Mayer.) *a*, Segmentation of the myelin; *b*, contents of sheath of Schwann reduced to granular mass; *c*, sheath of Schwann, containing only débris and nuclei; *d*, granular mass in the sheath in process of regeneration; *e*, regeneration of a single segment after degeneration.

a division of a nerve that are observed after its compression, with the difference that at the point of section the myelin runs out of the sheath of Schwann. The cut central end of the nerve becomes swollen into a bulbous extremity by a growth of connective tissue.

Whether a true union of the divided ends ever occurs is still a matter of uncertainty. The majority of authorities, following Ranvier, affirm that while a primary coaptation of the ends by an exudate which is secondarily transformed into connective tissue may occur and hold the ends in position, no true primary union of nerve-fibres is possible, and under all circumstances the degenerative process already described goes on to completion. Glück, however, claims to have observed an actual union of the two ends, with re-establishment of function, at a time too early to have admitted the occurrences of degeneration and regeneration ; and Wolberg, approaching the subject from the surgical side, and considering the results of nerve-suture, inclines to the same view.[1]

In the midst of such contrary statements, what conclusion can be reached as to the actual facts ? It seems evident that but one conclusion is certain, viz., that under different circumstances different processes occur. The various observers are equally trustworthy. It is impossible to choose one set of conclusions rather than another, or to rely wholly upon one series of experiments, however capable the observer may be, for they all rest upon repeated observations. It is undoubtedly true that in some cases the degenerative process, so graphically pictured by Ranvier, from the beginning segmentation of the myelin down to the final result in the connective-tissue strand, the relic of the empty sheath of Schwann, does go on ; while in other cases of a less serious nature the destruction is less complete, and there remains a fibre consisting of a sheath of Schwann, containing a granular mass which may be either an axis-cylinder or a mass capable of developing into an axis-cylinder under favorable circumstances. If this is the case, we

[1] See, also, Weissenstein, Brun's Klin. Chirurg., 1884, p. 310.

can affirm that brilliant surgical successes, with rapid restoration of nerve-function after suture, are possible when the partial degeneration is present, but are impossible when the total destruction of the nerve-fibre has occurred. And statistics show that there is a certain percentage of operations which fail of any result, though repeated on the same nerve.

It is evident that future research should be directed, not so much to determine which of the processes described occurs, as to settle under what circumstances the one is produced rather than the other. And such research will have an eminently practical bearing, inasmuch as it will also demonstrate under what circumstances nerve-suture is likely to be attended by success.

The Process of Regeneration.—After the process of degeneration has gone on for some time in the nerve-fibre, it may cease, and the process of regeneration may begin. With regard to the method of this process two widely divergent views are held. Ranvier and his followers claim that the new nerve is wholly a product of the central end of the injured nerve, growing out from it and making its way along the track of the peripheral end, which takes no active part in the process. Neumann and Mayer, on the contrary, believe that the regeneration goes on in the peripheral end of the cut nerve, segment by segment being formed successively, beginning at the point of injury and proceeding outward, the entire nerve being built up by the union of each distal segment with the one lying centrally to it, until this process has reached the end. These various views demand a more exact statement.

Ranvier describes several ways in which the new fibres issue from the central end.[1] He has seen the central end of an individual nerve-fibre become hypertrophied, and from this swollen part a single new fibre start out already medullated. He has also seen a single axis-cylinder grow out, and then divide into two, or even more, axis-cylinders, each of which develops into a complete medullated nerve; or from the swollen central end of a single

[1] L. c., ii., 42–67.

fibre several medullated and segmented fibres may originate, or one medullated fibre may be sent out with several non-medullated fibres coiled about it; or, lastly, a simple axis-cylinder branches out without a medullary sheath, divides into two, and each of these in turn divides into two, and so on until the sheath of Schwann, which had originally but one axis-cylinder within it, contains as many as eight or more (Fig. 4, *a*). These new fibres appear like a brush upon the end of the divided nerve, and wind about each other, forming a sort of plexus. By the aid of the cicatricial tissue, which usually joins the central end with the degenerated peripheral end, the bushy, branching fibres are directed outward toward the periphery. And when they reach the peripheral end of the cut nerve they insinuate themselves into the old remaining sheaths of Schwann, or between those sheaths, and grow on and outward till at last they reach the termination of the peripheral end, and the regeneration is complete. The terminal plates upon the muscles are renewed by a production of protoplasm in the plate. The description given by Neumann and Mayer is very different. It will be remembered that they describe, as the terminal result of the process of degeneration, a band of fibres each consisting of a sheath of Schwann containing a granular mass. The process of regeneration begins in this mass. Within it they have seen a narrow band of fine homogeneous substance appear, which has the structure of a rudimentary axis-cylinder (Fig. 3, *b*). This does not fill the sheath of Schwann, and is often pressed aside by the nuclei which lie in that sheath (Fig. 4, *c*). It is not at first continuous with the end of the old axis-cylinder, remaining in the central part of the compressed or divided nerve, but as it increases in definite structure it approaches this old axis-cylinder, and finally unites with it. At the point of union a ring of Ranvier is formed. As this axis-cylinder develops, a substance is gradually formed around it, which is stained by osmic acid. This increases in thickness as the protoplasmic mass and the nuclei diminish, until it finally forms a new myelin sheath. The new myelin sheath is never continuous with the old one in the central end of the nerve, since it is separated from that by

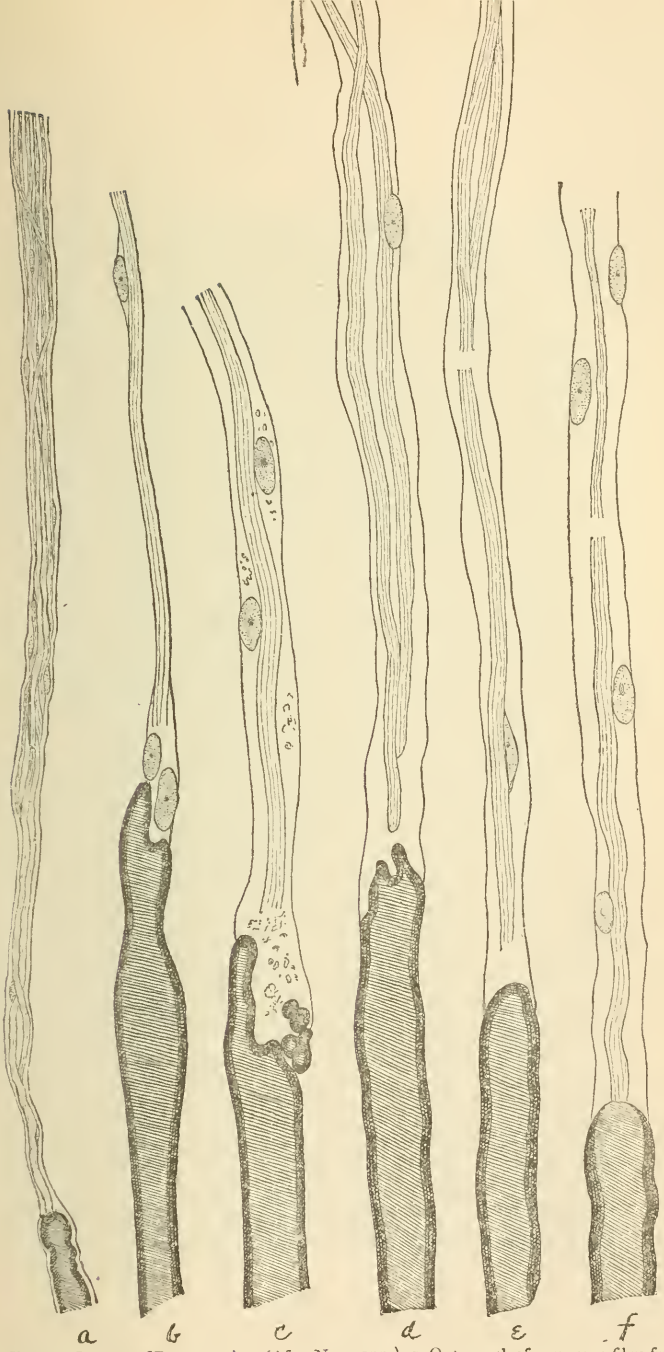

FIG. 4.—Process of Regeneration. (After Neumann.) *a*, Outgrowth of new nerve-fibre from central end, division into numerous axis-cylinders (Ranvier); *b*, development of axis-cylinder in peripheral end, the new fibre separated from the old by nuclei; *c*, ditto, with increase of protoplasm destined to become a new myelin sheath, overlapping of old myelin upon the new; *d*, two new fibres developing in a peripheral segment and ready to unite with the central end; *e*, two segments nearly developed, the central one in advance of the peripheral one; *f*, final union of the peripheral with the central segment, nuclei still remaining.

the ring of Ranvier; but often at first the older sheath seems to bulge out and encircle the new sheath, though this appearance is never permanent (Fig. 4, *c*). At the point of union of the new fibre with the old one, nuclei are often found; but these, like the others, gradually disappear (Fig. 4, *b* and *d*). Lastly, a new sheath of Schwann is produced around the new myelin sheath, and within the old sheath of Schwann. It presses aside the old sheath, together with such masses of protoplasm, drops of myelin, and nuclei as may remain, leaving them thus wholly outside of the new-made fibre, so that they coalesce with, and make part of, the endoneurium (Fig. 4, *e*). The new sheath of Schwann has but one nucleus in each segment, and presents the nodes of Ranvier at egular intervals.

Neumann has shown that this process goes on in every individual segment of the nerve-sheath, so that in segment by segment, proceeding toward the periphery, the regenerative change occurs, and as each segment approaches completion it joins itself to the preceding one (Fig. 4, *e* and *f*), until finally the entire nerve is re-established in its entire length. As the degenerative process begins in the segment nearest to the point of compression, so does the process of regeneration; and in some nerves the two processes may be seen going on together, the segments near the seat of injury being renewed while those at the periphery are still in a state of degeneration. The new fibres do not grow out from the old ones, as Ranvier describes in the regeneration of cut fibres, but protoplasm, with specific developmental properties, forms and differentiates the elements of the new fibre, and then unites it to the old one. The new fibres are, at first, somewhat smaller in calibre than the old ones (Fig. 4, *f*), but they gradually attain a normal size, and then the process may be said to be completed.

While Neumann would have the new fibres develop from the granular mass remaining in the old sheath of Schwann, Günther, Hjelt, and Weir Mitchell suppose that they may originate from the nuclei of the old sheath, or even from connective-tissue cells and neurilemma nuclei remaining in the connective-tissue strand, and to

this opinion Wolberg agrees. It is evident, therefore, that the process of regeneration is by no means accurately determined. But here again it is possible that conflicting statements may be explained. The method of regeneration may, perhaps, depend upon the exact stage of degeneration reached before it begins. If the product of degeneration is a simple band of connective tissue, it seems probable that the nerve-fibre will have to grow into it from a central origin, as in its original development in fetal life, unless the connective-tissue cells recently discovered are neuro-plastic cells, and have the power of producing new nerves, just as cells of periosteum may produce a new bone. If, however, when degeneration ceases, there remains a sheath of Schwann containing a granular protoplasmic mass, it is not at all improbable that that mass may be differentiated into an axis-cylinder and a medullary sheath, and joined to the old nerve-fibre, a process which has its analogy in the medullation of nerves in the embryonal state. If we admit, with Wolberg, that in some cases the axis-cylinder is not destroyed, the formation of new myelin is a rapid matter. That some such process as the one last described must occur in certain cases is certain, when the rapid recovery after minor injuries is considered, and when the results of nerve suture are taken into account; for in both these conditions the return of function occurs long before a new nerve-fibre, starting out from the old one, could have reached the periphery.

It is affirmed by Mayer that individual nerve fibres in normal nerves are constantly undergoing these processes of degeneration and regeneration, either because the necessary renewal of worn-out tissue takes place in this manner, or because slight injuries from pressure or over-strain are sufficient to start up degeneration in single fibres.

The process of degeneration in the nerves, consequent upon the destruction of the ganglion cells from which they arise (the so-called Wallerian degeneration) differs in no respect from that ensuing upon compression or division, excepting that in the latter case only the distal part of the divided nerve undergoes the patholog-

ical change, while in the former it is the entire nerve that is affected. And in the changes described in multiple neuritis the same progress of events and the same varieties of termination are observed.

When the cases of multiple neuritis are examined from a pathological standpoint several varieties may be distinguished. In some of the cases, and these are the most numerous, there is a parenchymatous inflammation in the nerve-fibre only visible to the microscope. At the outset of this inflammation the myelin sheath appears slightly swollen, is less homogeneous, and, from a difference of refractive power, is less translucent. It then becomes split up into segments of different length and form, the segmentation occurring preferably at the incisures of Schmitt, while the incisures at other parts disappear. Between these segments of myelin a finely granular protoplasm is seen, in which new nuclei are found. These nuclei probably are the cells of Rosenheim which have emigrated, although a few near the nucleus of the sheath of Schwann may be due to its division. In some fibres the axis-cylinder may still be preserved. In others it is broken at the same places as the myelin. At the next stage of the process the changes are more marked. The myelin is now reduced to a series of small globules surrounded everywhere by granular protoplasm, and in this protoplasm the nuclei are now very numerous. The axis-cylinder cannot be distinguished in the mass, as a rule, but occasionally a fine line is seen passing through the mass, which may be a remaining cylinder. The succeeding stage presents a different picture. While up to this time the size of the nerve-fibre has remained about normal and uniform, it is now seen to vary. At places the fibre is still wide and filled with a granular mass, at other places it is narrow, the mass having disappeared, leaving either a collapsed sheath or a sheath containing only nuclei here and there. In a few such narrow fibres there seems to be an axis-cylinder lying directly within the sheath of Schwann, and occasionally separated from it at various places by nuclei. But this appearance is rarely seen. As a rule, no trace of the axis-cylinder remains. As any single fibre may show at some places constrictions,

at others dilatations, the variation in its calibre is the most striking feature of this stage. In the terminal stage the calibre is uniform again, but is now everywhere reduced. The sheath of Schwann is empty, or contains only a little granular substance, and the nuclei are now less numerous than before. There is, in fact, only an atrophied tube with none of its original contents. These tubes, lying side by side, are folded and undulating, and appear like a strand of connective tissue.

These various stages of parenchymatous inflammation are to be seen in different fibres in the same specimen. Their appearance is identical with that observed in the course of degeneration of a nerve after compression, or after destruction of the spinal ganglion-cells. This has led such an accurate observer as Erb to advance the hypothesis that some slight changes in trophic cells in the spinal cord, not visible to the microscope, are present primarily, and that these changes are of a secondary nature.[1] But this cannot be admitted. Those who would explain the parenchymatous neuritis as secondary to changes in the cord, fail to explain why it should be limited, as it is, to the distal portions of the nerves, and not be continuous through the nerve-trunks and anterior nerve-roots into the anterior cells. They also offer no explanation for the cases in which the sensory, as well as the motor, nerves are affected, centrifugal degeneration of these nerves from spinal lesion being unknown. Strumpell, in meeting Erb's hypothesis, urges with reason that a parenchymatous neuritis has its parallel in other parenchymatous inflammations, and therefore does not need to be traced to any primary affection in trophic cells. He also mentions the existence of primary lateral sclerosis of the cord, similar in all respects to secondary degeneration of the pyramidal columns—yet now admitted to be a primary disease—as illustration of the fact that like changes are not always to be traced to one cause. It cannot but be admitted, therefore, that the first class of cases of neuritis must be considered as due to a primary parenchymatous inflammation in the nerve-fibres. This

[1] Erb: Neurol. Centralbl., 1883, p. 481.

form seems to be much more frequent in its occurrence than the first variety, and constitutes the lesion in the majority of the cases hitherto reported. The appearance in section is seen in Fig. 5, *b*. The appearances of single fibres are the same as those shown in Figs. 2 and 3.

A second class of cases presents a different appearance.

In this class the mere inspection shows the nerve to have been the seat of pathological changes, for it is either congested, swollen, and lacking in lustre, or it is yellow

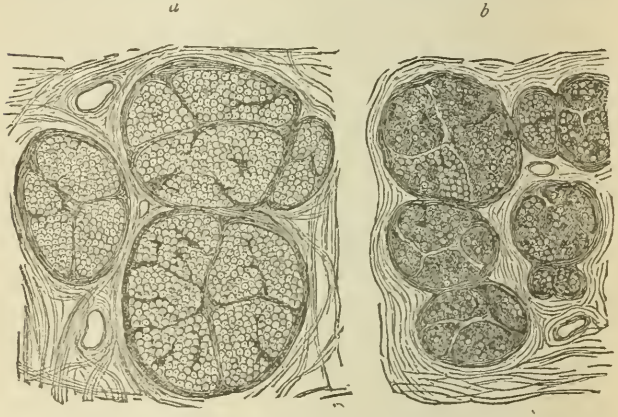

FIG. 5.—(After Joffroy.) *a*, Section through normal nerve; *b*, section through degenerated nerve in a case of multiple neuritis, bundles reduced in size, individual fibres degenerating, their place taken by débris.

and irregularly swelled by the accumulation of fat, or it is evidently reduced to a mere connective-tissue strand. Upon teasing the nerve it is at once clear, from its brittleness, that individual fibres are lacking in continuity and are changed in structure. And if it is examined under the microscope the exudation of serum and of lymphoid bodies, the great increase in the number of connective-tissue nuclei, the distended condition of the vessels, as well as the various appearances characteristic of nerve degeneration, are clearly seen. Here the inflammation is either originally an interstitial inflammation or, more probably, a diffuse one. It is possible that the degen-

erative processes in the nerves may have been due to the compression by the exuded products of inflammation within the nerve-sheath. In one or two cases where the patient died early in the disease, the nerve-fibres which lay near the vessels were affected to a greater degree than those lying deeper, and from this fact it was concluded that their degeneration was secondary. In other cases, however, all the fibres in a bundle are equally involved. Here it is probable that the inflammation was diffuse from the outset, parenchyma and interstitial tissue being affected simultaneously. The final result in either case is a diffuse inflammation. One marked feature in these cases is the large amount of fatty deposit found in the altered nerves. This is to be ascribed to the fact that myelin in undergoing degeneration breaks down into globules and undergoes a fatty change. It is itself allied to fat, and making up, as it does, a large part of the bulk of the fibre, it would be noticeable in the product of degeneration if it were not absorbed. In this form the absorption seems to be interfered with by the vascular condition, and hence the residual amount of fat is increased. The changes observed in the nerve-fibres are very similar to those seen in the first form of neuritis, and need not be described again.

It is to be noted that in both of these forms the pathological changes are always more intense in, and are occasionally limited to, the peripheral terminations of the nerves. The nerve-trunks may be slightly involved in their distal portions, but it is very rare to find any changes in them at their origin from the plexuses. And, as a rule, the spinal nerve-roots in cases of multiple neuritis are normal. In making autopsies upon such cases this fact is to be remembered, and the nerves are to be removed down to their finer branches in the muscles and fascia.

One additional pathological form must be mentioned, since it has been described by such a careful observer as Gombault.[1] It is the so-called segmental periaxillary neuritis. In toxic neuritis from lead-poisoning, Gombault found that the degenerative process was not uniform in

[1] Gombault: Archives de Phys., 1873, p. 592; also Arch. de Neurol., i., 1.

the entire length of a nerve-fibre. On the contrary, entirely normal segments alternated with the degenerated segments in the nerve. Mayer has noticed a somewhat similar condition (Fig. 3, *e*). The changes already described take place in the myelin sheath of the affected segment, even to its entire absorption, leaving the axis-cylinder in the sheath of Schwann. But as the adjacent segments are not involved, regeneration is thought to be more easily accomplished. Pitres and Vaillard [1] have noticed a somewhat similar condition in the neuritis occurring after diphtheria, although in their case the axis-cylinder as well as the myelin sheath was totally destroyed in many segments.

While it is, of course, impossible to describe definitely the process which goes on to complete regeneration of nerves in multiple neuritis, there is no reason to suppose that it differs in any way from that observed in experimental lesions.

The time required for the completion of the process will depend upon the severity and extent of the degeneration. When that is slight the recovery may be rapid, cases having been reported where a total cure took place in two months. As a rule, however, it is a slow process. The large majority of the cases on record required over six months for the complete regeneration, and in not a few cases from ten to sixteen months elapsed before the condition of the nerves was proved to be normal by the total disappearance of all symptoms.

A degeneration of the muscles proceeds hand in hand with that of the nerves. This is usually in the form of an atrophy, with fatty degeneration and an increase of nuclei. The muscle-fibres become thin, homogeneous, are easily split up, contain fatty granules, and finally, in place of the atrophic muscle, new connective tissue is found. When no regeneration takes place, a mass of connective tissue replaces the muscle, containing a few muscular elements.

With the end of 1883 we may consider the third stage

[1] Pitres et Vaillard : De la Névrite segmentaire, Arch. de Neurol., xi., 337.

of pathological discovery in the history of multiple neuritis as terminating. At that date the symptoms of the disease had been recognized as constituting a distinct clinical picture ; hypothetical lesions in the spinal cord had been abandoned, and the exact pathology of the disease had been ascertained. Since 1883 the final period in the development of knowledge of the affection has been in progress. About one hundred cases, of varying degrees of severity, have been observed (see Bibliography). The lesions described have been confirmed in all their details. The symptoms arising in the course of the disease have been subjected to careful analysis, together with their varied modes of combination. Certain diseases hitherto considered of spinal origin are found to be due to peripheral disease. Thus it has been discovered that a form of ataxia may occur from multiple neuritis of alcoholic or arsenical poisoning which closely resembles and was formerly confounded with tabes ; also, that some cases supposed to be anterior poliomyelitis must now be differently named. And as physicians review their records they find that former diagnoses require revision in the light of new discoveries, and that greater circumspection is to be exercised in differentiating spinal from peripheral affections. The grounds for such differential diagnosis will require attention in the next lecture.

And, what is perhaps of greater importance, the fact has been elicited that some combinations of symptoms, formerly supposed to be without a pathological basis, some of the so-called peripheral neuroses, really belong to this class of diseases. At present we have only time to allude to one or two forms of peripheral neuroses which must be removed from that unsatisfactory category of disease and be considered as peripheral neuritis.

And the first of these is the affection termed numb-fingers. This was first described by J. J. Putnam, of Boston, but met with instant recognition from neurologists and from general practitioners all over the world. It is a disease chiefly seen in women between the ages of forty and sixty, usually associated with dyspeptic or

uterine symptoms, but entirely independent of them. It begins as a tingling sensation in the ends of the fingers, felt at night, and sufficiently annoying to keep the patient awake. It then extends to the entire fingers, and may invade the hand, and is felt by day as well as by night. The fingers are so numb that all finer acts become impossible; the patient can no longer sew or knit, cannot be sure of holding anything securely, and finds herself unable to perform any delicate movement. Sometimes a slight degree of anæsthesia and analgesia can be discovered by ordinary tests, but often the disturbance of sensation is purely subjective. There is rarely any inco-ordination, and paresis is usually wanting. It may develop in the feet as well as in the hands, making walking more or less disagreeable, and adding to the discomfort of the patient. The affection is of indefinite duration; often subsiding quickly under treatment, sometimes baffling all attempts to arrest it. For a time it was considered a purely functional affection, then a spinal-cord disease;[1] but now, in the light of the parallelism between the symptoms mentioned and those which are characteristic of multiple neuritis, we cannot but consider it a slight form of this disease.

The second of the peripheral neuroses which must be referred to peripheral neuritis is intermittent paralysis. Cases of sudden paraplegia, lasting a few hours and passing off as rapidly as it appeared, have been observed too frequently to admit of any doubt. Westphal has described such a case in which no cause could be found. Gibney reported some cases presumably due to malaria. Others have thought the disease of functional character, either central or peripheral. But in the light of recent observations upon infectious cases of multiple neuritis it becomes evident that these sudden, transient paraplegiæ find their adequate explanation in such an affection.

And, lastly, there are numerous cases of indefinite nervous symptoms, pain of various kinds, formication, and odd sensations grouped under the indefinite term numb-

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[1] Ormerod, St. Bart. Hosp. Rep., 1883; Sandby, Lancet, 1884; W. Sinkler. Phil. Med. Times, 1884.

ness, flashes of cold and heat accompanied by actual changes in the temperature of the part, or only by apparent vascular irregularities, slight spasms or tremors, functional weakness, with sense of fatigue not reaching the grade of paresis, and many equally obscure manifestations of disturbed function in various parts of the body, which receive their best explanation in the theory of multiple neuritis.[1]

And since it was one of the objects of the founder of this lectureship to determine the true nature of such peripheral neuroses, it is with the greater interest that we examine the disease to which they must be assigned.

In the present stage of progress in the history of neuritis much attention is being given to the etiology of the disease. Cases which, from their causation, were formerly separated are now found to be closely allied in their pathology. Thus the forms of paralysis occurring after the ingestion of various poisons, such as arsenic, lead, bisulphide of carbon, and alcohol, are known to be due to a common pathological change. The various kinds of sensory and motor disturbance occurring as complications of the acute diseases, diphtheria, variola, typhoid and typhus fevers, and severe malarial fever, are traced to a lesion in the peripheral nerves. Tuberculosis is known to predispose to neuritis, and many cases formerly supposed, without question, to be produced by central or meningeal affections of a tubercular character are now assigned to a peripheral cause. It is a question whether syphilis will cause a simple degenerative neuritis, but syphilitic affections of the nerves are easily recognized and well known. Nor can the nervous system escape the action of those micro-organisms which are now recognized as the constant cause of many diseases. There is an epidemic form of multiple neuritis, fortunately not prevalent in this country, but occasionally imported here in the form of sporadic cases, known as kakke, or beriberi, the bacillus of which has recently been discovered and cultivated. And, lastly, there is a class of cases, of supposed spontaneous origin, in which cold or over-exertion are assigned as causes,

[1] Dana, Acroneuroses, N. Y. Med. Rec., July, 1885.

but which need further investigation in regard to their etiology.

It is evident from this array of causes that several conditions formerly separated from one another because of the different circumstances of their occurrence are really forms of the same disease. But while they may be brought together upon a pathological basis, and while all have many symptoms in common, each of the forms of neuritis presents certain distinct features. Without dwelling too long upon any single variety, it will be necessary to review the different classes of cases in order to obtain a clinical picture of each. And such a review, together with the analysis of the symptoms of multiple neuritis, the discussion of its diagnosis from other diseases, its prognosis, and the treatment, will occupy us at the next lecture.

Lecture II.

Mr. President, Ladies and Gentlemen of the Pathological Society:

In the last lecture we studied the history of multiple neuritis, and incidentally obtained some clinical pictures of various forms of this disease. We also discussed its pathology and its etiology. This evening we must look more carefully at the symptoms presenting themselves in the different varieties of the affection.

An etiological classification of the cases of multiple neuritis appears to be the one most useful to the clinical observer, and as the forms vary considerably in their symptoms, we shall perhaps arrive at a more definite knowledge of the disease if we consider each of the classes separately. It is possible to distinguish—

1. Toxic cases, due to poisoning by alcohol, arsenic, lead, and bisulphide of carbon.

2. Infectious cases, due to the direct action upon the nervous system of the infectious agents, producing diphtheria, variola, typhoid and typhus fevers, severe malarial fever, and tuberculosis; to which must be added the agent causing the epidemic form of neuritis known as kakke or beriberi.

3. Spontaneous cases, due to uncertain causes, among which cold, and exposure to damp and wet, and to overexertion, may find a place.

I. Toxic Cases.—(*a*) *Multiple neuritis, due to poisoning by alcohol.* While alcoholic paralysis has been universally recognized for many years, it is only within a short time that the symptoms and pathology have been brought into a logical connection. It is to Moeli,[1] of Berlin, and Dreschfeld,[2] of Manchester, England, that we owe the most important recent contributions to the knowledge of this form of neuritis, though the work of Henry Hun,[3] of Albany, and of Bernhardt,[4] of Berlin, cannot be passed over without recognition.

[1] Moeli: Charité Annalen, 1884.
[2] Brain, Nos. 26 and 32.
[3] Amer. Jour. Med. Sci., April, 1885.
[4] Zeitsch. f. Klin. Med., 1886.

It will be remembered that among the first cases of multiple neuritis in which an autopsy was made, viz., those of Lancereaux,[1] the disease was traced to alcoholism. Lancereaux deserves the credit of demonstrating the pathological basis of alcoholic paralysis, for the descriptions of its symptoms previously given by Jackson and Huss were not accompanied by post-mortem records. But Lancereaux's observations were for many years overlooked by physicians outside of France; and in England, where the disease is well known, it was ascribed, as lately as in 1883, to a spinal lesion.[2] But in 1884 Dreschfeld combated this theory and called attention to its true pathology, showing that cases formerly supposed to be spinal paralysis were really due to an affection of the peripheral nerves.

All observers have emphasized the fact, already noted by Jackson, that the disease is especially frequent among females. Males are not, of course, exempt from alcoholic paralysis, but in them the poison seems more liable to manifest itself by acute cerebral symptoms than by those of disease of the peripheral nerves. It is especially frequent among those persons in the higher classes whose nervous organism is highly developed, and who lead a comparatively inactive life. It seems not at all improbable that sedentary habits predispose an alcoholic drinker to this disease, and hence active workers, male or female, though taking an equally large amount of liquor as the luxurious drinker, escape. All alcoholic drinks are not equally prone to produce paralysis. It is the spirituous liquors—brandy, whiskey, gin, and rum, and the liqueurs, absinthe, vermouth, etc.—which are dangerous. And it is only after these drinks have been consumed in large amounts and for a considerable length of time that neuritis develops. Its onset, though often apparently very sudden, is usually gradual. For months the patient has suffered from chronic gastritis, insomnia, general neuralgic pains, or severe pains in the joints or limbs, and from tremor and a certain feebleness in movement, when all at once her legs give way beneath her, and after the

[1] Gaz. Hebdom., 1881.

[2] Wilks: Dis. of Nerv. System, p. 272.

sudden fall she finds herself unable to rise. Thus a patient of my own, after a year of such premonitory symptoms, was seized with paralysis quite unexpectedly when getting out of bed in the night. This paralysis soon becomes complete in the feet and legs below the knees, and may advance up the thigh. It next attacks the hands and forearms, and while in all extremities it is often greater in the extensors than in the flexors, in some cases both groups of muscles become entirely helpless. This has been the case in three patients under my care. The paralyzed muscles are flabby and soon become atrophied, they have no excitability to mechanical irritation, and the tendon reflexes are lost. They fail to react to a faradic current in the majority of cases, though occasionally a very strong current may produce a response. When galvanism is applied the reaction of degeneration is found to be present. No stimulus can be given to the muscle by sending a current through its nerve, and the positive pole produces more marked contractions with an equal current than the negative pole when placed on the muscle, and then it is only a slow or vermiform movement, not the quick jerk of health. It is also found that strong galvanic currents have to be used to produce any contraction at all. The paralysis of the muscles may advance rapidly in severe cases, involving the motor cranial nerves, the muscles of the trunk, and lastly the diaphragm, thus causing death. More frequently, however, it is arrested when only the distal parts of the extremities are involved, and then it gradually subsides until recovery is complete. The position assumed by the paralyzed limbs has been thought to be almost characteristic. There is dropped wrist, quite similar to that seen in lead-palsy, and also dropped foot, due to the falling forward of the foot from its own weight, since the anterior tibial muscles are weak. This deformity is increased by the fact that the patients lie in a recumbent posture with the feet extended; and when the flexors of the toes are but slightly affected, as sometimes is the case, their unopposed contraction serves to exaggerate the malposition. If there is entire paraplegia the legs and thighs may both be extremely flexed, so that the

heels touch the buttocks, but this is exceptional. While the dropped wrist is the usual deformity of the hands, cases are recorded in which the paralysis was limited to single muscles and to muscles supplied by single nerves. Thus Lilienfeld describes a weakness of the extensors of the thumb, fourth and fifth fingers, and Leudet mentions a paralysis of the ulnar nerve. In one case of my own the *main en griffe* was the deformity noticed, indicating a weakness of the interossei only. Such cases as those of Hun,[1] where a facial paralysis was seen, or of Lilienfeld,[2] where double abduceus paralysis occurred, are certainly rare, and still more so are those in which irregular, rapid pulse has been ascribed to an affection of the pneumogastric.

To the physician these motor symptoms, and the œdema, occasional lividity, profuse sweating, and glossy skin, so often associated with them, are very noticeable. But the patient suffers far more from the disturbances of sensation. In the description of Jackson the pains were graphically portrayed. They are the cause of terrible agony, sufficient to produce insomnia, and wearing seriously upon the endurance of the sufferer.

In addition to pain, hyperæsthesia is not infrequently observed. It is usually quite extensive in the legs, though in cases of poisoning by absinthe it has been limited to the soles of the feet. The muscles, as well as the skin, are sensitive to handling and to pressure, and marked tenderness in the course of the nerves is always elicited by examination. In one of my cases, soon after the onset, the patient could not bear to be touched or moved, though perfectly unable to help herself. Charcot goes so far as to say that muscular sensitiveness associated with flaccid paralysis is pathognomonic of alcoholism.

Paræsthesiæ are always complained of. Numbness, tingling, and formication are frequent. In one of my patients the sensation was as if heavy bracelets were around the wrists, and as if very tight drawers were on the legs. At other times she felt as if the limbs were

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[1] Hun : Loc. cit.

[2] Lilienfeld : Berl. Gesellsch. f. Psych. und Nervenkrankheiten, July 15, 1885; see also Richard Schultz, Neurol. Centralbl., Nos. 19, 20, 21, 1885.

swollen and as if the skin were about to burst. Such sensations may cease as the case increases in severity, and give place to a total lack of sensation in the parts. They return, however, with advancing recovery, and are among the last symptoms to disappear.

Abolition of tactile sense, and to some degree of muscular sense, is the rule after the paralysis is developed. Temperature-sense and the perception of pain are never wholly lost, but may be delayed in transmission. The anæsthesia may be limited to irregular areas, and may be only in the cutaneous distribution of one nerve, but is usually found over the entire distal part of the paralyzed limb. Usually the cutaneous reflexes are preserved. The loss of muscular sense is, in some cases, so marked a symptom, and one of such early occurrence, that Dreschfeld distinguishes a class of cases which he terms ataxic rather than paralytic. And this distinction is perfectly justifiable, for in many cases it is the inco-ordination which attracts the attention of both the patient and the physician. It is this class of alcoholic cases which may be mistaken for locomotor ataxia, and which have been named by French writers pseudo-tabes alcoholique.[1] But ataxia is not exclusively limited to this class of cases. It may be present in some degree in cases of paralysis, and during recovery from paralysis the deficiency in co-ordinating power may become evident, and appear to retard the progress of the case. Nor are the cases of ataxia, on the other hand, free from paralysis.

And this fact is proven by the observations of Westphal[2] and Charcot. For, by contrasting the walk of a true ataxic patient with that of an ataxic alcoholic patient, they have each discovered several points of difference. The ataxic patient throws the foot forward with undue violence, the toe lifted high in air, and brings first the heel down forcibly and then the entire foot. The alcoholic, however, has some weakness in the muscles of extension and cannot raise the toe. He therefore lifts the

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[1] Dejerine: Arch. de Phys., 1884.

[2] Westphal: Ueber eine bei chronischen Alcoholisten beobachtete Form von Gehstörungen, Charité Annalen, 1879. Charcot: Leçons, Progrès Médicale, 1886.

foot high in order to step over the hanging toe and not to trip on it, but the motion is made without undue force. He then throws the foot forward, in order to throw the toes up and get them out of the way as he brings the foot down to the floor. The motion is awkward and has an appearance of one stepping over high obstacles, but it is a voluntary attempt to remedy a deficient power—not the involuntary awkwardness of a man unable to manage strong muscles. There may be in both patients some tottering, and swaying when standing with the eyes closed, the so-called Romberg symptom is common to both locomotor ataxia and alcoholic neuritis. Dejerine describes the following case, in which the symptoms resembled those of tabes :

Observation VIII.—Male, forty-two, a hard drinker, complained for four months of shooting pains in the legs, and noticed a gradually increasing difficulty in walking, which was worse in the dark. He had also found his feet swollen at times, and had some difficulty in micturition. Examination on admission to the hospital showed a high grade of ataxia in walking, Romberg's symptom, loss of tendon reflexes, anæsthesia almost total of entire body, considerable analgesia with retardation of sensations of pain, temperature-sense being preserved, and loss of muscular sense in the legs. The muscles were slightly feeble and a little diminished in size. It could hardly be said that there was paralysis or atrophy. The diagnosis made was locomotor ataxia of an unusual type. The onset was so rapid, the anæsthesia so extensive and severe, the paresis of the limbs, though slight, so evident, that it differed from a typical tabes. But no other diagnosis could be made from the symptoms. The patient died of chronic nephritis six weeks after admission. The autopsy showed the spinal cord to be normal. But in the cutaneous, and to a less degree in the intra-muscular branches of the nerves, as well as in the distal parts of the nerve-trunks, a high grade of parenchymatous neuritis was found. The nerve-tubes had lost their double contour, the myelin sheath was segmented, the protoplasm was increased, the nuclei multiplied. The nerve-trunks on the plexuses and the nerve-roots were normal.

The nerves of the legs were more deeply involved than those of the arms.[1]

The second case, in a female forty-nine years of age, presented almost identical symptoms and lesions.

The special senses are occasionally affected in cases of alcoholic paralysis. Amblyopia has been observed, and also defective vision from central scotoma. The field of color-vision is often contracted, even when sight is preserved. There may develop a true optic neuritis, evident to the ophthalmoscope, and this may go on to optic-nerve atrophy.[2] Inequality of the pupils is frequently seen, as is also a moderate contraction of the pupil. All these eye symptoms, occurring as they may in a case of the ataxic variety, make a differential diagnosis from locomotor ataxia difficult. The Argyle Robertson pupil (which contracts in accommodation but not to light) has not been seen in alcoholic cases, while it is an early symptom of tabes.

One feature of alcoholic paralysis remains to be noticed, viz., the cerebral symptoms. These are hardly ever wanting. There is at first the excitement rising to the degree of active delirium, with illusions and hallucinations of the various senses; there is the insomnia which so soon exhausts the patient if it is not remedied; there is the loss of memory, especially of recent occurrences; and the lack of power of attention or concentration which prevents intelligent conversation. The indifference to bodily wants may be so great as to lead to uncleanliness, and since paralysis of the sphincter is the rare exception, incontinence is usually to be ascribed to the mental state. It is useless to attempt to get any reliable history of their illness from these patients. Their statements are unintelligible or unreliable. And here it may be well to notice a symptom first remarked by Strümpell.[3] These patients will relate occurrences as having happened recently with much elaboration of detail, when as a fact the story is entirely a product of their imagination. Thus

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[1] Dejerine: Arch. de Phys. Normale, 1884. For other ataxic cases, see Dreschfeld. loc. cit.

[2] Brissaud: Des paralysies toxiques, p. 31. Paris, 1886.

[3] Strümpell: Arch. f. Psych., 1883, xiv., 339.

one patient of my own, who had been confined to bed for many days, told me one afternoon that she had been out to see an eminent gynecologist during the morning; had gone to his office and waited for him several hours; had seen other patients there, and finally had been told by the doctor's brother that he would not return in time to see her, so she had come home again. And this was all related in apparent good faith, so that I have no doubt that she believed that what she said had occurred. With the possibility of such delusions in view, it is evident that the statements of these patients cannot be accepted regarding anything, especially as to their own history.

One patient, who was admitted to Bellevue Hospital during my service there, told me a different history of her case every day for a week, and it was only by interviewing her friends that the correct account was obtained.

The course of alcoholic neuritis is quite uniform. After a sudden onset the symptoms rapidly advance to a high degree, which is reached in a week or two from the beginning of the paralysis or ataxia. Then they may increase further, and cause death by respiratory paralysis. Usually they remain stationary for a time, and then gradually subside, the entire duration being from two months to a year. Individual muscles regain their power, tone, firmness, and electrical reaction slowly, and during recovery the tingling and numbness in hands and feet may be severe. In a few cases the muscles become contractured, and permanent deformities, only to be overcome by long-continued massage or by operative measures, develop. When the fact is considered that those who recover rapidly rarely fail to resort again at once to the use of stimulants, and thus expose themselves to the danger of a relapse, the ultimate fate of the chronic cases is hardly more serious than that of those who get well.

As examples of the paralytic form the following cases are cited:

Observation IX.—A baker, aged forty-seven, phthisical, a moderate drinker, had suffered for several years from numbness of the hands and legs; and for a year be-

fore the acute invasion of his illness from a slight weakness of the legs and uncertainty of motion in walking. This weakness became much worse during the spring of 1881, so that all summer he was confined to his room, though he managed to walk a little. In November, after an uneasy night, during which he was delirious, it was found that he could not move his legs at all, and that his arms were powerless. He was brought to the hospital November 25th, when a total paralysis, with atrophy and loss of tendon reflexes, was discovered in legs and arms, as well as slight disturbances of sensibility in the legs, consisting of a delayed sensation of pain. His mental condition was a weak one, but he answered questions rationally. The face was in no way affected. No involuntary evacuations. Electric examination showed a great diminution, and in many muscles total loss, of faradic excitability when the current was applied both to nerve and to muscle. With the galvanic current few contractions could be elicited by exciting the nerves, and by exciting the muscles the slow contractions characteristic of RD,[1] as well as the predominance of AnCC over KCC were present. The mechanical excitability of the muscles was not impaired. There were no pains. During the following three months there was little change in the condition, except that his mind was not clear, and that he told extraordinary stories of having been out walking, of having made purchases, etc., when, in fact, he could not move from the bed.

His pulse became more rapid during this time, averaging 120 per minute. In February, 1882, the legs had become œdematous, and there was marked analgesia in them, except on the soles of the feet, which were hyperalgesic; sensations of touch were, however, readily perceived. Though he had a sanguineous dysentery, there was no incontinence. On the 12th there developed suddenly great difficulty in respiration, due to suspension of all diaphragmatic breathing, and the next day he died. An ophthalmoscopic examination on the last day showed

[1] RD = reaction of degeneration; AnCC = anode closure contraction; KCC = cathode closure contraction.

an atrophy of both optic nerves on the outer side of the eyes.

Autopsy: Brain and cord normal. No macroscopic changes in the nerves. A microscopic examination, however, showed the presence of a very high degree of degeneration and degenerative atrophy in the nerve-fibres. The myelin had divided in large and small drops, containing fat-granules; the degenerated mass within the Schwann sheath, consisting of myelin and axis-cylinder, was seen in various stages of absorption, and in spots the empty Schwann sheath alone remained. No inflammatory exudation was present, and the endoneurium and perineurium appeared to be normal. The muscles showed evidence of degenerative atrophy. The anterior nerve-roots were normal.[1]

The lesion here resembled exactly that in Joffroy's case, already cited. It was similar in the following case:

Observation X.—Female, who had recently indulged in alcoholic excesses and had suffered from gastritis and delirium tremens; complained for some days of pain in her legs and numbness in hands and forearms. When seen by Dr. Webber the pains in the limbs were severe, and she could not turn in bed without help, her knees were drawn up, and attempts to straighten them caused pain. There was loss of power in extensors of the wrist and hands, and the fingers could not be shut perfectly. There was general tenderness over the muscles of the extremities, and pressure along the nerves was very painful. Her throat was sore, but she did not have diphtheria. She spoke in a whisper and with difficulty. Three days later anæsthesia of the feet and hands appeared, and the tendon reflexes were lost. The pain became so severe that morphine was given to control it. The paralysis became more complete, the anæsthesia increased, incontinence of urine and fæces developed, she appeared to be very feeble, and respiration became wholly thoracic. Seventeen days after admission to the hospital she died.

Autopsy showed brain and cord normal. The chief nerve-trunks of the extremities, and the phrenic and

[1] Strümpell: Arch. f. Psych., 1883, xiv., 2.

pneumogastric, were in a condition of degeneration, the distal ends being affected to a greater degree than the central portions. "The simplest change found was in the medullary sheath at Ranvier's constrictions, without break in the axis-cylinder, and without increase of nuclei." In some nerves extensive degeneration was found, the medullary sheath being broken up into fatty fragments, the axis-cylinder discontinuous or destroyed, and the number of nuclei increased. In the fibres which were most altered was found an increase of nuclei; and apparently small masses of protoplasm, not nuclei, were tinted in the midst of the granular débris. Nearer the root of the nerves axis-cylinders were more numerous. In the sciatic the sections from the highest part of the nerve, which had been preserved, were still diseased in a small proportion of their fibres.[1]

A very similar case, with the same changes in the nerves, is recorded by Müller.[2]

The following case, under my own care, terminated favorably :

Observation XI.—Male, aged thirty-six, a hard drinker, after an attack of gastritis became paralyzed suddenly in both legs. A few days after this, both hands became useless, so that he was completely helpless. His wife says that his memory is quite gone. On admission to Bellevue Hospital, September, 1880, seven weeks after the onset, he complained of pain in all four extremities, and of tenderness in the muscles and joints when these were handled. The upper extremities were almost totally paralyzed below the elbows, the only motion possible being a slight flexion of the fingers. There was wrist-drop on both sides. The lower extremities were totally paralyzed below the knee, and the feet hung down motionless. The muscles of the thighs were also affected, for although he could pull his legs up in bed, it was with great difficulty that they could be straightened out. All the paralyzed muscles were atrophied, and the faradic reaction was lost in the extensors and greatly diminished in the flexors. The galvanic reaction was

[1] S. G. Webber: Arch. of Med., 1884, xii., 33–49.
[2] F. C. Müller: Arch. f. Psych., 1883, xiv., 3.

not tested by me from lack of apparatus. Tactile sense was considerably impaired, but the senses of pain and temperature and muscular sense seemed to be natural. The knee-jerks were lost. When an attempt was made to have him stand, the feet were pushed out and the knees doubled under him at once. Fibrillary twitchings were very noticeable in the atrophied muscles, and a marked tremor of the tongue was seen. The facial and ocular muscles were not affected, and the special senses were normal. There was no incontinence of urine and no symptom of thoracic or visceral disease. His mind was much impaired. He talked in a rambling manner, laughed much, could not fix his attention, and his memory was so poor that no reliance could be put in his statements. Under treatment by iodide of potash, and complete cutting off of all stimulants, he began at once to improve. In two months from the time of admission he was able to walk, and one month later he was discharged perfectly well.

These cases are sufficient to present a clinical picture of the symptoms and course of the forms of alcoholic multiple neuritis. The differential diagnosis, elements of prognosis, and means of treatment will be considered when other classes of the disease have been studied.

(*b*) *Multiple neuritis due to poisoning by arsenic.*[1]— It has long been known that an occasional result of arsenical poisoning is the development of paralysis, but it is only within the past four years that the fact has been determined that the symptoms in these cases are due to an affection of the peripheral nerves. Had the fact of the peripheral origin of alcoholic paralysis not been already proven, it is probable that the theory so long in vogue, that arsenical nervous symptoms were due to spinal lesions, would still prevail. But there is such a similarity between the two sets of cases, that it is impossible to ascribe them to other than the same patho-

[1] Scolozouboff: Arch. de Phys., 1884, p. 323; Imbert Gourbeyer: Des Suites de l'empoisonnement arsenical, Paris, 1881; Seeligmüller: Deut. Med. Woch., 1881, p. 185; Lancereaux: Gaz. Hebdom., 1881, p. 719; Jaeschke: Thèse Breslau, 1882, Ueber Lähmung nach acuterar senik Vergiftung; Levin; Schmidt's Jahrbuch, Bd. 165, p. 239; Da Costa: Phil. Med. Times, 1881, p. 385; C. L. Dana: Brain, January, 1887; Brissaud: Paralysies toxiques, Paris, 1886.

logical condition. Autopsies in support of the position that the peripheral nerves are involved are few in number, but in several cases the lesion has been found. The observations on record of spinal lesions are, it is true, more numerous; but when these are compared it is found that different lesions have been discovered in different cases, so that there is no single pathological change in the spinal cord which is constantly produced by arsenic. Further, some of the changes described in experimental cases in animals (viz., vacuolization of cells) are due to imperfect hardening of the specimens. It must, therefore, be admitted that multiple neuritis may be due to arsenical poisoning.

The changes produced in the nerves are so exactly similar to those already described, that there is no need of a recital of the pathological process.

The nervous symptoms produced by arsenic have been thought to vary somewhat, according as the ingestion of the poison has been a sudden or a gradual one. Brissaud claims that if there is slow poisoning, as, for example, by the long-continued use of Fowler's solution, paralysis is rather the exception, and is not severe, it is diffuse and transient; while other symptoms, such as gastro-enteritis, trembling, delirium, and aphasia attract the chief notice. If there is acute poisoning from an overdose of arsenic, he holds, on the other hand, that paralysis ensues either during the period of active symptoms of poisoning, or soon after. The observations of other equally careful authors do not entirely support this view of Brissaud; for in two cases of Dana, one of acute, the other of chronic poisoning, very similar symptoms of paralysis and ataxia developed.

The description which has been given of alcoholic paralysis might almost be repeated for arsenical paralysis. There is the same limitation of the affection to the muscles of the distal parts of the extremities, the extensors being chiefly affected, and the weak muscles are flaccid, soft, and atrophied. There is a partial reaction of degeneration. The tendon reflexes are abolished; skin reflexes are preserved. There is often a marked tremor. The paralysis may begin either in the feet or

in the hands, is usually bilateral, but has been in four cases of the hemiplegic type. The same dropped wrist and dropped foot are seen as in alcoholic cases. Disturbances of sensibility are prominent symptoms; burning, tearing, shooting pains; formication, tingling, muscular, and arthritic pains and tenderness are associated with hyperæsthesia, and this may be followed by irregular patches of anæsthesia. The muscular sense is usually impaired, and so much so in some cases that an attempt has been made to establish a distinct class of cases as arsenical ataxia, or pseudo-tabes arsenicale. In these cases the inco-ordination of hands and feet, Romberg's symptom, and an awkward gait are very noticeable, so that tabes may be suspected until the history makes the causation evident. In a case of Dana's the patient could not tell the position of his limbs or distinguish between weights differing one to forty. In Scolozouboff's case the walk resembled that described by Westphal in alcoholic tabes, and was easily distinguished from a real ataxic gait. Seeligmüller and Dana, however, affirm that the gait in their cases was like that of locomotor ataxia. Occasionally œdema of the extremities, cyanosis, unusual sweating, and extensive desquamation indicate an implication of vaso-motor and trophic nerve-fibres. In the most severe cases contractures in a flexed position developed in the paralyzed limbs. The sphincters are never involved. The duration of arsenical paralysis is somewhat greater than that of ataxia. Either condition may last several months, but occasionally the recovery is complete in a few weeks. Frequently, after the power has returned, the patient suffers for months from numbness and tingling in the extremities, which is sufficient, as in a case under my own observation, to interfere with the finer motions, to disturb the sleep and cause constant discomfort. According to Gerhardt's statement (cited by Dana), ninety-seven per cent. recover wholly.

(*c*) *Multiple neuritis due to poisoning by lead.*—It is not my purpose to enter upon any description of the various forms of lead palsy, which are familiar to every practitioner. Nor is this the proper place for a discussion regarding the various theories of the pathology of

the disease. It is only necessary to call attention to the fact that there are now on record a number of autopsies in cases of lead paralysis in which the lesion has been found in the peripheral nerves. In a recently published case of Schultze[1] there was found a very marked atrophy and disappearance of nerve-fibres in the trunk of the musculo-spiral nerve, below the point where the branch to the supinator longus was given off. This decreased in intensity centrally, so that at the brachial plexus no anomaly was found. It increased in intensity toward the termination of the nerve in the muscles. The spinal cord was normal. This is simply a type of a number of recently published cases. On the other hand, there are numerous cases of this disease in which decided spinal lesions have been found—so numerous that many writers always ascribe the disease to destruction of certain groups of cells in the anterior cornua of the spinal cord. It must be admitted, therefore, that in lead we have a poison which, under certain circumstances, affects the spinal cord, and under other circumstances produces neuritis. It may be claimed that the same is true of alcohol and arsenic, and in showing that these poisons can cause neuritis there is no intention of defending the position that they never affect the central nervous system. But it is important to notice that the central organs are rarely involved. The limitation of the neuritis in lead palsy to the motor nerves makes the clinical picture in some cases resemble closely that of spinal disease. In illustration of this the following case is cited:

Observation XII.—A male, aged thirty-six, by occupation a dyer, had suffered from numerous attacks of lead colic, followed by general muscular weakness, which on one or two occasions was so great as to be a general paralysis rather than a simple weakness, and had finally developed a paralysis of the extensors of arms and legs, when admitted to the hospital, where he spent the last three years of his life. During this time his paralysis varied somewhat in degree, but was never entirely recovered from.

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[1] F. Schultze: Ueber Bleilähmung, Arch. f. Psych., xvi., p. 791.

The muscles affected were the extensors of arms and legs. These were much atrophied, and showed a loss of faradic reaction, and a characteristic change in the galvanic reaction, the anode producing stronger contractions than the cathode, and all contractions being slow. There was also a considerable degree of œdema in the extremities, especially in the legs. Spasms in the unparalyzed flexors occurred on one occasion for one day. The patient suffered also from the symptoms of cardiac disease and phthisis, to which he finally succumbed.

The autopsy showed no changes in the brain or spinal cord. The muscular branches of the peripheral nerves, although presenting no macroscopic changes, were found in a state of extreme degeneration when examined microscopically. Side by side with normal fibres were to be seen fibres, unstained by osmic acid, wholly destitute of myelin sheath. There was no fatty degeneration. Between the fibres a thick, finely fibrillary connective tissue, with small cells, was found. Transverse sections of the nerve-trunks showed a large number of small fibres scattered among the normal fibres, but not collected into bundles, and it was often difficult to distinguish naked axis-cylinders from connective-tissue fibres. The lesion was an increase in the connective tissue of the endoneurium with simple atrophy of the nerve-fibres of different grades.[1]

But in a certain proportion of the cases of lead palsy there are marked sensory disturbances, consisting of severe pains, and anæsthesia, with numbness. In these it is possible, especially if tenderness along the nerves and in the muscles is present, to make the diagnosis of multiple neuritis. The history of the following case, under my care, coincides so closely with that of other toxic cases of neuritis that the diagnosis could not be mistaken.

Observation XIII.—Male, aged thirty-eight, after suffering from an attack of lead colic, began to feel numbness and pain in his legs and feet, which soon extended to his hands and forearms. This steadily increased for two weeks, and to it was added paralysis of the extensors of

[1] Eisenlohr: Deut. Arch. f. klin. Med., 1880, xxvi., p. 543.

both hands and both feet, so that at the end of that time he was unable to use his hands or to stand. With the paralysis there was a rapid atrophy of the muscles, and a decline in the faradic excitability. The muscles became more and more tender, and the spontaneous pains gradually increased until it was necessary to use opium freely to quiet them. By the end of a month a well-marked anæsthesia had developed below the knees, and it was noticed that the atrophied muscles were in a constant tremor. There was wrist-drop and foot-drop on both sides, and all reaction to faradism ceased. It required a very strong galvanic current to produce contraction, and AnCC was greater that KCC. The knee-jerk was preserved. The muscular sense was impaired. For five months his condition remained stationary, in spite of treatment by strychnia, iodide of potash, massage, and electricity. Then a gradual improvement set in, the pains became less severe, sensation returned, the muscles regained their contour and strength, and finally the electric contractility returned to the normal standard. About a year from the onset of the symptoms, the recovery was complete.

(*d*) *Multiple neuritis from poisoning by sulphide of carbon*[1] *and from illuminating gas* has been suspected, and the similarity of the symptoms in such cases to those already described is quite remarkable. But as there are no autopsies to substantiate the theory, this cause is merely mentioned.

II. Multiple Neuritis consequent upon Infectious Diseases.—There are a number of infectious diseases which are especially liable to be followed by the development of nervous symptoms. These are diphtheria, variola, typhoid, typhus, and scarlet fever, malarial fever, and tuberculosis. The nervous affection usually appears shortly after the period of convalescence in the acute fever. The disease may consist of a simple paralysis of the muscles in the region of distribution of a single nerve. It may affect several nerves on both sides of the body symmetrically. It may even paralyze two or more

[1] Ross: Medical Chronicle, January, 1887.

limbs. It occasionally produces sensory as well as motor symptoms in a single nerve-trunk. It may even cause a general sensory and motor paralysis of as widespread and complex a kind as that produced by chronic alcoholism. Sometimes the symptoms are chiefly of a sensory kind, and consist exclusively of pains, numbness, and anæsthesia, or hyperæsthesia, in the legs, or of a loss of the muscular sense, in which case a true ataxia is the most noticeable symptom. Thus it is evident that the poison of an infectious disease may act as powerfully upon the nervous system as any other form of poison known.

It is only since the clinical pictures presented by multiple neuritis have been recognized that a question has arisen as to the part of the nervous system affected in these cases. Formerly all such phenomena were referred to central lesions; and undoubtedly in many cases this was justified, since autopsies are not wanting to prove that anterior poliomyelitis, diffuse myelitis, and hemorrhages into the cord and brain may follow the acute fevers. There are, however, many conditions which do not correspond to the types of disease produced by central lesions and which recover with a rapidity impossible were the brain or spinal cord involved. It was these cases which raised the question of some possible affection of the peripheral nerves. And careful investigation has been rewarded by the actual discovery of lesions in them.[1]

(*a*) Diphtheritic paralysis is probably more common than any other of these forms of neuritis. As is well known, it is usually the soft palate to which the paralysis is limited, and as a result difficulty in swallowing and in speech are the most prominent symptoms. Bernhardt has found[2] that in the large majority of cases of this kind there is a loss of the patella tendon reflex, but whether this indicates any general affection of the peripheral nerves he does not venture to state. The limitation of the paralysis to the palate has been explained by supposing that the poison of the disease has a direct action upon

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[1] Pitres et Vaillard: Rev. de Médecine, 1885, p. 986; 1886, p. 193 et p. 574, Des Névrites Périphériques chez les typhoides, les tuberculeux et les tabétiques; also, Arch. de Neurol., 1886, p. 337, De la névrite segmentaire.

[2] Virch. Archiv, 1885, Bd. 99, s. 393..

the terminal filaments of the nerves, which in this position are, as it were, dipped constantly in the poison. This theory is supported by a case in which paralysis of the abdominal muscles was associated with diphtheritic inflammation of the navel in a new-born child. But the more serious cases prove that through the blood the poison may be carried to nerves far removed from the seat of the diphtheritic inflammation.

And the following cases demonstrate the wide-spread symptoms which such general poisoning can produce, as well as the nature of the lesion.

Observation XIV.—Marie S——, aged thirteen, after suffering for a few days from a slight sore throat, found herself unable to see clearly because of the development of a paresis of accommodation. There followed a slowly progressing ataxia of the upper, and later of the lower, limbs, with uncertain gait, disturbance of touch, pain, temperature, and muscular senses, with delayed sensation of pain and loss of tendon reflexes. At first she felt shooting pains in the limbs, but later these ceased. There was at no time tenderness of the nerves to pressure. After three months bulbar symptoms appeared, atrophy, with paresis of the tongue, difficulty in swallowing, spasms of coughing, weakness of voice, and paralysis of the palate. The muscles of the hands began to atrophy about the time that the bulbar symptoms commenced, and soon were useless. In the atrophic muscles the faradic reaction was absent, the galvanic much reduced, and very slow. Nine months after the onset she died of pneumonia.

The autopsy showed the brain and spinal cord to be normal. In all the peripheral nerves of the extremities, as well as in the hypoglossal and recurrent laryngeal nerves, well-marked atrophic degeneration was found, with destruction of axis-cylinders and medullary sheaths, and with thickening and increase of nuclei in the connective-tissue sheaths.[1]

Observation XV.—Male, aged seventeen, after an attack of diphtheria grew gradually weaker and weaker, de-

[1] Kast.: Deut. Arch. f. klin. Med., 1886 Bd. xl., s. 41.

veloped paralysis of the uvula and paralysis, with some atrophy, in the muscles of all the limbs, and a slight paralysis of the face and tongue. There was no real ataxia, the muscular sense being normal, but the weakness of the muscles interfered with all accurate movement and caused trembling. The eyes were not affected. There was a slight decrease of tactile sense all over, but pain-sense was slow and imperfect. The reflexes were lost, electric reaction was normal in all muscles except the palate, in which RD was found. The sphincters were unaffected. The paralysis involved the respiratory muscles three weeks after the onset of the disease, and caused his death.

The autopsy showed extensive parenchymatous neuritis in all the peripheral nerves. The various stages of degeneration were found side by side in many nerves, and all degrees of degeneration were discovered. The nerves of the extremities, as well as the phrenics, showed these changes. Some nerve-fibres had simply lost their myelin sheath, others were reduced to a series of granular corpuscles ; others consisted only of the empty Schwann sheath, whose nuclei were numerous. In many nerves the lesion only involved a few segments, and was not continuous through the entire length. At places circumscribed thickening of the endoneurium produced nodular appearances in the course of the degenerated nerve. Here congestion and emigration of leucocytes were found. The perineurium was unaffected. In almost all the muscles of the extremities and trunk such degenerated nerve-fibres were found. The cutaneous nerves were similarly affected, but to a less degree. The nerve-trunks were but slightly affected, as were also the nerve-roots. The spinal ganglia and cord were normal.[1] The limitation of the degeneration to single segments has also been noticed by Gombault and by Pitres et Vaillard.[2]

Observation XVI.—A boy, aged eight, developed paralysis of the soft palate one week after an attack of diphtheria. This continued for one month, when there suddenly appeared a weakness of all the extremities, and

[1] P. Mayer : Virch. Arch., 1881, Bd., 85, s. 181–226.
[2] Arch. de Neurol., xi.

some disturbance of sight. Examination of the eyes showed a complete paralysis of all the recti muscles, with double ptosis, the accommodation and pupil reactions being preserved. The right side of the face was paretic. There was a very marked ataxia in all the limbs. It was with difficulty that he could carry food to his mouth, and he walked with much staggering, and with legs wide apart. Power and nutrition of the muscles was normal, and there was no change in their electric contractility. The tendon reflexes were absent, but the skin reflexes were preserved; and there was no disturbance of sensation or of bladder and rectal functions. The symptoms all increased for ten days, and then respiratory paralysis caused his death.

It was possible to examine the brain only. The small vessels and capillaries were engorged, and the perivascular spaces full of emigrated corpuscles, while here and there small capillary hemorrhages were found. The vessel-walls were not affected, and there were no thrombi discovered. The nuclei of the cranial nerves showed no changes. Both oculo-motor nerves were the seat of very great changes. The myelin sheath of individual nerve-fibres absorbed the carmine stain. In many fibres no axis-cylinder was to be seen. In others the cylinders were swollen or atrophied, presenting variations in size. The number of nuclei of the endoneurium was greatly increased, and among them large granular corpuscles were discovered.[1] The lesion, therefore, which had caused the ocular paralysis was a diffuse neuritis.

A similar affection of the ocular muscles, associated with general weakness, has been reported by Utthoff,[2] which developed two weeks after an attack of diphtheria in a boy, ten years of age, and who at the end of three months had completely recovered. In this case all reaction of the pupils in accommodation was lost, but the light reflex remained. The eye symptoms were attended by some paresis, especially in the legs below the knees—but the muscles were not atrophied, nor was their electric contractility altered. There was a very marked tenderness

[1] Mendel: Neurol. Centralbl., 1885, p. 128.
[2] Neurol. Centralbl., 1885, p. 125.

along the course of all the nerve-trunks. After recovery was complete the knee-jerk returned.

I have seen a case which resembled this quite closely, and inasmuch as paralysis of all the ocular muscles appears to be a rather rare affection, the case may be worthy of mention here.

Observation XVII.—A boy, aged thirteen, after an attack of diphtheria developed paralysis of the soft palate. One week after the paralysis of the palate he noticed a disturbance of vision, which proved on examination to be due to a total paralysis of all the muscles of the eyeballs. The pupil reflex to light was preserved; but as he could not converge the eyes there was no reaction in accommodation. This paralysis of the eyeballs came on within twenty-four hours, and at the same time he developed a very marked degree of ataxia. He swayed in standing with eyes closed, and his gait was exactly like that of a well-marked case of tabes. The motion of the hands was also disturbed, and all the tests demonstrated an extreme degree of inco-ordination. The knee jerks were lost; the skin reflexes were present. In addition to the ataxia there was actual weakness of the muscles, which was more evident in the arms and hands than in the lower extremities. He had formication in the legs, but not in the hands, and there were no lightning pains. He had great difficulty in swallowing solids as well as liquids, which seemed to point to some involvement of the muscles of deglutition as well as of the soft palate. This condition remained stationary for about three weeks, and then all the symptoms began to pass off gradually. His recovery was not complete until six months after the onset.

The prognosis in cases of diphtheritic paralysis is usually very good, the fatal cases here cited being rarities. The treatment consists in general tonic medicines, and the application of electricity to the limbs in the same manner as in other cases of neuritis.

(*b*) Neuritis following variola is a rare complication, and the following case is the only one on record in which an autopsy proved the seat of the lesion.

Observation XVIII.—A young man had varioloid in November, 1881, and while convalescing, six weeks later,

began to suffer from severe pains in his four extremities, especially in the joints of his arms, which were diagnosticated as rheumatic, although there was no fever. Soon after there followed a true paresis, with progressive atrophy of the muscles of forearms and legs. The muscles at the same time became very tender to touch or pressure. The tendon reflexes were much diminished. Reaction of degeneration developed in all the paretic muscles. The pains in the joints and limbs continued, but were less severe than at the outset. The sensibility of the skin was about normal. The nerve-trunks were tender to pressure. Profuse, offensive perspiration in all four extremities was a distressing symptom. There was no tendency to bed-sores, but an extensive pemphigus developed in the legs, and then the pains became more severe. The patient died in July, 1882, of pneumonia.

Autopsy showed the brain œdematous, and the cord in a state of hypostatic congestion. The pathological changes of importance were found in the nerves and muscles. The majority of the nerves of all the extremities were found in a state of degeneration and atrophy. There was marked degenerative atrophy and fatty degeneration of the muscles.[1]

(*c*) It is still somewhat a matter of conjecture whether cases of paralysis following typhoid, typhus, and malarial fevers are due to an affection of the peripheral nerves or of the spinal cord. The researches of Pitres and Vaillard[2] have shown that extensive degeneration of peripheral nerves is to be found in the bodies of patients who have died of typhoid fever, and they have also demonstrated that these fevers are often followed by local neuritis as a sequel. Cases of multiple neuritis with autopsies have not as yet been reported after typhoid or typhus; but Buzzard has recently recorded[3] two cases, following malarial fever, in which all the symptoms pointed to an affection of the peripheral nerves. In this country Gibney has described several cases of paralysis of the extremities, of sudden onset, rapid course, and prompt re-

[1] P. Grocco, Milano, 1885; Centralbl. f. Med. Wissen., 1885, p. 693.
[2] Rev. de Méd., Des Névrites périphériques, 1885, p. 980.
[3] Paralysis from Peripheral Neuritis, p. 104.

covery under large doses of quinine, which he considered malarial. Probably the following case, which was kindly sent to me by Dr. L. E. Holt, was of this nature :

Observation XIX.—Peter S——, aged ten, a healthy boy, was suddenly seized with a chill and fever, and with pains of a severe kind in both legs, associated with weakness so that he could hardly stand. The pain was referred to the sciatic nerves, and was accompanied by numbness and tingling of the feet and legs. There was marked tenderness both in the course of the sciatics and in the muscles of the thigh and leg. There was loss of tendon reflexes, preservation of skin reflexes, and marked paresis in all the muscles of the legs, so that he required help in walking. There was no ataxia, and no incontinence of urine. The symptoms lasted about twenty-four hours, and then decreased in severity gradually during the following day, but recurred on the third, and, after a remission, on the fifth day, with lessened severity. The periodicity of the affection and the rapid cure under quinine left no doubt as to its malarial nature. During the third day, at the time when I saw him, there was redness along the course of the sciatics, and in the region of the right median nerve, and extreme tenderness along these nerve-trunks, in addition to the symptoms mentioned. The electric condition was, unfortunately, not tested; but in Westphal's case faradic contractility was totally abolished during the attack.[1] In this case the active manifestation of the malarial poisoning was by a general neuritis. In Buzzard's cases the malarial attacks preceded the nervous affection. The tenderness in muscles and nerves left no doubt regarding the peripheral nature of the disease.

And here I may suggest that it is possible that many minor nervous affections of transient nature and of malarial origin, if more carefully examined, will be found to be due to some abnormal condition in the peripheral nerves. I allude not only to neuralgiæ, but also to visceral neuroses, whose clinical features have been so graphically described by Dr. Clifford Allbutt,[2] and to numb fingers

[1] Neurol. Centralbl., 1885, p. 287.

[2] Gulstonian Lectures, 1884.

and toes, and to hyperæsthetic and painful areas on the surface, and that host of symptoms of a local character so often carelessly ticketed neurasthenia and dismissed from consideration as soon as named. There is much in the study of the phenomena of neuritis to enforce this suggestion which cannot be developed here.

(*d*) We come, lastly, to those cases of multiple neuritis which occur in tubercular patients, or in those who have had syphilis. If a review of the cases of multiple neuritis already cited be made, it will be found that quite a number of the patients died of phthisis. This was true of the cases of Joffroy, Eisenlohr, Strümpell, Webber, and Müller. Oppenheim [1] has reported cases of multiple neuritis in tubercular patients which went on to recovery. The following case died:

Observation XX.—A young girl, who had had syphilis, and was suffering from phthisis, took a severe cold and began to have pains in the joints, weakness, and numbness of the lower extremities. The weakness of the legs increased rapidly, and was attended by rapid diffuse atrophy. The same symptoms soon developed in the arms. Four weeks after the onset partial RD was present in all the extremities, and total paralysis of the legs and nearly complete paralysis of the arms were present. In the course of the disease, which lasted five months, there was little pain, but great tenderness of the muscles and hyperæsthesia of the skin. The sensation was diminished in feet and hands, but this was slight in comparison with the motor symptoms. The paralysis finally attacked the trunk muscles and the diaphragm, while the beginning of rapid heart action was thought to indicate paralysis of the pneumogastric nerve. Great prostration, delirium, incontinence, bed-sores, and œdema of extremities, with fever, preceded death.

Autopsy: High degree of degeneration of the peripheral nerves, including the phrenic and pneumogastric, was found, with a moderate degree of atrophy of the muscles. Cord normal, anterior motor roots normal. The changes in the nerves were similar to those after

[1] Oppenheim: Zeitschr. f. Klin. Med., 1886, p. 230.

section, viz., a simple degeneration of the fibres with some increase in the connective tissue, but there were no evidences of changes in the vessel-walls, no infiltration with cells. The nerves contained very few normal fibres. The myelin sheaths were found in all stages of destruction, down to an entire absence of contents of the sheath of Schwann. Throughout the fibres fatty and granular masses were found. The sheath of Schwann did not, however, show an increase of nuclei.[1]

Such a case as this would have been ascribed, a few years ago, to a tubercular spinal meningitis, as would many of the other cases already alluded to. It is not my purpose to dispute in any way the fact that many cases of paralysis occurring in the course of phthisis are due to lesions of the central nervous system and its membranes; but I desire to emphasize the fact that a multiple neuritis may produce paralysis in tubercular individuals, and the importance of appreciating this possibility cannot be too strongly urged, since the treatment, as well as the prognosis, will differ widely according to the diagnosis made. It has been known for some time that local neuritis may complicate phthisis, and the researches of Pitres and Vaillard have recently confirmed this fact. But the complication of multiple neuritis is one not mentioned in the text-books, and yet is one which is by no means rare. It seems not unlikely that some of the nervous symptoms in consumption, formerly considered functional, may be traced to this affection.

Although syphilis has been described as a cause of neuritis, I cannot find that the lesion in the few cases examined has had the peculiar characteristics of syphilitic lesions elsewhere in the body, and therefore it seems to me doubtful whether we are justified in describing a syphilitic multiple neuritis. Further facts are needed to establish its existence.

(*e*) It only remains to consider briefly the epidemic form of multiple neuritis, and our study of the infectious cases will be complete.

In 1882 Professor Scheube, of Tokio, Japan, called

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[1] Vierordt: Arch. f. Psych., 1883, xiv., 3.

the attention of European physicians to the existence of a peculiar affection prevailing among the Japanese.[1] It was called kakke; from two Chinese words, *kiaku*, meaning legs, and *ke*, meaning disease. It had been known among the Chinese for centuries, being mentioned by name in Chinese medical books written two hundred years before the birth of Christ, and fully discussed by an eminent author in 640 A.D. It ceased, however, to prevail in China about two hundred years ago, and its ravages are now confined, according to Scheube, to Japan. There its importance is considerable, since it is so prevalent that in 1877 fourteen per cent., and in 1878 thirty-eight per cent., of the men serving in the army suffered from it. It is considered a miasmatic infectious disease by Scheube, although an eminent Japanese authority considers it due in some way to the diet of rice. That diet has something to do with its occurrence is proven by the fact, communicated to me by Dr. Wallace Taylor, that since wheat has been substituted for rice in the diet of some of the barracks and prisons in Japan the disease has been less common. It occurs in epidemics, but is always endemic in Japan. It does not attack Europeans. It affects females rarely, only nine per cent. of the cases being in women; and it is the youth of the land, between the ages of sixteen and twenty-five, who are attacked. Exposure to damp and cold in crowded dwellings, such as barracks, increases the liability to the affection. The majority of the cases occur during the hot months, but some are always under observation. This disease is not, however, confined to Japan. It has been observed for many years in the islands of the Pacific Ocean, in India, Ceylon, on the west coast of the Red Sea, in Borneo and New Guinea, in Brazil and Cuba, and its prevalence in the Dutch possessions in the China Sea has made it familiar to physicians from Holland who have visited these colonies. It is there known under the name of beriberi. It is endemic in these regions, but occasionally occurs as an epidemic. An interesting account of such an epidemic, occurring in

[1] Deut. Arch. f. Klin. Med., xxxi. and xxxii.

1882–83 in Manila, the chief city of one of the Philippine Islands, has been given by Dr. Koeniger.[1] It appears that in the fall of 1882 an epidemic of cholera occurred in Manila, of such severity that twenty thousand persons, in a population of four hundred thousand, were affected. As a precaution against this disease the native population lived for several months almost exclusively upon rice, refusing to eat fruit or fish, which are their other chief articles of diet. As the epidemic was subsiding a terrible cyclone devastated the city, destroying the light wooden houses, and leaving sixty thousand families homeless; and these poor people were exposed for several weeks to the inclemency of the weather, which at this time of the year is rainy. A few days after the cyclone the epidemic of beriberi began, and as the disease had never before appeared in Manila the unknown affection excited great alarm. This was increased by its fearful mortality, sixty per cent. of the early cases proving fatal. Europeans were exempt, with two exceptions, and the Chinese population did not suffer greatly, but among the natives the epidemic was wide-spread. Thus, in one suburb of Manila, of twenty-five thousand inhabitants three hundred died in the course of eight weeks. Men and women were equally affected, and persons of all ages, except young children were attacked. The disease terminated fatally in from ten days to five weeks after its onset; but as time went on the proportion of recoveries increased, and by the end of March, 1883, it had almost disappeared. The months from October to March are the dry, cool season in the Philippine Islands, although the climate is tropical. Exposure to heat could hardly be considered a cause of this epidemic, but whether the exposure to cold and damp, or the diet of rice, or the transportation of some infectious agent by the cyclone was the cause, gave rise to much discussion, and could not be determined.

Sporadic cases of beriberi, or kakke, occasionally appear in our hospitals, usually in the persons of Chinese or Malay sailors, or in the persons of travellers from tropical climates who have been exposed to the infection in the

[1] Deut. Arch. f. Klin. Med., xxxiv.

place from which they came. Three such cases have recently been reported by Dr. Seguin, of this city,[1] in patients who came from the West Indies, and a case observed in Bellevue Hospital, by Dr. J. West Roosevelt, was discussed in the Academy of Medicine recently. In 1881 a Brazilian naval vessel entered San Francisco with a large number of the crew affected by the disease. They were sent to the United States Marine Hospital, and attended by Dr. Hebersmith, who gave an interesting account of the circumstances leading to the development of the disease in the United States Marine Hospital Report.[2] From this report the following map is taken, which demonstrates the localities in which beriberi is endemic.

Only last year a commission was appointed by the Dutch Government to investigate the subject of its nature, and the recently published report contains the following statements:[3] The disease is caused by a micro-organism resembling the bacillus of splenic fever, though somewhat smaller, which colors with fuchsin and gentian-violet, and can be seen with a power of 560°. These bacilli are found in the blood, lungs, heart, brain, cord, and nerves of the patients, and can be cultivated outside of the body. The germs infect wooden dwellings chiefly. They may be conveyed by articles of clothing, and probably enter the body by the lungs. Direct contagion has not been observed. A potent predisposing cause to their reception in the body and to the development of the disease is lack of nutrition, consequent upon exposure to damp and to cold, and upon insufficient or bad food. It must, however, be added that a most thorough examination in Dr. Roosevelt's case, by Dr. Prudden, failed to reveal the presence of such bacilli.

In the light of these recent investigations a new view is taken of an epidemic of a peculiar kind which occurred in France in 1828. Buzzard has found an account of this, prepared by Graves, in which the symptoms are so fully

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[1] Medical News, Philadelphia, December 11, 1886.
[2] Marine Hospital Report. 1881.
[3] Deutsche Med. Woch., December 9, 1886 (an abstract only is given). See also Harada: Neurol. Centlbl., 1885, p. 326.

FIG. 6.—Sketch Map indicating the Geographical Distribution of Beriberi. (From United States Marine Hospital Report, 1881.)

detailed as to leave no doubt that it was an epidemic of multiple neuritis.[1]

The cases of beriberi are divided into two general classes, according to their severity.

There are, first, slight cases, in which the onset is gradual, being usually preceded by a little fever, coryza, and conjunctivitis, which cease when the actual symptoms commence. The patient first notices a weak and heavy feeling in his legs, and finds that he tires so easily that he cannot walk as much as usual. The tired feeling is soon associated with numbness and pain in the legs, and with a slight œdematous swelling. Then, if not before, palpitation of the heart, oppression and weight in the epigastrium, loss of appetite, and general malaise are felt, and the patient finds it necessary to apply for treatment. An examination then shows some diminution of power in the feet and legs, and also in the hands, with loss of tendon reflexes, and much tenderness in the muscles, which show a diminished electric excitability. There is never any ataxia, though the patient sways when his eyes are closed. There is discovered a slight degree of anæsthesia, of irregular distribution, chiefly in the legs and in the radial-nerve region on the forearms. Though the patients look pale, it is usually impossible to find anæmia by examination of the blood.[2] The circulation in the extremities is sluggish. The heart is irregular and rather rapid, and the œdema of the extremities indicates a failure of its power. Dr. Wallace Taylor[2] finds that a sphygmographic tracing is characterized by a sudden high upstroke in ventricular systole, by a precipitous descent from the apex of the percussion-wave, and by dicrotism. Beyond this point these cases, which make up the majority, do not advance. They usually recover in a few days, or at most a month; although a few become chronic and require several months before the cure is complete.

There are, secondly, severe cases. These may present three different types. There is the atrophic or dry type, in which, after an onset similar in nature to that in the slight cases, but much more rapid, the weakness devel-

[1] Buzzard: On Paralysis from Peripheral Neuritis, p. 37.
[2] Wallace Taylor, M.D.: Studies in Japanese Kakke. Osaka, 1886.

ops into a true paralysis, associated with marked wasting of the muscles and reaction of degeneration, with great diminution of galvanic excitability. Within a week the patient has to go to bed, and then the paralysis soon spreads from the legs to the arms, and may involve the trunk and even the face. The entire muscular system wastes away till the patient is a mere skeleton. In addition to the motor symptoms there is great sensory disturbance. The suffering from pain, paræsthesiæ, and general muscular tenderness is extreme, and the patient lies totally helpless and unable to tolerate the lightest touch. The skin may be glossy. There is usually some anæsthesia, but it is never complete, although it may involve the entire body. The temperature sense is seldom affected. Pain may be delayed in transmission. There are no gastric symptoms, and no œdema. Some cases prove fatal from general exhaustion, or intercurrent disease, but the majority recover after a convalescence which lasts a year or more, during which the muscular system is rebuilt.

There is, secondly, the hydropic or wet type. In these, heart-failure appears early, and is associated with a marked decrease of arterial tension and much œdema of the entire body, effusion into the cavities being added to that beneath the integument. The swelling of the edematous parts conceals the atrophy which is going on in the muscles, but this is indicated by the paralysis, which is as severe as in the preceding form, and it becomes evident during recovery when the œdema has subsided.

There is, thirdly, the acute pernicious type. In this all the symptoms of the two former types appear in rapid succession, and, in addition, gastro-intestinal symptoms and a suppression of urine combine to make the condition an alarming one. Effusions into the pleura and pericardium appear early. The pulse becomes small and irregular, and cyanosis indicates the heart-failure which precedes death.

In this form the disease may run its course in two weeks to a fatal termination. This was the form which chiefly prevailed in Manila, the cases of the atrophic form being the ones which recovered.

. The severity differs much in different epidemics, the mortality varying from two per cent. in Japan to sixty per cent. in Manila. It is usually not above three per cent. In all the forms there is some danger of a sudden heart-failure, and this is usually the cause of death.

The morbid anatomy of the disease is, primarily, a multiple peripheral neuritis, with, secondarily, numerous organic changes in various organs, none of which, however, are essential to the disease.[1]

While we cannot join in the wish expressed by an enthusiastic German author, that the disease may soon become a familiar one to those outside of Japan, we cannot pass it by without a brief reference, especially as it resembles in so many of its features, as well as in its pathological basis, the condition which has occupied our attention at present. And it may not be unprofitable to obtain a clinical picture which differs somewhat from that already viewed, in connection with the forms more familiar to us, in order to detect sporadic cases of the disease, if such should appear among us. As to its treatment, it may be mentioned that quinine failed to influence its course, and that heart-stimulants to combat the dangerous complications, hypnotics to counteract the pain and insomnia, and general tonic treatment have proved of the greatest service. Change of climate often is attended by recovery. In the stage of recovery electricity and massage have been employed with advantage.

III.—In a considerable number of cases of multiple neuritis no cause of the disease could be definitely ascertained. Great fatigue by walking, or by hard labor, and exposure to dampness and cold are assigned as causes in some of these cases, and may have acted as etiological factors. In others the disease seems to have developed spontaneously.

The following cases are cited as belonging to this class :

Observation XXI.—A perfectly healthy man, aged thirty-four, after a long and fatiguing walk was suddenly attacked with weakness of the legs, pain, and formication,

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[1] Scheube : Virch. Archiv, Bd. 95, s. 146.

which increased so rapidly that in two days he could not walk, and in eight days was totally paralyzed—legs, trunk, arms, face, and pneumogastric nerve being all involved. The paralysis was attended by severe pains, tenderness along the nerves, profuse perspiration, fever, and jaundice. Further, a diminution of sensibility, a loss of tendon reflexes, a loss of muscular sense, a rapid atrophy, with reaction of degeneration, were observed; and trophic disturbances, consisting of an abnormal growth of hair and pigmentation of the skin, as well as vasomotor disturbances in the form of œdema, appeared. There were no fibrillary muscular contractions, no increase of mechanical excitability, and no incontinence of urine or fæces. During the following four months a gradual improvement took place, which went on to final recovery a year and a half after the onset. The only anomaly at that time was an absence of contractility of the muscles to the electro-current, when excited through the nerve.[1]

Observation XXII.—A female, aged forty-two, previously in good health, was attacked, on October 1st, with pain in the back and in all four extremities, attended by great weakness, which increased so rapidly that on October 13th there was found to be an almost total paralysis of both legs, of the trunk, and of certain muscles of the arms. The pains were accompanied by a feeling of formication and a painful sensation of tingling. When first seen by Dr. Guttmann there was total paraplegia, slight movements in fingers and toes being the only motion possible; the muscles were all relaxed and flabby, were much atrophied, were tender to pressure, and a few of the smaller muscles showed slight diminution of faradic excitability. There was no disturbance of sensation. The tendon reflexes were abolished. The sphincters were not involved. The temperature was normal, but the pulse was unduly rapid. There were no bulbar symptoms. Under large doses of iodide of potassium and the application of faradism, the patient recovered completely in the course of three months.[2]

[1] R. H. Pierson: Ueber Polyneuritis Acuta, Volkmann's Klin. Sam., 1883, No. 229.

[2] Guttmann: Deut. Med. Wochenschr., 1884, No. 19.

In some of the cases of this form the early pains were located in the joints, and led to the diagnosis of rheumatism. The fact is merely mentioned to call attention to the possibility of mistake. A careful examination will never fail to correct it.

Symptoms.—Turning now to the consideration of the individual symptoms of multiple neuritis, and their course, we are at once impressed by their number and variety.

The *sensory symptoms* are the earliest to appear and the last to pass away. In the majority of the cases on record, from whatever cause, numbness, tingling, or formication usher in the disease. These forms of paræsthesiæ begin in the feet and hands, and extend to the knees and elbows. They may be associated with burning, stretching, boring, or tearing sensations, which distress the patient, especially during the onset. But all such sensations usually subside as the affection reaches its height. Their recurrence, as the case goes on, may be regarded as a favorable symptom, however annoying, for they frequently precede recovery, and are among the last evidences of the disease to disappear. Pain is usually present as well as paræsthesiæ. It may occasionally be sharp in character; but is usually moderate and not continuous. At times it may be lancinating, and so severe as to necessitate the use of morphia. But it is rarely as distressing as in cases of locomotor ataxia. Tenderness in the nerves and muscles is a constant symptom. It may be so marked that the limbs cannot be moved or handled, and thus it may interfere with the application of electricity and massage. When the tenderness and pain are referred to the joints, as not infrequently occurs in the early stage of the disease, the case may be mistaken for one of acute articular rheumatism, and if the joints are swollen or the limbs edematous the difficulty of diagnosis is greatly increased.

In addition to these subjective feelings some demonstrable disturbance of the various sensations is usually present. Hyperæsthesia to touch, and also to electricity, is not infrequently observed during the first few weeks. It is usually followed by some anæsthesia, although this rarely becomes complete. In some cases the loss of

tactile sense is quite evident from the outset, either limited to the cutaneous distribution of some special nerve, in which case oddly shaped areas of insensibility will be found, or, as is most often the case, about uniformly distributed over the distal parts of the extremities. When the anæsthesia is at its height the patient has difficulty in locating a touch upon the limb, even though he feels it. The transmission of pain and temperature-sensations is always delayed, but the impressions are usually felt quite acutely. The sense of pressure has been tested in only a few cases, and in those it was decidedly impaired. The muscular sense escapes any affection in some cases, but in others is the most profoundly disturbed of all the senses. When it is involved, the inco-ordination and ataxia are well-marked symptoms, and, as already stated, some of the cases have been mistaken for locomotor ataxia, because of the predominance of the disturbance of muscular sense.

These sensory symptoms are usually limited to the forearms and hands, and to the legs and feet. In a few cases they have involved the entire extremities, and even the trunk ; and one case of facial tingling, with anæsthesia, has been recorded.[1] The skin reflexes are usually preserved.

The special senses are rarely affected in multiple neuritis. It is true that optic neuritis has occurred in a few cases, and in two cases hearing as well as sight has been affected. These cases prove that no nerve can be said to be exempt from implication in this disease, but the liability to affection seems to be slight in the case of the nerves of special sense.

The *motor symptoms* are as marked and as important as the sensory. Paralysis, beginning as simple weakness, with a feeling of fatigue on any exertion, gradually increases in severity until at the height of the disease it becomes complete. It usually comes on rapidly, so that within two weeks the patient is helpless ; but it may be less sudden, and not deprive him of the power of walking and of using his hands for two or three months. In a

[1] Lowenfeld: Neuro. Centralbl., 1885, No. 7.

few cases a very acute onset is recorded, all the symptoms developing within three or four days. The distribution of the paralysis is not uniform at the outset. It may develop in the muscles supplied by a single nerve, and advance to others; it may begin in all the muscles of the legs, and then involve those of the forearms; it may commence in all four extremities at once. It is always more severe in the muscles which move the joints of the feet and hands, and the ankles and wrists. It rarely invades those which move the knees and elbows. When the disease is fully developed, all the muscles below the knees and elbows are much weakened or totally paralyzed. In a few cases those of the thighs and arms are involved also, and occasionally the muscles of the trunk and those of respiration become affected and the patient dies. When such a paralysis makes rapid progress and involves all the body, the case resembles Laudry's paralysis. But in multiple neuritis, as has been already stated, the disease does not creep up from legs to thighs, and then to trunk and arms, as in Laudry's paralysis. It spreads from feet to hands, from legs to forearms, and the trunk is invaded only at the end, and in Laudry's paralysis sensory symptoms are wanting. In some cases of multiple neuritis the cranial nerves become involved; those of the eye and of the face being most liable to invasion. It is only in fatal cases that the action of deglutition has been affected; and when the pneumogastric is invaded, and the heart becomes rapid and irregular, the prognosis is always grave. Spasms rarely occur.

The paralyzed muscles are relaxed, flabby, and atrophied; they may or may not lose their mechanical irritability, but their normal tone is always lost, and hence the so-called tendon reflexes are abolished.[1] To the electric current their excitability is very rapidly and markedly changed; but the conditions which have been observed are quite various. Sometimes there is a simple diminution of excitability, and then a very strong faradic or galvanic current is needed to produce contractions. Fre-

[1] A few exceptions to this rule are found in two cases recorded by Strümpell and Mobius, Munchener Wochenschrift, 1886, No. 34, and in Brit. Med. Jour., January 1, 1887.

quently all faradic excitability is lost, and then the muscles react to a galvanic current only. In this condition it may require a very strong galvanic current to produce contraction, and this fact is quite pathognomonic of neuritis. For an anterior polio-myelitis, where the muscles respond to galvanism only, it does not require a strong current to cause a motion until some months after the invasion. The action of the different poles is not uniform. In many cases the contraction of the muscle when stimulated with the positive pole is greater than when stimulated with the negative pole, and the contractions may be sluggish. Then the reaction of degeneration is present. But in some cases the normal condition is found, and the negative pole produces stronger contractions than the positive pole. If the muscles that are not paralyzed be tested, the same electrical changes may often be discovered in them. A loss of faradic irritability and a marked decrease in the galvanic irritability of the muscle and nerve are, therefore, important symptoms of multiple neuritis. And as the disease goes on to recovery a gradual increase in the galvanic irritability occurs, a fact which is often of much aid in prognosis if careful measurements of the strength of current used be made by the galvanometer. I am accustomed to record such measurements upon charts, and thus to obtain an electric curve for each muscle which is paralyzed.[1] These curves enable one to judge of the progress of the case quite accurately, and when the line is advancing steadily toward the normal point, after a great deflection or after a stationary level, the prognosis is very favorable.

As a result and accompaniment of the paralysis *abnormal positions* are assumed by the limbs. The dropped wrist and dropped foot are quite characteristic of multiple neuritis. But other deformities may be present. In a few cases there have been extreme contractures of all the extremities in flexed position. And not infrequently the appearance of the claw-shaped hand, and some one of the various forms of talipes, indicates a

[1] Electric Charts, Journal of Nervous and Mental Diseases, February, 1887.

serious shortening of one set of muscles, and corresponding weakness of its opponents. These deformities usually subside as the power returns, or if they do not they can be corrected by proper manipulation and by apparatus. In a few cases it has been necessary to resort to tenotomy, but a permanent deformity has not been recorded.

The *vaso-motor and trophic symptoms* are less constant than those already described. In some cases marked œdema has been an early and a permanent symptom. This may develop in the feet and hands, or may appear about the joints. It usually is temporary. The circulation is not impaired any more than is customary in a limb whose muscles are inactive, and coldness and cyanosis are rarely sufficient to attract attention. Sometimes profuse perspiration is a noticeable symptom, being limited to the paralyzed parts. It may be offensive, and by its evaporation always causes a complaint of coldness. In other cases glossy skin makes its appearance early, and remains until regeneration of the nerves begins. Its disappearance in one of my own cases was the first sign of recovery in the lower extremities. Other forms of trophic disturbance are rarely met with in multiple neuritis. And this is quite remarkable in view of the fact that it has been the tendency of late to refer such trophic affections as ulcerations, bed-sores, gangrene, pemphigus, and various eruptions to lesions of the nerves.[1] It is true that inflammations of the joints, resembling those appearing in acute rheumatism, sometimes occur at the onset of neuritis; but as they disappear quickly, while other symptoms remain, it is improbable that they are to be traced to the changes in the nerves. They may be due to the infectious agent, or to the same obscure cause which sets up the neuritis, or they may be evidence of an attack of acute articular rheumatism, which is in turn followed by neuritis; but they cannot be described as trophic symptoms of the disease, otherwise they would be more constant in their occurrence and more permanent in their duration.

[1] Raynaud's disease has recently been ascribed to neuritis. See J. Wigleworth, Brit. Med. Jour., January 8, 1887.

A negative symptom of some importance is the absence of any interference with the automatic acts controlled by the sphincters.

One word about the onset. Occasionally it is sudden and accompanied by a marked febrile movement, with chill, and temperature of 103° to 104.5° F. The fever may persist for several days, but usually subsides spontaneously and does not recur. In a few cases there has been a constant elevation of temperature from one-half a degree to one and a half degree above the normal; and an increase in the rapidity of the pulse throughout the disease has been noticed. A pulse of ninety need give no alarm, but if it runs up to one hundred and forty, and becomes irregular, there is reason to believe that the disease has attacked the vagus nerve, and then the prognosis becomes serious, though not by any means hopeless.

The duration of the disease varies considerably in different cases. An average of twenty-five cases gives seven months as the probable time required for complete recovery. But in these cases the duration varied from two months, in the most favorable, to sixteen months, in the most refractory.

The disease is more common in males than in females, excepting the form produced by alcoholism. Of 47 non-alcoholic cases, 33 were males.

All ages are liable to be affected, but in the records of multiple neuritis, excepting the form produced by diphtheritic poison, the cases recorded do not include any children.

That children may be affected is, however, not at all improbable. Dr. H. D. Chapin, of this city, has described four [1] cases of atrophic paralysis in children, in which the presence of sensory symptoms, pain and muscular tenderness, and the steady progress toward recovery pointed to the existence of multiple neuritis. The cases were such as are usually called infantile paralysis, but there were points of difference (viz., the existence of sensory symptoms) which removed them from this cate-

[1] Painful Paralysis in Children, New York Medical Record, January 15, 1887.

gory. Leyden called attention to the possibility of mistaking neuritis for poliomyelitis in his first article upon the disease, but he referred to adult cases rather than to children's cases. And if the surmise of Chapin should be substantiated by autopsy, it might be necessary to conclude that in children, as well as in adults, the majority of cases of supposed poliomyelitis which recover are cases of multiple neuritis.

In adults, out of 50 cases 6 were between the ages of 18 and 20; 8 between 20 and 30; 20 between 30 and 40; 8 between 40 and 50; 5 between 50 and 60 and 3 over 60 years of age.

Diagnosis.—While the individual symptoms occurring in the course of multiple neuritis are not different in character from those found in spinal-cord diseases, the diagnosis can usually be reached with very little difficulty when their combination, the causation, and the course of the case under examination are considered. The varying combination of the symptoms possible has been manifest during their description, and it is not my intention to review them again. There are, however, three combinations which resemble very closely, respectively, anterior poliomyelitis, locomotor ataxia, and diffuse myelitis, and to these attention must be directed.

Atrophic paralysis, with reaction of degeneration and loss of reflex is common to anterior poliomyelitis and some cases of multiple neuritis. In the latter a more gradual onset, preceded and attended by numbness and pain, tenderness in the course of the nerves, tenderness in the muscles, and the persistence of sensory symptoms after the invasion, will remove all doubt regarding the diagnosis. When these symptoms are not clearly marked, the distribution of the paralysis in symmetrically situated muscles, especially if these muscles are supplied by single nerves, and the further extension to muscles in other nerve domains, rather than the affection simultaneously of muscles which are grouped physiologically (*i.e.*, act together to perform one function), will point to neuritis. In neuritis the paralysis advances more or less gradually, while in acute poliomyelitis there is, after the onset, a subsidence of the paralysis in some of the muscles first in-

volved. And lastly, as the case goes on, a gradual complete recovery will be far more frequent if it was originally a case of multiple neuritis.

Ataxia, loss of knee-jerk, pain, and sensory disturbances, including a loss of muscular sense, Romberg's symptom, and optic neuritis are common to locomotor ataxia and multiple neuritis. In the latter the relatively rapid onset of the ataxia, which follows closely upon the sensory symptoms; the prominence of numbness and anæsthesia, rather than of lightning pains; the extreme degree of the anæsthesia and analgesia; the tenderness of muscles and nerves; the usual occurrence of some degree of actual paresis with atrophy and RD; and the absence of bladder and sexual symptoms will point inevitably to the diagnosis. Furthermore, the ataxic form of neuritis only occurs after poisoning with alcohol or arsenic, or as a sequel of diphtheria, and the establishing of the causation will aid the diagnosis. Here, again, the course of the case toward recovery, with return of the knee-jerk, will decide in favor of neuritis, if the diagnosis has not been reached in an early stage.

There are very few symptoms of diffuse myelitis which are not found in cases of neuritis. But cases of diffuse myelitis of the type of Duchenne—paralysie générale spinale subaigüe ascendante—are very rare, and indeed by Leyden it has been affirmed that all such cases are multiple neuritis. Other authorities dispute this assertion and leave us to establish points of difference. These are as follows: In neuritis affections of the functions of micturition and defecation do not occur. Girdle sensation is very rarely mentioned as a symptom; bed-sores and cystitis have not been observed. The advance of the paralysis is rarely from the legs to the thighs and trunk, and then to the arms; it is usually from the legs to the forearms, the trunk and thighs escaping, and, as a rule, the distal portions only of the extremities are paralyzed. If the muscles of the abdomen and respiration are involved, it is only in rapidly fatal cases. In neuritis there is usually some ataxia, and loss of muscular sense is quite evident; while in some, at least, of the cases of myelitis of Duchenne, there were no sensory symptoms at all. Fin-

ally, the tenderness of muscles and nerves, and the absence of tenderness to pressure or to heat in the spine, would decide in favor of neuritis. The diagnosis from meningitis of the cord, from tumors or hemorrhages into the cord, or from general paralysis of the insane, would rarely present any difficulty to one who was familiar with the symptoms in those affections, and who knew the prominent features of multiple neuritis.

No small difficulty may be encountered, however, in settling the question whether in a given case we have to deal with multiple neuritis alone, or with multiple neuritis which is complicated by myelitis. The importance of the question is evident, since the prognosis in the two conditions is very different; and the number of autopsies on record in which this complication has been demonstrated, though few, is sufficient to make a decision of the question necessary.[1] The following points may enable a determination of the question to be reached: (1) As long as a case is increasing in severity, or in the extent of the symptoms, no one can determine the extent of the lesion. It is only when its course has become stationary that the question of exact limitation will arise. Many cases remain practically without improvement for three or four months, and then gradually recover. A stationary condition alone does not, therefore, excite fear of a complication. But neuritis tends spontaneously to recover, the process of regeneration beginning soon after the degeneration has ceased. If, therefore, there appears to be no improvement of the condition after the fourth month, the probability is either that the cause of the disease has not been removed, or that a myelitis has developed and prevents recovery. (2) The course of a case of neuritis is quite characteristic, the symptoms reaching their maximum in a short time, and then subsiding. If, after a stationary period, further symptoms develop, we must believe either that the cause of the disease is renewed (*e.g.*, the use of alcohol) or that myelitis has begun. The symptoms of such a myelitis will be an increasing weakness and more rapid and pro-

[1] Oppenheim, Zeit. klin. Med., xi., 232; Leyden, Charité Annalen, 1878; Duménil, Gaz. Heb., 1861.

gressive atrophy of the muscles; a gradually decreasing degree of galvanic excitability in the paralyzed muscles; a loss of pain and temperature senses, which, as a rule, are not affected in neuritis; a decrease in the paræsthesiæ, and an increase in the degree of anæsthesia; the development of loss of control over the sphincters; the occurrence of bed-sores, furuncles, eruptions of a bullous nature; and the beginning of cystitis. (3) On the other hand, if the symptoms are gradually improving; if the power gradually returns; if the anæsthesia decreases, and is succeeded by paræsthesiæ, however disagreeable to the patient; if the galvanic excitability becomes gradually more acute in the muscles, so that the electric curve approaches the normal line; if the faradic contractility returns in the muscles; if the tenderness of muscles and nerves decreases; and if the glossy appearance of the skin disappears, it may be stated that no complication of myelitis has occurred, and that recovery, though possibly prolonged, will at length result.

Another question of diagnosis must also be considered. It is found that in no small number of cases of locomotor ataxia multiple neuritis develops as a complication. How can we determine when this complication exists? The symptoms of the two diseases may be so nearly identical that difficulty arises in distinguishing them. When they coincide it is equally difficult to determine to which disease any given symptom belongs. Pitres and Vaillard,[1] who have considered this subject most carefully, affirm that there is no constant relation between the severity of the central and peripheral lesions when they coincide. But there are certain symptoms which develop in some cases of tabes, but not constantly, which may be looked upon as accidental, and thus traced to neuritis. Such are the appearance of plaques of anæsthesia and analgesia of limited area, muscular weakness or paralysis of limited extent, trophic disturbances in the skin (*e.g.*, perforating ulcer), nails, joints (*e.g.*, Charcot's disease), bones (*e.g.*, spontaneous fractures), and teeth (*e.g.*, spontaneous falling out), and possibly the various crises referable to the

[1] Pitres et Vaillard: Des névrites périphériques chez les tabétiques, Rev. de Méd., 1886, p. 574.

viscera and larynx. In cases which competent observers have examined, peripheral neuritis has been found *post-mortem* in the nerves supplying the parts in which these symptoms appeared. It is, therefore, reasonable to conclude that in any case of locomotor ataxia in which these symptoms develop we have to deal with a posterior sclerosis which is complicated by a peripheral neuritis. And here again the distinction has a bearing upon the prognosis, for the symptoms of the accidental kind may pass off, while those due to the central lesion will remain. While these conclusions of the French authors are of importance, it must be noticed that their claim that trophic disturbances are due to a complicating neuritis is by no means substantiated by the history of cases of multiple neuritis, in which, as we have seen, trophic disturbances of the varieties mentioned do not often occur.

Prognosis.—The prognosis in multiple neuritis is good, provided the exciting cause can be removed. The only cases which form an exception to the rule are those whose constitutions are much impaired by excesses or by other diseases; those who have so far indulged in alcohol or are so completely soaked with arsenic or lead as to be unable to throw off the poison; and those in whom the disease begins with great suddenness, advances rapidly, and involves the phrenic and pneumogastric nerves. These cases either die of respiratory paralysis or of some complication. When a case has reached the stationary period the prognosis is generally favorable, and if the encouraging signs of recovery already mentioned begin to appear a cure may be promised. The possibility of the complication of myelitis must not, however, be overlooked, and if it occurs the prognosis becomes at once unfavorable. Even in serious cases of alcoholism, with gastro-intestinal and cerebral symptoms, it the acute stage is safely passed and all alcohol be removed from the patient's diet, recovery from very extensive paralysis will occur. The cases of diphtheritic paralysis and ataxia usually recover without treatment. The cases occurring with syphilis and tuberculosis are not so uniformly favorable, although the former usually respond promptly to specific treatment, and even the

latter may recover wholly. In epidemics, the percentage of mortality has been found to vary, but is usually low.

Treatment.—The treatment of multiple neuritis requires patience, but receives the reward of success. As we have already seen, the majority of the patients recover, and it is probable that, if the cause of the affection were removed and the patients placed in favorable circumstances, expectant treatment would alone be sufficient. It is, however, not advisable to let therapeutics play a passive part. The course of the disease can be altered and its duration much shortened by active interference. In the stage of invasion the free use of salicin, salicylic acid, or the salicylate of soda seems to have important results. These remedies cannot be said to act as promptly as in cases of acute articular rheumatism, but the consensus of opinion is that their effect in multiple neuritis is very marked. They should be given, as in acute rheumatic fever, in large doses until noticeable effects are obtained. They should be combined with the bromide of potash or soda, partly because these drugs counteract unfavorable symptoms produced by the salicin compounds, and partly because in the hyperæsthetic irritable condition attendant upon the invasion of the disease they are indicated. This condition may require stronger sedatives, and not infrequently morphine must be employed to give relief from the excruciating pains. The pains are often relieved by hot or cold applications to the limbs; but as the muscles are often exceedingly tender ordinary applications cannot be made. It is then advisable to use evaporating lotions, preferably those containing chloroform, which may be soaked into light cambric or gauze, and gently placed upon the limbs, which lie upon the softest pillows, or which may be more comfortable if the patient is put upon a water-bed. Applications of a five per cent. solution of carbolic acid have also been of use. If cool applications prove intolerable, heat may be employed. The limbs may be enveloped with cotton and covered with oil-silk, a light bandage keeping these in place; or they may be frequently bathed in hot water, and hot bottles placed against them, some soft substance

intervening. One of my patients found great relief from the paræsthesia by cold douches, while another preferred the use of hot water. It is best to let the patient decide as long as the application has to be made for the relief of pain. Gentle friction with oil of cocoanut often affords comfort. In the chronic stage, as we shall see presently, heat is to be preferred to cold. Cases which are distinctly syphilitic, if such occur, should be treated from the outset with inunctions of mercury and large doses of iodide of potash. I believe that both these drugs should be employed together, even in the tertiary stage of syphilis, and it is my experience that all syphilitic nervous lesions, whether central or peripheral, yield more promptly to their combined use than to the employment of either alone. Malarial cases must be treated with quinine or Warburg's tincture. In non-malarial cases quinine has proved of no avail. In cases which are due to poisoning of any kind the first necessity is to eliminate the toxic agent from the system, and the second to prevent any further ingestion of the poison. Iodide of potash aids in the elimination. The second indication is easily fulfilled when arsenic or lead are the toxic agents, but when the case is due to chronic alcoholism special precautions are needed. Alcoholic cases require from the outset special treatment. The condition at the time of the onset of the paralysis may be one verging upon delirium tremens. If all alcohol is suddenly removed, without due care to supply some other heart-stimulant and to secure the perfect nutrition of the patient, serious collapse may ensue. The first necessity is therefore to take care of the general condition of the patient. If this will admit of the immediate withdrawal of all alcoholic stimulation it should be done; if not, the alcoholic beverage must be immediately reduced in quantity, and as soon as possible wholly cut off. The use of milk diet, or kumyss, or pancreatized milk, or, if necessary, rectal alimentation will be followed by a gradual recovery of the power of assimilation, and as soon as the patient ceases to lose weight all alcohol may in any case be safely stopped; its elimination by the intestines and kidneys may be hastened by appropriate means, and cerebral symptoms, if they arise, may be

treated as in other cases of alcoholic intoxication. But it is in the chronic stage, when the patient is gradually recovering, that the vigilance of the physician is called into play to prevent a renewal of the poisoning. It is amazing that patients who know perfectly the injurious effect of alcohol upon them should insist upon getting it. But it is done. And when these patients are surrounded, as is often the case, by sympathizing friends, or servile domestics, or unscrupulous nurses, who do not appreciate the importance of total abstinence either for themselves or for the patient, they often succeed in baffling all attempts to deprive them of the favorite drink. It is only when they are watched constantly by persons who can be implicitly trusted, and who have sufficient authority to cut off all surreptitious supplies that the physician can feel sure that his commands are obeyed. And this precaution is by no means needless even when it is probable that family servants are trustworthy ; for the continued pleading and remonstrance of the patients may corrupt the best of attendants, especially if accompanied by threats of discharge at a future day. It is therefore necessary to place these patients under the surveillance of trained nurses from the start, or to remove them to an institution where they are under control.

In the chronic stage the drugs which are of greatest service are strychnia and arsenic. Strychnia may be given in doses of $\frac{1}{60}$ to $\frac{1}{20}$ gr. t.i.d., and it is well to combine it with phosphoric acid and the syrup of the hypophosphites. Arsenic may be used in tablets or pills containing $\frac{1}{50}$ to $\frac{1}{30}$ gr. t.i.d., or in Fowler's solution, five to eight drops t.i.d. The use of iron with these two drugs will be indicated in the majority of cases where there is attendant anæmia. In alcoholic cases both arsenic and strychnia may increase the mental irritability, but should be continued unless this becomes too great. I have seen benefit from both of these drugs, and think it well to employ them alternately, using each for about two weeks at a time.

The remedies used in the chronic stage have two objects : one is to increase the rate of repair in the nerves ; the other is to keep the nutrition of the muscles as good as possible. While the drugs mentioned probably meet

the first indication there are other remedies which meet both. These are massage, warm baths, and electricity.

The proper manipulation of the limb increases the circulation in it. The increase of circulation brings fresh supplies of material to the nerve which is undergoing repair; it also aids the nutrition of the muscle, which would otherwise be decidedly affected by the sluggish flow of venous blood, due to the lack of functional activity. As soon, therefore, as the active progress of the disease is checked and the muscular tenderness has sufficiently subsided to allow the limbs to be rubbed, this potent remedy should be employed daily.

Allusion has already been made to baths and douches in the early stage, for the purpose of quieting sensory symptoms. In the chronic stage the object is a different one. Like massage, warm baths and douches stimulate the circulation and aid the nutrition and reparative processes in progress. Hence they are to be used daily. And if the warm bath be given at night it will secure not only a local action but produce a general sedative effect, insuring quiet rest. In multiple neuritis as well as in many other nervous affections not due to anæmia, a warm bath at night or a warm douche to the neck and spine is far preferable to, and more efficacious than, the majority of hypnotic drugs in causing a good night's sleep.

The last agent to be mentioned is electricity. It is, however, among the first in importance, and in its use it is necessary to know what object is sought in its application. There is first the object of increasing the progress of nerve regeneration. This may be attained by the application of a constant galvanic current to the degenerated nerve, passing the current through the nerve in either direction, or in both alternately. A mild current should be employed, its strength being measured by a galvanometer. The strength of the current will depend: (*a*) on the size of the sponges placed upon the skin, (*b*) on the pressure upon the sponges, (*c*) on the resistance of the skin, and (*d*) on the number of cells of the battery used. If the sponges are two inches in diameter, six milliampères is enough; if they are three by five inches

in measurement, twenty milliampères should not be exceeded. If no galvanometer is used the strength of the current employed is uncertain. But it is to be remembered that the current grows stronger the longer it passes, since the skin resistance is gradually overcome ; and therefore, if the strength is measured in cells, the number of cells used should be decreased gradually during the application. As so few practitioners use a galvanometer it may be well to state that with large sponges (*i.e.*, three by five inches) wet with warm water a freshly filled bichromate of potash battery will give nearly one milliampère of strength for every cell used during the first three minutes, provided the sponges be put on any part of the body except the soles of the feet or the palms of the hands, and pressed firmly upon the skin. After the first three minutes the body resistance decreases, so that when twenty cells are used to start with, one should be cut off every half minute until the number is reduced one-third. The duration of the application should be about ten minutes to each limb. During this time the distal sponge should be passed over various parts, so as to include all the nerve-branches in the current ; the central sponge should be put over the nerve-trunk high up on the limb. The current should be begun and stopped gradually, and never suddenly broken ; applications may be made daily.

The second object to be obtained is to re-establish the conduction of impulses in the regenerated nerve. This is secured by the method just described. It may be attained by the use of faradism, the interrupted secondary current being sent along the nerves by placing one pole over the nerve-trunk and passing the other over the skin of the limb. The strength used should be just sufficient to be felt distinctly through the palms of the operator's hands. The third object sought is to maintain the nutrition and function of the muscles by exercising them, and thus preserving their normal irritability. This cannot be done by a faradic current as long as they do not contract to it. The galvanic current must therefore be employed. But now it is not a steady current which is needed, for this does not cause

any motion. It is only when the steady current is suddenly broken and renewed that the contraction occurs. Hence place one pole over the trunk of the nerve and the other upon the muscle, and with an interrupting electrode make and break the current at the pole which is on the muscle. The pole which produces a contraction with the least current possible is the one to be applied to the muscle. This is in RD the positive, in normal conditions the negative pole. Each muscle should be exercised for three or four minutes every other day. When electrical treatment is thus employed very marked improvement is observed, which can be measured accurately if a galvanometer is used, since every week will show a change of the strength of current needed to produce muscular contractions toward the normal.

The treatment must be kept up, in the chronic stage, until recovery is complete.

If contractures have occurred in the paralyzed limbs persistent massage may overcome them. If it does not, they are to be treated on general surgical principles.

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CEREBRAL LOCALIZATION

IN ITS

PRACTICAL RELATIONS.

BY

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While appreciating the honor of having been chosen to open a discussion upon Cerebral Localization in its practical relations, I am, at the same time, deeply impressed with the responsibility imposed by the choice. Difficulties beset my way; and not the least of these springs from the proportions to which the subject has grown. At the Seventh International Congress, in London, in 1881, the very existence of special cortical areas was under heavy fire; but the present discussion has been determined to other channels. In the few years which have elapsed since then great advances have been made, and while different interpretations of phenomena may be still worthy of argument, the guiding principles of localization are so firmly established that the physician and surgeon can use them without question for practical purposes—even to the extent of fearlessly invading with trephine and knife the deep recesses of the brain.

From the clinical and pathological observations of practical physical physicians sprang the great conceptions out of which have developed the science and art of localization. Gall,* from outward form and on uncertain grounds, located speech above the orbits; in 1825 its pathology and morbid anatomy were first clearly indicated by Bouillaud,† who held that in the anterior lobes of the brain resided the organ of speech; and Broca,‡ in 1861, from pathological observations, definitely placed the seat of articulate language in the gyre which bears his name. In 1864, J. Hughlings Jackson,§ suggested

* Gall et Spurzheim, Anatomie et physiologie du systême nerveux. Vol. i–iv. Paris, 1810–1819.

† Traité Clinique et Physiologique de l'Encéphalite, p. 284.

‡ Bull. de la Soc. anat., T. vi. Août, 1861.

§ London Hospital Reports, vol. i., p. 459, 1864, and Clinical and Physiological Researches on the Nervous System.

that certain convolutions superintended those delicate movements of the hands which are under the immediate control of the mind; and an observation of Hitzig* that certain ocular movements and other muscular phenomena occurred during the galvanization of the heads of patients, lead in 1870, to those researches with Fritsch, which have immortalized the names of both.†

In a national Congress it might not be out of place to review American work in localization; but time will permit me to recall only a few salient facts. The researches of S. Weir Mitchell‡ on the physiology of the cerebellum constituted an early and important contribution to encephalic localization. From numerous physiological experiments, chiefly on pigeons, both by methods of ablation and of chilling or freezing, he concluded that the cerebellum was a great reinforcing organ, capable of being more or less used in volitional muscular motion; but while believing this he was not prepared to assume that it had no other function.

In 1874, a committee of the New York Society of Neurology and Electrology, as the result of carefully recorded experiments, reported conclusions largely confirmatory of those announced by Hitzig. The committee tested also the effects of excitation of the dura mater.§

Dr. J. J. Putnam,‖ of Boston, in 1874, experimented with faradic currents on the cerebral cortex and the parts immediately beneath. He first found the centres for definite, and nearly or quite uncomplicated movements, and the minimal current strength that was necessary to produce these movements, after which, with a sharp knife he made a cut underneath these centres, leaving a good-sized but thin flap which contained these supposititious centres. Having done this, he found if he irritated as before, leaving the flap in situ, the movements did not occur. Turning the flap up, however, a slightly increased current strength produced the same muscular contractions. When the flap was turned back and adjusted, and the electrode applied on its surface as at first, the contractions were not produced. Three dogs were used in the experiments, which were made by Dr. Putnam at the Physiological Laboratory of the Harvard Medical College, with the assistance of Prof. H. P. Bowditch and Dr. William James. After they were made it came to Dr. Putnam's notice that from the same methods the same results had been obtained in the same year by another observer,

* Untersuchungen über das Gehirn.

† Ueber die elektrische Erregbarkeit des Grosshirns. Reichert and DuBois-Reymond's Archiv., 1870, No. 3.

‡ Am. Jour. Med. Sci., n. s., vol. lvii., 1869, p. 336.

§ New York M. J., 1875, xxi., 225–240.

‖ Boston M. & S. J., 1874, xci., 49-52. Ibid. 1879, c., 260–262.

Braun.* Dr. J. Burdon Sanderson,† also, in 1874, had announced the same fact.

The first reported physiological experiments on the human brain were those of Bartholow,‡ in 1874, who, using both a galvanic and a primary faradic current, passed insulated needle electrodes into the brain of a patient.

In any historical reference to American work the labors of Wood§ and Ott‖ on thermic phenomena must hold a high place.

In 1884, Starr,¶ in a review of American medical literature for twenty-five years before, found records of nearly 500 cases of local disease of the brain, some of great value ; such records have since increased and multiplied, and what is better, have improved in method and accuracy. The numerous contributions of Seguin here rank first. A brain tumor was removed by Hirschfelder and Morse,** of San Francisco, February 15, 1886, the fifth case of such operation. Of 63 cases of intracranial operations tabulated by Dr. Park, 17 have been reported by American neurologists and surgeons.

The surgical aspect of cerebral localization is naturally that which appeals to all as the most practical. In this field unprecedented therapeutic results have been achieved, the crowning triumph being the relief of that most agonizing of human diseases, tumor of the brain.

Fascinated by these achievements, we incline to pass by the results elsewhere wrought—in psychological medicine and medical jurisprudence ; in general symptomatology and diagnosis ; in medical therapeutics and technique. I may, however, be allowed to devote to these a few fleeting words.

Cerebral Localization and Insanity.

Bevan Lewis,†† in 1883, pointed out some of the directions in which studies in cerebral localization might advance our knowledge of insanity, but to those I can scarcely more than allude. He held that the localization of cerebral function was the outcome of the great princi-

* Eckhard's Beiträge zur Anatomie und Physiologie, vii., 2. Also: Centralblatt. Berlin, June 13, 1874.

† Proc. Royal Soc., June, 1874.

‡ Am. J. M. Sc., Philada., 1874, n. s., lxvii., 305–313.

§ Fever: A Study in Morbid and Normal Physiology. Smithsonian Contributions to Knowledge. November, 1880.

‖ Jour. Nerv. and Ment. Dis., April, 1884. Philadelphia Med. News, July, 1885. Jour. Nerv. and Ment. Dis., vol. xiv., No. 3, March, 1887. Ibid. No. 7, July, 1876, p. 428. Ibid., vol. xiii., No. 2, February, 1888.

¶ Am. J. M. Sc., Phila., 1884, n. s., lxxxvii., 366–391.

** Pacific M. and S. J., San Francisco, 1886, xxix., 210–216.

†† Brit. M. J., London, 1883, ii., 624–628.

ple of evolution carried to its logical issues ; that the alienist should rivet his attention upon the changes undergone by the material substrata of mind ; that he should strictly and closely study the objective manifestations of mental activity ; that he should learn to examine the various limited lesions of the cortex as to area, depth, localized atrophy, relative bulk of convolutions, and tracts of ascending and descending degeneration.

Numerous isolated cases have been reported in which special mental phenomena have accompanied lesions and defects localized in particular regions—cases of lesion of the frontal lobes with affection of the intellect ; of other cortical lesions with disturbance of a speech and real or apparent mental impairment ; of others with hallucinations, visual, aural, tactile, olfactory, and gustatory; of delusion, hallucinatory or otherwise, with arrested or aberrant development of fissures and gyres. In particular, a considerable collection of visual hallucinations and delusions with localized lesions have been reported. Sir J. Critchton-Browne,* Spitzka,† and others, have contributed valuable localization observations from studies in general paralysis of the insane.

Mickle‡ has shown that lesions of the cortical sensory centrea of the cerebrum are connected in an intimate way with the production of most of the hallucinations in progressive paresis ; that from the cerebral localization point of view use may be made of the distribution of the cerebro-meningeal adhesions and the cortical changes associated therewith ; and that in all cases of visual hallucinations the angular gyre is not affected in the marked manner one would anticipate, on the theory that it is the sole cortical visual centre ; nor in cases of auditory hallucinations is the first temporal, viewing it as the sole cortical centre. The morbid anatomy of progressive paresis, he therefore believes, fails to support the exclusive view that these gyres are the sole centres of sight and hearing. The supra-marginal convolution is affected more than the angular in those with visual hallucinations, and the adhesions are often well marked on the posterior parietal lobule. The second temporal gyrus seems to suffer more than the first in cases with auditory hallucinations taken collectively.

Trephining has been performed in many cases of insanity during the last few years, a fair percentage of the operations having been guided at least in part by the principles of localization.

* West Riding Reports, vol. vi., p. 170.

† Insanity: Its Classification, Diagnosis and Treatment. Article on Paretic Dementia.

‡ Jour. Men. Sc., Oct. 1881 ; Jan. and April, 1882.

Two of the recent cases of brain operation, reported by Bennett and Gould* and by Macewen,† open a possible new field for surgical interference in insanity. In the case of Bennett and Gould, the patient had received a violent blow on the right side of the head and had a scalp wound without apparent injury to the skull. Pressure on the cicatrix caused the sensation of a flash of light followed by unconsciousness for one or two seconds. The patient had no paralysis, loss of sensation, or other symptoms, but was subject to left unilateral convulsions with loss of consciousness, commonly followed by violent mania. The attacks were usually preceded by a bright red flash of light, and were succeeded by what appeared to be threatening visual hallucinations. The scar was over the region which corresponded with the angular gyre. A large trephine opening was made by Mr. Gould, bone and dura mater were removed, and exploration was made in different directions in the brain, but nothing abnormal was detected. Five months later the patient was apparently well having had no attack during that time, although for six years before he had had on an average one fit a week. After his recovery he seemed to forget all about the hallucinations. Dr. Bennett in another case, observed similar hallucinatory phenomena, and after death the angular gyre was found to have been injured. Such cases are of importance as opening the question of the propriety of excising cortical areas as a method of treatment in insanity as well as epilepsy, when certain subjective phenomena such as hallucinations of sight and hearing can be given a local habitation in the brain.

Macewen's case was one of psychical blindness. The patient had received an injury about a year previously and suffered from deep melancholy, and strong homicidal impulses directed against his family and relieved by paroxysms of pain in the head of indefinite seat. Prior to receiving this injury he was perfectly free from such impulses and had led a happy life with his family. Behind the angular process was a slight depression, which could not account for his symptoms. No motor phenomena were present, but on minute inquiry it was discovered that immediately after the accident and, for about two weeks subsequently, he had suffered from psychical blindness. The angular gyre was exposed for operation, and it was found that a portion of the internal table of the skull had been detached from the outer, and had exercised pressure on the posterior portion of the supra-marginal convolution, while a corner of it had penetrated and lay imbedded in the brain. The bone was removed from the brain and re-implanted in proper position, after which he became

* Brit. Med. Jour., Jan. 1, 1887. † Lancet, August 11, 1888.

greatly relieved in his mental state, though still excitable. He made no further allusions to his homicidal tendencies.

Cases of double personality and double consciousness, and of unilateral hallucination, like the following reported by Magnan,* may eventually receive their proper interpretation through investigations in localization. Magnan holds that there are hallucinated individuals who hear on one side agreeable things and on the other side unpleasant. He had had under observation four cases of this kind, of which one was reported in detail. The case was one of primary monomania, complicated with epilepsy. On the right side disagreeable statements were made ; on the left ambitious ideas were conveyed. These latter hallucinations were obviously secondary to the first. He concluded, first, that these unilateral hallucinations on opposite sides were independent of local lesion ; that they did not differ from other hallucinations ; that they proved the double action and functional independence of the two hemispheres ; that analogous phenomena were noticed in hypnotic states ; and that their existence demonstrated the action of separate sensorial centres in the cortex.

Contributions of Cerebral Localization to General Medicine and Therapeutics.

The vast improvements in precision both in examining and describing the symptoms of nervous disease ; and in making and recording the results of autopsies have been largely due to the stimulus to exactness which has been given by the science of cerebral localization which has at its very foundation topographical precision.

The contributions of cerebral localization both to general and local symptomatology, if carefully brought together, would furnish material for an elaborate address. A flood of light has been thrown upon the nature of epilepsy, or rather epilepsies. Many old differential symptoms, some of them once regarded as pathognomonic, have been swept away, and better and surer criteria have been substituted in their place. The clinical teacher no longer announces that unconsciousness is the one sure sign of epilepsy, and the preservation of consciousness of hysteria ; but the question of consciousness becomes a relative one in the consideration of both diseases. We are slowly getting the data for a really scientific classification of epilepsy into reflex, toxic, cortical, bulbar, and spinal. As Mr. Horsley has recently shown, it is no longer necessary to consider hystero-epilepsy, epileptiform seizures, laryngus stridulus, and eclampsia as altogether apart from epilepsy.

* Journal de Médicine de Bordeaux, Sept. 30, 1883.

Not a few symptom-groups or symptoms formerly not understood at all, and some of them regarded as independent diseases, have been given their proper positions; such affections, for instance, as athetosis, tetany, and some spastic diseases of children. Vagueness has given place to clearness with reference to such affections as cerebral softening; and new light has been thrown upon such common and important diseases as tubercular meningitis, particularly as it affects the convexity of the hemispheres.

Now and then a new experiment or observation on cerebral localization has let in the light upon some obscure symptom or condition known to the physician. That peculiar perversion of sensory localization known as allochiria was noticed, for instance, by Horsley and Schäfer,* as the result of lesions produced by them in the limbic lobe.

Something has been accomplished with reference to the action of drugs on localized cerebral areas. I might point to the investigations of Albertoni† as to the augmentation of the excitability of the cortex by atropine, and the action of bromide of potassium in reducing the same excitability, a conclusion which has since been confirmed by Rosenbach and others, and is in accord with all clinical experience; to the work of Luciani and other Italian observers on chinconidine and pyrotoxine as epileptogenic agents; and to the experiments of Tamburini, Seppilli, Hitzig, and Franck into the effects of anæsthetics and narcotics on critical areas. Franck‡ has thoroughly investigated the effects of curarization on cortical excitability, and some of his results may prove of medico-legal importance in the study of masked or hidden epilepsy. Danillo, Magnan, and Franck have made important observations on absinthine epilepsy.

Experiments and discoveries like those of Eulenberg and Landois,§ Wood,‖ Ott,¶ Richet,** Aronsohn and Sachs,†† Wood, Reichert and Hare,‡‡ and Girard,§§ on the existence and phenomena of heat centres in the brain, have been of practical value in throwing light on

* Phil. Trans. Royal Soc. of London, vol. clxxix, (1888), B. pp. 1–45.

† Cortical Epilepsy. Experimental Researches. Synthetic Review. By Greuseppe Seppilli, M.D., Alienist and Neurologist, January, 1885. Translated by Joseph Workman, M.D., from the Rivista Sperimentale, 1884.

‡ Leçons sur les Fonctions Motrices du Cervean, Par le Dr. François-Franck, Paris, 1887.

§ Compt. rend. Acad. de Sc. Par., 1867, lxxxii., 564–567. ‖ Op. cit. ¶ Op. cit.

** Bulletins de la Société de Biologie, March 29, 1884.

†† Deutsche Medicinische Wochenschrift, No. 51, 1882, and Pflüger's Archiv.

‡‡ Therapeutic Gazette, vol. ii., 3 s., No. 9, September, 1886, p. 577.

§§ Arch. de Physiol., norm. et path., Paris, 1886, 3 s., viii., 281–299.

the mechanism of fever, and on the action of special drugs and different modes of treatment on forms of high temperature. I will refer very briefly to some of the experiments and inferences of these observers, simply to show their practical tendencies.

Wood, for instance, holds that with the facts of his experiments in mind, the theory of a causation of fever becomes very plain. "It is simply a state in which a depressing poison or a depressing peripheral irritation acts upon the nervous system which regulates the production and dissipation of animal heat; a system composed of diverse parts so accustomed to act in unison continually in health, that they become as it were one system, and suffer in disease together. Owing to its depressed, benumbed state, the inhibitory centre does not exert its normal influence upon the system, and consequently tissue change goes on at a rate which results in the production of more heat than normal, and an abnormal destruction and elimination of the materials of the tissue. At the same time the vaso-motor and other heat dissipation centres are so benumbed that they are not called into action by their normal stimulus (elevation of the general bodily temperature), and do not provide for the throwing off of animal heat until it becomes so excessive as to call into action by its excessive stimulation even their depressed forces. Finally, in some cases of sudden and excessive fever, as in one form of so-called cerebral rheumatism, the enormous and almost instantaneous rise of temperature appears to be due to a complete paralysis of the nervous centres presiding over heat production and dissipation."

Girard,* as the result of certain experiments on rabbits, concludes that the cerebral centre of thermo-genesis is in the corpus striatum. Lesion of the median portion produced well marked increase of heat, and this was not the result of spasm of vaso-constrictor nerves of the skin. Exciting the region electrically caused a notable increase of heat, showing that this resulted from excitation and not from paralysis. Similar excitation caused increase of urea, indicating an increase of combustion in the organism, which was accompanied by considerable emaciation. Girard believes this apparatus or centre increases the heat under excitation, and notably influences the regulation and production of heat; also that artificial increase of heat is not identical with that of fever. Increased production, and at the same time diminished dispersion of heat, from the body are, according to his view, the two conditions essential to fever.

One of the latest contributions of Ott is on the heat centres of the cortex cerebri and pons varolii. He found that when in his experi-

* Gazetta Degli Ospitali, Aug. 17, 1887.

ments upon rabbits, a puncture was made just in front of the ear into the cortex, there ensued a fugitive rise of temperature ; and this observation led him to try in cats the effects of removal of areas of the cortex in this and other regions. A point at the juncture of the supra-Sylvian and post-Sylvian fissures was found to have the highest thermic value. Other parts of the brain, with the exception of the cruciate centres, had but small effect upon the temperature. The rise of temperature after injury to the Sylvian centres was from three to four degrees, and continued till the death of the animal, which was usually about the fifth or sixth days. The calorimetric investigations showed that either immediately, or at the end of twenty-four hours, the heat production and heat dissipation were increased ; after that they usually fell below normal, although the temperature remained elevated, with a weight decreasing daily. He believes that this increase of heat production was not due to secretory changes ; as pulse and pressure both rose for a short period, and then fell to a certain extent below normal, although the temperature was then rising.

The mechanism of temperature production, according to Ott, is : (1) Thermotaxic centres, cruciate and Sylvian of Eulenberg and Landois ; (2) Thermotaxic and thermo-genetic centres—the centre about Schiff's crying centre, and the extra striate, (Sachs and Aronsohn), and the thalamic centres ; (3) Thermogenetic centres—spinal centres.

"It is probable," says Ott, "that after injury to the cortical heat centre, the basal and spinal thermogenetic centres are temporarily permitted to obtain the upper hand, but that shortly the other cortical heat centres bring the thermogenetic centres into subjection, and thus reduce the heat production. In the case of lesion of the basal and spinal thermogenetic centres, for a short period they primarily overcome the cortical centres, but finally succumb to the domination of the thermotaxic centres of the cortex. In other words, the Sylvian and cruciate centres constantly antagonize the basal and spinal thermogenetic centres. It is also probable that under certain impulses the cortex and basal centres combine together to antagonize the spinal thermogenetic centres. It would seem that an injury to the thermotaxic or thermogenetic apparatus sets up a fever which is primarily accompanied by increased production and dissipation ; but they soon fall below normal, whilst the fever continues till the lesion is repaired. This would lead to the belief that in continued fever the generation of a ptomaine is continuously carried on for some time, and thus keeps up the fever."

Scarcely anything as yet has been contributed by these investigations to the surgical aspects of the question ; but a case reported by Mr. Page* has at least some suggestive value, and is the only one to

* Lancet, London, 1887, ii., 1216.

which I will allude. A man, from a fall, had a wound one inch in length in the right parieto-occipital region. He was put to bed and became dull and apathetic; his temperature rose until it had reached 105° F., but otherwise he presented no symptoms that could be determined. Trephining was performed over the posterior part of the temporo-sphenoidal lobe. The patient's high temperature rapidly subsided and he recovered without other symptoms.

Before leaving the consideration of these questions of general symptomatology and therapeutics, it might be well briefly to refer to what has been accomplished in cerebral localization with reference to some of the organic or involuntary functions. Are there circumscribed localized areas in the cerebrum which are capable of producing certain so-called organic or involuntary effects, or effects which may be classed as somewhere between the purely voluntary and the involuntary? In other words, to put the question in its simplest expression, have we centres—comparable to those which give definite motor reactions—for such functions as those of respiration, heart-action, vascular tone, oculo-pupillary movements, the secretion of sweat, saliva, and bile, or the excretion of urine? No one has studied this subject with the thoroughness and originality of François Franck in his great work on the motor functions of the cortex, to which reference has already been made. His conclusions are based chiefly upon the results of irritation of various regions of the brain. He found excitation of the brain in various regions of the cortex efficient to produce organic and partly organic manifestations, but such areas were not circumscribed and invariably the same. Changes in respiration, arrest or increase of the movements of the heart, flushing or paling more or less local or general, suppression or increase in the flow of saliva, sweat, bile, urine, etc., could all be brought about by experimenting upon the cerebral cortex of dogs and monkeys. Such results, however, he does not believe should be regarded as simple reactions comparable to the definite movements caused in face or limb by irritation of the centres assigned to these parts. They are complex results more comparable to the reflex effects produced from irritation of sensory surfaces anywhere. He shows that the suppression of the cerebral region, whose excitation so clearly produces organic effects, does not cause the loss of the function put into action by the excitation.

These views are probably correct, in the main, although they may receive some modification with increase of knowledge upon this subject. Regions of the brain in the process of evolution have been differentiated into definitely localized centres of representation in pro-

portion as the functions represented have become more and more volitional, more and more under the control of the individual. We can never probably have localizations for organic manifestations which will be available, for instance, for the purposes of the surgeon. A closer and fuller study may show the truth to be largely the same even for the so-called thermic or heat centres. It is altogether doubtful whether we have distinct vaso-motor cortical centres comparable to the simple centres for motion. In reference to this question, Franck says that all localizations of this kind ought to be renounced. The cortical surface, he says, agrees in a certain degree with the sensory surface, and does not contain the vaso-motor centres any more than the organic centres, whatever they may be; the cortex fills the roll of separation, and not that of a productive organ of visceral reactions. The true vaso-motor centres are contained in the bulb and spinal cord. They receive the cerebral excitations as they receive the peripheral excitations, and react in both cases in a reflex manner in consequence of a similar mechanism.

Such localizations as those of Christiani* of higher respiratory centres must not be regarded in the same light as motor, visual and other independent simple localizations. This investigator, as the result of a series of experiments on rabbits and dogs, believed that he had found higher respiratory centres, three in the basal ganglia; first an inspiratory one, chiefly reflex, at the bottom of the third ventricle; second, one, also inspiratory, at a point between the anterior and posterior corpora quadrigemina; third, an inspiratory and inhibitory centre at the entrance to the aqueduct of Sylvius. He also discovered, anterior to the inspiratory centre in the third ventricle, a coördination centre.

To speak of emotional centres in the same sense that we do of motor, visual, or auditory centres, is also unphilosophical. In certain organic brain lesions, says Pontoppidan,† emotional manifestations such as laughing or crying appear without a cause; or an emotional cause produces undue effects, a pain, for instance, produces laughter. Such symptoms are usually met with in disease of the pons and oblongata. The investigations of Pontoppidan seem to show that the centres affected in such cases are those in the vicinity of the vaso-motor centre in the pons. He describes in detail three such cases. In the first, any question caused the patient to laugh; in the second, laughing or crying occurred indiscriminately when any attempt at conversation was made by the patient; in the third, fits of laughter occurred without any apparent cause—the mere entrance of any one into the room would

* 36 DuBois Arch., 1884. † Centr. f. Nervenheilk., 1887.

produce one. In two of these cases autopsies showed the existence of apoplectic clots in the crus cerebri and pons Varolii, and other symptoms of pons disease were present.

In the nervous wards of the Philadelphia Hospital are several cases similar to those described by Pontoppidan, other symptoms pointing also to disease of the pons. Such facts, however, do not indicate the existence of a special centre for emotion, comparable in any true sense to the circumscribed centres of the cerebral cortex; but rather point to the fact that in the pons-oblongata we have crossing and interblending the various tracts, ascending, descending and transverse, which unite the higher regions of the nervous systems to those lower centres which energize the nerves and muscles concerned in the expression of emotion, or join together these lower centres and the cerebellar hemispheres.

Cerebral Localization in its Relations to Surgery.

Let me now turn to the surgical aspect of this great subject—the surgical aspect in so far as it concerns the neurologist; it is upon this that the attention of the medical world is riveted to-day.

In this portion of my remarks, I will consider (1) the forms of disease and injury in which cerebral localization is a valuable aid to diagnosis; (2) the parts of the brain accessible to surgical interference, and the topographical diagnosis for these accessible areas, with some sources of error in diagnosis.

The neurologist is now constantly called upon with the surgeon for the relief of intra-cranial affections long held not to be amenable to treatment, and scarcely worthy, from a practical point of view, of diagnosis. My remarks must be chiefly concerned with questions of diagnosis.

Examination of medical literature shows that operations upon the brain, guided by localization, have been for tumor, cyst, fracture, abscess, hemorrhage, and discharging cortical areas.

Brain Tumors.

It may be broadly affirmed that brain tumors should be removed by operation when their exact position can be diagnosticated, when they are in accessible areas, when they are solitary, and when they are not of enormous size. Of Dr. Park's 63 cases, 11 are cases of tumor, and 12 of cyst; of 17 operations by American surgeons, 5 have been for tumor. In this connection I will only stop to give some facts and draw some inferences from personal experience; it is often wise to review personal experience

even if in so doing we sometimes awaken vain regrets. I have notes of 20 cases of brain tumor with autopsies, most of which have already been published in some form. Hale White's* cases numbered 100; and Seguin and Weir,† combining the statistics of White and Bernhardt, tabulated 580 cases. Twenty cases are comparatively few, but such a list has the advantage of thorough personal knowledge. Of these 20 cases the locations were as follows: Prefrontal lobe, 2 cases; posterior portion of second frontal gyre, 1 case; motor (Rolandic) zone, 6 cases; superior parietal lobule, 1 case; temporal lobe, 2 cases; cerebellum, 2 cases; mid-base and corpus callosum, 1 case; pons-oblongata, 4 cases; optic thalamus, 1 case.

Twelve out of the 20 cases were in areas accessible to operation; one of the accessible cases was multiple. Of the 11 accessible cases left, 4 were fibromata, 3 gummata, 2 tubercular, 1 a carcinoma, 1 a glioma with intercurrent hemorrhage. In neither of the 2 tubercular cases would operation have been successful because of the diffusion of cerebral tubercular disease. The carcinoma and glioma would probably have given only temporary success. Of the 7 cases left, all could probably have been removed successfully by operation at some stage of their growth; although in 3 of the cases, at the time of death, the tumors were of such size, and the break-down of brain-tissue in their neighborhood was so great, that the operation then would probably not have resulted in success. In at least 4 of the 20 cases, operation at any time before death would, in all probability, have been wholly successful. Is it any wonder that vain regrets for lost opportunities sometimes arise?

I favor the removal of old gummata, and this opinion is based upon considerable experience. Again and again I have seen such growths resist the most active and persistent anti-syphilitic treatment. It is probable that one reason why they will sometimes not yield to medicinal means is because in the progress of their growth they have obliterated blood vessels and become practically inert foreign bodies. Bergmann and White oppose, and Seguin favors the removal of gummata.

Cranial Fractures.

Localization rules are sometimes of value, even in cases of visible and easily detectable fractures, with lacerations, scars, clefts, depressions or ridges. These rules may be called in to clear up obscure points. Often in cranial fractures the extent of unseen damage cannot be told

* Guy's Hosp., 1884–85, 3 S., xxviii.

† Am. Jour. Med. Sci., July, August and September, 1888.

by the position and character of visible lesions. Numerous cases have been reported in which the operators would have been misled by trusting to external evidences alone; but in which by calling in the established facts of localization to assist they were able to place the trephine over the best spot for operation. Examination of surgical literature also shows that in many cases, demonstrated by autopsies, if the rules of localization had been properly applied, the site of hidden fractures either of the internal table or not, could have been determined and operations performed to the great benefit of the patient.

The best point for trephining in cases of fracture is not always the place of the greatest depression or cleavage, or over the centre of a large scar. In fracture cases the symptoms of dural irritation will often be prominent, and, particularly when the injury is over the motor area, may confuse the picture of spasm which is presented. The spasm may be dural or reflex, rather than cortical, or may have a mixture of reflex and cortical characteristics; and hence may be on the same side as the lesion or general, and thus involve the mind of the diagnostician in some doubt. An abscess resulting primarily or secondarily from a fracture may be so situated, or may have so enlarged, that localization rules alone can determine the best site for trephining. According to Jacobson,* out of 70 cases of middle meningeal hemorrhage a fracture was present in 62; so that in the majority of instances both fracture and hemorrhage must be taken into account.

Intra-cranial Abscess.

The question of intra-cranial abscess as well as fracture will be fully treated by Dr. Park and I will therefore say but little about these subjects. The cases of abscess in which localization rules have given the most brilliant results have been those in which without external evidences, a position for operation has been fixed. Several brilliant operations guided by cerebral localization have been recently reported, one of the most striking of these by Ferrier and Horsley.† This patient first complained of pain in the left ear, later a discharge, first clear and then of blood occurred. He became stuporous, had pain in the left side of the head, forehead, and back of the eyes, and photophobia. Later he became delirious and showed relative weakness of the right side of the face, a peculiar form of aphasia, and slight paresis of the right upper limb especially of the hand and digits. He had well marked optic neuritis, with a small hemorrhage over the right disc, and a small band below that of the

*Guy's Hosp. Rep., 1884–85, 3 S., xxviii, 147–308.

†Brit. Med. Jour., March 10, and March 24, 1888.

left. His speech disturbance was peculiar. He was able to sit up in bed and talk, but his words were incoherent and for the most part unintelligible. He appeared to understand simple questions, but at other times seemed confused and unable to understand. He called things by wrong names. When asked to read a few sentences from a journal, the words he uttered had little or no resemblance to those before him. In addition to the involvment of the auditory centres there was probably here also a fracture between the receptive and emissive speech regions. Mr. Horsley operated for the locality determined by Dr. Ferrier and himself; about five drachms of pus were removed and the patient recovered. Dr. Ferrier refers to other operations reported by Gowers and Barker, Greenfield, Schondorff, and Truckenbrod, the first two having been cases of abscess in the temporal lobe diagnosticated without external indications.

Intra-cranial Hemorrhage.

In a large number of cases of intra-cranial hemorrhage trephining has been performed, successfully or unsuccessfully. I have collected many of these cases, but cannot refer to them here except in the most general way; they constitute in themselves material for a lengthy paper. During recent years some important operations for such cases of hemorrhage have been guided by the principles of cerebral localization. Dr. J. B. Roberts, of Philadelphia, in his monograph on The Field and Limitation of the Operative Surgery of the Human Brain, of American authors has most thoroughly discussed the questions of operative interference in these cases of intra-cranial hemorrhage, as well as in fractures, abscess, tumors, and other lesions.

Intra-cranial hemorrhage may be (1) supra-dural; (2) sub-dural; (3) cortical or sub-pial; (4) intra-cerebral, that is, into the basal ganglia, capsules, or both, or into the centrum ovale. The first two forms are commonly due to lesions of the meningeal arteries, chiefly the middle meningeal, and are frequently associated with fracture, and occur from injury. Cortical or sub-cortical hemorrhage has its source in the cerebral arteries proper, most frequently in the cortical system of the middle cerebral.

These cerebral arteries have also a central or ganglionic system of branches, independent of the cortical, and it is from this arterial network that the ganglionic or capsular hemorrhage occurs. Hemorrhage into the centrum ovale may occur from the terminal vessels of either the cortical or ganglionic system.

Hemorrhage from contre-coup often calls for the application of the principles of localization. In cases of contre-coup the lesion, however, is often a form of bruising of the brain and its membranes with but little hemorrhage, for which trephining would be of no especial service, and it is important to distinguish such cases from those in which a genuine hemorrhage is present.

The forms of hemorrhage most amenable to topographical diagnosis and operative procedure are from the meningeal arteries proper and from the cortical system, that is, supra-dural, sub-dural and cortical. True cortical hemorrhage is comparatively rare, and meningeal hemorrhage comparatively frequent. Sometimes, instead of coming directly from a meningeal artery, the bleeding may be from the diploe of the fractured skull.

According to Kronlein,* the most frequent site of intra-cranial hæmatoma is the middle fossa of the skull, such lesion being usually limited in front by the lesser wing of the sphenoid, and behind by the margin of the petrous portion of the temporal bone, because of the adherence of the dura mater at these places ; below they reach nearly to the foramen spinosum, and above to the squamous suture, sometimes crossing the latter. The effusion is always thickest at the site of the rupture.

The symptoms of middle meningeal hemorrhage and supra-dural clot are both general and localizing. The general symptoms are such as loss of consciousness, and, in cases of traumatism, an interval of consciousness before the appearance of pressure symptoms ; change in temperature, usually elevation ; somnolence, stupor or coma ; slow pulse, sometimes becoming frequent at last ; slow, labored respiration ; vomiting. A small hemorrhage may give rise to few, if any, serious general symptoms.

Supra-dural Hemorrhage.

The symptoms of extravasation, when the hemorrhage is supra-dural are chiefly general. Contra-lateral paralysis, however, when the bleeding is over the motor area, may serve as a broad localizing indication when external appearances are wanting. Certain other phenomena are also usually present.

Unilateral affection of the pupil is often a sign of the utmost importance, particularly if, says Jacobson, one pupil is found widely dilated, the other being natural or contracted in size, and if the dilatation be on the side of the face corresponding to the injured side of the head. Mr. Jonathan Hutchinson has particularly studied and discussed

* Quoted by Jacobson.

the importance of this valuable symptom, and in honor of him Jacobson proposes to call it the "Hutchinson pupil." Hutchinson regards the symptom as due to direct or indirect compression of the third nerve. The pupils also furnish valuable indications as to the probability of recovery. The more dilated, insensitive and immovable they are, the less favorable the prognosis.*

Of the many cases of supra-dural extravasation which have been reported, in very few have the symptoms been studied closely.

Sub-dural Hemorrhage.

Sub-dural, or intermeningeal hemorrhage, if extensive, gives general symptoms much like those which are present in supra-dural clot, namely, loss of consciousness, changes in temperature, pulse and respiration, vomiting, etc. A sub-dural clot will usually to a greater or less extent bruise, and possibly even tear the brain surface. Spasm due to irritation of the motor cortex may be present, as well as dural or reflex spasm. Paralytic symptoms will be definite and pronounced if the lesion is in the motor region. Cheyne-Stokes breathing may or may not be present. The following are condensed notes of three out of a number of cases of this kind, of which I have collected the histories.

The first is a case of unilateral meningeal hemorrhage with contra-lateral symptoms, reported by S. N. Townsend Porter.† The patient was a woman admitted to the hospital unconscious, with Cheyne-Stokes respiration, which became stertorous and puffing. Paresis of arm and leg on left side, mouth slightly drawn to the right side, and left naso-labial fold almost obliterated.

* Recently I had the opportunity of seeing an instructive case of supra-dural clot in the Philadelphia Hospital, in the wards of my colleague, Dr. F. X. Dercum. I will only refer to the case briefly, as it will doubtless be more fully reported by Dr. Dercum. The patient was a plethoric young man who came into the hospital without history, having been found in a stable insensible. Temperature, 95° F.; respiration stertorous —in breathing only the right nostril dilated and the right side of the mouth puffed; pulse weak, intermittent. The patient was insensitive to all impressions. Both arms and legs were spastic; the former drawn upwards and across the chest; the latter extended, the feet turned somewhat inwards. Occasionally jerking movements of both arms occurred. The head turned toward the right; the right pupil was dilated and dilating while the patient was under my observation; there was also right external strabismus. The patient died a few hours after these observations were made. The autopsy showed a bruised appearance of the skin about an inch above and to the right of the occipital protuberance. No depressed fracture was present, but a slight cleavage of the external table of the skull, and an extensive radiating or stellate fracture of the inner table. An immense supra-dural clot was found covering the lateral aspect of the parietal, and largely of the occipital lobe. The clot was back of the motor area.

† St. Louis Med. and Surg. Jour., 1887, vol. lii., p. 76–78.

Only moved the right extremities; the head turned toward the right. Feeble convulsion lasting three minutes at night. Both sides affected, but left much less so than the right. Next day head and right eye showed marked deviation to right side. A clot was found between dura and pia weighing 170 gmms ($5\frac{3}{10}$ ozs.). It covered almost the entire right hemisphere. Gyri of right side were slightly flattened and of pinkish hue. Puncta vasculosa marked. The ruptured vessel not found.

The second is a case of inter-meningeal hemorrhage with general symptoms, reported by Clemen.* Female, 67 years. Intense headache, chiefly frontal; worse from 8 to 10 P. M. At times wakeful and restless for days together, and then would become drowsy and semi-unconscious. No motor paralysis; incontinence of urine: general hyperæsthesia; cerebral breathing at times; sometimes twitchings of flexors and pronators of both forearms. Old bloody intra-meningeal effusion was found between dura and arachnoid, over both hemispheres, extending into the middle posterior fossæ of the skull on the right side; only in the middle fossa on left side the clot was thickest in convexity. Also some adhesive meningitis, supposed to have been due to slow simultaneous multiple capillary hemorrhage.

Dunn† has recorded the details of a case of clot over the motor area causing rhythmical motions of the other side of the body. The patient was a female 73 years old. Congestive apoplexy. (?) Fair recovery in a few days. Second attack during the night. Regularly recurring rhythmical movements of the left side of the body. Sensation and consciousness were normal. The right side of the body could be moved at will. Articulation impossible. Incontinence of urine and fæces. The movements of the body continued during sleep, and gradually lessened, leaving the leg on the fourth day and arm on the fifth. A clot as big as a hen's egg was present on the right side of the brain: this was superficial, reaching from the pre-central gyrus to the occipito-parietal fissure, and from the longitudinal fissure to the temporo-sphenoidal lobe.

Cortical Hemorrhage.

Sub-dural or inter-meningeal hemorrhages are frequently also cortical, that is, they invade or involve the pia-arachnoid and cortex. Occasionally, however, cases of intra-cranial hemorrhage occur which may be more particularly classed as cortical or sub-pial. These are usually limited in size, and often take place from arterioles or capillaries. A case reported by Horsely‡ illustrates what is meant by one of the forms of true cortical hemorrhage.

This was the case of a man who had been suffering from tubercular disease of the bone for some months, and suddenly developed symptoms of thrombosis of the longitudinal sinus with cortical epilepsy as the result. The case is interesting not only as one of a peculiar form of cerebral hemorrhage, but also because of its teachings with reference to the area for the turning

* Medical Press and Circular, 1886, vol. i., p. 335–336.

† Jour. of Am. Med. Ass., 1886, vol. ii., p. 75–76.

‡ Brain, April, 1888.

of the head and eyes to the opposite side, and, at the same time, the anterior limit of the upper limb area, together with the special representation of the segments of that limb at the anterior part of the region devoted to it. "The movements observed were first turning of the head to the left; then raising the arm at right angles to the trunk in complete extension, with extreme extension of the wrist and interosseal position of the fingers; gradual turning of the head to the right, and subsequently the rest of the body involved in the spasm."

Thrombosis of the sinus and veins was present and caused the following lesions. "*Right hemisphere.*—The surface of the hemisphere appeared perfectly normal, except in the neighborhood of the blocked frontal vein before described. The posterior sixth of the middle frontal convolution in its whole breadth was the seat of a hemorrhagic extravasation. The ascending frontal convolution was highly congested, especially in its anterior border; the membranes also of the superior frontal sulcus were congested along its posterior third, and there was a slight hemorrhagic extravasation in the outer border of the middle third and the superior frontal convolution of this side (the right). *Left hemisphere.*—There was a dark black hemorrhagic focus occupying the anterior half of the middle third of the superior frontal convolution for half its breadth. This, the only lesion in the left hemisphere, was situated at the highest point of the area for the head and neck in the left hemisphere."

Small, superficial, cortical extravasations of this kind are to be localized by the rules and principles for irritative and destructive lesions of the brain surface of whatever character.

Intra-cerebral Hemorrhage.

Intra-cerebral hemorrhage will next engage our attention. Of course a hemorrhage may take place anywhere within the cerebrum—in the pre-frontal, postero-frontal, parietal, occipital or temporal lobe, but we cannot stop here to differentiate between the varieties of hemorrhages occurring in these positions. The remarks upon the localization of lesions of any kind in these locations will in large part apply to hemorrhage. In this connection the discussion will be largely confined to those varieties of intra-cerebral hemorrhage which are most common, and which might be said to have become almost classical—the cases of hemorrhage into or near the great ganglionic masses.

Commonly intra-cerebral hemorrhage occurs, as Gendrin and Charcot * have pointed out, not in the body of either the caudate or lenticular nucleus, but rather just in contact with the external surface of the lenticular ganglion. Not infrequently small hemorrhages occur in these positions. When a large hemorrhage occurs, it forces its way

* To Charcot we are indebted for our most exact knowledge of this branch of the subject of localization.

especially in a transverse direction, tearing through and pressing aside the brain substance, the greatest compression taking place towards the lateral ventricle because the resistance is least in this direction. Symptoms of both destruction and pressure abound in such cases and are sometimes hard to separate. Sometimes the hemorrhage breaks through the ganglia and the internal capsule and inundates the ventricles.

The central branches of the middle cerebral artery play the most important role in such hemorrhages. Charcot * has indeed proposed to call one of the branches of this middle cerebral artery "the artery of cerebral hemorrhage." This vessel after having entered the third segment of the lenticular nucleus traverses the superior portion of the interior capsule and then enters the body of the caudate ganglion. In rare cases the surgeon might trephine successfully for intra-encephalic hemorrhage. This must be done, if at all, at a point where it has been determined by pathological observation that the hemorrhage in its enlarging waves outwards usually comes nearest the surface, or would be most easily reached and relieved. The cases of hemorrhage in which the ventricles are broken into and inundated would probably be benefited only very rarely by operation, but no harm could be done in such an almost necessarily fatal case.

Intra-cerebral hemorrhage may occur in any one of half a dozen positions with reference to the three great ganglia at the base of the brain, and the internal or external capsule. With our present knowledge the exact position of some of these hemorrhages cannot from any localizing data be accurately determined. It remains true now, as stated by Charcot ten years ago, that lesions confined to any one of the gray central ganglia when the internal capsule is not involved, do not give any special diagnostic features. We have no characteristic symptoms based upon a knowledge of the functions of these ganglia. Certainly a hemorrhage or other lesion cannot yet be very positively determined as limited to either the caudate or lenticular body or the thalamus.

With reference to hemorrhage without ventricular inundation several locations in or near the ganglia may be diagnosticated. If the hemorrhage has occurred at a position corresponding to the anterior half, or perhaps two-thirds of the lenticular ganglion and internal capsule, the chief effect is the production of motor paralysis of the opposite half of the body with symptoms of the acute apoplectic attack, which symptoms are practically the same for all

* Lectures on Localization in Diseases of the Brain. Translated by Edward P. Fowler, M.D., New York, 1878, p. 73.

the non-ventricular varieties. If the hemorrhage has occurred so as to be related to the posterior third of the capsule, where it lies chiefly between the lenticular body and the thalamus, paralysis both of motion and sensation of the opposite side of the body will be the great feature. When the extreme posterior limit of the internal capsule and ganglia are the seat of extravasation, contralateral hemianæsthesia without hemiplegia will be present; but this variety is comparatively rare. Many facts with regard to the regional diagnosis of such hemorrhages have been given by Charcot. It does not come within the purpose of my paper to discuss the exact arteries affected, and various other collateral matters anatomical and pathological, but I wish simply to give the persisting diagnosticating features of these forms of hemorrhage; and the symptoms usually observed at the time of the apoplexy. The latter are loss of consciousness, more or less complete according to the extent of the hemorrhage; stertorous respiration, sometimes so far as the mouth is concerned, one-sided; sometimes also Cheyne-Stokes; temperature at first lowered and afterwards rising; pulse sometimes slow and full, sometimes weak and intermittent. Conjugate deviation of the head and eyes may be present but is not invariable; it is usually away from the side of the paralysis. It is not infrequently somewhat difficult to determine the full extent and character of the paralysis and loss of sensation, if this also be present, in these cases of apoplexy. Careful inspection of the face, however, will usually show some drooping on the side of the paralysis and some pulling to the other side. Watching the limbs, the unparalyzed members will be seen to be used by the patient occasionally. The paralyzed extremities when taken hold of are usually limp and offer no resistance, while a certain amount of resistance is offered by the limbs of the other side even though the patient may be unconscious. My experience has shown me that cases of even somewhat extensive extravasation into the capsules and ganglia, differ considerably in the amount of paralysis produced. A fuller knowledge of intra-cerebral localization may eventually throw light upon these differences. In general terms the paralysis of the limbs is usually much more complete than in cases of cortical lesion.

The following notes of a recent case of intra-cerebral hemorrhage restricted to the internal capsule and ganglia will serve to illustrate one of the forms of hemorrhage. The patient, a man 62 years old, was admitted to the Hospital in a nearly unconscious condition. When first admitted he had some use of all his limbs; but he gradually became worse and in the course of twelve hours could not respond intelligibly to anything that was said to him, but even then he could be aroused so that he would open his eyes and look

around for a few moments, and then sink again into a stupor. When able to speak his articulation was thick and indistinct. For at least twelve hours he certainly understood what was said to him. His breathing was puffing and gradually became more stertorous. It never assumed the true Cheyne-Stokes type, but showed an occasional tendency to do this. After he had become totally unconscious a few conditions were positively determined. The mouth was drawn slightly but distinctly to the left; his right arm was paretic; the right leg was helpless and spastic. The left leg also remained nearly all the time as if powerless, and it was difficult to determine any difference as to loss of power of the two extremities. He had not true conjugate deviation of the head and eyes, although his head at times showed a tendency to turn to the right. The pupils were equal and slightly dilated. Knee-jerk was present and marked on the right, diminished on the left. His head temperature, taken once at a spot corresponding to a point just below the middle of the horizontal branch of the Sylvian fissure, was 96.2° on the left, and 100.4 on the right. The patient lived six days from the time of his admission. His body temperature when first taken was 96°. It rose the second day to 101°, and from that time on until his death, ranged between 99° and 103°, being at the highest point at the time of death. He developed pneumonic symptoms three days after admission.

At the autopsy, on exposing the left lateral ventricle a nearly black, irregularly shaped spot was seen reaching across the caudate nucleus where it begins to curve around the thalamus. This appearance indicated a recent clot which had not quite broken into the ventricle, still having a thin roof formed by a layer of the caudate body. The ganglia and capsules were studied by transverse sections. The anterior limit of the extravasation was towards the median line of the brain, and was three-fourths of an inch from the head of the ganglia. Its posterior limit, a narrow wedge, was one-third of an inch in front of the posterior extremity of the thalamus. The blood was still fluid, and the parts involved by the clot were chiefly the middle portions of the lenticular body and internal capsule, and an external anterior segment of the thalamus. The pia mater of the convexity was œdematous and opaque, in spots and patches hyperæmic, and Pacchyonian granulations were exuberant. The blood vessels were highly atheromous. The kidneys showed interstitial nephritis. One lung was nearly solidified, and a patch of consolidation the size of a lemon was found in the other.

In this case the hemorrhage probably occurred slowly and most likely at the site of an old cyst. In cases of rapid hemorrhage in the same locality, all the general symptoms such as loss of consciousness, changes in respiration, temperature, etc., would be more sudden and complete. If breaking into the ventricles should occur it would become more profound and threatening.

In this case, as in others, I made some experiments to determine whether the extravasation could have been reached by trephining. A needle or trocar passed through the upper portion of the third tem-

poral convolution, or at the line of junction of the second and third, about 3 inches back of the anterior extremity of the temporal lobe, in a direction forward and downward reached the clot at a distance of about an inch from the surface. It would be necessary if trephining was attempted to thus enter the temporal lobe, low down and well back so as to avoid the Sylvian fossa and island of Reil. In a highly vascular territory like the Sylvian fossa the cortical vessels are large and near their origin from the middle cerebral and internal carotid arteries, and if in operating this fossa was carelessly penetrated, more harm than good might be done to the patient. The peculiar position in which the ganglia and capsules are located with reference to the Sylvian fossa, the island, and the descending horn of the ventricles, would constitute one of the chief sources of difficulty in attempting to trephine for intra-cerebral hemorrhage. Still the operation is not impossible, and we will probably eventually learn exactly how far it can be resorted to with advantage, probably only in a very limited number of well chosen cases.

Intra-cerebral Hemorrhage with Inundation of the Ventricles.

What now are the symptoms of intra-encephalic hemorrhage with ventricular inundation? Whether this form of hemorrhage is or is not susceptible of improvement by operative interference, its diagnosis has considerable negative practical importance. I have, for instance, known the diagnostic question chiefly discussed in an important case to have been, whether the patient was suffering from a hemorrhage which had burst into the ventricles, or from supra-dural or sub-dural clot of immense size. Certainly as I have seen the cases there are striking points of resemblance between some cases of ventricular and some of meningeal hemorrhage; but the points of difference are sufficient to separate the varieties if we are sufficiently careful and minute in our study.

In the Philadelphia Medical Times, for October 23, 1880, I published a history of an interesting case of hemorrhage into the basal ganglia followed by effusion of blood into and beyond the ventricles, and I have studied and made autopsies upon other similar cases. In the case reported, the patient, a man 63 years old, while eating his dinner suddenly fell unconscious; his breathing became puffing, and marked right-sided paralysis was at once observed. The right arm and leg were powerless, and inspection showed that both the upper and lower muscles of the face were paralyzed. The right eye remained partly open and the mouth was pulled decidedly to the left. Two hours after the attack it was noted that he was profoundly unconscious; his face was pale; the right eyelids did not quite close; the pupils were sluggish

but equal, the eyes were directed straight forward; conjunctival reflex was present; the mouth was drawn slightly to the left; the right nostril was more dilated than the left; no sensory responses could be obtained; the skin reflexes were marked and somewhat exaggerated on the left side of the body, the triceps reflexes were well marked but the knee-jerk was not examined. General inspection showed but little difference in paralysis between the limbs of the right and left side; but closer examination revealed a more profound paralysis of the right than of the left limbs; he occasionally moved the left arm and leg, and a tendency to contracture was present on the right side. Tremulous and spasmodic movements occurred on both sides of the body, but were a little more marked on the right than on the left. The pulse on the left side was comparatively full and strong; on the left feeble, frequent, and irregular. The temperature was taken several times in both axillæ, and varied between 99° and 101.2°, but with no uniformity as to the two sides. A marked difference between the head temperature of the two sides was noted, the right Rolandic station giving temperature of 102, the left only 99.2. The breathing passed through three periods, at first it was puffing, soon Cheyne-Stokes, and two hours before death regular but constantly feebler and shallower. When of the Cheyne-Stokes type, the period of nearly regular breathing lasted from four to five minutes, the apnœal stage only from eight to fifteen seconds. When breathing began after the apnœa it presented an ascending character, but the apnœal stage began very abruptly. He died about twelve hours after the stroke, and before death the paralysis of the limbs and face became absolutely general. The pupils became more dilated but not unequal.

Autopsy.—Resting the brain on its convex surface, large masses of dark blood could be seen occupying the central region of the base from the pons to the optic chiasm; the blood enveloped the cranial nerves in this area, and infiltrated the membranes and the spaces beneath them far out into the Sylvian fissure. Hemorrhagic foci were found here and there in the pia of the cerebellar hemispheres, the substance of which showed a few bloody points. The fourth ventricle was filled and distended with dark blood; its floor showed a very slight depression or splitting at the upper part; the aqueduct of Sylvius was very greatly dilated. The lateral ventricles which were entered from below, were filled with blood; their cornua were also enormously distended with blood. The septum lucidum, fornix, corpus callosum and commissures were broken down, and the lateral and third ventricles had become one cavity engorged with blood. The anterior extremity of the left optic thalamus and the cue-portion of the caudate nucleus were broken through. The hemorrhage had apparently taken place either from one of the lenticulo-optic or one of the posterior internal optic arteries.

Certain points of difference are to be noted between this case, and the previous one in which the hemorrhage did not reach the ventricles, as, for instance, the more sudden and profound unconsciousness, the complete unilateral paralysis which soon became general, the absence of all sensory response, the tremulous and spasmodic movements of both sides of the body, and the peculiar Cheyne-Stokes breathing.

I have examined the specimen from one case of secondary ventricular hemorrhage in which the primary extravasation took place in the centrum ovale of the parietal lobe, the blood breaking through the root of the ventricle ; but usually the secondary ventricular flooding takes place in the manner and from the direction indicated in the account of the case just given.

Of primary ventricular hemorrhage I have had no experience. "Primary ventricular hemorrhage," Gowers says, "causes symptoms which may, from the first, closely resemble those of the secondary form, but more frequently the onset resembles that of hemorrhage into the substance of the brain, in the presence at first of unilateral symptoms. Prodomata are rare, but headache is occasionally met with, very variable in seat, character, and duration. The onset may be (1) By sudden apoplexy, deepening rapidly ; death may occur in a few hours. (2) By apoplexy with hemiplegic symptoms, or with convulsions. (3) In the very rare slow hemorrhage, hemiplegia first occurs alone, loss of consciousness only supervening after a few hours. Hemiplegia occurs because the blood is effused into one lateral ventricle, and causes paralysis on the opposite side by the compression of the motor path or centres. When the effusion is rapid, and both lateral ventricles quickly become distended, the unilateral symptoms quickly give place to general relaxation of the muscles and loss of all reflex action. Rigidity is often met with, but less frequently than in the secondary form ; it is usually bilateral, sometimes one-sided, and occasionally involves only the muscles of mastication ; it is often intermittent. Convulsions are also frequent, occurring in at least a third of the cases, sometimes general, sometimes affecting only the paralyzed side, or only part of it. In cases of slow onset, speech is often lost before consciousness. The power of swallowing usually persists until the apoplexy becomes profound. The temperature resembles that of other forms of cerebral hemorrhage. The malady is usually fatal, but recovery has occurred, as is proved by old and altered clot being sometimes found in the lateral ventricles, but it is possible only when the hemorrhage is small in quantity and the symptoms are slight and equivocal." The fact that recovery has occurred in such a case is a reason for considering the practicabillty of trephining.

Tapping and draining the ventricles have been performed though rarely ; but in the future, with the comparative immunity from danger in our present methods of attacking the brain, may be resorted to much more frequently. The ventricles can be reached with pre-

cision at several points, best probably from an anatomical and surgical point of view, by way of the posterior horn, or perhaps where the lateral ventricle and the middle and posterior horns diverge. Besides blood, effusions into the ventricles may be also either serum or increased cerebro-spinal fluid, or pus from an abscess.*

Various practical questions arise in connection with the subject of trephining for intracerebral clots, particularly when deeply situated. It has been suggested that it might be impossible to remove the extravasation on account of its having formed a firm coagulum. It does not always do this. Within one week I saw two cases of intracncephalic hemorrhage, in one of which the cavity was filled with a firm clot and in the other the blood was entirely fluid although the patient had been dead more than twenty-four hours. Why this difference should occur I do not know, but it is a fact well known to surgeons that in hæmatocele, no matter where situated, when not in contact with the air, the blood is sometimes coagulated, and sometimes is not. Even though the blood has coagulated it might in some cases be removed by carefully enlarging the opening made by the knife to reach the seat of hemorrhage with flat retractors, and then extracting the coagulum in fragments with forceps or a spoon. The bleeding in case of cerebral hemorrhage is probably stopped because of the retraction of the vessel and the forming of a small coagulum in it, but of course the danger of producing a fresh or renewing an old hemorrhage should be considered. If such operations are resorted to, care should be taken not to move the patient more than is absolutely necessary.

* Since the meeting of the Congress Dr. W. W Keen, of Philadelphia, has proposed tapping and draining the ventricles as a definite surgical procedure, describing an operation for this purpose. He says: "As we now open the belly and drain in tubercular peritonitis with such remarkable success, I would propose that we do precisely the same for the brain. That it may be done with precision and without serious injury to the cerebral tissues the history of the present case, I think, abundantly shows; that it is even *more* urgently necessary in the brain than in the chest or belly seems clear when we consider the relative effects of pressure in the two cases. In the chest or belly the walls are more or less yielding or spongy, to a large extent. They can bear great and long continued pressure but with little damage to their ultimate integrity, or to life, if the pressure be relieved within any reasonable time.

Not so in the cranium. The walls are rigid bone, and the brain can undergo but little pressure, and for a brief time (except it be gradual, as in chronic hydrocephalus) without inviting death. The fatal issue is so uniform that *any* means that holds out a reasonable hope of relief, even though it involves great risk to life, should at least be tried; and the proposal in the present paper seems, at least, to involves but a moderate danger to life with a reasonable probability of success." (Medical News, December 1, 1888.)

Cortical Epilepsy without Gross Lesion.

In cases of cortical epilepsy when the symptoms indicate a discharging lesion of a localized cortical area, operation is justifiable whether or not the probability of a gross lesion can be made out. Hughlings Jackson in the course of a discussion of a paper on brain surgery, read by Mr. Horsely* at the meeting of the British Medical Association, at Brighton, in 1886, strongly advocated the cutting out of the part of the cortex which represented the peripheral parts first in the spasm, whenever the spasm began very locally and deliberately, and when the fits were often repeated. He advocated this, no matter in what condition the brain cortex might be found. He considered it quite certain that epileptiform seizures would be impossible in such a case if enough of the so-called motor area were removed. He believed it better to have some permanent paralysis than to be subject to fits, some becoming universal. This advice has already been acted upon by Horsley,† Keen,‡ Lloyd and Deaver,§ and Hearn and the writer. The most interesting case of the kind yet reported is that of Lloyd and Deaver. Macewen rather advises against this operation, particularly if large wedges of brain tissue are to be taken out, but I believe it to be good practice, even some permanent paresis being preferable to epileptic attacks with their destructive effects on the brain.

Accessible Areas of the Brain.

More and more has that region been narrowed which cannot be reached by the venturesome surgical explorer. The lateral aspect of the pre-frontal lobe, the entire motor area, the superior and inferior parietal lobules and the upper temporal region can, of course, be attacked with the greatest facility. In the regions difficult yet possible of access, lesions of large size and of displacing character will be more readily reached. The orbital surfaces of the pre-frontal lobe can be reached and large displacing lesions removed by trephining low down in the frontal bone. In Durante's case, the tumor removed occupied the left anterior fossa of the cranium. Almost the entire temporal lobe, with the exception of the parts bordering on the mid-brain is accessible. The occipital lobes have been operated upon successfully. With care the great median fissure may be entered for lesions of the marginal convolutions and limbic lobe. The longitudinal sinus has been successfully plugged and ligated. The

* Brit. Med. Jour., Lond., 1886, ii., 670–675.

† Ibid., Lond., 1887, i., 863–865.

‡ Am. Jour. Med. Sci., vol. xcvi., No. 4, Oct., 1888.

§ Ibid., vol. xcvi., No. 5, Nov., 1888

outskirts of the ganglia have been approached, and the ventricles have been pierced. Even a tumor situated on the intra-cranial portion of the auditory and facial nerves can probably be reached and removed. Suckling and Jordan,* Bennett May,† Horsley,‡ and Weir have looked during operation with the eyes of the flesh on the foramen magnum itself. Absolutely inviolable then are only the middle region of the base, and its bordering convolutions, the corpora quadrigemina, and pons-oblongata.

In the accessible areas of the brain are (1) regions in which an absolute localization can be made by positive symptoms ; and (2) regions in which a close approximate localization can be made by positive symptoms, combined with methods of exclusion and differentiation. Under the first head, come the motor, visual, and motor speech areas and tracts ; under the second, the cerebellum, the pre-frontal, and the temporal lobes, with their more or less positively determined functions. The areas for general sensation are still doubtful, but will be considered.

Motor Localization. Researches of Ferrier and of Horsley and Schäfer.

Motor localization has become almost an exact science. Properly interpreted, the phenomena produced by irritative and destructive lesions of the cortical motor area can be relied upon to lead the neurologist to a precise topographical diagnosis, with as much certainty as the stethoscope for cardiac diseases guides the thoracic diagnostician.

The latest physiological researches bearing upon this are those of Horsley and Schäfer.§ They give a new diagrammatic representation of the subdivisions of the motor area in the monkey, both upon the lateral and median aspects of the hemisphere, to which I call attention (Fig. 1 and 2). Excitation of the external surface of the hemisphere in the hands of these experimenters yielded results which were generally similar to those described by Ferrier, which they extended and confirmed, but with some extension as to detail. Comparison of these diagrams with the earlier diagrams of Ferrier, will show the direction in which recent experimentation has added to our precision in motor localization. (Fig. 3, 4 and 5).

In glancing at these more recent results in motor localization, I cannot refrain from paying a passing tribute to the enduring value of

* Lancet, October 1, 1887.
† Lancet, April 16, 1887, vol., i., p. 768.
‡ Brit. M. J., 1887, vol. i., 865.
§ Op. Cit.

the researches of Dr. Ferrier. Their accuracy and reliability are shown by the fact that the results obtained, even as to detail, have been in the main confirmed by the most careful later investigators. With reference to certain questions in dispute, as for instance, the situation of the area of representation of movement of the head and eyes in the second frontal convolution and adjoining regions the existence of distinct centres or areas for the senses of touch, pain, and temperature; and the relation of the so-called angular gyrus to vision, his positions have not been seriously disturbed; at the most, it has only been necessary to modify and enlarge his views, as, for example, to admit the part taken by the gyrus fornicatus in sensation, and of the occipital lobe in vision.

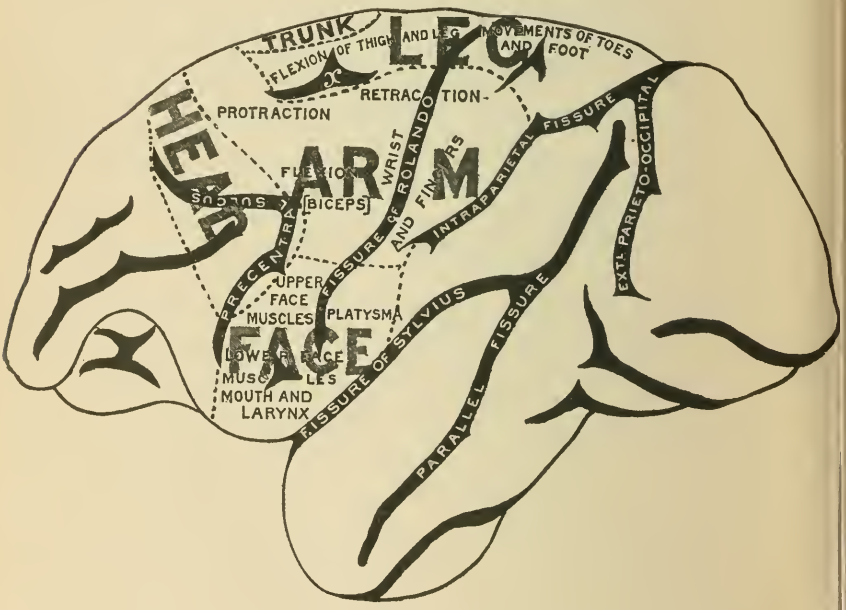


Fig. 1.—Lateral Surface of Brain of Monkey (Horsley and Schäfer).

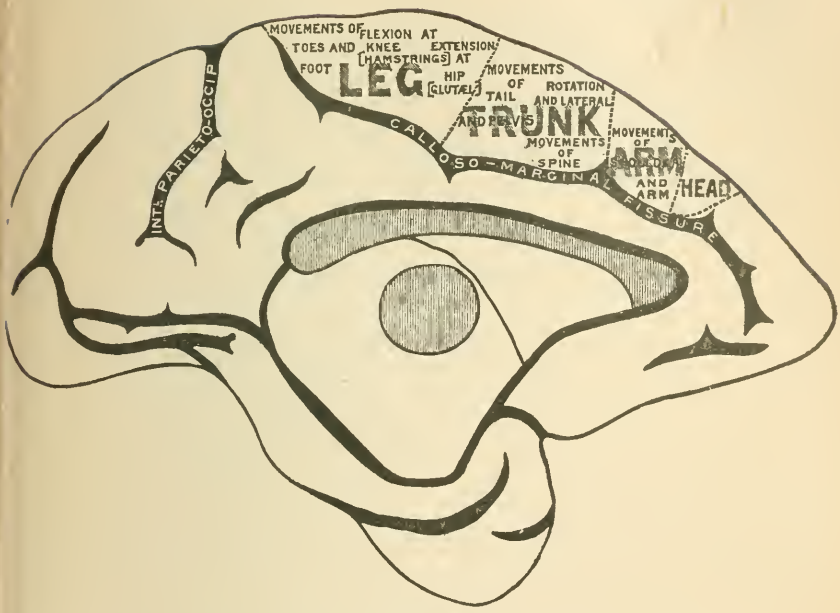


Fig. 2.—Median Surface of Brain of Monkey (Horsley and Schäfer).

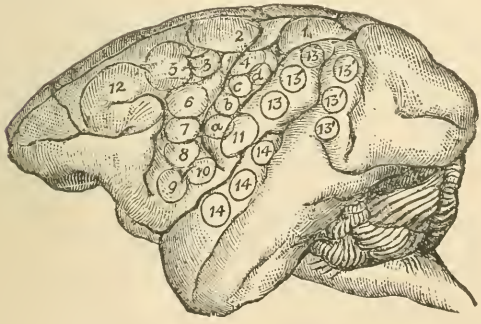


Fig. 3.—Lateral Surface of Brain of Monkey (Ferrier).

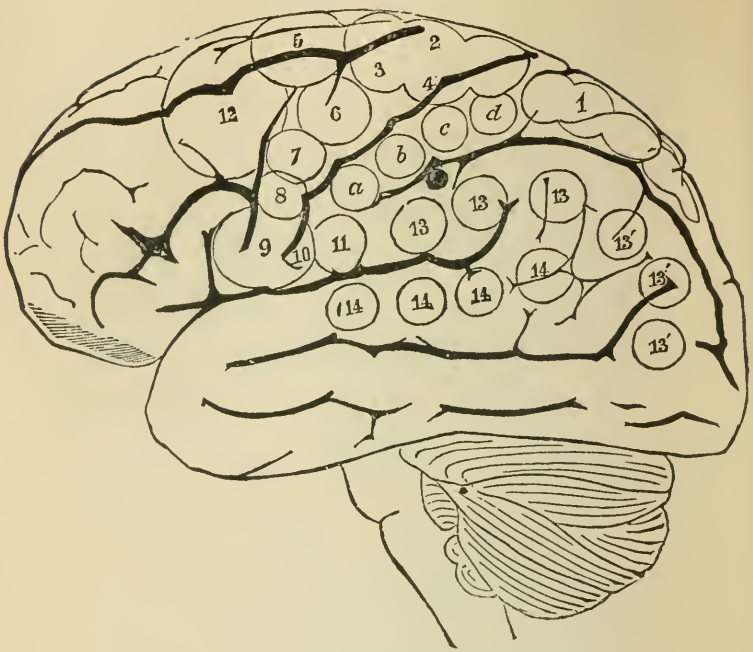


Fig. 4.—Lateral Surface of Human Brain (Ferrier).

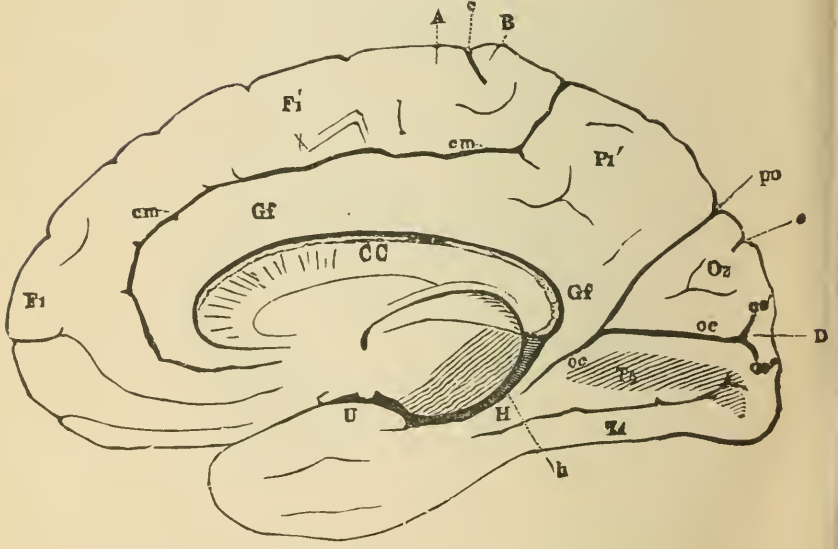


Fig. 5.—Median Surface of Human Brain (Ferrier).

On the diagrams of Horsley and Schäfer are placed the names of the zones and centres as determined by them. Below are given the explanations of the circles numbered on the diagrams of Ferrier representing both the monkey and the human brain. The numbering of the centres or areas is the same for both. The diagrams and descriptions are taken from Ferrier's treatise on the *Functions of the Brain*.

(1) placed on the posterior central and postero-parietal lobule, indicates the position of the centres for movements of the opposite leg and foot, such as are concerned in locomotion.

(2), (3), (4), placed together on the convolutions bounding the upper extremity of the fissures of Rolando, include centres for various complex movements of the legs and arms, such as are concerned in climbing, swimming, etc.

(5), situated at the posterior extremity of the superior frontal convolution, at its junction with the ascending frontal, is the centre for the extension forwards of the arm and hand, as in putting forth the hand to touch something in front.

(6), situated on the ascending frontal, just behind the upper end of the posterior extremity of the middle frontal convolution, is the centre for the movements of the hand and forearm, in which the biceps is particularly engaged, viz: supination of the hand and flexion of the forearm.

(7) and (8), centres for the elevators and depressors of the angle of the mouth respectively.

(9) and (10), included together in one, mark the centre for the movements of the lips and tongue, as in articulation. This is the region, Ferrier says, disease of which on the left side causes aphasia, and is generally known as Broca's convolution. (It will be seen later that I regard these as oro-lingual centres, but place another propositionizing speech centre in advance of this area.)

(11), the centre of the platysma, retraction of the angle of the mouth.

(12), a centre for lateral movements of the head and eyes, with elevation of the eyelids and dilatation of the pupil.

(a), (b), (c), (d), placed on the ascending parietal convolution, indicate the centres of movements of the fingers and wrists.

Circles (13) and (13), placed on the supra-marginal lobule and angular gyrus, indicate the centre of vision, which includes also the occipital lobe.

Circles (14), placed on the superior temporo-sphenoidal convolution, indicate the situation of the centre of hearing.

The centre of smell is situated in the uncus gyri hippocampi or hippocampal lobule. (Fig. 5, V.)

In close proximity, but not exactly defined as to limits, is the centre of taste.

The centre of touch is situated in the hippocampal region (Fig. 5, H) and gyrus fornicatus (Fig. 5, Gf).

Physiological Experiments on the Human Brain.

New clinico-pathological facts, obtained from surgical operations, and justifiable physiological experiments made upon the brain during

such operations, have all helped to more accurately fix the sub-areas of the motor zone. In a number of operations on the motor cortex, weak faradic currents have been used to accurately localize and define the centres sought. In four instances I have seen experiments of this kind, and in one had excision of the cortex performed through the indications thus offered. I have also had the opportunity of observing the effects of faradizing the white matter beneath the excised human cortex. Brief reports of such experiments occur in accounts of operations by Horsley, Keen, Weir and Seguin, Lloyd and Deaver, and others. Horsley first resorted to this means of diagnosis nearly five years ago. The neurologists are thus to some extent repaying in kind the gifts received from physiology. Such experimentation is not only justifiable, but sometimes demanded in the interest of the patient. Gentle faradization of the human cortex does no harm, although it is not so certain that this is true of the application of the galvanic current. The light thrown upon disputed questions by close repeated examinations made after operations will be referred to later.

Boundaries of the Motor Areas.

Let us now glance at the boundaries of the various motor areas—in front, behind, above, below. The anterior branch of the Sylvian fissure, extended mentally, may be regarded as defining the anterior limit of the motor area, including the centres for emissive speech and for the head and eyes. The area which represents the movements of the face is somewhat accurately limited in front by the precentral fissure ; but the movements of the upper extremity have their representation more forward of this line, as do also those of the lower extremity. In front, indeed, the region for the representation of the upper limb extends into the mid-frontal gyre for perhaps one-fourth of its antero-posterior extent ; blending in the anterior portion of this forward extension with the region for the head and eyes.

The inter-parietal (intra-parietal) fissure is usually regarded as forming the posterior limit of the motor area. This large fissure runs upward and backward across the parietal lobe. It is doubtful whether in man the whole of the superior parietal lobule or convolution is concerned with motion, and hence the so-called retro-central fissure is perhaps the more probable posterior boundary of the true motor region ; the postero-parietal area being concerned, in part, at least, with sensation.

This so-called retrocentral fissure (Fig. 6, Rc) is practically very constant in the human brain, and has been regarded by Wilder and others as a distinct sulcus. I have in a few instances seen it of nearly the

same length and depth as the central fissure itself. It is regarded by some as a secondary upward extension of the anterior extremity of the inter-parietal fissure. It generally runs parallel with the upper two-thirds of the central fissure, very clearly bounding behind the posterior central gyre. For practical purposes of operation, at least, this retro-central fissure may be regarded as the posterior boundary of the motor area, rather than the inter-parietal fissure as commonly described. This would leave a distinct postero-parietal region on the lateral surface of the brain in *man*, of uncertain function—a region included between the retro-central fissure in front and parieto-occipital behind.

The horizontal *branch* of the Sylvian fissure forms, as is well known, the inferior boundary of the motor region.

Until quite recently the longitudinal fissure or median edge of the hemisphere was generally regarded as the superior boundary of the motor area, but the investigations of Horsley and Schäfer have shown that this area extends over the edge of the hemisphere into the so-called marginal convolutions on the mesial aspect of the hemisphere, as represented in the diagram (Fig. 2).

As these results are not generally known, it might be well to quote from these authors their general conclusions as to motor representations in the marginal gyres.

"Looking, as a whole, at the results of stimulation of the excitable portion of the marginal gyrus," they say, "it would appear that the application of the electrodes at successive points from before backwards produces (1) movements of the head; (2) of the forearm and hand; (3) of the arm at the shoulder; (4) of the upper (dorsal) part of the trunk; (5) of the lower (pelvic) part of the trunk; (6) of the leg at the hip; (7) of the lower leg at the knee; (8) of the foot and toes." These movements, they say further, in a foot note, are the primary movements, but, as will be seen from previous descriptions, they are almost invariably complicated by secondary movements, which are usually the primary movements produced by excitation of the adjacent parts. The part of the marginal convolution which is concerned with the movements of the leg and foot is that portion which is often known as the para-central lobule.

Diagrams of the Areas and Sub-Areas of the Human Brain.

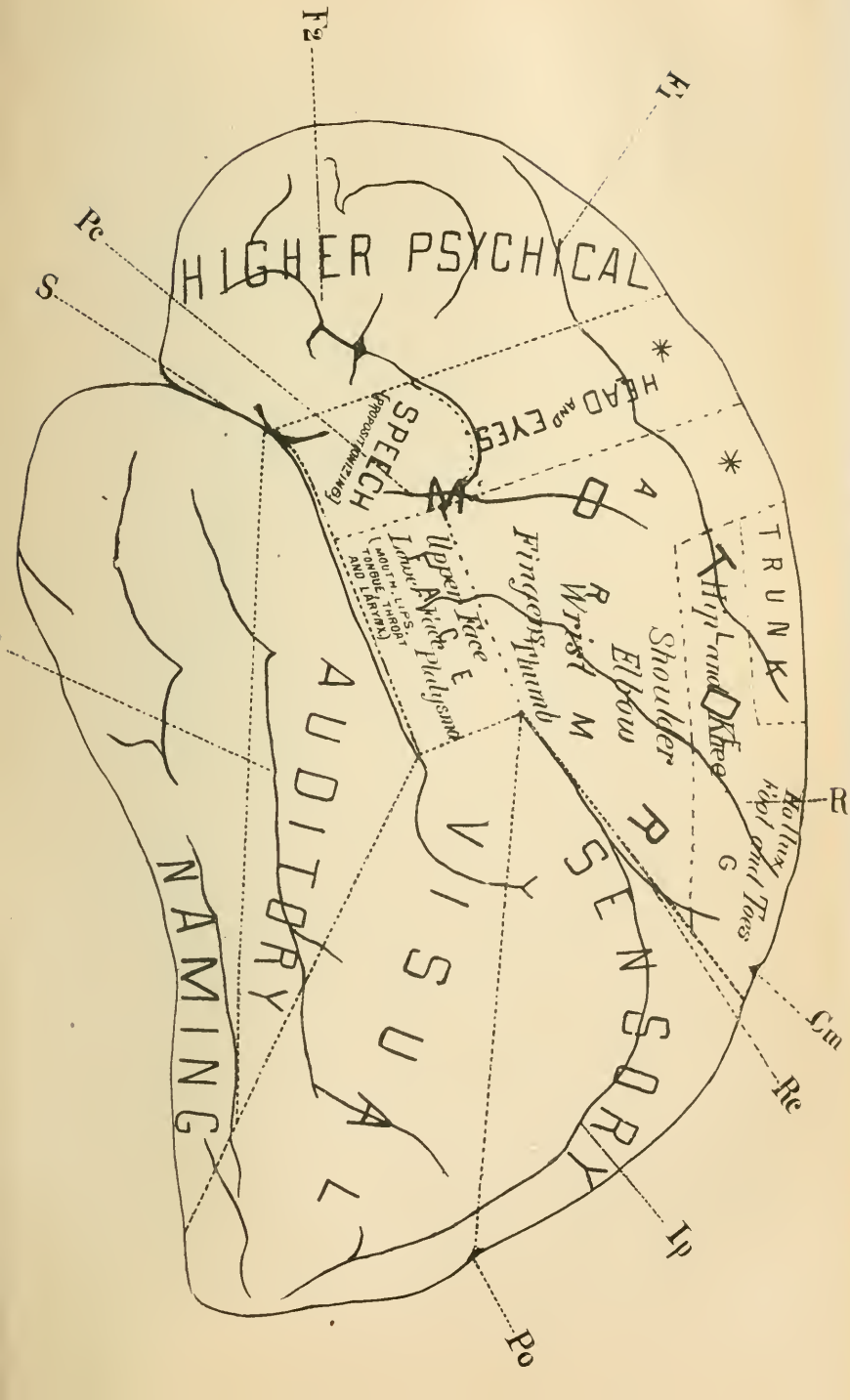
Based upon the investigations of Ferrier, Horsley and Schäfer, and others, and upon a study of cases, personal and collected from the literature of the subject, the diagrams (Fig. 6 and 7) have been made to approximately represent the areas and sub-areas or centres in the

motor zone. In addition, as far as possible, I have indicated areas or centres for other functions—speech, vision, hearing, etc.—so as not to necessitate the repetition of diagrams.

These diagrams (figs. 6 and 7) approximately indicate the views held by most localizationists, as the result of experiment and its confirmation or modification by clinico-pathological observation. They represent the division of the lateral and median surfaces of the cerebrum into higher psychical, motor, sensorial, visual, auditory, olfactory, and gustatory areas; also the subdivision of the motor area into sub-areas, for speech, the head and eyes, the face, arm, leg, and trunk; and the further subdivision of these sub-areas into centres for certain specialized movements of the face, arm and leg. The diagrams for the motor sub-areas are based upon the diagrams and researches of Horsley and Schäfer, but with some modifications as to extent and arrangement. Although a large portion of the paper immediately following is devoted to a consideration of the division and subdivision of the cortex into areas and centres of representation, it will probably serve a good practical purpose to give here immediately in connection with the diagrams a general description and explanation. Only certain main fissures have been indicated by lettering, so as not to confuse: *S*, fissure of Sylvius. *R*, fissure of Rolando or central fissure. *Pc*, pre-centralor vertical frontal fissure. *Rc*, retro-central fissure, sometimes regarded as a secondary branch of the inter-parietal. *F1*, first or superior frontal fissure. *F2*, second or inferior frontal fissure. *Cm*, calloso-marginal fissure. *Ip*, interparietal fissure. *Po*, parieto-occipital fissure. *T1*, first temporal or parallel fissure. *Ca*, (fig. 7) calcarine fissure.

The pre-frontal lobe, that portion of the brain anterior to the universally recognized motor region, has been designated as the higher psychical area. This term is certainly open to objection but it is difficult to substitute it by any appropriate general expression. All portions of the brain are concerned with processes of mentation, but this pre-frontal region, as Ferrier and others have shown, seems to be related to the highest mental processes, its lesions causing, when sufficiently extensive, a mental deterioration which is essentially or mainly a defect of the faculty of attention.

The motor area on the external surface of the hemisphere is made to include the posterior portions of the first, second, and third frontal, and both ascending or central convolutions, but not to reach backwards so as to take in the superior and inferior parietal convolutions. The sub-divisions of the motor zone into sub-areas and centres are indicated by the wording on the diagram, and are explained more at length in the body of the paper. Following Horsley and Schäfer's conclusion from physiological experiment the areas for the arm and for the head and eyes are made to extend forward and upward to the median edge of the hemisphere, but few if any clinico-pathological observations support this view, which is based upon physiological experiment and is probably correct. While, therefore, the portions of the first frontal convolution marked with asterisks * * may be regarded as theoretically included in the areas for the arm, and for the head and eyes, we are not justified for operative purposes in extending these areas above the first frontal fissure. No sub-divisions of the head, arm, trunk and leg areas in the marginal convolutions on the mesial surface of the hemisphere have been made in the diagrams, as these could only be so far our present knowl-


F2
F1
Pc
S
HIGHER PSYCHICAL
SPEECH
(Motor Centre)
HEAD AND EYES
A
TRUNK
Hip
Knee
Ankle and Toes
Shoulder
Elbow
Wrist
Fingers
Thumb
Upper Face
Lower Face
(Mouth, Lips, Throat, Tongue, Larynx)
M
G
SENSORY
VISUAL
AUDITORY
NAMING
R
Cm
Re
Ip
Po

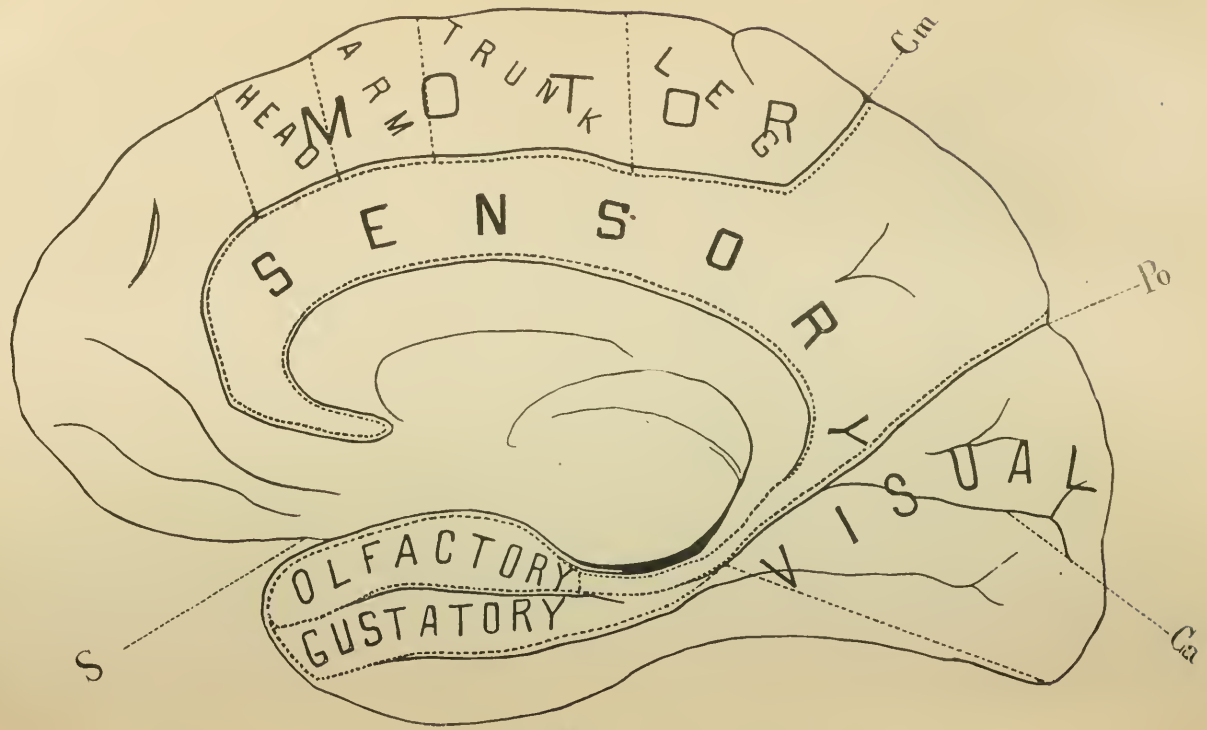


Fig. 7.—Areas of the Mesial Aspect of the Cerebrum.

edge goes, a reproduction of the subdivisions given by Horsley and Schäfer in their diagram (Fig. 2).

By the sensorial area is meant that for the senses of touch, pain and temperature, and modification of these senses, and it has been made to include the gyrus, fornicatus, hippocampal convolution, precuneus, and also portions of the superior and inferior parietal convolutions. This sensorial area has therefore been extended to the external surface of the cerebrum so as to include the general postero-parietal region. This keeps the motor and sensory areas distinct and is based upon the reports of cases with autopsies in which marked disturbances of sensation have been present, although experiments on the lower animals do not seem to have differentiated a sensory area in this lateral external region of the brain. The more elaborate development of the human brain in this region must not be lost sight of in considering this question. It is probable that the exact limitation of the area of common sensibility in the cerebrum has not yet been determined; but anatomical and morphological observations as well as clinico-pathological facts, point to the separation of this sensoral area from the motor region by the great calloso-marginal fissure on the median surface, and on the lateral aspect by it and the so-called retro-central fissure, *Rc*, the parieto-occipital fissure sharply demarcating it behind.

The visual area is represented in the two diagrams so as to take in all of the occipital lobe and adjoining portions of both the temporal and parietal lobes including the so-called angular gyre. Such a delimitation brings into fair accord the findings in reported autopsies, and the researches of Ferrier, Munk, Schäfer and others.

Auditory localization is still in an uncertain state, but limited pathological evidence favors localizing this faculty, as Ferrier advocates, in the first temporal convolution and probably also in the adjoining second temporal.

The views of Ferrier have been accepted as to olfactory and gustatory localization, according to which the centre for smell is located in the uncinate gyrus, and the sense of taste is closely related to that of smell, and may therefore be provisionally placed in the adjoining fourth temporal convolution. Possibly it is farther back in the temporal lobe than has been indicated in the diagram.

An area including the middle region of the temporal lobe—the third temporal convolution, and adjoining portions of the second and fourth has been designated provisionally as the ideational centre or region. This is in accordance with the views of Broadbent,* Kussmaul,† and some others. I believe the ground taken by these authors is a correct one. The only question in my mind is as to the exact localization of these centres for which they claim a dwelling place somewhere on the sensory, or receptive side of the nervous system. A consideration of the differentiation and localization of an ideational or conceptional area in the cortex comes up more particularly in discussion of disturbances of speech, and will doubtless be treated of fully by Dr. Starr, whose views may differ from mine. It is necessary, however, briefly at least, to discuss the question in attempting a division of the surface of the brain into general areas, and therefore I touch upon it in this connection. This region is, according to Kussmaul, that por-

* Brain, January 1879.

† Ziemssen's Cycl. Pract. Med., Am. ed., vol. xiv.

tion of the cellular net-work of the cortex in which ideas are produced as a result of impressions of the most varied description made on the senses (object-and-word-images). According to Broadbent also, the formation of an idea of any external object is the combination of the evidence respecting it received through all the senses: and for the employment of this idea in intellectual operations, it must be associated with and symbolized by name. The structural arrangement connected with this process he supposes to consist in the convergence from all the perceptive centres of tracts to a convolutional area which may be called the Idea Centre or Naming Centre. This, he believes, is on the sensory, afferent or upward side of the nervous system; its correlative motor centre being the propositionizing centre, in which names or nouns are set in a frame-work for outward expression, and in which a proposition is realized in consciousness or mentally rehearsed. The destruction of this centre among other things would cause the loss of the memory of names or nouns. As a provisional guess, Broadbent placed this centre in an unnamed lobule situated on the under surface of the temporo-sphenoidal lobe, near its junction with the occipital lobe, as he believed, fibres from all the convolutions in which perceptive centres have been placed by Ferrier, converged to and end in the cortex of this region. It would certainly seem probable that either in this middle temporo-occipital region, or in the insular or retro-insular convolutions, this conceptional, ideational, or naming region is located. Let no one be misled by this use of the terms ideational, conceptional, etc., and charge that it is an attempt to locate the mind in a limited region of the cerebrum. It is only an effort towards a more thorough understanding of the mechanism of thought and speech. A very careful study of the entire subject of speech disturbances, including an analysis of cases already reported, will, I think, be convincing as to the necessity of a higher area for speech and thought, intermediate between the sensory or receptive centres, and the motor or emissive.

From a study of these diagrams it will be seen that it might be practically convenient to sub-divide the brain into five lobes, four of these, at least, according to the great general functions subserved, these lobes having in nearly all directions well defined fissural boundaries. (1) A *higher psychical or inhibitory lobe*, in front of the basal and anterior branches of the great Sylvian fissure and on the median surface in front of the anterior bend of the calloso-marginal fissure. (2) A *motor lobe*, including the posterior parts of the first, second and third frontal, both ascending or central convolutions, and the adjoining marginal gyres on the median surface. (3) A *lobe for general or common sensation*, including the gyrus fornicatus, the hippocampal convolution, the precuneus, and the postero-parietal gyres. (4) A *lobe of the special senses*, including the whole of the occipital and temporal lobes. (5) The island of Reil or insular lobe.

Differing views have been advanced as to the function of the island of Reil. With its adjoining parieto-temporal convolutions, it forms a distinct lobe, and is, as is well known, sometimes called the central

lobe or, the lobe of the insula. Its importance and size are possibly often not fully considered by the physician and surgeon, owing to the fact that in the average human brain it is so thoroughly concealed by the overhanging fronto-parietal convolutions and the temporal convolutions overlapping from below. The position, relations, and considerable size of the insula can be best seen in some of the brains in which development is arrested or aberrant, as in those of the negro, some criminals, and in the idiotic. Thus studying the lobe, it can be seen to be a great intermediate or binding lobe, probably connecting the other lobes of the brain so that their associated and related functions may be properly performed, and also for the same purpose uniting the ganglia with the different lobes.

Case of Trephining for Cortical Epilepsy.

Before entering upon the discussion of the subdivisions of the cortical motor zone, I will give the details of a case in which the principles of localization were called in to determine the position of operation. One object of introducing the history of the case here is because in fixing the position for excision of the cortex the faradic current was used, and certain results were obtained which assist in indicating the exact site of certain sub-centres of the motor zone, as, for instance, those for turning the head, for extension and flexion of the fingers and hand, and for drawing upwards and outwards of the angle of the mouth.

M., 14 years old, when two years of age had a series of convulsions coming and going during twelve hours, and followed by a stupor which lasted several days. Six years later he again had a series of severe spasms, the epileptic status continuing for several hours; he had a third similar attack about one year later. Since, during the past five years, he has had fifteen to twenty spasmodic seizures, the intervals between them having grown shorter, so that recently they had only been a few weeks apart. Before the convulsions he was usually nervous and excitable, and on coming out of them nearly always complained of pain above and somewhat in front of the left ear. He had always been of an excitable temperament; and unusual excitability was noticeable during the six years between his first and second attack of spasm. He was, however, a bright boy, of good disposition, affectionate and careful of himself, and his general health, as a rule, was excellent. He was seen in consultation with Dr. Wilson Buckby. Twelve days before he went into a severe convulsion, and from that time he had not spoken, and had had violent spasms with intervals, in the course of twenty-four hours having ten or more distinct paroxysms. In the intervals between the attacks, he was sometimes stuporous and some-

times in a condition of excitement, but his mind was continually clouded so that he did not appreciate his surroundings.

In every seizure of the series of spasms the convulsive movement began the same way; the fingers of the right hand first flexed, then flexion took place at the wrist and elbow, and the parts remaining flexed, soon the whole arm, forearm and hand were drawn upward and somewhat outward. As one of his family expressed it, "his right arm was drawn until it looked like a chicken's wing." His face and head, after the movements in the upper extremity were well under way, were drawn a little to the right, his leg at about the same time, as nearly as could be determined, taking part in the spasm, semi-flexing at the knee, and the toes and foot contracting. The signal symptom was always the same, namely, a movement of flexion of the fingers; and the spasm was always first and most marked in the right upper extremity; it was commonly unilateral, but sometimes became general. Between the paroxysms his right arm was often the seat of a tremulous vibratory movement. Examination in the interval between two seizures, showed slight paresis of the lower portion of the right side of the face, and more marked paresis of the upper extremity, particularly of the forearm and hand. This was always more decided after each convulsive attack. Although right-handed, he constantly used the left hand in preference to the right. Tactile sense could not be closely studied, but he undoubtedly appreciated sensations of touch, pain, temperature. Knee-jerk was somewhat exaggerated on the right side.

After several consultations it was decided to trephine, and if no gross lesion was discovered, to excise the cortex of the area or centre for the fingers and hand in the left hemisphere, because of the invariability with which the spasmodic symptoms began in the fingers and hand of the right side.

The trephining was performed July 28, 1888, by Dr. W. J. Hearn. At the operation were present, besides the writer and operator, Drs. W. Buckby, R. B. Burns, J. H. Lloyd, A. H. P. Leuf, W. M. Coplin, M. Imogene Bassette, J. C. Cooper and C. P. Noble. The head was shaved and prepared anti-septically. A sublimate solution was used and great care was taken with the instruments, sponges, etc. The line of the fissure of Rolando was determined by the methods of Hare and Thane, and a point was selected for the centre of the first trephine at what was considered to be the junction of the arm and face area, about three-quarters of an inch in front of the fissure of Rolando. A large horseshoe flap was made, its convexity backwards. Two trephine openings were made and bone cut away until finally the opening measured in its greatest diameter, which was from above downward, 2½

inches, and 1½ inches in its greatest width. The long axis of the opening was nearly in a line with the general direction of the fissure of Rolando. The region intended to be exposed was the lower two-thirds of the arm area, the upper anterior portion of the face area, the hinder upper part of the speech area, and a posterior strip of the area for movement of the head and eyes ; the convolutions uncovered were, therefore, presumably nearly the lower halves of the two centrals, the posterior extremity of the second frontal, and the posterior superior corner of the third frontal. No lesion of the bone or of the dura mater was found. On raising the flap of the dura mater, the pia arachnoid in the lower half of the opening was decidedly oedematous. No gross lesion could be found on inspection and close examination in the pia mater, cortex, or sub-cortex.

Careful examinations were made with the faradic current applied to the cortex with the view of locating the proper centres for excision. Four distinct responses in the shape of definite movements were obtained after several trials ; these were (1) in the most anterior position at which movements resulted distinct conjugate deviation of the head to the opposite side ; (2) a little below and behind this point, drawing of the mouth outwards and upwards ; (3) above this spot for movements of the angle of the mouth, about half an inch, extension of the wrist and fingers was produced ; (4) behind and above the latter point, distinct flexion of the fingers and wrist. Continuing and increasing the faradic application at this last determined point, the fingers, thumb, wrist and forearm were successively flexed, and the whole extremity assumed the " wing-like " position. The order of events, according to three persons who were present, and who had observed the patient's spasms, being exactly that which had been noticed in the beginning of his convulsive seizures.

As no gross lesion was discovered on careful examination and exploration, excision was performed of the cortex and sub-cortex so as to include the area excitation of which by faradism started the spasmodic movements of the fingers and wrist. The operation was concluded after the usual manner. The patient recovered from the operation without serious symptoms. In three days his mental condition was much improved ; the restlessness, irritability, and semi-maniacal condition which had been present before the operation passed away. His aphasia persisted. He had distinct paresis of the fingers and wrist, including the thumb, slight clawing of the fingers and bending of the wrist being present. This gradually improved.*

* January 3, 1889, it is reported to me that this patient has had no spasms since the operation. He has regained almost entirely the use of his right hand and arm. He is still aphasic, although he has acquired the use of a few words.

Subdivisions of the Motor Area.

In the area for face, head, arm, leg and trunk, the neurologist should be able to locate for the surgeon, through a study of motor phenomena, at least seven or eight different sub-areas ; and in order to do this it is imperative for him to have exact knowledge, not only of the anterior and posterior limits, but also of the horizontal subdivisions of this zone. Too much stress cannot be laid upon the proper separation of the region into horizontal levels ; for, as Horsley* has well put the matter, the variation in the representation of motor function is greater in passing over the motor area from above downwards than from before backwards.

Horsley suggests the horizontal subdivision of the motor area mentally by means of certain sulci and their imaginary extensions. The imaginary extensions backwards of the superior and inferior frontal sulci through the central or Rolandic fissure subdivide with approximate accuracy the pre-central or ascending frontal gyre into three areas or zones from above downwards, namely, for the lower extremity, upper extremity, and face. According to Horsley, also, a line drawn forward from the anterior lower end of the intra-parietal sulcus will mark distinctly the division between the representation of movements of the upper limb and of the face behind the fissure of Rolando. This suggestion, however, is not as good a one practically as that with reference to the two frontal sulci. In the human brain, at least, the lower end of the intra-parietal sulcus is by no means fixed; it is often as low down as the end of the central fissure. It is better simply to place the posterior part of the area for the face in the lower third or fourth of the posterior central convolution.

The old method of subdividing the motor zone was by cutting the fissure of Rolando into thirds, and locating a circular or elliptical area over each of these thirds on both sides of the fissure—and upper area for the lower extremity, a middle one for the upper, and a lower one for the face. Such a sub-division is not now exact enough for accurate topographical diagnosis for operative purposes.

Instead of subdividing the central or Rolandic fissure into thirds, it is better perhaps to divide it into fourths, placing the area of representation for the lower extremity in the first fourth ; that of the face in the lower fourth, and the areas for the upper extremity include the second and third fourths. This makes the diagrammatic method of representation correspond more closely to the results of recent investigations, as the vertical extent of the arm region on the

* Am. Jour. Med. Sci., April, 1887, p. 342–369.

lateral aspect of the hemisphere is about twice as great as that for the leg and somewhat greater than that for the face. The fissure of Rolando does not extend usually as far as the Sylvian fissure, and therefore making the junction of the third and last fourths of the former fissure the upper boundary of the face area, gives this area a greater height than that for the leg, but not as great as that for the arm.

Although time will not permit lengthy consideration, it will be interesting briefly to discuss some of the ascertained facts with reference to the sub-areas or centres in this wonderful motor region.

Subdivisions of the Face Area.

In the first place, the face area is best subdivided into an upper and a lower sub-area. In the upper sub-area movement of the opposite angle of the mouth and of the lower face generally, are represented. In three cases during operations on human beings, I have observed faradization of the anterior superior portion of this face area produce contraction of the opposite angle of the mouth and face. It is probable that in the extreme upper anterior portion of this area, immediately adjoining the area for the head and eyes, is a sub-centre for such movements of the upper face as contraction of the frontalis and orbicularis palpebrarum muscles. Such a centre does not come out clearly as the result either of physiological investigation, or the experiments of disease, because associated movements are apt to remain even after destruction of a centre for such movements on one side of the brain. Of the face area Horsley and Schäfer say that it is physiologically remarkable, that many of the movements which result from its excitation are apt to be executed bilaterally, which is only exceptionally the case with excitation of the other areas (except that of the head and eyes). Excitation of the upper third or half of the area, they continue, causes winking or closure of the eyelids, elevation of the ala of the nose, and retraction and elevation of the angle of the mouth.

An observation of Dr. Berkeley, of Baltimore, helps to fix with positiveness the exact location of the cortical centre for the movements of the angle of the mouth, chiefly performed by the zygomatic muscles, in this upper anterior portion of the general area for the face at a point about opposite the usual position of the fissure between the middle and inferior frontal convolutions. Gowers, in his Manual of Diseases of the Nervous System, page 663, has a cut from a photo-

graph furnished by Dr. Berkeley, showing a small focus of softening in the ascending frontal convolution at this point. This very circumscribed focal lesion caused persistent clonic spasm, chiefly of the zygomatics. My observation on the case trephined by Dr. Hearn, confirms this position for the centre for this movement, as gentle faradization of the cortex at this spot caused distinct drawing of the mouth upwards and outwards. The centre for the orbicularis palpebrarum, as stated is doubtless in close proximity, probably just above the position of the centre for the angle of the mouth. While it is not usually the case, paralysis in the upper distribution of the facial nerve sometimes does take place as the result of cortical lesion. In one of my cases of tumor in the posterior portion of the second frontal convolution, and causing some destruction by the invasion of the ascending frontal, distinct lagophthalmus of the opposite side was present. No lesion of the cranial nerves at the base was present. In this case ptosis was present on the other side, that is, on the side of the lesion. The fact that in facial spasm whether secondary after a peripheral facial paralysis, or primary from nerve or central lesion—the orbicularis palpebrarum and zygomatic muscles usually act together so that the eye is closed or partly closed at the same time that the angle of the mouth is drawn upward, and the naso-labial furrough deepened, is clinical evidence in favor of the close proximity of the zygomatic and orbicularis palpebrarum centres in the cortex. Close examination of cases of hemiplegia and monoplegia will often show weakness of the movement of closure of the eyelids, in the paralyzed side, a paresis which would not be observed by a careless observer.

The lower two-thirds of the face-area may be divided into at least two parts, an anterior and a posterior. As the question of the exact function of this lower anterior portion of the face-area is one of considerable importance, and one about which some doubts still exist, I will briefly detail some of the facts with reference to this sub-centre. Beginning with the most recent contribution to the question, it may be first stated that Dr. Felix Semon, working in conjunction with Professor Horsley,* found that the lower end of the ascending frontal gyrus anteriorly is also excitable ; an effect being produced upon the glottis by its excitation, viz : to bring about phonatory closure of the vocal cords.

A fair amount of other evidence has been collected to show the existence of a cortical centre for laryngeal movements. In 1877,

* Phil. Trans. Royal Soc., vol. clxxix., 1888.

Seguin* reported a case of left hemiparesis without loss of consciousness but with impairment of speech, and also of phonation, the patient after the attack never being able to control the pitch of the voice, apparently from a lack of proper action of the muscles of the pharynx and larynx. Autopsy showed the surface of the right third frontal convolution degenerated, being yellow, tough, and elastic. The same change to a less extent was found in the same location on the left side. From such observations a motor centre for laryngeal movements has been sought for in the posterior extremity of the *right* third frontal (homologous with the speech centre on the left side, in right-handed persons).

Krause,† in the laboratory of Professor Munk, in 1883, investigated this question experimentally. On excitation of the cortex he noticed rise of the larynx, and movement of the vocal bands to a position midway between expiration and phonation, lifting of the palate, contraction of the constrictor pharyngis, and movements at the base of the tongue. With extirpation experiments he found that eight dogs had lost the power of barking, on attempting which they uttered only a hoarse whine or made a sound.

Delavan,‡ in 1865, contributed a paper on laryngeal cortical centres, in which he records some valuable facts and refers to the observation of Seguin, Krause and others.

Garel§ read before the French Society of Otology and Laryngology, in April, 1886, an interesting communication on the laryngeal cortical centre, and vocal paralysis of cerebral origin, in which he reported a case with the details of an autopsy and a sketch of the locality of the lesion. The inferior portion of the precentral gyre on the *right* side was slightly adherent to the meninges. The membranes being stripped the surface beneath presented a light yellow discoloration. At the foot of the third frontal gyre were two points of red softening, but there was no lesion of the anterior portion of the third frontal. On section, these lesions were found to involve only the cortical substance, at the upper part only very slightly invading the white. The lesion of the precentral penetrated slightly into the white substance. It would seem from these experiments and observations that a centre for the movements of the larynx and throat is in the extreme lower anterior portion of the pre-central convolution, and that it is probably better differentiated in the right than in the left hemisphere.

* Referred to by Delavan in Med. Rec., N. Y., Feb. 14, 1885.

† Cited by Horsley and Schäfer and Delavan.

‡ Med. Rec., N. Y., Feb. 14, 1885.

§ Annales des Maladies de l'Oreille et du Larynx. Tome xii., 1886, p. 218.

Somewhat numerous pathological observations corroborate the existence of Ferrier's oro-lingual centres also in the lower anterior portion of the face area, probably a little behind the centres for the throat and larynx. In these oro-lingual centres are located particularly the representation of the movements produced by the orbicularis oris, and of protrusion of the tongue. Recently a case of typical oro-lingual paresis with involvement of this region has been observed by me, some details of which will be given later when speaking of the alleged sensory functions of the motor cortex. Pathological observations also somewhat numerous have confirmed the position of Ferrier's centre for movements performed by the platysma myoides muscle in the face area behind the Rolandic fissure.

In the hinder lower portion of the face area is probably represented opening and shutting movements of the mouth, and retraction of the tongue.

Intra-cerebral Facial Tracts.

The existence and location of separate intra-cerebral facial tracts is a subject bearing a direct relation to that of the cortical areas and sub-areas for the face, and also a matter about which our knowledge is scanty.

Kirchoff,* in 1881, reported the case of a man aged 24, who had several attacks in which he became giddy, had convulsive tremors, lost power of speech, was unable to swallow, had profuse salivation, and drawing of his face to the left. Examination showed that he articulated with difficulty; labials and gutturals especially were troublesome; linguals he spoke with comparative ease. The lips were moved little in speaking; he could not whistle, but was able to approximate the lips. Saliva flowed from the mouth and there was excessive secretion of tears. The tongue was not protrusible more than one centimetre from the mouth, and it moved clumsily in the act of biting. At the time of examination swallowing was unimpeded, but the glottis was closed tardily. The patient often laughed without occasion. There was disease of the mitral valve. Ten days before death his face was suddenly drawn to the right, and his left arm and leg became powerless. Convulsions occurred from time to time up to his death.

The post-mortem revealed embolic softening of the posterior two-thirds of the right corpus striatum (caudate nucleus), the underlying

* Archiv. f. Psych. Bd. XI,, and Brain, July, 1881.

internal capsule, the outer segment of the lenticular nucleus, the claustrum, external capsule and island of Reil. The focus of softening in the lenticular nucleus was distinguished from the other softened portions by being surrounded by a wall of compact sclerosed tissue. Careful microscopic examination failed to show any disease of the medulla or pons. The author atributes the glosso-labial paralysis to the lesion of the lenticular nucleus; and the hemiplegia to the quite recent lesion of the caudate nucleus, internal capsule and other parts. Cases of bilateral affection of the face, tongue, and throat, caused by unilateral lesion of the cerebrum, are rare; the author cites two, recorded by Lepine and Magnus respectively.

Ross,* also reported a case of brain disease simulating bulbar paralysis in which the lesions were cerebral, in the ganglia and alongside of them in the capsules. In 1880, I observed a third similar case at the Philadelphia Hospital.

Hobson,† in 1882, reported a case without autopsy—the main symptoms being left hemiparesis or paralysis, or paralysis of the tongue, difficulty of deglutition, speechlessness, clenching of the jaws; the patient had one inarticulate sound for everything, and a slight sound on laughing. In 1882, Ross,‡ in an interesting paper on labio-glosso-pharyngeal paralysis of cerebral origin, traversed the literature of the subject, giving also some interesting original observations.

Subdivisons of the Arm Area.

The subdivision of the area for the upper limb, according to Horsley, is for the shoulder in the upper part, the elbow next below and behind, the wrist next below and in front, the thumb lowest and behind. In the area just above the superior frontal sulcus the movements of the lower and upper limb are absolutely blended, most markedly in the hinder sixth of the superior frontal gyre. Sometimes an epileptic fit from a lesion centred here begins by complicated or generalized movements of both extremities on one side.

At various points on the posterior central convolution, Ferrier, it will be remembered, fixed centres, excitation of which caused flexion of the thumb and fingers and firm clenching of the fist, with the synergic action of the wrist and fingers, but he did not differentiate centres for different flexors and extensors. From my own observation, I believe that the centres for movements of extension of the fingers and wrist are a little anterior and below those for flexion of the same parts.

* Diseases of the Nervous System, vol. ii.

† Brit. M. J., April 29, 1882.

‡ Brain, July, 1882.

In one of Keen's operations* the position of the hand centre was fixed by means of the faradic current. The fissure of Rolando was determined by both the methods of Hare and Thane. The trials with the faradic currents were made according to the determination of Dr. Keen, on both the post-Rolandic (post-central or ascending parietal), and pre-Rolandic (pre-central or ascending frontal) convolutions, and also the posterior extremity of the second frontal convolution. Excitation of the post-Rolandic convolution produced no effect. On touching the cortex with the electrodes at a position which apparently corresponded to the anterior portion of the pre-Rolandic convolution just back of the precentral fissure, movements of the wrist and fingers were produced. The hand moved in extension in the mid-line and to the ulnar side at different touches, the fingers being extended and separated. Above the region in which these movements were obtained, application of the current caused movement of the left elbow, both flexion and extension, and of the shoulder, which was raised and abducted. Below the region where the hand movements were excited the application of the current produced an upward movement of the whole of the left face. In the cases of Hearn and the writer, reported above, the exact movements described by Keen were produced, that is, the extension at the wrist and separation of the fingers ; also, below the spot where these movements were produced, an upward and outward movement of the face, or rather angle of the mouth. In our case, however, elbow and shoulder movements were not produced except as a secondary result. We obtained, however, a primary movement of flexion of the fingers and hand to which Keen does not refer, and which presumably was not produced. This movement resulted from touching with the electrodes a spot a little above and behind the place where the movements of the extensors were caused. Continuing and increasing the strength of the faradic applications at this point, flexions took place in succession of the fingers and thumb, and at the wrist and elbow. Keen estimated the portion of the convolution as containing the hand centre as about 1¼ inches long, and he places the centre for the wrist and fingers in the pre-Rolandic gyrus, its lower limit being at three-eighths of an inch above the temporal ridge, and its upper end where it fused with that of the elbow 32 millimetres higher up. The shoulder he placed still higher, while the centre for the upper face was in the same convolution below. These results correspond closely with those of Horsley. These facts of experiments on man would seem to uphold the view that the motor zone, in man at least, is much more extensive in front of than behind the

* Trans. of Am. Surg. Association, vol. vi., 1888, and also in the Am. Jour. Med. Sci., November, 1888.

fissure of Rolando. These results of Keen were all produced by excitation of the cortex anterior to the fissure of Rolando, supposing, of course, his determination of that fissure to have been correct. As nearly as I could determine the location of my own results were the same.*

I was present at the operation in the case of Lloyd and Deaver.† By following Reid's and Horsley's lines, an area was exposed which was supposed to be on both sides of the Rolandic fissure, about the junction of the middle and lower thirds of the central convolutions. The faradic current was then used to identify locations. When the electrodes were applied to a point which was supposed to be just back of the fissure of Rolando, the movements which occurred were in order turning of the thumb on the palm, flexion of the fingers, flexion of the wrist extending to flexion of the elbow. At a point in front and below faradic stimulation caused marked contraction of the face muscles of the opposite side. "The mouth began to contract and was drawn to the left with a tremulous motion, and soon the tongue began to protrude toward the left corner of the mouth. Soon the left thumb began to be contracted and adducted into the palm ; then the fingers contracted into the palm, and about the same time the face muscles began to contract more actively, while the head was drawn to the left, and the left eyelid began to work. At the same time the hand was gradually closed, and contraction of the forearm and arm began, while the latter was drawn from the side to an angle of forty-five degrees (deltoid action), and contractions of the biceps occurred. At no time in the course of these faradic applications, anywhere within the area exposed by the trephine and forceps, did any contraction of the leg muscles occur."

Subdivisions of the Leg Area and of the Trunk Area.

The movements of the lower extremity are represented in the upper portion of the motor area, and the adjoining marginal convolution ; probably hip and thigh movements on the lateral and mesial aspects of of the hemisphere near the median fissure, well formed in the area, and

* October 4th, 1888, since the meeting of the Congress, Nancrede of Philadelphia, before excision of the cortex fixed the position of the thumb centres by means of the faradic current. The patient suffered from convulsions which began with strong flexion of the right thumb, followed by extension of the wrist and fingers, pronation of the forearm and hand, flexion at the elbow, powerful flexion and rotation of the head to the right, thrusting out of the tongue between rigid jaws, and coincident with all conjugate ocular deviation to the right. The spot at which the faradic current was applied was estimated by Nancrede, to be from below upwards in the second fourth of the ascending parietal convolution.—*Medical News, November* 24, 1888.

† Since reported in the Am. Jour. Med. Sci., November, 1888.

movements of the leg and toes farther back on the lateral aspect and also in the para-central lobule, and marginal convolutions of the median surface.

A narrow strip of the anterior portion of the leg area appears both from the results of experimentation, and of pathological and surgical observation to be a trunk-area, this being larger proportionally on the mesial than on the lateral aspect of the hemisphere, as represented in the diagrams. Horsley and Schäfer occasionally obtained movements of the trunk when the electrodes were applied to the lateral surface near the margin of the hemisphere. On the adjoining mesial surface, however, excitation produced rotation and arching of the lower spine and the pelvis, and extension of the hip, movement of the tail to the opposite side, and flexion at the knee.

Horsley* says that at the summit of the ascending frontal gyrus begins the representation of the lower limb only, the primary movement being that of the hallux. He describes a case of traumatic epilepsy the primary movement consisting of flexion of the hallux followed by the gradual flexion of the rest of the lower limb, and that followed by successive invasion of the rest of the lower body in the usual order. A dense and cystic cicatrix was found at the upper end of the ascending frontal gyrus. In another case, in which a tumor was removed, and with it the cortex in front of the upper end of the fissure of Rolando, the only permanent complete paralysis of the lower limb was that of the hallux. In one of my own cases a small gumma involved the upper fourth of the ascending frontral, and a smaller segment of the ascending parietal, crossing the upper extremity of the Rolandic fissure. This patient had severe attacks of left sided spasm beginning with twitchings in the left toe and foot ; she also had partial paralysis of the left leg and arm, most marked in the leg. The leg area, however, as shown by Horsley and Schäfer, is largely situated upon the mesial surface of the hemisphere. According to these authors, the excitation takes effect chiefly upon the ankles and digits, producing most commonly flexion of the foot with flexion of the digits. The most marked movement in front of the upper end of the Rolandic fissure is flexion of the leg at the knee, with the addition, when the electrodes are applied more anteriorly, of flexion at the hip.

Area for the Movements of the Head and Eyes.

I cannot agree with Seguin in the recent paper by Weir and Seguin† that the centre for ocular movements is quite certainly not in the second frontal gyre as claimed by Ferrier and Horsley. Much is in favor of the view that it is situated in this neighborhood.

* Am. Jour. Med. Sci., April, 1887. † Op. cit.

In one of the cases of Horsley, in which operation was performed at the point of the meeting of the areas for the movement of the trunk, protrusion of the upper limb, and turning the head and eyes, the aura was contraction of the abdominal muscles followed by turning of the head and eyes to the opposite side. Other cases have been reported in which turning of the head was the starting point of the spasm. In some cases, at least, when the aura or signal symptom can be most certainly shown to be the turning of the head and eyes to the side opposite to the supposed site of the lesion, the probabilities are that the focus or primary seat of the irritation is from a lesion in this oculo-motor region. The fact that cortical oculo-motor palsies are not present as a persistent condition even when we have definite lesions of the second frontal gyre is not an argument of weight against the existence here of oculo-motor centres. Such persistent oculo-motor paralysis was not present in one of the best defined cases of lesion in the second frontal gyre ever reported, a case occurring in my wards at the Philadelphia Hospital. Such symptoms do not persist because of the automatic nuclear mechanism of the cranial nerves related to these centres.

In the last edition of Ferrier's Functions of the Brain, he adheres to his views as to the position of the oculo-motor centres, and gives some new experiments bearing upon the subject. Irritation of the base of the superior and middle frontal convolutions in monkeys, gives rise to lateral movements to the opposite sides with dilatation of pupils. The expression assumed by the animal is that of attention or surprise. The same movement, however, as Ferrier himself states, also occurs along with other special reactions, on stimulation of the angular gyrus and superior temporo-sphenoidal convolution more especially. With the latter is associated pricking of the ear from stimulation of the auditory centre. Ferrier argues that although the effects are the same the causes are different. Stimulation of visual and auditory centres attract attention movements, the same as would result from stimulation of the motor centres for those movements. Destruction of the oculo-motor centres of Ferrier, according to some experiments, causes conjugate deviation towards the side of the lesion. Bilateral destruction of these centres for the first day caused inability to turn the head and eyes, but the animal recovered. Horsley and Schäfer, and Ferrier also, got no motor or sensory symptoms from lesion of the pre-frontal lobes, except in one case in which the paralysis of the lateral movements of the eyes following the lesion of the post-frontal centres having completely disappeared, the destruction also of the pre-frontal regions caused rapid oscillations of the head, apparent in-

ability to turn the head except en masse with the trunk, and drooping of the right eyelid. These facts, according to Ferrier, show that the pre-frontal regions belong to the same centres as the post-frontal, just as the occipital lobes belongs to the visual centres.

Attempts have been made to remove the post-frontal as well as pre-frontal region. The animal could not maintain the upright position or move its head and eyes laterally. The eyes were kept shut except on cutaneous or other sensory stimulation. Some microscopical examinations of degenerations of tracts seem also to prove that the post-frontal regions contain the oculo-motor centres. Descending sclerosis from the innermost or mesial bundles of the internal capsule, does not extend below the pons, but probably into the oculo-motor nuclei.

Horsley believes that the focus of representation of the movement of the turning of the head and eyes to the opposite side is in the middle frontal gyre, but also that these movements have a much more extensive representation.

"It must be left, for the present," he says, "an open question as to how far the representation of this important and interesting conjugate movement extends *forward* in the frontal lobe. A definite answer can only be given when the homologies between the sulci and the frontal lobe in the Macaque monkey and man have been thoroughly determined. That this area of function is continued over the margin of the hemisphere into the marginal convolution, has already been shown by Professor Schäfer and myself. * * * *

"In every instance the head and neck are turned to the opposite side, and in some parts there is produced at the same time or later, conjugate deviation of the eyes."

Horsley and Schäfer in their contribution to the Philosophical Transactions, speak as follows with reference to this area: "The *head-area or area for visual direction* comprises an oblong portion of the surface of the frontal lobe, extending from the margin of the hemisphere, round which it dips for a short distance, outward and somewhat backward to the upper and anterior limit of the face-area. Posteriorly, it is bounded by the arm-area, and in front by the non-excitable portion of the lobe. It extends, therefore, in front as far as the extremity of the precentral sulcus, and it includes the middle part of the frontal lobe above the antero-posterior limb of the sulcus, the part included in the angle formed by the antero-posterior and vertical limbs of the sulcus, and perhaps a small portion of the ascending frontal gyrus, close to the vertical limb of the same fissure. The effects produced by excitation of this are similar to those described by

Ferrier as resulting from excitation of the rather more limited area marked 12 in his diagrams, viz: opening of the eyes, dilatation of the pupils, and turning of the head to the opposite side, with conjugate deviation of the eyes to that side. If the electrodes are applied near the angle of the precentral sulcus, the ears are frequently also retracted."

In the patient referred to in whose case trephining was performed a weak current applied forward of the position at which movements of the fingers and hand were produced, caused distinct deviation of the head to the opposite side. As nearly as could be determined the electrodes were applied over the extreme posterior portion of the second frontal gyre.

Conjugate deviation of the head and eyes, when a persistent or permanent symptom, is most likely to arise from lesions of the pons, cerebellum or cerebellar peduncles.

I have thus tried to indicate the recognized centres and sub-centres of the motor zone. To such great works as that of Ferrier on the *Functions of the Brain*, and to such monographs as those of Horsley and Schäfer, Horsley and Beevor, and Seguin, I must refer those especially interested in obtaining fuller details.

Overlapping Areas.

Some of these areas it will be seen apparently overlap each other, so far as their cortical representation is concerned, hence giving positions for trephining in some cases over the border of two adjoining areas. It might be said that with a large trephine it will not be necessary to separate and localize so many areas, as an opening 1½ inch or 2 inches can be made, and even this can be enlarged by the rongeur until a suspected lesion is reached, but this is a crude method in these days of precision. Even in cases of comparatively large lesion, the complete success of the operation will depend somewhat upon the first position in which the opening is made. The ideal position would of course be one that corresponded to the centre of the lesion.

Wonderful indeed is this motor zone of the cerebrum, a marvellous mosiac of centres of function, wrought from the great conceptions and priceless labors of the artists of our own guild ; a mosiac, to each block, angle and jointure of which the neurologist can point the surgeon and say, cut here or there, or touch not this or that.

Different Classes of Localizing Symptoms, their Characteristics and Comparative Value.

The neurological diagnostician must make use of his knowledge of these areas after a definite plan, if he wishes to turn it to the best account.

When localizing lesions he must go beyond even the important distinction advanced by Brown-Sequard, and very properly insisted upon and elaborated by all subsequent writers upon localization, namely, the differentiation between symptoms of irritation and those of destruction. He should appreciate the possibility of six classes of symptoms presenting themselves for his consideration, namely, those of (1) local irritation, (2) local destruction, (3) local pressure, (4) invasions by lesions growing from adjacent areas to those under determination ; (5) local instability, (6) reflex action at a distance.

In this connection I will only treat broadly of a few points, as the necessity for this subdivision of symptomatology becomes apparent when considering localization in special regions. In the motor zone the symptom of irritation is especially spasm; but irritation symptoms may occur in other localities. In the visual, aural, olfactory, gustatory, or cutaneous areas they may take the form of hallucinations or other perversions of the senses. Symptoms indicating destruction are, in the motor areas, paresis or paralysis, and in other regions such manifestations as hemianopsia, word or mind blindness, word deafness, anæsthesia, analgesia, anosmia, etc. Pressure and invasion symptoms may, of course, be indicative of irritation or destruction, but are considered by the clinician in their relations to special areas under process of determination. Invasion symptoms will at first commonly be phenomena of irritation, and later both of irritation and destruction. By symptoms of instability I refer to those manifestations which occur as the result of discharging cortical areas without demonstrable gross lesions. Symptoms of reflex action will occur mostly in connection with lesions of the cranial or other nerves, and of the cerebral membranes, particularly the dura mater. They will receive particular attention when discussing some of the sources of error in motor localization.

Certain characteristics, both general and special, of cortical spasm should be well understood. These have been best studied by a few observers, such as François-Franck and Horsley. In Franck's great work the peculiarities both of cortical, sub-cortical and capsular spasm have been determined by electrical experimentation, and are carefully described and graphically represented, the phenomena having been en-

registered. Horsley, practically concurring with Franck, enumerates these characteristics as the presence of a period of latency, then tonic spasm, then clonic spasm, arrest of respiration with cyanosis and salivation.

A study of the initial symptom or sign in a case of irritative cerebral lesion, and also of the serial order of phenomena, may be of the utmost importance. Seguin has proposed to call this initial symptom the "signal symptom." Horsley's view of the manner in which movements are represented in the motor cortex is that in any given part of the cortex as minute as can be examined experimentally, there is represented a definite movement or combination of movements, being the primary movement and elicited by minimal stimulation only; and that secondary movements are due to the subsequent invasion by the discharge of nerve energy of portions of the cortex which lie nearest to and are in close relation with the parts stimulated. The primary movement gives the signal symptom of Seguin, and the secondary movements represent the "serial order" of phenomena.

The signal symptom in Jacksonian spasm has already been made use of in a number of cases to guide the surgeon in part or whole in selecting the site for operation.

In one of Horsley's cases there was first tonic extension and clonic spasm of the right lower limb. "The right upper limb was then slowly extended at right angles to the body, the wrist and fingers being flexed; the fingers next became extended, and the clonic spasms of flexion and extension affected the whole limb, the elbow being gradually flexed. At this time, spasms in the lower limbs having ceased, those in the upper limb continued vigorously. The spasm gradually affected the right angle of the mouth, spreading over the right side of the face, and followed by turning of the head and eyes to the right."

In another case first came "clonic spasmodic opposition of the left thumb and forefinger. The wrist next, and then the elbow and shoulder were flexed clonically, then the face twitched and the patient lost consciousness. The hands and eyes then turned to the left, and the left lower limb was drawn up. The right lower limb was now attacked, and finally the right upper limbs. Paralysis of the left upper limb frequently followed a fit. At frequent intervals every day the patient's thumb would commence twitching, but the progress of the convulsion could often be arrested by stretching the thumb and applying a ligature."

In another case by the same surgeon the spasm was ushered in with a desire to defecate, sometimes with sharp pain in the left side of the

belly. Then followed tightness of the throat, and sometimes spasmodic cough. Then the head and often the eyes turned to the right; the right arm was jerkily protruded, and the patient became unconscious. All the limbs became powerfully flexed, as a rule, but the lower limbs were frequently extended.

Weir and Seguin, Keen, Lloyd and Deaver, the writer, and others have taken advantage of the signal or initial symptom in fixing a site for operations, and thus, either with or without gross lesion, hand centres, thumb centres, face centres, etc., have been excised.

Even movements of the trunk have been used to guide operation by Horsley.* "As regards the trunk muscles," he says, "much might be said, but reference for detail is invited to the above mentioned paper in the Proc. Roy. Soc., 1885. It is, however, worth while pointing out, psychologically speaking, that there is scarcely ever performed a highly purposive act by the trunk muscles only. The movements of the trunk are simply subordinate to the purposive movements of the limbs, and consequently we should not be surprised to find, as in this case, how extremely small a portion of the cortex is sufficient for primary representation of this part of the body. An illustrative case of the position in the human brain of the areas we have just been considering, is that of a case in which a man had been a victim of traumatic epilepsy for many years due to a small punctured fracture of the skull, the said fracture being demonstrated externally by a minute depression three or four millimetres broad. The puncture had caused splitting of the inner table, laceration of the dura mater, and partial destruction of the subjacent cortex, so that at the time of operation (eleven years later) there was found a rough ring of bone on the inner surface of the skull around the centre of the fracture, from which a sharp and corrugated fragment, one cm. long by five mm., broad projected downward, together with a flap of entangled and torn dura mater, into the wall of a small cystic cavity in the cortex just above the junction of the middle and posterior thirds of the superior frontal sulcus. This fairly extensive lesion, which was freely removed (the result being cure of the epilepsy), was thus situated at the point of meeting of the area for raising with protrusion of the upper limb, and of that for turning the head and eyes to the opposite side of the body. The existence of such a lesion was diagnosticated from the fact that the course of events in the epileptic fit began with an aura of contraction of the abdominal muscles, this was followed by turning of the head and eyes to the opposite side, and then there occurred the raising of the upper limb. The exemplifications of the topographical relations of these centres was thus faithfully demonstrated."

* Am. J. Med. Sc., vol. xciii., n. s. 1887, p. 367.

Sub-cortical Lesions and the Intra-cerebral Tracts.

It will be well to say a word or two here about the diagnosis of sub-cortical motor lesions—tumor, cyst, hemorrhage or abscess—which has practical importance, not only for its own sake but chiefly because, in some instances, the question of proceeding with an operation might depend largely upon the supposition of a lesion being sub-cortical. In the case of Weir and Seguin, after the flap of the dura mater was reflected and the brain exposed, nothing abnormal was seen on the exposed surface, and the finger at first recognized no tumor nor abnormality; but at the depth of nearly an inch a small growth was found. If the probability of the presence of a sub-cortical lesion had not been fully considered in this case the operation might have been absolutely fruitless. After a somewhat elaborate study of the question of the diagnosis of sub-cortical tumor, Seguin concludes that in favor of a strictly cortical or epi-cortical lesion are these symptoms, none of them having specific or independent value: "Localized clonic spasm, epileptic attacks beginning by local spasm, followed by paralysis; early appearance of local cranial pain and tenderness; increased local cranial temperature. In favor of sub-cortical location of tumor: local or hemiparesis, followed by spasm; predominance of tonic spasm; absence, small degree, or very late appearance of local headache, and of tenderness to percussion; normal cranial temperature."

The neurologist will probably in time be able in some cases to diagnosticate with sufficient accuracy for surgical purposes, lesions so situated as to destroy intracerebral traits in various regions of the brain. Studies of the different forms of aphasia demonstrate the truth of this proposition. As the various sensory and receptive centres concerned in the production of speech are situated in the parieto-temporal and temporal regions of the brain, the tracts connecting these areas with the motor or emissive speech regions, both for proposition and utterance, must lie in a space of a few inches from before backwards and from above downwards in the region bordering or lying within the Sylvian fissure.

Starr* has brought together in compact form some of the most important facts bearing upon the physiology of the intra-cerebral tracts, drawing largely upon Nothnagel, Charcot, Strumpfell, Flechsig, Edinger, Exner and Spitzka. At least three sets of fibres are to be distinguished in the centrum ovale, namely, the projection, commisural, and association systems. The projection system joins the cor-

* Med. Record, Feb. 13, 1886.

tex with parts of the nervous system below; the commisural system corresponding areas of the two hemispheres; the association system different convolutions of the same hemisphere. The investigation of these different systems is an intricate study, still involved in much obscurity; but it does not come within my province to consider it in this paper except in the most practical way, in connection with the localization of gross lesions.

A careful, elaborate, clinical study of hemiplegias, monoplegias and aphasias, will eventually enable us to separate with considerable certainty lesions of the cortex from those of the centrum ovale, capsules, and ganglia. We will do this by relating the symptoms found not only to lesions of the cortex and the projection system of fibres, as is too commonly the restriction placed on our studies in this direction, but also, to lesions of commisural and association fibres. I am convinced that a lack of consideration of these commisural and association fibres is at the bottom of much of our confusion in analyzing certain cases. Very few lesions are absolutely cortical. Many of those which are generally regarded as cortical, involve to a greater or less extent the sub-cortex. As every convolution of the cerebral surface is connected with some other, and probably with many other convolutions, some association fibres must nearly always be destroyed in these cases.

Differential Diagnosis, particularly of Jacksonian Epilepsy—Dural and other Reflex Epilepsies.

Sufficient diagnostic difficulties are still present to make it important in the light of the tremendous impetus towards operations to carefully examine all questions of differential diagnosis. We should know, in connection with cerebral motor localization, whether certain affections do not simulate cortical epilepsy so closely as to sometimes endanger exact diagnosis. In certain motor and especially spasmodic affections, for example, we have striking resemblance between affections clearly of reflex origin and those as demonstrably central. Trigeminal epilepsies, whether dural, facial, dental, nasal, pharyngeal, laryngeal, or of whatever local origin, may cause unilateral convulsions or even monospasm. Brown-Sequard has contributed largely to our knowledge of this subject both in his early and recent researches. During a few years stimulated by practical specialism, much work in the direction of diagnosis and treatment of reflex epilepsy has been done, some of the best of it by members of the Associations represented in this Congress. The same conclusion might be arrived at for all, as that of

Boucheron * with reference to aural epilepsy, namely, that spasm may proceed from lesions of the ear, eye, nose, pharynx, larynx, face, scalp, or dura mater, and may present all the clinical varieties of epilepsy, or even a form of hystero-epilepsy ; and that the point of origin of these disorders is intense excitation of a sensory nerve.

Dural epilepsies are especially worthy of attention. During a recent operation in one of the Philadelphia hospitals a faradic current accidentally applied to the dura mater, almost instantly produced spasm which invaded the whole body. Dupuy† has published various papers regarding irritation of the dura mater causing muscular movements, claiming that his results are constant when the animal experimented upon is not in a state of anæsthesia incompatible with the manifestations of animal life, and when it has not lost too much blood. Brown-Sequard and Burdon Sanderson have recorded similar phenomena; and the facts of these experiments have been used as arguments against cortical localizations. The Committee of the New York Society of Neurology and Electrology, in 1874, found that galvanization of the dura or other sensitive parts produced by reflex action, muscular twitchings, oftenest on the same side of the body. Duret‡ has given particular attention to the rôle played by the dura mater in the production of sensory, spasmodic, and other phenomena. He does not, however, with Dupuy, hold that the fact of the production of spasms in this way in the least invalidates the doctrine of cortical motor localization, but that a clear differentiation between dural and cortical spasm can be made. Bochefontaine has shown that irritation of the dura mater determined cries of pain, and general movements more or less energetic ; and also that mechanical irritation of the sensitive points of the membrane produced, in certain conditions, movements limited to one or several parts of the body, the movements of the limbs on the same side being more energetic than those on the opposite side. Franck has made careful comparison and contrast of cortical epilepsies and those which are reflex and toxic, including those which are due to irritative lesions of the dura mater. In one of his experiments§ he produced an epileptic seizure from mechanical irritation of the dura, and among other things noted was that at the moment of the irritation of the dura mater the muscles of the face of the same side were attacked with violent convulsions. Attacks followed, as many as nine in twenty-five minutes, all clonic and generalized.

* Compt. rend. Acad. de Sc. Paris, 1887, cv., 944–947.

† Examen de quelque points de la physiologie du cerveau. (Thèse inaugurale, Paris, 1873). Also: Experiences sur les functions motrices du cerveau, 1888. Compt. rend. des séances de l'Acad. des Sci.

‡ Sur les Traumatismes Cérébraux, and Brain. April, 1878. § Op. cit., p. 470.

The following is an abstract of the record of this experiment :

" Experiment No. 45 (Jan. 7, 1879) with M. Senna of Coimbæ. Reflex attack of epilepsy (excitation of the dura mater) commencing on the side irritated.—State of disease.—Arrest of the salivary flow in the attacks.—New series of reflex attacks by incision in the skin.—Circulatory modifications in this form of epilepsy.

" A young dog, spaniel of large size, very vigorous. The motor zone was exposed at the right side while under the influence of a slight anesthetic of chloroform ; a large opening was made in the frontal sinus in order to discover the excitable region, the crucial edge forward.

" It was proposed to study at the same time with salivation, the modifications of the heart and of compression, in their connection with the cortical origin of convulsions ; but the animal was taken with a reflex attack of epilepsy under the following conditions : The dura mater had been cut all round the trephining point ; a fragment remained adherent to the anterior inferior angle of the wound and caused a slight flow of blood somewhat interfering with the experiments at excitation. At the time when an attempt was made to stop the flow of blood with a piece of medicated cotton, the simple friction of the strip of dura mater provoked a series of violent convulsive attacks, having their point of commencement in the muscles of the face and neck on the *same side* (contrary to the epileptic attacks of cortical origin which *always* commence in the opposite side of the body, and severely in the corresponding muscles to the cortical centre excited.)

" The first attack was exclusively clonic, very violent and generalized.

" During several minutes spontaneous attacks succeeded, separated from one another by a few seconds only ; at the fourth attack the convulsive movements and the salivary escape were simultaneous. It was remarked in the first periods of the attack the salivation came on slowly ; it appeared in this fourth attack only thirty-two seconds after the commencement of the clonic movements.

" The animal had nine grand attacks in succession in twenty-five minutes. Then exhaustion came on and it was quiet, the respiration rapid, the heart beats quick, and the arterial pressure much diminished.

" It was left to repose for half an hour, then wishing to apply a manometer to the femoral artery, an incision was made in the skin of the thigh. At this moment a new convulsive explosion came on and without pauses, in the same register as had been made in the former attacks." * * * * *

The nerves of the dura mater spring from the fifth pair, and are distributed nearer to the internal than to the external surface of the membrane, which explains why some lesions of the dura are more likely than others to lead to spasm. The difference depends, in part, at least, on the site and intensity of the lesion with reference to the internal and external aspects of the membrane. Sub-dural hemorrhage is more likely to give rise to reflex spasms than extravasation between the membranes and the skull, unless the blood tears through the membrane. A spicule of bone, in like manner, driven

into the dura is more likely to cause reflex dural spasms than a depressed fragment; while a tumor arising in the membrane is more likely to bring about the same result than an exostosis, or a neoplasm growing from the agglutinated membranes into the brain substance, as is so often seen in intra-cranial growths.

I have notes of five cases in which operations have been performed for epilepsies apparently reflex in character. In two of these spicules of bone were removed from the dura mater. These cases bear out to some extent the views of the existence of distinctive characteristics for reflex epilepsies, but also point to certain resemblances to cases of cortical epilepsy. In one case in which fracture was present in the left frontal region, anterior to the motor area, the patient had convulsions at irregular intervals of weeks or months; usually having sharp pain at the seat of the scar before the seizure. He had no loss of sensation nor paralysis. His convulsions were frequently unilateral; I saw him in one which was confined entirely to the left side, and began in the left leg. This case was trephined for me by Dr. W. J. Hearn, of Philadelphia, and a spicule of bone dissected from the dura mater. In another case the patient was trephined by Dr. J. W. White at my request, for a fracture from a pistol-shot wound just above the right temple. Nearly three months after the injury he began to have spasms; and had had about seven seizures in all. In a convulsion which I witnessed he was completely unconscious; his body was twisted somewhat to the right; his face and all his limbs as well as his head and trunk taking part in the spasm which was tetanic in character. In another case in which an operation was performed for me by Dr. Hearn, the spasm seemed to show a somewhat confusing admixture of what might be termed dural and cortical characteristics. Notes on this case were furnished to Dr. J. B. Roberts, and were published by him in his pamphlet on the Operative Surgery of the Human Brain. In a fourth case seen with Dr. L. W. Steinbach, the patient had been subject to convulsions which seemed to date back to an injury to the head; he had a scar and apparently a depression of the skull over the frontal region. Pressure on this scar brought on a unilateral, largely tetanic convulsion on the same side as the scar. A flap including the scar was lifted, and trephining was performed, but nothing abnormal was found in the inner table or in the dura. The scar was excised. In a fifth case, the patient had convulsions, sometimes on one side, sometimes on both, and these could be brought on by pressure on a scar left by an old sabre cut. The cicatrix was cut out and the patient recovered, at least, he remained for several months in the Hospital without attacks, although before the operation he had been having them at frequent intervals.

With Franck I fear we are not always able to make a trenchant separation between cortical and reflex epilepsies; but a few points may be indicated. In reflex epilepsy the attack does not begin with brusque tetanization as in the case of cortical disease. If the reflex epilepsy has a tonic period it rises slowly to its maximum. In cortical epilepsy the convulsion begins without exception on the side of the body opposite to the side of the brain excited or irritated; in reflex epilepsy, or at least in dural and perhaps other forms of trigeminal spasm, it frequently begins on the same side as the focus of irritation. Unfortunately we have not here a radical difference as it may begin on either side in reflex cases. In the reflex cases if the spasm begins locally or unilaterally, there is not likely to be a definite initial or signal symptom and serial order of movement; one half of the body usually plunges immediately into spasm.

True Jacksonian epilepsies are, I believe, sometimes reflex in origin; that is, they become established as the result of intense persistent peripheral irritation, dural, dental, palmar, etc.; and even after the source of irritation is removed the cortical discharges continue. Herein perhaps lies the explanation of Jacksonian spasm in which gross lesion is not discovered, and herein also sometimes is to be found justification for operation for the removal of cortical discharging areas, even when such lesion is not present. Such a method of origination of cortical epilepsy is in accordance with physiological principles. Meynert,* in the development of his idea of a projection system, has perhaps more clearly than anyone else made apparent the method in which this result may be brought about. Movements which were originally reflex in character may after a time result from cortical impulses. In the normal brain no reflex actions can be performed without exciting to action secondary volitional movements which no longer requires the stimulating influence of a reflex action. Some of the observations and experiments in hypnotism, as those of Heidenhain† in particular, also throw some light upon the manner in which reflex epilepsies may develop into true organic cortical disease. The phenomena of unilateral hypnosis are particularly interesting in this connection. When certain definite cutaneous surfaces are irritated, certain muscles and groups of muscles related to these areas can be brought into isolated or successive action; stroking the ball of the thumb, for example, causes adduction of the thumb towards the palm; or stimulating the skin over the sterno-mastoid causes the head to assume the wry-neck position.

* Psychiatry, a Clinical Treatise on Diseases of the Fore-Brain. Translated by B. Sachs, M.D.

† Hypnotism or Animal Magnetism. Translated by L. C. Wooldridge, M.D.

A case reported by me in 1880,* is interesting in connection with this question. It was one of epilepsy clearly Jacksonian in type, and as clearly due to a fibroma involving a nerve trunk on the palmar surface of the hand. The patient, 15 years old, had had the seizures since the age of 4 years, they coming on after an injury to the hand at the situation of the fibroma. After removal of the growth, she had spasmodic attacks of the same type as before the operation, but less in frequency for a year, after which she rapidly improved, and I have been recently informed has had no spasms for nearly two years.

A description of the usual character of the attacks shows that they were distinctly Jacksonian. The description will be quoted at length because of the importance of the matter under discussion.

The distal phalanx of the ring finger of the right hand was first flexed; secondly, a few spasmodic movements of flexion would occur in this finger; thirdly, the other fingers and thumb of this hand would begin to twitch convulsively—the second phalanges would be flexed, the last extended; fourthly, the clonic convulsive movement would extend to the right hand, forearm and arm, and simultaneously the muscles of the lower part of the right side of the face would become affected with spasm, a tremor also appearing in the tongue during this period; fifthly, the right arm and leg would now become affected with a clonic spasm, causing them to assume positions of flexion, the head, neck and body being drawn by the spasm at the same time to the right, a condition of pleurosthotonus being, in fact, produced. The seizures would pass off with a very severe jerking movement of the right shoulder, and a renewal of the twitchings of the muscles of the right angle of the mouth. These movements of the shoulder and mouth would sometimes occur only once, just before the close of the attack; more frequently, however, they would take place two or three times in succession. Occasionally the patient would bite her tongue during the paroxysms. She apparently was never entirely unconscious during the attack, no matter how severe it might be. During the height of the convulsion, if her hand was pressed too hard she would manage to gasp out, "Don't," or to make some other exclamation.

Unilateral Nervous Affections in Bright's Disease.

Considerable evidence has accumulated to show that affections of the nervous system, strictly limited to one-half of the body, occur during the course of some forms of Bright's disease. In this country Dercum† has reported cases of hemichorea, hemiplegia and unilateral convulsions. Raymond,‡ Chantmesse and Tenneson§ reported series

* Phila. Med. Times, December 18, 1880.

† Jour. Nervous and M. D., vol. xiv., No. 8, August, 1887, p. 473.

‡ Thèse pour le Doctorat en Médecine, 1878, Versailles, and Rev. de Méd., Sept. 1885.

§ Rev. de Méd., Nov. 1885.

of cases of unilateral affections, chiefly hemiplegia and epilepsy apparently of uræmic, or at least renal origin. In not one, according to the reporters, could the trace of a strictly focal lesion be discovered. Chaufford * reports a highly interesting case under the title of uræmic convulsions of the Jacksonian form.

Hystero-Epilepsy and Jacksonian Epilepsy.

Some cases which seem to be clearly forms of hystero-epilepsy closely resemble organic epilepsy of the Jacksonian type. Hystero-epileptic attacks, it is well known, can be produced by irritation of the hystero-epileptogenic zones, described by Charcot, Richer, and others, which are evidently analogous to the epileptogenic zones of Brown-Sequard.† Almost every form of spasm in localization and extent can be found in the descriptions of hystero-epilepsy. Features of distinction are, however, present. Undoubtedly one reason for the similarity between spasmodic affections reflex, hysterical, toxic and cerebral, lies in the fact that in these cases, whatever may be the starting point, central areas are discharged and give definite character to the convulsions. Horsley speaks of hystero-epilepsy as a cortical disease, but this view cannot be upheld for all cases, if he means by this that the spasms are usually the result of cortical discharge. They are rather sometimes bulbar or spinal, cortical inhibition being removed.

The difficulties of making a diagnosis between grave hysteria or hystero-epilepsy, and cerebral tumor or other organic lesion with apparent or real Jacksonian symptoms, is sometimes great, and was strikingly shown in a case seen by me in consultation with Dr. J. M. Barton, of Philadelphia. This patient, a married woman, 35 years old, came under observation in the spring of 1886. She had been in bed eight weeks, and had taken no part in her household affairs for several months. Her sickness began with complaints of headaches and feelings of slight numbness in the left hand and arm; she would occasionally drop things from this hand. She soon developed analgesia and anæsthesia in the left arm and leg, and sometimes in the face; this varied in severity. Her mental condition gradually changed, she became irritable, absent minded, and lacking in attention and judgment. She had at times hallucinations of sight, which usually occurred after lying down, these often taking the form of animals, as cats, mice, etc., disappearing around corners. Hearing was good in the left ear, but in the right was diminished; careful examination showed no external or middle ear disease.

* Arch. Gen. de Méd., Paris, 1887, ii., 5–19.

† Lancet, Lond., 1886, ii., 1211–1213, Abstract of Brown Lectures.

She suffered almost continuously from headache, which she described as violent and agonizing, and which she localized mostly in the fronto-parietal region ; and a cranial area over the right motor zone was very tender to pressure and percussion. Nausea and severe vomiting came on late, and both headache and vomiting were accompanied by vertiginous sensations. A curious symptom was a constant diarrhœa, the patient having six to fifteen passages a day, and sometimes as many as twenty-four. She also often spat a bloody fluid from the mouth. Her appetite and sleep were much impaired, and she lost considerably in weight.

Ophthalmoscopic examinations made in March, 1886, by Dr. W. W. McClure, gave the following results : *Right eye:*—The pupil more active than the left ; media clear ; slight physiological cupping and venous pulsation ; no arterial movement observable. Slight choroidal change in pigmentation to the temporal side of the nerve ; outline of nerve above not defined, but merged with the retina. Macula good. Hypermetropia. *Left eye :*—Pupil more dilated and less active than in the right eye. Appearances much like those seen in the other eye, but worse. Evidences in color and margin of slow chronic inflammation. Macula good. Right eye, vision, $\frac{20}{20}$; left eye, vision, $\frac{20}{40}$.

After her illness had continued several months, the patient began to experience at times attacks of flexure of the fingers and thumb of the left hand, and cramp-like feelings in the forearm and arm. Later these spastic attacks began in the left foot, causing the great toe to be flexed upward and the other toes downward, with also some cramp feelings in the left leg.

A number of consultations were held with reference to this patient with Drs. J. B. Roberts and C. B. Nancrede, who took the view that the case was probably hysterical, and treatment was eventually suspended.

The patient's general health improved ; she gained in weight and attended to household and business affairs, never, however, getting entirely rid of her main symptoms, and recently some of these have returned with renewed vigor. She has fallen several times in public places and at her own home, appearing to become entirely unconscious. The attacks are preceded by a thrill passing up the left leg, and twitching of the left arm ; the latter continuing through the entire seizure and being the only muscular movement noticed.

Visual Localization.

Next to determinations of the motor zone visual localizations are the most conclusive, and this in spite of the hard-fought battles of the

physiologists over the cortical sight areas. Clinical medicine and pathology have here come bravely forward to clear away the storm. A few well reported cases of hemianopsia with autopsies, as those by Jastrowitz,* Haab,† Huguenin,‡ Monakow,§ Seguin,‖ Hun,¶ Fere, Keen and Thomson, seem to settle beyond doubt the connection of the cuneus and adjacent region with the retina and simple visual sensation.

Jastrowitz has recorded a case of paresis of the right leg, arm, and face, with a peculiar form of aphasia. The patient was unable to read and write connectedly; he could not understand written words. The history does not relate definitely whether or not he understood spoken words. Right hemianopsia was also present. The autopsy showed tumor of the left occipital lobe and precuneus.

Seguin, in a contribution to the pathology of hemianopsia of central origin, in many respects the most valuable publication on the subject which has yet appeared, collected forty cases with autopsies, and five traumatic cases without autopsies. Eleven of these were cases of hemianopsia due to lesions of the white substance of the occipital lobe. Sixteen were cases of cortical lesion, or of lesion limited to the cortex and the white substance immediately subjacent; and four of the sixteen (those of Haab, Huguenin, Fere, and Seguin referred to above) are what might be termed conclusive cases as to the question of the location of at least a portion of the cortical visual centre in man; as in them the lesion was circumscribed and occupied nearly the same place in the occipital lobe. They would, at least, seem to settle definitely that in the cuneus and its immediate neighborhood the visual half centre for retinal sensations is located. Owing to their importance I will give briefly from Seguin a condensed abstract of these cases.

Haab's case was a man, 68 years old who had an attack of temporary paresis of the left extremities. The patient complained that he could not see to his left with his left eye—though his right eye was normal. No anæsthesia; intelligence was normal, hearing good. Central vision—1 (H.2). There was left homonymous hemianopsia, the limit reaching quite (?) up to fixation point. In the right fields color perception was good. Optic nerves presented a "senile grayish color." The autopsy showed the extremity of the right hemisphere

* Centralb. für prakt. Augenheilk, vol. i., December, 1879, p. 254.
† Klinische Monatsblätter f. Augenheilk, xx., 141, 1882. ‡ Ibid.
§ Archiv f Psychiat, u. Nervenkrankeiten, Bd. 16, S. 166.
‖ Jour. of Nerv. & Men. Dis., vol. xiii., No. 1, January, 1886, 1–38.
¶ Am. Jour. Med. Sci., vol. xciii., January, 1887, 140–168.

5 mm. shorter than its fellow and a depression in the right occipital lobe, the pia hanging loosely over a cavity containing clear fluid. The patch was mostly upon the mesial aspect of the hemisphere (including apex). It occupied the site of the fissure hippocampi, and extended beyond it above and below. The white substance was but slightly injured.

Huguenin recorded the case of a girl, aged 8 years, whose chief symptoms were headache in paroxysms; later, frequent vomiting, sleep broken; severe convulsions which frequently recurred; increasing dementia; slight neuritis with some swelling. It was noticed after some months that the patient held her head obliquely to the left. Examination revealed left homonymous hemianopsia, the only symptom indicating a focal lesion of the brain. The patient died of broncho-pneumonia. At the autopsy two tumors were found in the brain; one at the apex of the left frontal lobe. The second tumor lay in the mesial aspect of the right occipital lobe, projecting a few mm. above the level of the brain, firmly adherent to the pia and only slightly to the dura. Its length was 3 cent., height 3 cent., thickness 2.5 cent.—mostly buried in brain substance. It lay directly over the sulcus hippocampi, extending to either side of it. The base of the occipital lobe was not involved.

Fere reported the case of a female, aged 52 years, who in November, 1883, had a sudden apoplectic attack followed by right hemiplegia. She had partial and slight right hemianæsthesia to cold and pain. Hearing, taste and smell were normal; and typical right lateral hemianopsia, the vertical line passing through the point of fixation, was present. Autopsy showed a yellow patch destroying the greater part of the left cuneus and encroaching somewhat on the adjacent occipito-temporal convolution, the fifth temporal convolution of Ecker. No other lesions were present.

Seguin reports one personal case. The patient, a man 46 years old, consulted him first in January, 1884, for insomnia and dyspepsia. He was treated for various symptoms with varying success. December 5, 1884, he had an attack in which he complained of numbness in the left cheek, arm and face, and most marked in the hand and foot. He had no distinct hemiplegia, and no hemianæsthesia, but thought that tactile sensibility as in passing his fingers over objects, was somewhat duller. He could not see objects on his turning his head and eyes, and testing revealed left lateral hemianopsia, with a vertical division line not including the point of fixation. Central vision was good. He continued with varying symptoms until May 17, 1885, when he died. During his long illness he had attacks of acute hallucinatory

mania, both aural and hallucinatory illusions being present. He had chills, high fever, and sweats which followed no distinct periodicity. Previous to his death his speech was sometimes somewhat difficult to understand. The hands both showed disorders of movement, choreiform tremors, and in the left hand slight ataxia and larger motion. The hemianopsia persisted to the last unchanged. The central vision remained good. The brain lesion which doubtless caused the hemianopsia was found on autopsy to be a large focus of yellow of softening, evidently an old patch, involving the basal part of the cuneus, the fourth and fifth temporal convolutions of Ecker, and a part of the hippocampal gyre. Other lesions were present in the brain, but as this case was in harmony with others Seguin regards it very properly as of great value.

In his table in the paper referred to on cortex hemianopsia, Seguin has included five cases of traumatic hemianopsia due to injuries of the occipital region of the skull, and lesion of the subjacent brain. I will refer to only one of these cases, that of Keen and Thomson, which is of historical as well as of scientific interest. Through the courtesy of Dr. Keen, I have examined a cast of the head of this patient. The case was reported in the photographic Review of Medicine and Surgery, February, 1871 ; also in the Medical and Surgical History of the War of the Rebellion, Part 1, p. 206–207. It is also referred to in Flint's Physiology of Man, vol. v., p. 41–42, 1874, as one of the first cases to show that the filaments from the optic tracts on the two sides are connected with distinct portions of the retina. The patient was kept under observation off and on for a number of years, and was examined and exhibited by Dr. Seguin at the time of the reading of his paper. His hemianopsia was found unchanged twenty-three years after the reception of his injury. The patient died within a year or two, but unfortunately no autopsy was secured. He was hit on the head by a minie ball at the battle of Antietam, September 17, 1862. The wound of entrance was in the middle line 1¼ inch above the external occipital protuberance ; it made its exit about two inches to the left of the middle line, and three above the wound of entrance. He noticed impairment of vision two days after the injury. About ten days afterwards he had an attack of loss of consciousness with some paralysis of the right arm and right leg, which lasted some two or three months. His memory was imperfect for some months, but he had no aphasia. In walking he was very giddy, and noise and laughter would hurt him. His mental and physical powers gradually grew better. He was seen and examined by Drs. Keen and Thomson, in December, 1870. Among other conditions he was found to have a

complete lateral hemianopsia. Upon testing the field of vision it was found to be divided in each eye by a line passing through its centre in a vertical direction—total blindness existing to the right, and perfect vision to the left of this line. Ophthalmoscopic examination showed no pathological appearances. When Dr. Seguin examined this man in 1885, he presented no distinct paralysis, no anæsthesia, no aphasic symptoms. His tongue deviated a little to the right, and the grasp of the right hand was a little lighter than that of the left. A rough test with a small white object at 18 inches showed right lateral hemianopsia, with line passing outside of point of fixation, and a darkened area in the left upper temporal quadrant. Pupillary reaction was normal. Examination of the fundus showed blood vessels of normal size; outer temporal quadrant of each disc whiter than normal; left a little whiter than the right. An experiment on the cadaver showed that the track of the ball was such that it must have injured the optic fasciculus on its way to the cuneus.

The most important case which has been recorded since the paper of Seguin is that of Hun, in which a defect in the fields of vision involving the lower left quadrant of each eye occurred with atrophy of the lower half of the right cuneus.

The patient, a man aged 57, in 1869, had a severe attack of double pneumonia, and during the year following slight attacks of vertigo while walking, which were attributed to weakness. From 1877 until his death, he was troubled by slight deafness and by more or less roaring in his ears. In 1882 he had a large carbuncle on his neck, and after that time he seemed less vigorous than before. Early in December, 1884, he was examined by Dr. Merrill, who found normal reaction of the pupils, normal appearance of the fundus, vision and color perception perfect, but a defect in the fields of vision involving almost the whole of the left lower and the peripheral portion of the left upper quadrants in each field. The defect was somewhat more extensive, especially as regards the upper quadrant, in the left field of vision than in the right.

From this time the condition of the patient did not change materially. He continued to be very nervous, and at times irritable and suspicious. He exhibited no paralysis of motion or sensation. His memory was weak in regard to names; he often called the same person by several names in the course of conversation; while in other respects, as in recognizing faces, his memory was excellent. He slept but little, and in his sleep there was much twitching of the limbs. In 1885 he had a severe attack of angina pectoris, and for two weeks he could walk only a short distance without bringing on an attack of the pain. In

February, 1886, he had his most severe attack of angina, lasting several hours, and from this attack until his death, he was scarcely a day without pain in the precordia or in the arm. During the last month of his life he vomited often. He complained of increasing dullness of vision and of greater angle of obliteration, and was much troubled by a new building near by appearing to be out of line. On May 7, 1886, while quietly walking in the street he sank gently to the ground and died.

At a point on the median surface of the right occipital lobe was complete atrophy of the cerebral convolutions, only a trace of them remaining as a delicate gray gelatinous fringe. This atrophy was strictly limited to the lower half of the cuneus; being bounded below by the calcarine fissure, in front by the parieto-occipital fissure, and above by a curved line which started from the parieto-occipital fissure and arching backwards across the middle of the cuneus terminated at the posterior border of the median surface close to the calcarine fissure. The white matter underneath the point of atrophy was softened to a depth of about one-third of an inch. There was no deposit of pigment in the neighborhood. The corresponding point on the left occipital lobe showed no atrophy, nor did any of the other cerebral convolutions. Sections through the brain substance, the optic thalami, and the other ganglia at the base revealed nothing abnormal. The optic nerves and tracts showed no microscopic atrophy or degeneration. No microscopical examination was made.*

Views of Physiologists as to Visual Localization.

The view of Ferrier as expressed in the latest edition of his *Functions of the Brain*, is that the angular gyres maintain relations with the areas of clear vision, and as a matter of course especially with the macula luteæ.

This physiologist has modified his earlier views in so far that he no longer localizes the visual centres in the angular gyres to the exclusion of the occipital lobe; but believes now that the visual centres

*At a recent meeting of the Philadelphia Pathological Society, I presented the brain of a man who had been blind more than twenty-five years—how much more could not be positively ascertained. Both occipital lobes were unquestionably small. The cuneus on each side was small, the first occipital convolution of Ecker (superior external pli de passage of Gratiolet, and par-occipital of Wilder) showing lack or arrest of development. The second and third occipital convolutions of Ecker, especially on the left, presented a narrow, dwindled appearance. In another brain of an old woman, blind for at least thirty years, similar gross appearances of arrested development in the occipital region were present.

embrace not only the angular gyres, but also the occipital lobes which together he terms the occipito-angular regions.

Recently considerable activity has been exhibited in the investigation of visual centres.

From a long series of experiments upon the monkey's brain, on which he was engaged with Mr. Victor Horsley during more than two years, Schäfer* writes as follows :

"With regard to vision our experiments were not conclusive. We found that extensive lesions, both of the occipital lobe and of the temporal lobe, were invariably followed by visual disturbances, taking the form, when the operation was confined to one side of the brain, of bilateral homonymous hemianopsia ; but in nearly every case the hemianopsia was merely temporary, and after a certain time we could not in our monkeys obtain any distinct evidence of the persistence of the visual defect. The most marked results of this kind were obtained when the occipital lobes were the seat of the operation, extensive unilateral lesions in this region producing hemiopia, and bilateral lesions producing amblyopia ; but in neither case were the symptoms permanent, and after a time the animals, so far as we were able to determine, could see as well as their intact fellows. In one case only did the hemiopia persist, and this was one in which, after a bilateral lesion of both occipital lobes had been carried out and the temporary blindness thereby produced had been recovered from, the angular gyrus of one side was destroyed. This second operation, made upon the animal in which the occipitals had already been extensively destroyed *without* permanent blindness, did produce a condition of hemianopsia which lasted until the animal's death some three months later. We were of opinion at the time that this instance might warrant us in taking up a position similar to that of Luciani and Tamburini, and intermediate between those of Ferrier and Munk—the former of whom originally denied the participation of the occipital lobe in the visual perceptive function, and still appears to regard it as subordinate to the angular gyrus ; whereas the latter would localize those perceptions entirely in the occipital lobe, and deny all participation of the angular gyrus. But we made only four experiments upon these regions, and in none of them was the removal of the occipital lobe complete, as was proved by post-mortem examinations of the brains. They were not, therefore, decisive against Munk's statement, that persistent hemiopia or blindness follows extirpation of one or both occipital lobes alone, and it became necessary to pursue further inquiries in order to test its accuracy."

In conjunction with Dr. Sanger-Brown, Schäfer also experimented upon the angular gyre and upon the occipital lobes. Destroying one angular gyrus as completely as possible with the actual cautery they could discover no defect of vision, no loss of movement of the eyes or eyelids, and no anæsthesia of the corneal conjunctiva. A week later the angular gyre of the opposite was destroyed, also with negative results.

* Brain, London, January, 1888.

In illustration of the effects produced by complete removal of the occipital lobe, and that alone Schäfer gives two instances, in one of which the operation was unilateral, in the other bilateral.

"In the monkey upon which the unilateral operation was performed, the left occipital lobe was removed by a vertical incision carried along the line of the parieto-occipital fissure. That the removal was exact and complete was confirmed on post-mortem examination, some eight months after the establishment of the lesion, when it was seen that the whole of the occipital lobe, and only this lobe was involved, the angular gyrus being quite intact and normal, and the surface of the section looking as fresh, and showing as clearly the distinction of grey and white matter, as if the operation had just been performed. The result was the immediate establishment of bilateral homonymous hemianopsia, which persisted the whole time the monkey was kept alive. Objects so placed that their images fell upon the left half of the retinæ were taken no notice of: a threatened blow coming from the right-hand side of the mesial visual plane was winced at or avoided; currants strewn upon the floor were only picked up towards the left side, the animal working in that direction. In the case of the monkey with bilateral operation the result was total and persistent blindness."

The views of Hun* are that the convex surface of the occipital lobe, particularly of the left side, is associated with complete visual perception and recognition; and also that the so-called left angular gyre is essential for the memory of the appearance of words, lesions of it causing alexia and agraphia.

I shall not attempt to analyze, criticise or reconcile these various and varying views. They agree, at least, in showing the production of hemianopsia from lesions of the cuneus and adjacent occipital lobe.

The general visual zone, which has been determined can probably be compared as to subdivision with the general motor zone. As the motor zone has been subdivided into areas of representation, not only for the leg, trunk, arm, face and speech, etc., but also into areas or centres for parts of the leg, arm, face and speech, so efforts, partly successful are now being made to subdivide the visual zone. The retina so far as its connection with the central cortex is concerned, can be subdivided into segments probably of a somewhat regular shape. At first studies in hemiopia and hemianopsia seemed to show that the only definite connection was between the halves of the retinæ and cortical centres, but the latest observations indicate that quadrants, and probably even smaller portions of the retinal expansion are related to separate areas in the brain. The macular region almost certainly has its special cortical centre. The tendency of investigation, both experimental and pathological, is to show that the whole of the occipital lobe, and adjoining portions of the parietal and temporal lobes,

* Op. cit.

are in some way concerned with vision, as I have tried to indicate in the diagram (Fig. 6).

With reference to visual localization, the importance of confirming physiological experimentation by careful clinico-pathological observations cannot be overestimated ; these observations are here even of more importanoe than in motor localization. The movements of a limb or a portion of a limb, can be studied with considerable accuracy in the lower animals as well as in man ; but in investigating sight, the other special senses, or general sensibility, in the lower animals, we are confronted with special difficulties and sources of error. This is perhaps more strikingly true of hearing, taste and smell, than of the other senses but it is true for all. Schäfer referring to a monkey from which he believed he had eradicated both angular gyres, states that for three or four weeks the animal failed to see objects which were just below or to one side of its eyes, and even at the time of writing there appeared to be complete absence of vision in the antero-superior and lateral portions of the retinæ ; but it was difficult to prove this because the monkey had acquired the habit of rapidly directing his head and eyes so as to use the central parts of the retinæ.

Operations guided by Visual Localization.

In spite of the conflict of views with reference to visual localization, sufficient has been determined to indicate several positions for operation guided chiefly by visual symptoms.

If a patient has lateral homonymous hemianopsia as the special localizing symptom, operation should be performed with the view of reaching the cuneus behind the position of the parieto-occipital fissure. If, without hemianopsia, the patient fails in intelligent recognition of things and words, the aim should be the lateral occipital convolutions and the angular gyre, which is adjacent, or indeed may be situated in the occipital lobe. When, with lateral homonymous hemianopsia, the patient also has hemianæsthesia, the lesion is probably in the tracts between the cuneus and the primary optic centres, large enough also to involve the sensory tracts. Such a lesion would probably best be reached beneath the position where, on the lateral aspect of the hemisphere, the parietal, occipital and temporal lobes come together. Hemianopsia is sometimes a late symptom, the result of invasion of the visual region from other localities ; such a case will be referred to later when speaking of large lesions of the temporal lobe.

I have made various examinations of the human brain on recent as well as older specimens in order to determine, if possible, where the optic radiation proceeding from the corpora genticulata and corpora quadrigemina enter the hemisphere to proceed to the cortical centre of vision ; in part with a view of determining the relations of the angular gyrus, cuneus, and adjoining occipito-temporal region to this place of entrance, and therefore the position and direction of the intra-cerebral tracts. Undoubtedly, so far as the human brain is concerned, both the angular gyrus and the occipital lobe are so placed with reference to the geniculate bodies and corpora quadrigemina that they are anatomically and morphologically in relation with these radiations. Schäfer * gives a view of a longitudinal section of the hemisphere of a monkey which he believes demonstrates that fibres proceeding to the angular gyrus are not involved in lesions of the occipito-temporal regions. Whatever may be true of the monkey, this is certainly not true of the human brain.

Wernicke's Hemiopic Pupillary Inaction.

The great practical importance of some of the studies which have grown out of cerebral localization is seen in the fact that the simple but valuable discovery in semiology, Wernicke's hemiopic pupillary reaction, enables us with a flash of the ophthalmoscope to throw the lesion producing hemianopsia back of the primary optic centres. With a normal retina uniform pupillary reaction occurs no matter at what position the ray of light strikes upon it. Seguin † clearly explains this hemiopic pupillary reaction, or inaction, as he suggests to call it, in one of his papers on hemianopsia. In a word, the test depends upon the fact that the hemiopic part of each retina being physiologically inert, fails to receive any impulse from the light which is thrown on it. When the lesion is in the cuneus or occipital lobes, hemiopic pupillary reaction is not observed. When the lesion, producing hemianopsia involves the optic tracts, there is from an early period hemiopic pupillary inaction as well as partial nerve atrophy. A full discussion of this subject will be found in the paper of Seguin, and the original memoir of Wernicke.

* Brain, July, 1888.

† Jour. of Nerv. and Ment. Dis., vol. xiv, November and December, 1887, 721–737, and Fortschritte der Medicin, I. Heft 2, 1883, cited by Seguin.

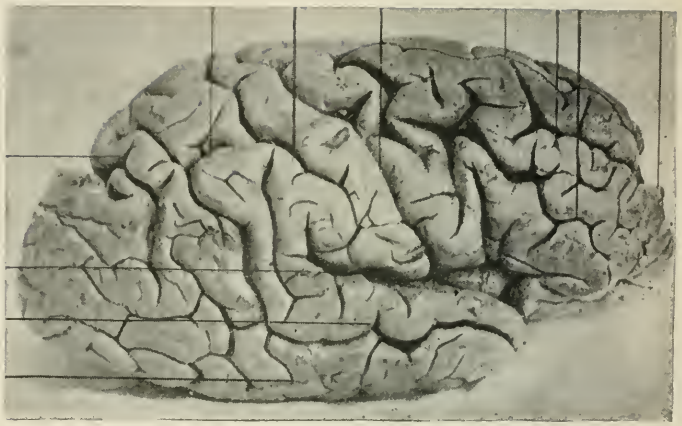


Fig. 8.—Brain of Delusional Monomaniac.

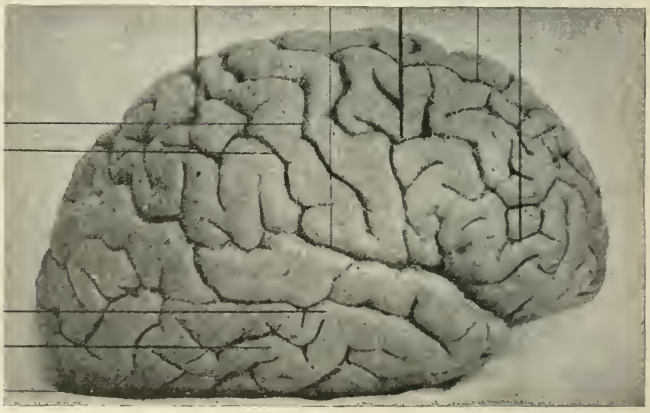


Fig. 9.—Chinese Brain.

Morphological Peculiarities of the Human Brain.

It is a question whether we are in a position for practical purposes to make any distinction between the angular gyre and the external occipital convolutions ; nor is it absolutely necessary that this should be done in trephining with a large instrument.

Morphological peculiarities of the human brain have an important bearing on this subject. In 1880,* I called attention to this matter and particularly with reference to the so-called angular gyre, referring to the brain of a white man in which the first temporal sulcus extended back of the transverse occipital and to within half an inch of the longitudinal fissure. The angular gyre is usually regarded as the arch composed of the posterior uniting portions of the inferior parietal and superior temporal gyres. It has seemed to me that these morphological aberrations are especially marked in the parieto-occipital region, so much so in some cases as to lead us to use great care in determining by external methods the position of the cerebral areas.

It will only be necessary to call attention to one or two striking peculiarities in the brains examined, mostly in the insane and in individuals of low organization.† In a case of delusional monomania, for example, the occipital fissure showed itself and in its communications and surroundings, marked ape-like conditions, such as might have been very deceptive if exposed by trephining (Fig. 8).

In the other hemisphere of the same case the cuneus was small, and the first temporal fissure presented an unusual appearance, being divided about its middle by a comparatively large gyre. In another case, in the right hemisphere, the second temporal fissure was short, and communicated with a very well defined almost vertical fissure, which separated the temporal from the occipital lobe. The first temporal, as well as the second, communicated with this fissure, the vertical extension of the first being, in fact, continuous with it. The cuneus was distinctly smaller and of a different shape from the cuneus of the right hemisphere. In another case, the first temporal fissure was remarkable in that it was completely confluent with the Sylvian, extending from the point of the confluence as a deep, well defined trench backwards and upwards nearly to the sagittal fissure, crossing and confluent with the interparietal. In a Chinese brain (Fig. 9) studied by Dr. A. J. Parker and myself, a remarkable peculiarity of the brain was that in the right hemisphere the first temporal fissure, beginning near the anterior extremity of the temporal lobe, passed

* Phila. Med. Times, April 24, 1880, 366–370.

† Jour. Nerv. and Ment. Dis., vol. xii., September and October, 1886, 517–553.

backward across the entire extent of the parietal lobe and over the median edge of the hemisphere for the distance of half an inch, terminating in the precuneus just in front of the parieto-occipital fissure.

This is an interesting field, and has some practical importance. Even the Sylvian, the central, and the parieto-occipital fissures, have considerable variations, the first in its horizontal level and extension, and the others in their antero-posterior position. I have reported one case in which the Sylvian fissure took a position much more vertical than usual and reached nearly to the longitudinal fissure. As a rule, variations, such as those referred to here, for fissures like the Sylvian, would not confuse us in operating after the methods of Broca, Thane, Reid, Horsley, and others ; but they are certainly worthy of some consideration.

Localization of Cutaneous Sensations.

With reference to the localization of the centres for cutaneous sensations—touch, pain, temperature, etc.—we are acquiring more positive knowledge and may be able in time to use such knowledge to guide in surgical operations. The evidence, physiological, clinical, and pathological is becoming stronger in favor of the existence of a zone for these general sensations separate from the motor areas.

Various, indeed, have been the views held with reference to the localization of these sensations. Luys, largely from anatomical studies, made the thalamus a centre of sensibility, subdividing it into four special centres—olfactory, optic, acoustic, and for general sensibility. Ferrier, regards the thalamus as in some way connected with sensation, but believes that differentiated areas for sensation also exist in the cortex. He holds that it is probable that the thalami are especially related to the sensory tracts, and the striate bodies to the motor tracts ; that these ganglia represent in a subordinate manner all the sensory and motor tracts of the cortex. The thalamus is a centre of conveyance, or an interrupting ganglion in the course of the sensory tracts. The experiments of Monakow led to the conclusion that different portions of the thalamus are related to cortical areas. Fournier believes that sensory fibres terminate in the thalamus. Crichton-Browne regards this ganglion as a great centre of general sensibility ; but Flourens, Longet, Schiff, and Tamburini, among others, have assigned to it motor functions. The weight of the evidence both from experiment and disease connects the thalamus with various forms of sensation. It is probably a halting place where sensory impressions going from the periphery to the cortex undergo some transformation. It is nec-

essary, at least, briefly to thus refer the functions of the thalamus as some authorities are inclined to regard it as the sole cerebral region for sensibility.

From analogy and general principles it is altogether probable that we have above the ganglia a separate localization for the different forms of cutaneous sensibility. As Ferrier puts it, up to the point of radiation towards the cerebral cortex the sensory paths have been proved to be entirely differentiated from the motor, and it is therefore extremely unlikely that the two should become jumbled together indiscriminately in the cortical centres. We have not only the well known differentiation between sensory and motor nerve roots and tracts in the spinal cord, oblongata, pons, and crus cerebri; but abundant evidence as to the separation of sensory from motor tracts in the internal capsule. The very simple but natural question may be asked: where do these paths lead if not to special cortical areas for sensation? Cases have been reported by Demeaux (quoted by Ferrier), by F. Müller and others, in which autopsies have shown hemianæsthesia to result from lesion of the hinder third of the internal capsule or adjoining portions of the corona radiata.

Veyssière produced hemianæsthesia on the opposite side of the body by dividing this portion of the capsule in animals. We not infrequently see hemiplegia and hemianæsthesia united in the same patient as the result of a capsular lesion, and numerous autopsies have shown that the posterior and middle parts of the capsule are in these cases involved in a destructive lesion. Cases of hemianæsthesia from lesions both of the thalamus and lenticular nucleus have been reported, but in very few has the lesion been strictly localized to these ganglia, and even in these it is a question whether the hemianæsthesia resulting has not been due to pressure on the internal capsule.

Recently I made a post-mortem examination on a patient who died at the Philadelphia Hospital, a woman 47 years old. She had had many symptoms which were supposed to be hysterical. On the morning after her admission she had a fit or seizure, the character of which was not closely observed. Afterwards, however, she was paralyzed and anæsthetic over the right half of the body and limbs, her speech also being thick, but consciousness not lost. Partial anæsthesia was present over the left half of the body. Knee-jerk was increased, and ankle clonus was present on both sides. She improved rapidly but remained in bed two weeks. Two days before her death she had an apoplectic attack and became completely paralyzed and hemianæsthetic in the face, limbs, and trunk of the left side. The autopsy showed a

foyer of softening in the right hemisphere, including the entire breadth of the internal capsule, two-thirds of the adjoining lenticular nucleus, a small anterior external portion of the thalamus, and a narrow strip of the caudate nucleus where it curves around the thalamus. Careful transverse sections showed that the anterior boundary of this focus of softening was 1¼ inches behind the head of the caudate and lenticular bodies, and that it extended from before backwards about ⅝ of an inch. It was so situated that a transverse section immediately in front of the thalamus marked its anterior boundary. The softening of the narrowed caudate nucleus was all that was observable in opening the ventricle. The left hemiplegia and hemianæsthesia might very properly be attributed to the complete transverse destruction of the internal capsule. A large recent embolus was found in the right middle cerebral artery. It is difficult to account for the temporary right hemiplegia and hemianæsthesia, except on the theory of hysteria or functional disturbance, as no lesion of the cerebral hemisphere on the left side was discovered.

The fibres of the sensory tract are probably separate from the motor even in the centrum ovale.

"The third set of fibres of the projection system," says Starr,* "includes those which lie just posterior to the motor tract, and which pass inward from the parietal convolutions. These take a similar course to those of the motor tract, and fill up to a considerable extent the space between it and the radiation of the visual tract, toward the occipital lobe. They are mingled with fibres which pass to the optic thalamus, but are separable from them, as Edinger has shown, in fœtal brains, and may be traced down through the capsule to the tegmentum of the crus, where they divide into a portion going to the leminscus, and a portion going to the formatio reticularis. This set of fibres conveys the sensations of touch, pain, temperature, and muscular sense, and lesions in its course will cause disturbance of these sensations. Like lesions in the motor tract, the rule obtains that the nearer the cortex the more likely is the lesion to cause an affection of a single limb, while the nearer the capsule the more likely is the symptom produced to be hemianæsthesia."

Ferrier in his earlier experiments found that lesions involving the horn of ammon and hippocampal convolution caused impairment or abolition of tactile sensibility on the opposite side of the body, and located the cortical centres for this form of sensibility in the hippocampal region. In experiments with Professor Yeo, in 1884, he established lesions in the hippocampal regions in ten monkeys, and in

* Medical Record, Feb. 13, 1886.

five of these in both hemispheres, and these lesions showed that tactile sensibility was in every case impaired or abolished, according to the amount of destruction of the hippocampal and temporo-sphenoidal region. It was also established, however, that a very extensive lesion might be made in one or both hippocampal regions without producing permanent anæsthesia.

Munk* claims that the area of common sensation—including sensation of pressure, location of a limb, muscular sense, and touch—lies in the central region, including the anterior and posterior convolutions and adjacent cortex. He divides this area into special centres for the hind leg, fore leg, head, eye and ear muscles, neck and body; holding that these coincide with but are somewhat more extensive than the corresponding motor centres for these parts.

The sensations of touch, according to Luciani, are perceived in the central convolutions, and therefore lesion of these cause anæsthesia as well as paralysis. The tactile area includes the parietal convolutions also, but does not reach the occipital and temporal lobes. This author holds that all sensations appear to have a common zone in the parietal region, and lesions in this common zone may cause disturbance of all the senses.

It is worthy of remark that the physiologists and others, who contend that the sensory portion of the cortex coincides with the motor, also hold that it extends beyond into adjacent cortical regions, as witness the views of Munk, Tripier, Moeli, and others, cited by Starr in his elaborate paper.

Ferrier's explanation† of what he regards as the errors of observers like Schiff, Munk and others who believe that sensory areas coincide with motor centres, is reasonable. "The conclusion that tactile sensibility is lost or diminished after the destruction of the cortical motor area," he says, "is based on defective methods of investigation and erroneous interpretation of the reactions of the lower animals to sensory stimulation. Though an animal does not react so readily to sensory stimulation of the paralyzed side it does not follow that this is due to diminished or absent perception of the stimulus. An animal may not react, or react less energetically, to a sensory stimulus, not because it does not feel it the less, but because it is unable or less able, to do so from motor defect. It is astonishing what apathy or indifference some animals display towards certain forms of stimulation, such as gradually increasing pressure on the fingers or toes

* Pflüger's Arch. f. Physiologie. Quoted by Starr, Jour. Nerv. and Ment. Dis., vol. xi., No. 3, July, 1884, 327–407.

† Functions of the Brain.

which one would regard as well calculated to elicit reaction or signs of uneasiness. Unless the stimulus is of a nature to at once excite attention, or to evoke reflex action, it may appear to be altogether unperceived. All that the experiments of Schiff and Tripier demonstrate is that motor reactions are less readily evoked on the side opposite the cortical lesion. But the same thing occurs in cases of purely motor hemiplegia in man."

On anatomical and morphological grounds Broca holds that the callosal and hippocampal regions constitute together a distinct lobe of the brain, the falciform or limbic lobe ; also I believe erroneously, that this lobe is connected entirely with the sense of smell. So far as the brain of man is concerned, in this lobe should probably be included the entire precuneus or quadrate lobule on the mesial aspect of the hemisphere and the adjoining portion of the superior parietal convolution on the lateral aspect of the hemisphere.

The most important recent contribution to cortical sensory areas, is certainly that of Horsley and Schäfer.* Hemianæsthesia, partial or complete, and contra-lateral, resulted from destructive lesions of the limbic lobe. They found that any extensive lesion of the gyrus fornicatus was followed by hemianæsthesia more or less marked or persistent ; sometimes the loss of sensation involved almost the whole of the opposite side of the body ; sometimes it was localized either to the upper or lower limb, or to a particular part of the trunk. They did not, however, succeed in establishing the relations between special regions of the body and the parts of the convolutions which had been destroyed.

"These experiments were frequently, but by no means in every case, complicated by the presence of a certain amount of motor paralysis, chiefly, if not entirely, affecting the muscles of the leg. We have no doubt that this condition was always due to a lesion (accidentally produced during the operation, or subsequently, as the result of interference with the circulation) in the leg-area of the marginal convolution. Now in one or two of these cases the anæsthesia affected chiefly the paresed limb, and it might be argued by those who, like Schiff and Munk, hold that the excitable areas of the cortex are concerned with the perceptions of sensory impressions from the corresponding regions of the body that the loss of sensibility was due to lesion of the motor-area. But against this argument we may not only put forward those experiments in which there has been no accompanying paralysis, but also others in which the hemianæsthesia has been well marked in the upper limb and upper part of the trunk, while the lower (paresed) limb has exhibited no diminution of sensibility whatever." * * *

* Phil. Trans. Royal Soc., 1888.

"From which it would appear that while the whole limbic lobe may be concerned in the perception of sensory impressions, the part played by the gyrus fornicatus, at least, as regards cutaneous sensibility, is more important than that played by the hippocampal portion of the lobe. But until it has been possible entirely to destroy the whole lobe upon both sides of the brain it is impossible to determine whether it is assisted in its functions by any other portion of the cerebral cortex." * * *

"The results of our experiments upon the limbic lobe seem to point to the conclusion that this portion of the cerebral cortex is largely, if not exclusively, concerned in the appreciation of sensations, painful and tactile. This is an extension of the view put forward by Ferrier, who was inclined as the result of his own experiments, to limit that function to the hippocampal region. Dr. Ferrier was good enough to assist at some of our experiments upon this part, and has fully accepted the conclusions to which they point."

Starr, from a series of American cases of cortical lesions of the brain,* and also from a study of the sensory tracts,† concluded that the various sensory areas lie about and coincide to some extent with the various motor areas for similar parts; that, in other words, the Rolandic region is a sensory-motor region, the sensory area, however, including to some extent the gyres of the adjacent postero-parietal lobe. Collections of cases such as these cannot, however, overcome positive evidence of decided destructive lesions of the cortical motor centres, without any disturbance of touch, pain or temperature, or even of the muscular sense; and a close study of the cases of cortical lesion in which both motor and decided sensory phenomena have been present, will not bear out fully the view of Starr.

One of Starr's tables contains 41 cases showing cortical lesions of the brain with sensory symptoms. That the sensory area lies about the motor region might even from these cases be conceded, but that the two coincide has been by no means proved, particularly if the postero-parietal region is not regarded as a necessary part of the motor area. Reviewing the 41 cases, in only one-fifth to one-sixth are the lesions restricted to parts of the brain in front of the central fissure, although in many of the others both the anterior central and posterior central gyres are involved. In more than 30 of these cases the reported lesions involved in part or whole the postero-parietal region, and posterior central gyre. Four of my cases are included in the table, and this has led me to look over not only these but also

* Am. J. M. Sc., Phila., 1884, n. s., lxxxvii., 366–391.

† Jour. Nerv. & Ment. Dis, vol. xi., No. 3, July, 1884, 327–407.

other of my cases of cortical lesion with sensory phenomena ; and from doing this the conclusion has been reached that they do not support the doctrine that sensory and motor areas entirely coincide.

In the case of a fibroma which involved the first and second frontal gyres, and anterior segment of the gyrus fornicatus and corpus callosum, anæsthesia of the conjunctiva, with conjunctivitis and corneitis were present. In a patient with a gumma involving the upper fourth of the pre-central and a smaller segment of the post-central gyre, hyperæsthesia was marked, with also crural Jacksonian epilepsy, but the lesion grazed the sensory area. A carcinoma of the middle portion of the posterior central gyre and the upper part of the inferior parietal lobule was accompanied with wide-spread destruction of the corona radiata, and impaired sensibility of the limbs of the opposite side with other phenomena was present. Owing to the posterior extension of this growth and the large sub-cortical destruction, this lesion could certainly not be claimed to teach anything with reference to the coincidence of motor and sensory areas. In another case of tubercular tumor in the motor area, the interior of the hemisphere was broken down, and hemianæsthesia, at first partial but later complete and persistent, was present.

Numerous observations have shown me that hyperæsthesia is a common phenomenon in cases of diffused cortical tubercular meningitis, and this hyperæsthesia may involve any or all the extremities or the face. This fact is not to be explained as simply from irritation of the pia mater ; for, on the other hand, I have observed cases of localized lepto-meningitis in which both hyperæsthesia and even pain in the head were absent. Such cases also cannot be fully explained by tubercular deposit in and inflammation of the dura mater. Although dural meningitis may cause headache of great severity and sensory phenomena in the domain of the fifth nerve, it should not be held responsible for hyperæsthesia of the extremities. The hyperæsthesia in these cases is best explained by irritation of sensory cortical areas.

Bernhardt * has recorded the case of a woman, 41 years of age, who after an apoplectiform attack had hyperæsthesia of both lower extremities ; also paresis of the right arm, lower face, and foot; spasm involving the right hand, arm, and face ; weak reflexes of the right leg ; confusion, excitability and aphasia. The lesions were a tubercular growth in the upper extremity, lateral and median portion of the left posterior central convolution and in the precuneus ; a similar growth in the middle portion of the posterior central gyre ; and a tuber-

* Arch. f. Psych., iv., Heft 3, 1874.

cle the size of a pea at the anterior extremity of the corpus striatum. The hyperæsthesia in the lower extremities might be explained by the tumor in the great median fissure. Hitzig* quotes from Löffler, the account of a soldier who received a gunshot wound on the top of the head, the dura mater being pressed inward by the depressed bone at a point which corresponded with the upper ends of both central gyres. Both legs were hyperæsthetic and also paralyzed. Such symptoms can be explained by pressure on the paracentral gyres of both sides, and direct or inflammatory irritation of the neighboring sensory cortex below or behind.

Bramwell† has reported a case of incomplete paralysis of the left extremities and of the face, with disturbance of sensation of both the arm and leg ; with also atrophy of both optic nerves and other sight disturbances. The autopsy revealed a tumor with softening of the surrounding brain in the right superior and middle parietal convolutions, and a part of the first occipital convolution.

Brill‡ records the history of a patient with right-sided anæsthesia and paresis, the former more marked than the latter. In a few weeks hyperæsthesia developed, with also restlessness, tinnitus, beclouded mind, slight amnesic aphasia, and color blindness, the patient not being able to tell green from red, although his field of vision was not limited. A triangular focus of softening was found in the left cuneus, most of the cuneus being involved.

Fortunately I had the advantage a few hours before delivering this address of hearing one of the best papers yet written on the cortical localization of cutaneous sensations, read by Dr. C. L. Dana before the American Neurological Association, and since published in the Journal of Nervous and Mental Disease for October, 1888. Dr. Dana discusses the three views with reference to this localization : (1) That the centres for these senses are entirely separate from motor areas. (2) That the cutaneous sensory centres are more or less identified with the motor centres ; and (3) That both the motor zone and the limbic lobe are concerned in the representation of cutaneous sensation. His paper deals principally with pathological and clinical data. Altogether he collected 142 cases, including four personal observations. He concludes, among other things, that the clinical and pathological evidence collected by him shows that the motor areas of the cortex contain also the representation of cutaneous sensations ; and that cutaneous anæsthesia of organic central origin is always limited to or more pronounced in certain parts. The same objections apply to most of his cases as indicated for those of Starr.

* Op. cit.

† Lancet, September 4, 1875, ii., 346.

‡ N. Y. Med. Record, vol. xxii., 1882, July 15, 81–82.

Dana collected for this paper 20 cases in which the gyrus fornicatus or hippocampal convolution were more or less involved, and in none of these, he says, was any anæsthesia present that could be fairly attributed to the lesion. Even a casual study of these cases seems to me to show their weakness for the support of the position taken that the limbic or falciform lobe is not concerned with sensation. In nearly one-third of these cases some disturbance of sensation was present. Three of the remaining cases were demented, and presumably any investigation into cutaneous sensation would have but little value; one case presented no symptoms.

What Ferrier says of defective methods of investigation and erroneous interpretation of the reactions of the lower animals to sensory stimulation may be applied with some additions to the study of cutaneous sensation in cases of cerebrel disease in man. Numerous sources of error are present, not only in investigating but also in reporting the results of investigation.

To test cutaneous sensibility requires time, patience, and method. It is often tedious and monotonous; but it must be done carefully or the results, particularly for tactile sensibility, are worthless. Patients cannot often be depended upon, as with reference to such tests the greatest differences in the personal equation are found. Some patients have a fashion of stopping to think or weigh their answers when being tested for cutaneous sensation. This often renders the answers worthless; the response should be given promptly.

One of the commonest methods of investigating cutaneous sensibility is that of Weber, which depends upon the fact, or the alleged fact, that the distance apart at which two points can be discriminated is much the same for the same local areas in different individuals. Tables have been published which are supposed to show the least normal distances at which two points can be distinguished. This method, if very carefully applied, is sufficiently correct for many cases, but often the results obtained from such testing are utterly worthless. I have known not only patients with real or alleged loss of sensation, but perfectly healthy individuals to give diverse and confusing answers when tested with the æsthesiometer. Some of the records of the presence of anæsthesia or analgesia in reported cases have doubtless been made improperly as the result of carelessly testing the power of the patients to discriminate between two blunt or sharp points.

Another source of error, particularly in recording cases, is in deciding that objective insensibility exists from the statements of the pa-

tients with reference to their feelings. Whatever may be the explanation, it is true that many, though by no means all, patients suffering from motor paralysis the result of a cortical or sub-cortical lesion, complain of certain parasthesias. Spontaneously, or in response to questioning, they will refer to a paralyzed member, or a portion of it, as feeling heavy or numb, or cold, or as if asleep ; but often in these cases careful and elaborate testing for true depression or loss of sensation will fail to reveal its presence in the slightest degree. I have been for some time engaged in minutely studying cases of hemiplegia, monoplegia, and aphasia with a view of determining in the most detailed manner the exact movements, or forms of speech, impaired or abolished, and also whether or not in these cases localized defects of sensation were present. The investigation is a clinical study in localization, the value of which will be much enhanced by the number of autopsies obtained in the cases studied, but even without such autopsies some results both curious and valuable can be obtained. A single case will serve to illustrate the point which I wish to make about some sensory investigations and records. A young man, with a history of rheumatism, and several heart murmurs, was admitted to the Philadelphia Hospital three weeks after a sudden attack of right-sided hemiplegia with aphasia. On admission he had recovered almost entirely from the paralysis of the arm and leg, but decided facial paralysis and slight aphasia, or rather ora-lingual paresis, were still present. His motor symptoms were studied with minuteness and will be published in a paper in course of preparation. On inquiring as to sensory disturbance he said that he had had for several days some pain in the leg and arm, and a feeling of numbness or as if the parts were asleep, in the right face, arm and leg. This sensation had disappeared entirely from the leg and arm with the abatement of the motor paralysis, but he still complained of decided numbness in the right cheek. The most careful tests were made as to the senses of touch, pain, and temperature, but not the slightest loss could be determined. He was examined by gently touching the cheek with the finger and a feather, with blunt and sharp points, with hot and cold water, with different weights and different amounts of pressure, and by the method of Oppenheim, namely, that of touching symmetrical spots on both sides of the median line at once, and observing whether he appreciated the touch on both sides equally.*

* The patient recently died. At the autopsy, a focus of strictly cortical, yellowish softening was found involving the lower extremities of the central convolutions both on their external and Sylvian surfaces, and a spot one-half an inch in diameter about the middle of the internal portion of the island of Reil. The softening reached into the

Some light has been thrown on the disputed question of the existence of sensory centres in the motor cortex by careful examinations of patients after operations, particularly when definitely determined gyral areas have been cleanly excised. In one of Horsley's cases, in which the scar tissue and surrounding healthy brain substance was excised, after the operation the patient had at first, coupled with some motor paralysis, a loss of tactile sensibility over the dorsum of the two distal phalanges of the fingers, and could not tell the position of any of the joints of the fingers, thus showing apparently some loss of tactile and muscular sense ; but as Horsley remarked it was possible that some of the fibres of the corona radiata coming from the gyrus fornicatus may have been injured. Both motor and sensory paralysis disappeared in the course of two months.

In Weir and Seguin's case of right-sided Jacksonian epilepsy and paresis—in which a small sarcomatous growth was cut out of the white substance below the posterior edge of the second frontal, and the anterior edge of the pre-central gyres—careful tests were made over a period of nearly seven months after the operation. They showed at first slight apparent dulness of tactile sensibility in some parts ; retention of pain, and temperature, and muscular sense, with motor paresis and speech defect. In later tests the patient no longer felt numb ; could tell the lightest touch on the fingers and hand ; and with eyes closed could distinguish consecutive contacts with a coarse bed cover, a thin handkerchief, and a sheet of paper ; he could also recognize slight difference of weight. Nearly seven months after the operation co-ordination of the hand was perfect ; the patellar reflex normal and equal on both sides; there was no wrist reflex ; the senses of touch, temperature normal ; he could distinguish differences of weight of only a few grains, and was fully conscious of all passive movements.

In the case of Lloyd and Deaver, also one of brachio-facial spasm and paresis, reported at the meeting of the American Neurological Association during the session of the Congress, no gross lesion was found, and the facial and arm centres were carefully excised, with a resulting cure of the spasms. On several occasions, with Dr. Lloyd, I tested the conditions as to motor power, sensibility, and the reflexes in this patient, with results which have been reported by Dr. Lloyd.

fissure of Rolando, and also into the precentral fissure, taking in a posterior, inferior strip of the second frontal convolution. Its greatest height was 1¼ inches upward from the Sylvian fissure, its width along this fissure was 1¼ inches. The lesion did not reach to the anterior branch of the Sylvian fissure, its anterior limit being one-fourth of an inch behind this fissure.

This patient blindfolded could instantly recognize the slightest touch on all points of the affected side ; even light breathing upon his hand was at once detected ; pain and temperature sense were normal ; and he could discriminate between weights. If objects were placed in his paretic hand he often failed to recognize what they were, but apparently because he was not able easily to grasp and run his fingers over them and thus take in their form and bulk.

These experiments, which should be repeated with the greatest care whenever opportunity offers, seem to uphold the doctrine of pure psycho-motor centres rather than the sensory, motor or mixed theory. They certainly are not in accord with the view that the motor zones contain centres of the muscular sense ; or that they are areas for pathic or thermic sensibility, or places of confluence of excitations, or centres acted on at a distance by inhibition. The temporary disturbances in sensation may be due sometimes to destruction of association fibres between related sensory and motor areas.

Bechterew* maintains with reason that the loss of sensation in animals who have had the motor area of the convolutions destroyed is apparent and not real ; that they cannot withdraw the irritated extremity though they feel the pain, because they have no control of the muscles. He also considers the loss of the muscle sense only apparent because if the animal's paws be placed in an uncomfortable position, its failure to be removed is due to motor inability rather than impaired muscle sense. The animal will move away if irritated on the affected limb, thus showing that it feels but cannot withdraw the limb except by moving the entire body. He concludes that tactile sensation is behind and external to the motor area, and the centre for muscle sense and pain is at the beginning of the fossa of Sylvius.

In several cases of surgical operation on lesions of the motor zone, it has been claimed that the presence of sensory symptoms supports the opinion that the motor areas subserve a sensory as well as a motor function ; the case of Macewen,† for example, of proto-spasm of the hallux preceded by sensory impressions and followed by paralysis, in which a tubercular nodule, was found in the upper part of the post-central gyre. Such a case may prove contiguity but not coincidence of motor and sensory areas.

The neurologist and surgeon must therefore depend on motor symptoms alone in fixing the site for operation in cases in which motor symptoms are definite. When positive sensory symptoms are present, they should regard these as indicative of the extension of the lesion

* Neurol. Centralbl., Leipz., 1883, ii., 409–414.

† Lancet, Lond., August 11, 1888.

towards either the limbic lobe, or the posterior parietal convolutions; or the involvement of the fibres going or coming from these gyres in the corona radiata.

We have no clinical or pathological data with reference to the so-called muscular sense of positive practical value to the physician or surgeon. The hypothesis that the cortical motor zone is rather a zone for muscular sense has little to support it either in physiology or in clinico-pathology. I can see no reason for requiring a muscular sense entirely distinct from other acknowledged forms of sensibility. Some facts have been brought forward which are supposed to indicate that a separate cortical area for registering impressions of muscular sense exists in the parietal lobe behind the motor zone, probably in the inferior parietal lobule; but these observations are not convincing; they certainly cannot yet be made of value in topographical diagnosis for surgical purposes.

The conclusion is warranted that there is a region for general sensation, including touch, pain, temperature, and possibly pressure, and location of a limb, which can be divided into special sub-areas for the various distinct portions of the body, and that these regions lie along side of and have close anatomical and morphological relations with corresponding motor areas, but that they are not identical with them. From an anatomical and morphological point of view, and from the facts of physiology and pathology, no part of the brain is more likely to contain these differentiated areas for sensation than the gyrus fornicatus, the hippocampal gyre, the precuneus, and the postero-parietal convolutions.

The Pre-frontal Lobe and the Cerebellum.

For the pre-frontal lobe and the cerebellum our diagnostic guides are gradually becoming more definite. We may not be able with the same absolute confidence from positive symptoms and signs to indicate lesions in these localities as we can point to the motor and visual zones, but we often can make the topographical diagnosis with sufficient certainty even for surgical purposes by combining various modes of investigation, as: (1) By a few positive localizing symptoms; (2) By the general symptoms—such as, in brain tumor, for instance, choked disc, secondary atrophy, headache, vomiting and vertigo; (3) By excluding lesions of the motor, speech, visual and auditory areas, and their association tracts; (4) By special pressure and invasion symptoms—invasion by lesions growing from adjacent areas to those under consideration.

Lesions of the prefrontal lobe, although this is one of the so-called latent districts of the brain, have in a large percentage of the carefully

studied cases shown distinctive manifestations. The symptoms are largely psychical, and unfortunately the physician is not usually well trained to study such phenomena. Mental disturbances of a peculiar character occur, such as mental slowness and uncertainty, want of attention and control, and impairment of judgment and reason ; closely studied, the inhibitory influence of the brain both upon psychical and physical action is found to be diminished. Memory is not seriously affected although a continuous train of thought cannot well be followed, and complex intellectual processes cannot be thoroughly performed. The results of experiments upon lower animals have not been very helpful towards determining the existence of prefrontal lesions, because psychical phenomena cannot be studied with accuracy in animals below man. Ferrier,* however, found after removal of the prefrontal lobe a decided alteration in the behavior of animals, difficult precisely to describe. They had apparently lost the faculty of intelligent observation. Horsley and Schäfer,† Hitzig,‡ and Goltz§ have also observed apparent mental changes. Phenomena such as these do not measure in usefulness for the average diagnostician with such positive objective manifestations as hemianopsia or Jacksonian spasm, but they should be valuable aids in the hands of close observers. Among pressure and invasion symptoms motor aphasia, nystagmus, contra-lateral paresis, and unilateral convulsions often occur late, particularly in cases of tumor and abscess. Given these symptoms, if now lesions of motor and visual regions can be excluded, it remains only to differentiate the lesions of the cerebellum and possibly some lesions of the temporal lobe.

In cerebellar cases, as shown by the studies of Nothnagel,‖ Seguin,¶ and others, the most positive symptoms are usually choked discs, optic neuritis or secondary atrophy, occipital headache, sometimes increased by percussion or pressure on the occiput or neck, vomiting, often apparently causeless in character ; typical cerebellar titubation, or other disorders of motion or gait ; nystagmus and conjugate deviation ; and among pressure symptoms one-sided paresis, occasional anæsthesia, sometimes disturbance of temperature, pulse, respiration, and deglutition.

Some lesions of the cerebellar hemispheres as in cases reported by Hun** give no localizing symptoms.

* Functions of the Brain. † Op. cit. ‡ Quoted by Ferrier.

§ Quoted by Ferrier.

‖ Topische Diagnostik der Gehirnkrankheiten, p. 78. Berlin, 1879.

¶ Jour. Nerv. & Ment. Dis., vol. xiv., No. 4, April, 1887, 217–235.

** Albany Medical Annals, May, 1888.

It might be of value to attempt a contrast of the usual symptoms produced by lesions in these localities. In pre-frontal disease psychic symptoms of the peculiar character just described, are usually present; in cerebellar affections they are as commonly absent, although irritative lesions of any part of the encephalon may, of course, cause emotional mental disturbance. The station or gait is not affected in pre-frontal cases : in cerebellar lesions, either typical titubation or some form of ataxia or staggering is usually present. Cerebellar titubation is characteristic of a destructive lesion of the middle lobe of the cerebellum, and when lesions of the cerebellar hemispheres are present, this results from pressure or encroachment. Bulbar symptoms and symptoms of pressure on the bulbo-spinal tracts, both sensory and motor, are somewhat frequently present in cerebellar lesions, particularly late in the history of a lesion of large size. With pre-frontal lesions of large size, the pressure and invasion symptoms are more likely to be disturbances of speech or smell, facial, brachial, or aural paresis, and unilateral spasm. Carefully recorded cases show that nystagmus, and conjugate deviation may be present in both pre-frontal and cerebellar cases ; but in the pre-frontal cases the latter is more likely to be towards the side of the lesion presumably destructive rather than irritative ; in cerebellar cases more probably the reverse. Headache is more likely to be frontal in the pre-frontal, and occipital in the cerebellar. Elevation of local surface temperature, and tenderness on percussion, have some, but no great value in differentiation.

Auditory Localization.

While auditory localization remains in a somewhat uncertain state, on the whole, the evidence is in favor of the localization of the cerebral centres or area, of hearing in the temporal lobe, and probably in its upper portion, that is, in the first or second, or in both the first and second, temporal gyres. The most recent and interesting discussion of this subject has arisen from the reports of the results obtained by Schäfer in his experiments on special sense localization in the cerebral cortex of the monkey. Dr. Ferrier * in reviewing the paper of Prof. Schäfer, attacks the results obtained and the conclusions drawn by the latter, and contends stoutly for the localization of the centre of hearing in the temporal lobe, and in accordance with his original view, more especially in the superior convolution of that lobe. Dr. Starr, in his consideration of aphasia, will doubtless discuss at length the question of auditory centres, naming centres, speech association

* Brain, January, 1888.

tracts, and speech centres, and I will, therefore, only briefly and for the purpose of completeness refer to this portion of the subject. Schäfer claims to have more or less completely destroyed the superior temporal gyre on both sides in six monkeys, and in one of the animals to have removed every trace of the convolution on both sides. In these cases he reports that hearing was not permanently affected, so far as it is possible to determine in monkeys, and concludes that the auditory faculty is not localized in the superior temporal convolution. In one experiment of Horsley and Schäfer, the superior temporal convolution was almost completely removed on both sides; in this case the animal appeared to hear quite distinctly, so far as it went the experiment being at variance with the results of Ferrier and those of Munk. The experimenters, however, say that they do not claim to have obtained direct corroboration or refutation of the views of Ferrier, but regard the question as still open.

Ferrier reaffirms his position, and reproduces with some additional facts, his original experimental evidence in favor of auditory localization in the temporal lobe, and particularly in its superior convolution. Munk, who is quoted both by Ferrier and Schäfer, extended the area for perception of auditory impressions over the entire temporal lobe; and Luciani believes that lesions of the temporal lobe in dogs produce abolition or impairment of hearing.

Gowers* refers to an interesting case of extensive tumor in which the oldest part was beneath the first temporal convolution. In this case convulsions, commencing by an auditory aura referred to the opposite ear, were a very early symptom. In another case of tumor of the first temporal convolution and Sylvian fossa, unilateral convulsions were preceded by a loud noise as of machinery. He notes that the loss of deafness in such cases is not permanent; that perfect compensation seems to be possible, presumably by the corresponding centre of the opposite side; and that each auditory nerve must be structurally connected with both hemispheres, although only the connection with the opposite hemisphere is habitually in functional action.

The pathological evidence with reference to the existence of auditory centres is not extensive or very decisive. That complete deafness from cerebral disease may occur probably requires, as Ferrier states, the existence of symmetrical lesions in both temporal lobes. It has, however, been clearly established by reported cases, that the so-called word-deafness may result from lesion of the upper convolution or two upper convolutions of the *left* temporal lobe. Cases in

* Manual of Diseases of the Nervous System, p. 454.

which the power to understand spoken words has been lost or impaired with lesions, in part at least, in the temporal lobe have been reported by various observers as, for instance, by Seppilli, Monakow and Amidon.

Ferrier * refers particularly to two cases in support of his position, one reported by Shaw† and one by Wernicke and Friedlander.‡ Shaw's case, a woman, aged 34, two months before her admission into his asylum, lost power in the right arm, and soon after had a sudden apoplectic seizure resulting in loss of speech and deafness. The loss of power in the right arm soon passed off. She became incoherent, more or less maniacal and demented. On admission she was found to be perfectly deaf and blind. She died of pneumonia a year afterwards. Post-mortem examination showed complete atrophy of the angular gyri and superior temporo-sphenoidal convolutions of both hemispheres. The gray matter of the atrophied regions had entirely disappeared, leaving the outer layer attached to the pia mater, with a cavity underneath formed at the expense of the gray matter. The cranial nerves were normal in appearance, the optic nerves showed increase of the connective tissue septa, atrophy of the nerve fibres, and spaces filled with a colloid-like material.

Wernicke and Friedlander's case, a woman, aged 43, had never suffered from deafness or affection of vision, and was attacked June 22nd, 1880, with right hemiplegia and aphasia. She remained in the hospital until August 4th, when she was discharged. At this time the patient could speak but unintelligibly, and was sometimes believed to be intoxicated. She not only could not make herself be understood but she could not understand what was said to her. She was received into the hospital again on September 10th, with slight paresis of the left arm. The right hemiplegia had entirely disappeared. The patient was looked upon as insane, and was absolutely deaf so that she could not be communicated with. She died of an attack of hæmatemesis on the 21st of October. An extensive lesion was found in each temporal lobe, invading the superior temporal convolution on both sides. The rest of the brain and the cranial nerves exhibited no abnormality. It was proved that the patient had previously enjoyed excellent hearing. Her total deafness occurred rapidly in connection with the other indications of cerebral disease.§

* Brain, xii., London, April, 1888, p. 18.

† Archives of Medicine, Feb. 1882.—Abstract in Brain, vol. v., 1882–83, p. 430.

‡ Fortschritte der Medicine, Band I., No. 6, March 15, 1883.

§ With Dr. Roland G. Curtin, of Philadelphia, I exhibited at the Philadelphia Pathological Society, the brain of a man for many years a deaf mute. A description of this brain, with illustrations, will be published with the reports of some other specimens referred to in this paper, in a forthcoming number of the University Magazine. On

Large Lesion of the Temporal Lobe.

With a large lesion of one temporal lobe, as tumor, hemorrhage or abscess, the diagnosis is best made by a careful consideration of pressure and invasion symptoms in addition to those which are strictly localizing in character. A single case recently published by Dr. Bodamer and myself, will perhaps best illustrate this mode of diagnosis. The chief symptoms were severe headache, more localized in the temporo-frontal region; pain on localized percussion; impairment of sight and hearing; choked discs; dilatation of the right pupil, and three days before death paralysis of the left arm and paresis of the left leg, and aphasia. The autopsy showed a large lesion centred in the middle of the right second temporal gyre; it was a vascular glioma, having beneath and partly around it a cavity containing detritus, and a large and evidently recent clot. It occupied a large portion of the interior of the right temporal lobe, but was strictly limited to it.

Macewen reports an operation on a somewhat similar case, of a lesion definitely localized in the temporo-sphenoidal lobe. A patient exhibiting symptoms of cerebral abscess had, on the left side ptosis, stabile mydriasis, paresis of all the ocular muscles, with the exception of the external rectus, without external squint; on the right side, paralysis of the facial muscles, which retained emotional expressions to a slight degree, and power to close the right eyelid by an effort of will, although it remained partially opened during sleep. He had, also, paresis of the right arm, which during the few hours he was under observation before operation, had amounted to distinct paralysis. The leg remained normal, and there was no diminution of cutaneous sensibility.*

the whole, the gross appearances could be regarded as favoring strongly Ferrier's auditory localization. The first temporal convolution of the left hemisphere was narrow and lacking in gyral elaboration; it was apparently distinctly atrophied or arrested in development. The first temporal convolution of the right hemisphere was smaller than usual, but it did not present the marked smoothness and diminution in size shown by the corresponding convolution of the other side. The brain was compared directly with half a dozen other specimens, normal and abnormal, and was examined by several brain anatomists and morphologists, who agreed with me as to the striking appearance of lack of development of the first left temporal. Other peculiarities of the temporal, third frontal, and central convolutions, the island of Reil, etc., were noted, and will be given in the description of the specimen.

* While this paper is passing through the press, through the courtesy of Dr. H. C. Wood, of Philadelphia, I have had the opportunity of seeing a case of large lesion of the right temporal lobe which shows how active symptoms of such lesions may be absent for a long period, or if present how they may differ from the pressure and invasion symptoms in the two cases above given. Dr. Wood will publish a full account this interesting and important case. The patient had had for many months general

Tumor of the Facial and Auditory Nerves.

At the meeting of the Neurological Society, of London, March 15, 1888,* Dr. Sharkey read notes and showed the brain of a case of tumor of the auditory nerve. During the discussion of this specimen, Mr. Horsley stated that the tumor might have been removed by an operation he had already advocated—namely, incision of the tentorium and ligature of the lateral sinus. I have made two dissections with a view of determining whether a tumor in the intra-cranial course of the facial and auditory nerves can be removed, and have concluded that it could, and best, not by operating above the tentorium as I understand Mr. Horsley's suggestion, but by operating below the tentorium, and then pushing aside or excising an outer segment of the cerebellar hemisphere.

Olfactory Localization.

The location of the cortical centre of smell is still uncertain, but the pointings are all towards the region of the uncinate convolution and its immediate vicinity. Zuckerkandl, of Gratz, in 1887, published a monograph on the olfactory centre, the work being anatomical and physiological, not clinical. The anatomical portion is largely comparative, considering the brains of twelve varieties of animals besides man. One chapter describes the cornu ammonis region, another the olfactory lobes of animals and man, etc. The writings of Burdach, Treverinus, Huguenin, and Ferrier are largely quoted. Zuckerkandl

symptoms of brain tumor, such as headache, choked discs, vertigo, etc., but no paralysis; and no loss of hearing that could be detected by ordinary tests. During the absence of the patient from the city, left homonymous hemianopsia developed, with pronounced contraction of the other visual fields. Wernicke's hemiopic pupillary inaction was carefully tested for, but was certainly not present. Operation was performed by Dr, D. Hayes Agnew for Dr. Wood, and a cyst in the right occipital lobe was discovered and removed. After death a large fibro-glioma was found occupying almost the entire second and third temporal convolutions, and also invading the central portion of the first temporal, the superior aspect of which was somewhat flattened by pressure. The fourth and fifth temporal convolutions were apparently not greatly encroached upon, the uncinate convolution escaping entirely. The patient had no loss of smell. The occipital cyst was evidently a radiation or a secondary result of the growth, due to softening from obliteration of blood vessels. This case emphasizes the great importance of carefully studying pressure and invasion symptoms with reference to such comparatively latent regions as the temporal, (particularly the right temporal), and pre-frontal lobes. The pressure in this case was evidently not exercised in the same direction as in the two cases referred to above. In a lesion of a so-called latent region, the pressure and invasion symptoms will depend both upon the nature of the growth, and on the particular direction in which it happens to develop.

* Lancet, London, April 7, 1888.

believes, but, I think without reason, that the entire limbic lobe is the seat of olfactory sensation. Among other cases, he has collected two in which infants born without the olfactory lobe on one or both sides, showed arrested development of the horn of Ammon, and in both of which the gyrus fornicatus and hippocampus were small.

Zuckerkandl, in this view, is simply following Broca, who divided all animals into osmatics and anosmatics, or good smellers or bad smellers, and believed that the whole of the falciform or limbic lobe was the cerebral organ of smell. I cannot here go into the anatomical and physiological arguments bearing upon this subject. To one familiar with brain anatomy a knowledge of the relatively large size of the gyrus fornicatus and hippocampal region in an animal like man, in whom the sense of smell plays so comparatively an unimportant part, is an argument of some weight against the view of Broca. In osmatics, however, the hippocampal lobule or region of the amygdala—the uncinate convolution so-called—is very large while in the anosmatics it is comparatively small. Ferrier's view is therefore probably correct that the only relationship which undoubtedly exists is between the olfactory bulb and the anterior portion of the hippocampal convolution. The anterior commissure has an anterior and posterior division, which connect respectively the olfactory bulb and the region of the hippocampal lobule and nucleus amygdala, a fact which also tends to prove that the anterior portion of the hippocampal convolution is the cortical organ of smell.

Electrical irritation of the hippocampal lobule or uncinate gyrus in monkeys, cats, dogs, and rabbits furnished Ferrier with significant indications of subjective olfactory sensations. This reaction, the same in all, as described by Ferrier, was a peculiar torsion of the lip and nostril on the same side. The experiment in which Ferrier produced destructive lesions of the hippocampal lobule, were, on the whole, unsatisfactory, but to a certain extent supported the view that this region contained the centre for smell.

Few autopsies have been recorded in which loss or diminution of smell has been present as the result of cerebral lesion. Ferrier, however, refers to cases reported by W. Ogle, Fletcher and Ransome, of the occurrence of the loss of smell in the left nostril with right hemiplegia and aphasia ; and he also alludes to cases reported by Hughlings Jackson. From a study of these cases he believes that we have reason to regard anosmia as probably due to softening of the region of the hippocampal lobule. The connections of the olfactory tract are with the hemisphere on the same side. In some cases of cerebral hemianæsthesia, as in the hysterical and alcoholic forms, impairment

or loss of smell is present and seems to be contra-lateral. This, Ferrier believes, may be explained on the supposition that the defect was due to an anæsthetic condition of the nostril, either from lesion of the fifth nerve or of the centres for cutaneous sensation in the cerebral hemisphere. Close testing in such cases would probably show that the olfactory sense was not absolutely abolished.

Various cases have been reported with autopsies in which the presence of an olfactory aura has been accompanied by a lesion of the temporal lobe, and these cases, on the whole, point to the lower convolutions of this lobe as the probable seat of the centre of smell.

Hughlings Jackson has written various papers showing the great importance of studying all forms of aura in order to be able better to localize the lesions in the encephalon. As early as 1876, and again in 1879, he considered the varieties of epilepsy in which not only crude sensations or warnings of smell, taste, etc., were present, but also a more elaborate mental condition which he speaks of as the "dreamy state." As theoretical as such discussions may seem at first sight, he clearly shows that they are of practical value not only in leading to an early recognition of epilepsy, but also in localizing lesions in either epileptic or epileptiform cases.

I have had several epileptic patients in whom the attacks were initiated by an odor usually offensive. Jackson* gives interesting histories of three such cases in which attacks were ushered in by a crude sensation warning of smell, accompanied sometimes by other warnings as epigastric sensations, and the dreamy state. These cases are of great clinical interest but are not accompanied by autopsies. He refers, however, to the necropsy of a woman who had had paroxysms with the dreamy state and crude sensation warnings of smell. She had left hemiplegia and double optic neuritis. The autopsy showed a tumor in the right temporo-sphenoidal lobe.

Allan McLane Hamilton† has reported the following case of softening of the temporo-sphenoidal lobes, in a woman of forty years. The patient suffered from the age of ten from epileptic attacks, which occurred four or five times a year, and consisted of general convulsions. The first attack occurred after a fall when she struck her head, and was unconscious thereafter for some hours. No scar was visible on the head. She always had an aura of a peculiar character before the attack. She suddenly perceived a disagreable odor, sometimes of smoke, sometimes of a fetid character, and quite uncomplicated by other sensory warnings. She died of phthisis.

*Brain, July, 1888. † Am. Jour. Med. Sci., April, 1884. Quoted by Starr.

On post-mortem examination the dura mater was thickened and opaque in spots, and at the base was adherent to the temporo-sphenoidal lobes. The adhesion of the membranes was most marked on the right temporo-sphenoidal lobe somewhat posterior to its apex. At this point a decided shrinkage of tissue was discovered with depression, the induration involving the uncinate gyrus and parts of the adjacent convolutions. The basal ganglia and motor tracts were normal, and the olfactory nerves were not involved.

Worcester* reports the case of a farmer, aged 30, who had had epilepsy for two years before admission to the hospital. The case presented no special features until January 26, 1878, when after a severe convulsion the man remained in a state of alarming collapse. Radial pulse was almost imperceptible, surface cold, and there appeared danger of immediate dissolution. He rallied somewhat under stimulants, but remained for three days in a stupid condition unable to be long out of bed. Shortly after the attack slight innervation of the right side of the face was observed, but only when the muscles were called into action as in talking and smiling. On February 11th, he regained his ordinary mental condition. No paralysis was discovered except as above mentioned, and no impairment of sensibility except a transient numbness of the hand at times. For several days hallucinations of smell—at first constant, afterwards transitory, were present. Once he imagined the room was full of smoke. He fancied at times there was an odor like the vapor of alcohol passing quickly. He thought this took the place of a convulsion. No test was made of his sense of smell. No marked changes occurred until his death on February 28th, after a series of tonic convulsions, with marked opisthotonos, affecting chiefly the muscles of the back.

The autopsy revealed, on inspection of the inferior surface of the brain, a small red spot of softening at the most prominent point of the left gyrus uncinatus. The brain was not opened until it had been hardened in alcohol. A focus of softening existed in the white matter of the anterior part of the left temporal lobe, extending to the surface, externally, and internally involving the pes hippocampi in the floor of the descending cornu of the lateral ventricle. The portion of the hippocampus major not discolored, was swollen and softened. A very small focus of softening, without discoloration, about the size of a large pea, was found in the white matter of the frontal lobe on the same side. No other gross lesions were discovered, but the perivascular spaces were very generally dilated, so as to give thin sections of the brain a worm-eaten appearance.

* American Journal of Insanity, for July, 1887.

Gustatory Localization.

Our knowledge of a cerebral centre for taste is even more unsatisfactory and undecided than that for an olfactory centre. Morphology, anatomy, physiology and pathology combine to indicate that this centre is probably situated close to and in the same lobe of the brain as that for smell. The experiments of Ferrier seemed to show that affections of both taste and smell were evidently connected with lesions of the hippocampal lobule and its neighborhood. "It was noted in connection with electrical irritation of the lower extremity of the temporo-sphenoidal convolutions in the monkey, and of the same region in the brain of a cat, that the movements of the lips, tongue, cheek-pouches and jaws were occasionally induced—phenomena which might be regarded as indications of the excitation of gustatory sensation. This interpretation receives support from the above described results of destructive lesions; and we have, therefore, reasonable grounds for concluding that the gustatory centres are situated at the lower extremity of the temporo-sphenoidal lobes, in close connection with those of smell. This would enable us to explain the occasional occurrence in man of anosmia and ageusia as the result of severe blows on the head, especially the vertex. A blow in this region causes counter-stroke of the base of the brain, particularly in the region of the olfactory centres."

Dr. James Anderson* has recorded a case of epilepsy in which, from symptoms, ocular and cerebral, detailed in his report, he correctly predicated tumor and its position. The patient's dreamy state was associated with a rough, bitter sensation in his mouth. It is the only case published, according to Jackson, in which a necropsy has been had revealing any local morbid changes in a case of the variety of epilepsy mentioned. Dr. Anderson refers to a case, closely like that of his own patient, recorded by Mr. Nettleship. In the report of this case, however, the dreamy state was not mentioned; there was a crude sensation warning in the patient's fits—a sudden feeling of suffocation in the nose and mouth.

As our special subject is the practical relations of cerebral localization, I cannot forbear to recall here the advice of Hughlings Jackson† on the great practical importance of the close study of epileptic seizures. These remarks are made in connection with the discussion of the different varieties of aura—crude warnings of smell and taste, intellectual aura of the dreamy state, temporary word-blindness or

* Brain, October, 1886. Quoted by Hughlings Jackson in Brain, July, 1888.

† Brain, July, 1888.

word-deafness, noises, flashes of light, hallucinations, etc. No better neurological work, Jackson holds, can be done than the precise investigations of epileptic paroxysms. The efforts should be to describe *all* that occurs in the paroxysms. Epilepsies are as numerous as are paroxyms beginning with different warnings. The warning is the first event from or during the onset of the epileptic discharge ; it is the clue to the seat of the discharging lesion. Using the term suggested by Seguin, I might interpolate here that it is the sensory "signal symptom." The discharging lesions have as many different seats as there are different warnings of the paroxysms.

"Before we can make good generalizations," says Jackson, "we must carefully analyze. To group together as 'visual warnings' color projections, apparent alterations in the distance of external objects and 'dreamy states' with definite scenes, is generalizing without previous analysis, and is an attempt to organize confusion ; they are exceedingly different things. He who is faithfully analyzing many different cases of epilepsy is doing far more than studying epilepsy. The highest centres ('organ of mind'), those concerned in such fits, represent all, literally all, parts of the body sensorily and motorily, in most complex ways, in most intricate complications, etc. A careful study of many varieties of epileptic fits is one way of analyzing this kind of representation by the 'organ of mind.' Again, it is not, I think, an extravagant supposition that there are, after slight epileptic fits of different kinds, many temporary morbid affections resembling those persistent ones produced by destructive lesions of different parts of the cortex. To illustrate for a moment by epileptiform seizures ; there is temporary aphasia after some fits beginning in the face or hand (more 'elaborate' utterances, I think, when the exact starting point is in the ulnar fingers) ; this is the analogue of aphasia from a destructive lesion (softening, etc.). To return to epilepsy. There is, I am convinced, in, or after, certain paroxyms of epilepsy temporary 'word-blindness ;' certainly in one patient of mine who had a 'warning' by noise. I could not make out that this patient was at the same time 'word-deaf,' but thought his temporary deafness was ordinary deafness. Still there may have been word-deafness. In another patient who called his attacks 'losses of understanding,' there was clearly both 'word-deafness' and 'word-blindness,' with retention of ordinary sight and hearing ; this patient's attack used to begin with a warning noise, but he has recently had his 'losses of understanding' without that warning."

Jackson holds that there is some local disease in every epilepsy, some pathological process productive of high instability.

His views on the arterio-cortical pathology of some varieties of epilepsy or epileptiform seizures are of great importance to those who are concerned not only in locating the site of a discharging lesion, but also in deciding whether such lesion shall be removed by operation. Sometimes, in the cases already operated on, even when the most careful, and doubtless accurate, localization has been made no gross lesion has been discovered, and yet even in these cases a true gross lesion may have been present—if the plugging of arterioles can be regarded as a gross lesion. Jackson believes that most cases of epilepsy proper are due to the plugging of arterioles. His views upon this question are full of suggestive value. "Centres of taste and smell," he says, "lie, according to Ferrier's localization, in the region of the posterior cerebral artery, whilst, still according to his localization, the centres for hearing and part of the centre for sight (angular gyrus) lie in the region of the middle cerebral. Hence, if arterial plugging be the pathology, it may be that we have different varieties of epilepsy proper, according as arterioles are plugged in different vascular regions. The variety of epilepsy I am remarking upon in the text may be owing to morbid changes in the district of the posterior cerebral. But tumors would grow regardless of vascular regions. I suggest that cases of epilepsy with mixed warning (of smell or taste along with the warnings of color) are more likely to be owing to tumor or other gross organic disease."

Handicapped by the embarrassing proportions of my subject, I have imperfectly presented it for consideration ; but trust that my remarks may open lines of discussion to those here present far better fitted than I to enter the lists in such a debate. These practical discoveries in cerebral localization, with the achievements of antiseptic surgery, constitute the grandest triumphs that adorn the history of our noble science and art of medicine.

CONTENTS.

SPINAL LOCALIZATION

IN ITS

PRACTICAL RELATIONS.

PART I.—GENERAL CONSIDERATIONS.

SPINAL localization, particularly in its practical relations to surgical procedure, is one of the topics of the hour which has not yet been fully discussed in any of our Philadelphia medical societies. Although its proportions do not approach those of cerebral localization, the subject has sufficient literature and interest to allow of lengthy treatment and open a wide field of discussion; but it will be necessary for me to be brief in order to cover the ground.

Spinal trephining is by no means a recent operation; it has an ancient history both as regards suggestion and actual performance. Horsley* speaks of it as known to surgery since the time of Heister (1757), but Lidell attributes the suggestion to Paulus Ægineta. An operation of M. Louis, performed in 1762, is sometimes referred to as the first instance of spinal resection, but, as stated by Ashhurst and Lidell,† it was not a resection at all, but merely an extracting of some loose fragments of bone on the fifth day in a case of gunshot fracture involving a dorsal vertebra, an operation which was performed twenty-four times during our civil war with satisfactory results.

* *Med.-Chir. Trans.*, vol. lxxi., Lond., 1888, p. 383.

† "Intern. Encycl. Surg.," vol. iv.

Genuine spinal trephining was first performed by Henry Cline, June 16, 1814, for fracture of the twelfth dorsal vertebra. Horsley, writing last year, cites Erichsen as stating that it has been performed in all about thirty times; but Ashhurst, about the same time or earlier, tabulated fifty cases. Of these fifty cases, thirty-three died, seven were not benefited, four were relieved, two recovered, and in four the result was unknown. I have collected fifteen cases in addition to those in Dr. Ashhurst's table, and doubtless a few others have escaped me. In addition to these I have personal knowledge of three other cases in which spinal trephining has been performed, but in which the accounts of the operations have not yet been published.

While we have here, however, a record of sixty-eight cases in which the operation of spinal trephining has been performed, a close study of the details of these cases shows that very few operations were guided strictly by the rules and principles of spinal localization. External pointers, as deformity, depression, or swelling, were commonly present. Gowers and Horsley's case of spinal tumor is perhaps the most brilliant example of localization, and vies with the cases of Macewen and Abbe in brilliancy of result.

Many experimental investigations of the functions of the spinal cord have been performed by Brown-Séquard and a host of physiologists, but the first experiments to determine the exact functions of the horns of the cord at various levels were those of Ferrier and Yeo,* in 1880. Pathological observations had been made prior to this time and have been since, and are largely confirmatory of the results obtained by these investigators. In 1884, Starr† collected most of the work on

* "Proc. Royal Soc.," No. 212, and *Brain*, July, 1881, vol. iv. p. 223.

† *Am. Journ. Neur. and Psych.*, vols. ii. and iii. p. 443.

this subject up to that date. Ross* has worked in the field of sensory localization to good purpose, contributing an excellent paper on the "Segmental Distribution of Sensory Disorders." Much material of a highly practical character will be found embodied in Gowers's "Manual of Diseases of the Nervous System" and in Seguin's contributions to localization in the "American System of Practical Medicine." Recent valuable papers, partly neurological and partly surgical, on spinal localization are those of Thorburn.† This writer records nineteen cases of injury to the cervical region of the spinal cord, studied with the view of determining the exact spinal localization as indicated by such injuries. He has also contributed a valuable article on injuries of the cauda equina. Osler‡ has published a brief paper on lesions of the conus medullaris and cauda equina, and on the situation of the ano-vesical centre in man. Other papers on allied topics have been contributed by Bernhardt, Oppenheim, and Kirchoff, who are cited by Osler.

It is important to closely study every case of supposed spinal focal lesion, whether from injury or disease. Reports of such cases, particularly when accompanied by autopsies, will continue to be useful until a number of points now obscure become clear; but even without autopsies they may be of great value if minute and accurate. Some of the papers on cerebral localization, most valuable because of their suggestiveness, have been purely clinical and largely speculative; as, for example, some of Hughlings Jackson's and Broadbent's on affections of speech. One of the most instructive articles on spinal localization, that of Remak§ on the localization of the

* *Brain*, January, 1888.

† *Brain*, January, 1887; January, 1888; and October, 1888.

‡ *Med. News*, vol. liii., December 15, 1888.

§ *Arch. f. Psych.*, ix.

lesion in anterior poliomyelitis is based entirely on clinical cases. From a careful study of many cases, Remak shows how various groups of muscles are frequently affected together, and, reasoning back to the lesion, concludes that disease in a certain segment or adjoining segments of the spinal cord will produce paralysis in a group of muscles which act together. He made some highly-interesting observations with reference to palsies of particular muscles or muscular groups. These investigations will be referred to again later in this paper.

NOTES OF CASES OF SPINAL FOCAL LESION, WITH AUTOPSIES.

Let me first refer briefly to a few cases of spinal focal lesion, with autopsies, occurring in my own experience, briefly commenting on them with reference to the possibility of successful operation.

While it has been my fortune to see a large number of patients suffering from intraspinal focal lesions, I have made comparatively few autopsies on such cases. I find among my notes, however, records of six post-mortem examinations, three of which have been reported. Of the six cases, two and perhaps three might have been trephined with some show of success. One of these was an injury in the lower dorsal region in which the lamina of the twelfth dorsal vertebra had been broken and driven inwards, a case similar to one of the operative cases reported by Macewen, similar even to the vertebra injured. A bony mass was present about half an inch long by one-quarter of an inch wide in the external layer of the dura, just at the lower end of the spinal cord. This mass, which was like the bony concretions sometimes found in the falx cerebri, could have been removed by operation, after elevation of the depressed and broken spinal arch.

In another case operation might have been of benefit if performed immediately after the accident. The most important features of this and the first case were as follows: A history of severe injury to the middle and lower spine; this was followed at once by pains in the back and limbs, spinal rigidity, anæsthesia, and paralysis of the lower limbs and of the bladder and bowels. The condition remaining was one of motor paralysis, wasting, contraction, depressed electro-contractility, coldness and lividity, peculiar zones of anæsthesia, and abolition of the tendon reflexes in anæsthetic regions. In this second case, at the upper edge of the twelfth dorsal vertebra adhesions were found which continued down the canal for three or more inches. At the commencement of the cauda equina, which was about the centre of the adhesion, caries of the first lumbar vertebra was found, the portion involved being the inner surface of the laminæ close to the junction of the body. Above and below the caries the adhesions were most persistent. The conus medullaris, cauda equina, and membranes were a glued mass. For an inch or two more above and below this the membrane was considerably thickened, a deposit being present of a calcareous nature.

In a case of caries and dural tumor, of which I exhibit specimens, the patient might perhaps have been benefited by relief of pressure. This patient, a man 72 years old, was admitted to the Philadelphia Hospital with a history that he had been lame in his left leg, the result of a fall, since he was ten years old. No relatives or friends could be found, and, as his mind seemed somewhat clouded, it was difficult to obtain from him any statement of value as to his past condition. It was learned, however, that three or four months before admission he had had an attack in which he suffered from diarrhœa and fever; that he became weak in

both legs, but largely recovered power in them, until four weeks before admission, when he became a complete paraplegic.

He had no paralysis of the face and arms. His left leg was shorter than his right, and was considerably atrophied, measurements showing about an inch difference between the limbs. Motor paralysis was complete in both legs, but he had control over the bowels and bladder. Tactile sensation was diminished in both legs, and on his trunk to a line on a level with the third dorsal spine behind and the fourth rib in front. Pain and temperature sense were preserved. He complained of a belt-like feeling around the waist at the highest level of the anæsthesia. He had a sacro-ischiatic bedsore nearly central in position. He died a few days after admission to the hospital. The brain showed no gross lesion. On opening the spinal canal, when the line of the fourth dorsal vertebra was reached the dura mater was found to be strongly adherent, requiring dissection, and a small tumor or enlargement was attached outside of this membrane on the lateral aspect of the cord. It was about three-quarters of an inch in length and one-third of an inch in thickness. Possibly the cord was slightly diminished in general bulk from compression at this point, but it was not abnormal in consistency, and no hemorrhage nor inflammation were present. The body of the fourth dorsal vertebra throughout was found to be eroded and in a carious condition. For a little distance below the periosteum was detached and the bone partly exposed. The disease of the bone had destroyed nearly the whole of the interior of the body of the vertebra. Below the index-finger could be passed into the cavity, while above was a shell or bridge of bone. The vertebra was not displaced. The intervertebral cartilage between the fourth and fifth dorsal vertebræ was also necrosed. The upper lobe of the right lung

was strongly adherent to the bodies of the third, fourth, and fifth dorsal vertebræ. A communication existed between the space in the decayed vertebra and the pleural sac.

In two cases of syphilomata of the dura mater of the cervical region, operation would have been useless from the nature, extent, and high level of the lesions.

The sixth case was one of hemorrhage into the cauda equina, the symptoms being similar to those in the second case referred to above. The question whether in such a case early trephining at the lowest point of the lumbar region would have been feasible, and would have offered any prospect of relief, will be considered later.

SPINAL OPERATIONS GUIDED BY LOCALIZATION. SPINAL FRACTURES AND DISLOCATIONS.

Trephining the spine in whole or in part, guided by the rules of localization, may be resorted to for fracture, dislocation, tumor (including certain hypertrophic inflammatory pachymeningeal lesions), hemorrhage, abscess, caries, and neuralgia.

Page* has said, with reference to fracture and dislocation, that trephining of the spinal column can be of no advantage; but this is certainly far from being true of the former, and even in some cases of dislocation the operation may be of assistance to the surgeon in bringing about reduction. Other eminent surgeons, as Lidell and Ashhurst, hold that more lives are lost than saved by interference. Whether operation in cases of fracture and dislocation will or will not succeed is largely a question of exact diagnosis of the extent and location of the injuries. When, for instance, it can be demonstrated

* Heath's "Directory of Surgery," p. 134, quoted by Horsley.

by close study that the brunt of the injury has been borne by the spines and laminæ, operation should be at once performed, and even when in doubt it is better to explore rather than to hesitate in the face of paralysis or death.

An interesting recent successful case is one performed in 1887 by an English surgeon, Mr. Jones, reported by Thorburn in *Brain* for January, 1888, and also later, as to result, in the *British Medical Journal* for October 20, 1888.

The patient, a collier, aged 15, was injured by a heavy stone striking him on the shoulders and back. He received a fracture of his right femur, and was also instantly rendered paraplegic. The limbs presented a position of partial paralysis, with wasting, lost faradic contractility, absent knee-jerk, changed skin reflexes, paralysis of the bladder, cystitis, and priapism. Sensation was blunted or lost over special nerve areas in the lower limbs. The third lumbar spine presented a prominence. To the left and above was another bony prominence. Trephining was performed. The detached spine of the second lumbar vertebra was removed, and showed a gap between the arches of the second and third, filled with dense cicatricial tissue. By means of bone forceps the arch of the second lumbar was now almost entirely removed, exposing the membranes of the cord, which had obviously been compressed by it. Around these membranes was also cicatricial tissue, which was not interfered with. Twenty months after the operation the patient was exhibited at the Manchester Medical Society by Mr. Jones. The condition of the patient was then very satisfactory. Sensation was normal in both legs, which were, however, much wasted, and there was evidence of degeneration in the muscles of the left limb. The movements of the knee-joint were normal, but those of the

ankle were limited to extension, flexion being practically abolished. He had perfect control over his bladder, and could urinate normally. He was able to walk three miles with short intervals for rest. When standing he was obliged to lean on some support unless he bent his knees. He was gradually improving.

An equally interesting, but unsuccessful, case, operated upon by Mr. Hardie, is also reported by Thorburn.* The patient was a man 38 years old. The operation was performed October 26, 1887. The patient had a fall of six feet from a wagon, and immediate paralysis without loss of consciousness followed. He had pain across the shoulders and shooting down the arms to the elbows, and arching backward of the cervical vertebræ. The lower limbs, abdomen, and thorax were absolutely paralyzed. His respiration was diaphragmatic, with sense of dyspnœa and cough. All the muscles of the right upper extremity were paralyzed, and the extrinsic muscles of the right upper limb also paralyzed,—the pectorals, spinati, latissimus. The neck muscles were normal on both sides. The muscles of the left upper extremity escaping paralysis were the biceps, supinatus longus, deltoid, supra-spinatus, and infra-spinatus. Both palpebral fissures and pupils were smaller than usual. Anæsthesia extended as high as the third rib in front and the sixth cervical spine behind; on the right side there was sensation over the region of the deltoid and slightly beyond it, and on the left over the deltoid and down the outer side of the limb to the thumb. No hyperæsthesia was present. Knee-jerk, plantar, cremasteric, gluteal, and epigastric reflexes were absent. The temperature was normal. The urine was retained, the penis turgid. His pulse was 66, with a marked respiratory

* *Brain*, October, 1888.

wave. A vertical incision about four inches long was made over the cervical spines, with its centre opposite the fifth. The muscles being cleared from the laminæ, an interval of about a quarter of an inch was found between the fifth and sixth spines, and the fifth vertebræ appeared to be slightly displaced forward. The laminæ of the fifth and sixth vertebræ were removed by bone forceps and the dura mater exposed, presenting a perfectly normal appearance without any trace of hemorrhage. The patient died the next day. The heart-beats, which were very feeble and infrequent, continued for about ten minutes after respiration had ceased, but stopped at 3.35. The disk between the fifth and sixth cervical vertebræ was found to be ruptured, the former bone projecting very slightly forward. No fracture was discovered. The dura mater was uninjured, but the cord was flattened opposite the seat of the injury, and was much contused for about an inch above and below, containing hemorrhage in its substance and in the central canal ; elsewhere its structure was normal.

In lesions of the cauda equina which cause compression, whether fracture, hemorrhage, or abscess, but particularly fracture, trephining is undoubtedly indicated in most cases, and in some, with our present light, it is imperatively demanded. This view I take from personal experience of cases with autopsies, and of others which I have studied clinically. The same view is strongly taken by Thorburn,* who shows that some of the objections which are valid against trephining higher in the vertebral column, do not apply so fully to injuries of the cauda equina. He advocates trephining in injuries of the cauda equina, always bearing in mind the following conditions :

" 1. We must be sure of the localization.

* *Brain*, January, 1888, p. 406.

"2. We should, if possible, wait for a reasonable period, say six weeks, before operating, and should then do so only if the patient shows no signs of spontaneous recovery.

"3. Should the paralysis of the bladder be early followed by severe cystitis, and should we suspect secondary renal troubles, what are we to do? On the one hand, not to operate probably means death; and on the other, the risk of operation is enormously increased. Further experience may enable us to settle this problem, but at present the outlook appears to be sufficiently gloomy. A third course has suggested itself to me, but I have had no opportunity to test its results,—viz., to drain the bladder by means of a suprapubic cystotomy. The object of draining the bladder is obvious, and the suprapubic appears preferable to the perineal route, because we are thereby enabled to construct our fistula through parts which are not anæsthetic and predisposed to slough. If we could thus prolong life for a time, we might afterwards proceed to operate upon the spinal column with fair hopes of a successful result, and encouraged by the great improvement effected in the case of R. M. C., and the cure obtained in a case recently published by Lauenstein, in which there was complete paralysis of the lower limbs, etc., from a dislocation of the twelfth dorsal vertebra, and where a perfect cure resulted from the removal of the misplaced arches. It would appear, therefore, that whether 'trephining the spine' is or is not justifiable in cases of injury of the cord, it is certainly the proper treatment to pursue in those of the cauda equina, and hence the importance in such cases of making an exact diagnosis."

SPINAL TUMORS.

In most cases of tumor of the spinal cord or its envelopes, operation would not for several reasons be completely successful. It

would, however, be entirely successful in some, and I have come to the conclusion that almost all cases should be trephined for the chance of success, or to relieve pain and diminish the effects of pressure. When the growths spring from the bone or dura mater the chances are, of course, the best.

Even if the neoplasm is situated in front, or surrounds a large part of the cord, trephining might be successful, for during operation the lateral and even the anterior aspects of the cord can be explored, the latter certainly by cutting one or two spinal nerves. This can be seen by examining the cord in position in a fresh specimen. With a little care, using a blunt hook, the cord can be rolled and lifted so as to examine it on almost every side in position. Erosion or caries, with perforation of the body of the vertebræ, can also be determined by careful explorations with properly-curved probes thrust around and beneath the exposed cord.

Three operations for spinal tumor, all of them successful, have been reported by Macewen,* Gowers and Horsley,† and Abbe.‡ The case of Macewen was a boy, 9 years of age. The operation was performed May 9, 1883. The main symptoms were complete sensory and motor paraplegia, with incontinence of urine and fæces. Angular curvature was present, most marked between the fifth and seventh dorsal vertebræ. The laminæ of the fifth, sixth, and seventh dorsal vertebræ were removed. A fibrous neoplasm attached to the theca, between it and the bone, was dissected off. After this, cord pulsations, which had been absent, returned. Twenty-four hours after the removal of the pressure the limbs had lost their livid color and were distinctly warmer; the spastic rigidity had

* *Lancet*, August 11, 1888.

† *Brit. Med. Journ.*, January 28 and June 16, 1888, and *Med.-Chir. Trans.*, vol. lxxi.

‡ *N. Y. Med. Rec.*, February 9, 1889.

greatly lessened. The sense of tickling had returned to the soles, and that of touch had improved. After eight days the first return of movement was observed; soon after he had perfect control over his sphincters. Six months subsequently he was able to go about without support, and five years afterwards he walked three miles to pay the doctor a visit. He attended school regularly, joined in all the games, including foot-ball, and said he felt quite strong. Charcot has pointed out the occurrence of these connective tissues in cases of caries, and one of the specimens exhibited in connection with this paper is a tumor of this character.

The case of Gowers and Horsley was a man, aged 42 years, and the operation was performed June 9, 1887. He had suffered for three years from localized pain beneath the left scapula. The pain varied considerably. He showed great mental irritability and hypochondria. Four months before operation first the left and then the right leg became weak, and gradually there was complete paraplegia. He had motor and sensory paralysis up to the level of the sixth or seventh dorsal nerves, with intense spasm in the legs, with foot clonus and rectus clonus. Urine was retained and he had some cystitis. He suffered with severe pain around the trunk, which was greater on the left side, and increased to agony by any movement. Caries and syphilitic disease of the spine were excluded. The spines and laminæ of the fifth, fourth, and third dorsal vertebræ were removed, and a fibro-myxoma, about the size of a filbert, was discovered and removed. Mr. Horsley reported, January 4, 1888, that the pain was for a time slightly relieved, but again and again returned with great severity, and the power of motion was only slowly and intermittently regained during the first three or four weeks. The local pain gradually diminished and motor power grad-

ually returned. The surgical result was excellent. He reported, June 12, 1888, that the patient had lost the agonizing pain, and at the same time gradually recovered motor and sensory power, as well as the control of the bladder and rectum. He remains in perfect health.

One of the most recent contributions to the subject of spinal trephining, that of Dr. Robert Abbe,* is a case of extradural tumor of the spine, with complete paraplegia, in which the operation resulted in recovery. The patient was a young man, aged 22 years, and the operation was performed on May 26, 1888. The first symptom was pain in the back; swelling of the soft parts two inches wide by three long. There was disturbance of tactile sensibility in both legs, followed by gradually-increasing paresis. The line of hyperæsthesia was between the anæsthesia and the normal skin. He had uncontrollable twitching of the legs, which were cyanotic when dependent. The spine became rigid and very painful; the hyperæsthesia, anæsthesia, and paralysis deepened. He suffered with girdle pains about the limiting line of the disease. He had incontinence of urine and fæces. Active hectic was present. The spines and laminæ of the eighth, ninth, and tenth vertebræ were cut away, and a dense mass of tissue and desiccated pus was removed by scraping. Sensation began to return on the eighth day; in the fourth week he began to move his left leg and toes on the right. The fever and pain disappeared, and in six weeks he moved both legs. He improved continuously, and in four and a half months he moved, pushing a chair. He was exhibited to the Surgical Society of New York eight months after the operation, when he walked well without support and was stout and heavy.

* *N. Y. Med. Journ.*, November 24, 1888, and *N. Y. Med. Rec.*, February 9, 1889.

In the disease described by Charcot and Jeffroy as hypertrophic internal pachymeningitis, operation for the removal of the lesion might in some cases prove successful. Usually after the disease has existed some time the lesion becomes practically a fusiform tumor of considerable size. It might be objected, perhaps, that the disease is inflammatory in character; but, on the other hand, it is chronic, often of slow development, and sometimes almost stationary. No harm could result in a disease always one of great suffering and eventually fatal, and great relief and possibly a cure might sometimes be effected.

INTRASPINAL HEMORRHAGE.

As within the cranium, so within the spinal canal, hemorrhage may be of four varieties as to location,—namely, (1) supradural or extrameningeal; (2) subdural; (3) subarachnoid or subspinal; and (4) intraspinal,—that is, into the spinal cord itself. The hemorrhage may be posterior, anterior or lateral, or antero-lateral, postero-lateral, etc.; but commonly it will surround the cord; if intraspinal, it may be into the anterior or posterior horns, the central gray matter, or columns, but usually it will be diffused through the cord. With reference to the question of trephining it is only practical to consider supradural or subdural hemorrhage.

A net-work of large blood-vessels lies between the dura mater and spinal bone, and from this supradural spinal hemorrhage usually occurs. Such an extravasation may be minute, of moderate size, or very extensive. The symptoms of supradural hemorrhage will vary, of course, with its location and extent. The history is generally of a sudden attack, often after a fall or exhibition of violence of some kind.

Hemorrhage between the dura mater and the bone probably usually finds its way down

the whole length of the spinal canal to the beginning of the sacral canal. The space surrounding the cauda equina in the lumbar canal is comparatively large, and the much-narrowed sacral canal is placed at a slightly different level, so that much of the effused blood would probably accumulate in the lumbar region. The fat and connective tissue between the theca and the bone are loosely distributed in the extradural space, and differ considerably in amount in different individuals, so that in some cases more than others much of the blood would be entangled and coagulated in the meshes of the connective tissue and fat near the seat of extravasation. The subdural space, although much smaller than that outside of the dura, is a smooth serous cavity, and therefore much of a hemorrhage at any level would also probably descend into the region of the conus and cauda equina.

I do not know of any reported case in which trephining has been resorted to simply for the relief of intraspinal hemorrhage. In a case of resection at the Bellevue Hospital, New York, by Dr. Stephen Smith,* it is reported that notwithstanding from eight to twelve ounces of extravasated blood, having a dark color, escaped from the spinal canal after the depressed bone had been extracted, the compression of the cord from extravasated blood and the paraplegia steadily crept upward, and finally caused death by asphyxia. The case is reported to show that the operation of trephining the spinal column or resecting the vertebræ would not relieve the spinal cord from compression when it was exerted by the extravasation of blood.

The diagnostic symptoms of meningeal hemorrhage as given by Gowers and others, and according to my own experience, are as

* Lidell, in the "Intern. Encycl. Surg.," vol. iv. p. 379.

follows: (1) sudden and violent pain in the back, less or more diffused; (2) pain along the course of the nerves passing through the membrane near the extravasation; (3) abnormal sensations,—tingling, etc., and hyperæsthesia, referred to the same parts; (4) spasm involving vertebral and other muscles supplied by affected nerves and also sometimes the muscles supplied by the cord below the seat of the hemorrhage; (5) sometimes general convulsive movements; (6) sometimes spasmodic retention of the urine; (7) consecutive paralytic symptoms, but not usually complete paralysis.

Some points of differential diagnosis between meningeal hemorrhage and extravasation into the substance of the cord should be borne in mind. Symptoms of irritation, such as pain, hyperæsthesia, paræsthesia, and spasm, in meningeal hemorrhage are usually immediate or very early, and may precede paralysis, which is commonly not complete. In hemorrhage into the substance of the cord paralysis may be very complete at first, or rapidly become so, and symptoms of irritation may be very largely wanting. Hemorrhage may, and not infrequently does, involve not only the membranes but also the substances of the cord, giving complex symptoms.

In some cases of intraspinal hemorrhage, whether primary or secondary, trephining might be performed in two places in order to make sure of a good result,—namely, at the supposed seat of the extravasation, and at the lowest portion of the lumbar spine, where, as already indicated, much of the extravasated blood would gravitate. Kronlein, it will be remembered, has recommended that in cases of cranial meningeal hemorrhage, two trephinings should be performed if one is not successful, and several cases of failure in trephining for intracranial hemorrhage have been reported, which would have been successful

had the second operation been performed. In like manner, two trephinings of the spine, or a single trephining at the lowest possible place in the lumbar region, might prove successful where failure would otherwise result.

The somewhat frequent occurrence of spinal hemorrhage, with fracture of either the body, spines, or laminæ of the vertebræ, must not be overlooked. The diagnosis of the exact height of the lesion and the decision as to trephining may often have to be settled negatively or positively through a consideration of the question of the probability of a double lesion in cases of accident,—namely, both fracture and hemorrhage. The fracture and the hemorrhage may be situated at different levels, or if the hemorrhage occurs primarily at the seat of the fracture it may find its way down the canal.

I might give many illustrations gathered from medical and surgical literature of intraspinal hemorrhage in which operation would probably have proved successful, but I will confine myself to two, one twenty years old and the other a recent case.

Jackson* reported an interesting case of localized spinal apoplexy, which with our present lights might perhaps have been relieved and a life saved by careful trephining. The patient was a bright girl, 14 years old. While dressing her fingers felt weak. The next day she had a similar weak feeling in her hands. One day later she was unable to move her arms except at the wrists. Later, the intercostal muscles did not act quite freely, and she seemed to lie heavier in the bed. Moist crepitant râles, with a little cough, developed. On the fifth day after the first symptoms, careful examination clearly demonstrated great loss of power in all the voluntary muscles of respiration and in those muscles of the arm, back, and chest supplied by the

* *Lancet*, July 3, 1869.

cervical nerves. The diaphragm became fixed, and there was slight lividity about the cheeks, with a fall in the natural temperature. Sir W. Jenner, who was called in consultation, diagnosticated a clot in the cervical portion of the spinal cord. Death soon took place without pain. The whole cervical portion of the spine, but particularly in front and to the left, was embedded in an oblong clot of dark venous blood outside the membranes. The clot ceased at the seventh cervical vertebra.

In this case, as the reporter remarked, the effusion probably took place very gradually, had room to extend itself, and coagulated very slowly and imperfectly. Until the phrenic nerves were interfered with nearly every symptom might have been attributed to hysteria.

The following case was reported by Dr. Arlidge to the Staffordshire branch of the British Medical Association.* The patient, 44 years old, a drinking man, was admitted to the infirmary with complete motor paralysis of the legs, and some weakness in the arms, where, too, formication and numbness were experienced. Sensation was normal, and the legs were painless and their reflexes abolished. His only complaint was of dorsal pain. The bladder was distended, and prior to admission had been emptied by the catheter, but there was no dribbling of urine. Some rhonchi were noted in the upper part of the left lung ; heart healthy ; bowels confined for four days ; the cerebrum undisturbed. The first temperature taken was 103.6°, but the next day it fluctuated between 105° and 106°, and rose to 107.4° the day preceding his death, on September 26, and was accompanied by some delirium. A post-mortem examination revealed a copious hemorrhage

* *Med. and Surg. Rep.*, March 23, 1889, vol. lx. No. 12, p. 370.

within the meninges of the spinal cord, extending downward from the last cervical vertebra for about six inches. The blood was coagulated. No ruptured vessels were found in the cord itself, and no cerebral disease, although the dura mater was very adherent to the skull. There was no fracture of the spine, and no caries of any vertebra.

CARIES AND ANGULAR CURVATURE.

The question of operation for the relief of angular curvature of the spine is one of considerable importance and not to be dismissed hastily. Such an operation has been, until lately, commonly regarded as useless or worse. Its dangers have been overrated, although they are doubtless greater somewhat than most cranial operations. Macewen has reported an extraordinary series of six cases, in which elevation of the posterior laminæ of the vertebræ was performed,—in four with recovery. In favor of operation are,—(1) the fact that by thus allowing the cord to expand backward a present serious paralysis may sometimes be relieved without imperilling the ordinary chances of recovery by anchylosis; (2) when an approximate cure has been effected by anchylosis, but a paralysis or contractures remain, the latter may sometimes be relieved and improved; (3) secondary degeneration of the cord may sometimes be prevented or arrested; (4) when the paraplegia is produced by the connective tissue or other tumors which sometimes accompany angular curvature, rather than by the latter, such growths can be removed. Against operation are,—(1) that cases can sometimes be cured without such heroic procedure; (2) that caries often involve the body of so many vertebræ that operation is often useless or worse.

Trephining in such cases has been performed twice at the Philadelphia Hospital by Dr. White in cases of Dr. Dercum.

A case is reported by Mr. G. A. Wright,* of a boy, 7 years old, who was operated upon January 26, 1888. Disease of the spine was observed one month before operation, with marked angular curvature in the mid-dorsal region, with some lateral thickening. Paresis of the lower limbs was present, and the patient grew gradually worse. Before the operation he had complete paralysis of both lower extremities, with incontinence of urine and fæces. Anæsthesia to the level of the eleventh rib on the right side, and on the left decrease and finally loss of sensation, developed, the anæsthesia extending from the toes to the level of the pubes; from thence to the line of the eleventh rib a condition of analgesia existed. The muscles of the lower limb were in a state of tonic contraction, with marked flexion. The plantar reflexes were exaggerated; cremasteric very feeble, and deep reflexes could not be obtained. His feet were slightly swollen and cyanotic. Until late the symptoms were more marked on the right side than on the left. External and internal treatment were used without avail. Three laminæ were removed in the dorsal region. The theca of the spinal cord exposed was surrounded by a buff-colored, tough, leathery substance, which was cut away with the scissors. The cord did not appear to pulsate, but no point of constriction could be found. Some improvement took place in voluntary motion and sensation during a month following the operation. Later, the area of anæsthesia increased, and the condition of the patient became about the same as it was before operation.

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* *Lancet*, July 14, 1888.

SPINAL ABSCESS.

Spinal abscess, like brain abscess, is usually a complication found in connection with disease of the bodies of the vertebræ, and localization rules will not commonly be of much value in making a diagnosis. Pus at any level within the spinal canal will, like blood, usually find its way to the lumbar region. In rare cases suppuration is confined to the space between the dura mater and bone, and when our diagnostic acumen becomes greater we will doubtless in some of these cases be able to trephine with advantage to our patient. Pachymeningitis, with or without suppuration, is generally due to traumatism or vertebral caries, but occasionally cases appear to arise as primary or idiopathic.

In this so-called idiopathic suppuration in the spinal dura mater, trephining low down to give exit to the pus might in rare cases prove a useful procedure. Such a case is that reported by Dr. Robert Maguire.* Other cases have been reported by Mueller, Spencer,† and others.

In Spencer's case of idiopathic inflammation of the spinal dura mater, the patient's illness began with pain in the feet and knees and in the small of the back, frequently shooting down the back to the legs. He had fever and diarrhœa. There was great tenderness on pressure over both lumbar regions. The surface of the body was covered with copious acid sweats. Temperature elevated; pulse rapid, wiry, regular; tongue dry, white fur in the centre, red at the tip and edges. Breathing became rapid and somewhat painful, with a dry, hacking cough. The patient became delirious and

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* *Lancet*, July 7, 1888.

† Ibid., June 14, 1879.

his pain in the back very severe, but no paralysis until late. The neck was very stiff. There was a peculiar purplish mottling on the chest and abdomen. He became steadily weaker and died. The whole spinal canal was filled, from within two or three inches of the head down to the sacrum, with thick, creamy pus of a light yellow color and containing numerous shreds of lymph. The pus was found to lie outside the dura mater, between it and the periosteum. The pus had found its way out of the canal through the intervertebral foramina, and surrounded the spinal nerves for some distance. It had also passed out through the laminæ and infiltrated the connective tissue between the muscular structures on each side of the spine, forming collections of various sizes, the largest opposite the lumbar vertebræ. These were not abscesses, but infiltrations of pus into the interstices between the muscles. The tissues around these collections of pus were softened and the spinal nerves were laid bare. The most noteworthy of these collections was one opposite, and to the right of, the sixth and seventh dorsal vertebræ; the pus had here found its way under the costal pleura and projected into the thorax as a tumor, the size of a large hen's egg, lying close to the spine. In this case trephining low down as well as operations in the external abscesses might have been performed.

NEURALGIA.

In rare cases of intractable neuralgia trephining and section of the nerves close to the spinal cord may be tried as a last resort. This operation has been performed in one case by Dr. Abbe,* of New York, on the recommendation of Dr. C. L. Dana. The patient, a man 44 years old, was operated on Decem-

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* *N. Y. Med. Rec.*, February 9, 1889.

ber 31, 1888, and January 2, 1889. His symptoms began with throbbing pain on the posterior surface of the right forearm above the middle; later, paroxysmal pain, giving a peculiar twitching sensation in the thumb, index, and middle finger; after some months there was complete disablement of the hand, which he kept in a stiff position with the fingers semiflexed. The forearm and hand were slightly emaciated, with atrophy of the muscles in the interosseous spaces. The posterior, interosseous, and ulnar nerves were stretched without any improvement. The arm was amputated about the humeral insertion of the deltoid, but without abatement of pain. The morphine habit had been contracted. He had twitchings and tonic contractions of the muscles of the trunk; also the Brauch-Romberg symptoms; he swayed in standing and had a tendency to fall to the right. Knee-jerks were exaggerated, and there was ankle clonus in the right leg. The right shoulder showed muscular atrophy, and stiffness in the neck muscles. A small tender neuroma of the muscular nerve was removed without relief. The right half of the laminæ of the third cervical to the first dorsal spine was removed. Both motor and sensory roots of the sixth and seventh cervical nerves were cut square across outside of the dura. The posterior roots of the seventh and eighth cervical nerves were cut off close to the posterior columns. The patient made great improvement.

PART II.—THE FACTS AND RULES OF SPINAL LOCALIZATION.

In a paper on "Cerebral Localization,"* I directed attention to the possibility of six classes of symptoms presenting themselves for the consideration of the neurological diagnostician,—namely, those of (1) local irritation, (2) local destruction, (3) local pressure, (4) invasion by lesions growing from adjacent areas to those under determination, (5) local instability, (6) reflex action at a distance. All or any one of these classes might require to be considered in localizing a spinal lesion, but the last three are not relatively of as much importance in spinal as in encephalic diagnosis ; and, in order to restrict the present paper to reasonable limits, the discussion of this part of the subject will practically include only a study of symptoms of irritation, destruction, and pressure. Irritative, destructive, and compression lesions, whether intracranial or intraspinal, give rise mainly to three great classes of diagnostic phenomena,—reflex, sensory, and motor. Vaso-motor, trophic, visceral, thermic, psychic, and some special symptoms, have at times considerable importance, but are generally secondary in value for ordinary practical purposes to the three classes mentioned.

Under reflex phenomena are usually included the cutaneous reflexes,—scapular, epigastric, abdominal, cremasteric, gluteal, plantar, etc. ; and the so-called tendon reflexes,—wrist-jerk, knee-jerk, ankle-jerk, ankle-clonus, perineal-jerk, etc. Under the same head also come such phenomena as the involuntary reactions of the rectum, bladder, and sexual organs. Reflex symptoms may be, as might

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* *Transactions of the Congress of American Physicians and Surgeons*, First Triennial Session, Washington, p. 238, 1888.

be expected from the functions of the spinal cord, of decided localizing value in spinal lesions, whereas for brain lesions they are usually only of general significance and value. Such reflexes as the scapular, epigastric, abdominal, cremasteric, gluteal, plantar, and rectal may, for instance, greatly help to fix the height of a lesion, as may also such phenomena as knee-jerk, muscle-jerk, and ankle-clonus, but the latter are sometimes in spinal as in cerebral cases only of general significance.

Some of the so-called reflexes are a guide to definite spinal segments. While, however, it is important and a great aid to prompt diagnosis to recognize quickly certain marked isolated symptoms, as disturbance of particular reflexes, on the other hand it is essential that such phenomena should not be misinterpreted. Exaggeration of certain reflexes, as, for instance, the ankle-jerk, knee-jerk, the cremasteric-jerk, etc., indicates not merely disease producing irritability of the centres for these phenomena, but, it may be, rather disease or compression in the cerebro-spinal axis anywhere above these centres. Abolished knee-jerk may be indicative of extensive degenerative disease of the spinal cord, as in posterior sclerosis; or, it may, in much rarer cases, indicate limited disease of the particular segment of the cord related to the patellar tendon. Westphal,* for instance, has reported the autopsy of a case of dementia paralytica, in which the patellar reflex had been absent on the left side only; and in which sections of the cord showed extensive sclerosis of the posterior root-zones at the junction of the lumbar and dorsal regions on the left side, while on the right was only a slight beginning of the sclerotic process.

Under sensory phenomena it is necessary to consider clearly the particular variety of sensation lost or perverted, the exact cuta-

* "Ann. Univ. Med. Sci.," vol. i. p. 95, 1888.

neous areas affected by increase, depression, or perversion of sensation, the nerves involved, the situation of their nuclei in the cord, and the relations of their origin to the intervertebral foramina. These sensory phenomena include the symptoms,—pain, hyperæsthesia, anæsthesia, and paræsthesia.

The great motor symptoms are paresis or paralysis, spasm, contracture, and tremor. In studying motor phenomena, we must consider not only the muscles affected by loss or exaltation of power, the exact nerve-supply to such muscles, and the intraspinal course of these nerves, but also the situation of the nuclei of these nerves in certain segments of the spinal cord, and even the particular cell-groups to which such nuclei belong. The value of a study in spinal localization depends largely upon the *exactness* with which we differentiate such phenomena and relate them to their lesions.

A good practical point is the importance of some single striking fact in spinal function in localization diagnosis for surgical or other purposes. That the phrenic or diaphragmatic spinal centre is situated in the fourth, or in the third and fourth cervical segments, should, for instance, be taken into full consideration in operations in this locality, great care being taken not to bruise or deeply injure the cord, or to explore with needles or by incision a region where such a vital centre has its abiding-place. Respiratory and cardiac phenomena occur with lesions at and above the phrenic centre; pupillary changes when the cord is affected above the level of the second dorsal nerve or segment; while extreme trophic or vaso-motor affections of the skin and its appendages are only present when the central gray matter, somewhat posterior to the central canal, is diseased.

My main object in this portion of the present paper is to give the methods of referring or relating sensory, motor, reflex, and other

phenomena, which are distributed in limited regions of the body, to the particular levels of the spinal cord in which they have their source or termination. It is necessary, for both the physician and surgeon, to have quick and ready methods of determining the position of a lesion in the spinal cord by a study of the symptoms presented. The study is a broad one, and, in considering it, a paper might be almost indefinitely extended ; it is necessary, therefore, to seize upon salient features and methods of presentation.

SOME POINTS IN COARSE SPINAL ANATOMY.

Certain well-known anatomical facts should always be borne in mind when considering for practical purposes the question of spinal localization. We should remember that the spinal cord proper is only from seventeen to eighteen inches long, while the spinal canal to the last lumbar vertebra is about twenty-three inches, and, if the sacral portion and coccyx are included, four inches more should be added. The cord, therefore, is much shorter than the canal, and usually reaches only to about the second lumbar vertebra. As already indicated in Part I., one of the most important and most available positions for successful spinal surgery, in cases of localized lesion, is the region of the cauda equina, most of which is entirely below the spinal cord. We have, in other words, a comparatively extensive intraspinal region in which the lesions are, strictly speaking, peripheral, and the diagnosis, both local and general, must be made with this fact fully recognized. That the spinal cord does not fill its canal transversely is also a familiar fact, the significance of which should also always be before us. The cord is, indeed, suspended very loosely in the canal. It follows, therefore, that many of the intraspinal lesions which are operable are membranous or bony in origin, and in symptomatology are,

in part, at least, peripheral, as they involve nerve-trunks in their intraspinal course, however long or short this may be. Every one who wishes to become practically familiar with spinal anatomy for surgical purposes should study a series of transverse sections showing the relations of the spinal cord to its enclosing envelopes, membranous and bony. Even the dural sheath is much larger than is necessary for its contents. I have already referred to the fact growing out of such anatomical studies of the spine, that the spinal cord can be lifted with care so as to be the subject of operation on any of its surfaces, anterior or lateral, as well as posterior. A consideration of all the questions relating to the spinal membranes and spaces will eventually enable us to separate with fair accuracy the lesions which are supradural, subdural, and strictly speaking spinal.

For various reasons, operations for localized lesions, next to the cauda equinal region, probably offer more chances of success in the dorsal spine than elsewhere, owing to the less size of the cord and the less danger in operating. In the cervical and lumbar enlargements the greater bulk of the cord and the closeness of the nerve-roots is always to be considered. From one part of the cord, the smallest in transverse section, some of the largest nerves of the body originate. This is the narrowed lowest extremity of the cord, the conus medullaris, in which the lower sacral nerves have their roots. It is a region small in general bulk, because, owing to its position, it contains few conducting tracts, although relatively a large amount of gray matter. Lesions of this conus medullaris, embedded in its net-work of great nerves, can be accurately made out by symptoms, and its actual and relative size to the membranes and spaces of the spine should therefore always be kept in view.

The intraspinal course of the spinal nerves

should be absolutely familiar. Nearly all of the nerve-roots descend to the foramina, from which they emerge to be distributed to various parts of the body. The lowest nerves have, of course, the longest intramural course. Some facts about the relations of spinal segments to the vertebræ, foramina, and nerves are, doubtless, known to all, but are probably not much considered. The segmental nature of the spinal cord will be referred to presently at some length. The term spinal segment is used to indicate the limited vertical portion of the cord from which arises each pair of spinal nerves. In the cervical region are eight of these segments, but only seven vertebræ; hence the eight pair of spinal nerves emerge from their foramina in such a manner that, although they all descend a little in their course, they emerge after the first, so that the segments are located opposite spines and bodies of the vertebræ which do not correspond in name. The third cervical segment, for example, according to Gowers, is about opposite the first cervical spine, and its nerves emerge between the second and third cervical bodies. The eighth cervical segment is about opposite the sixth cervical spine, or the junction of the sixth and seventh, and its nerves emerge between the seventh cervical and first dorsal body. In the dorsal region the segments are the longest of the entire cord, and they are not situated numerically opposite the spines and bodies of the same name in any single case. The first dorsal segment is nearly opposite the seventh cervical spine and body; the second dorsal nearly in a line with the first dorsal spine and body. The tenth, eleventh, and twelfth dorsal segments are much shorter than the segments from the first to the ninth, and these three segments are included chiefly between the eighth and tenth dorsal spines and bodies. The entire eleven spinal segments, from the first lumbar to the coccygeal, inclusive, are

crowded vertically between the eleventh dorsal and second lumbar spines and bodies, a space of only about two to three inches.

Commonly, we speak only of two sets of nerve-roots,—the anterior or motor, and the posterior or sensory,—the great discovery of Sir Charles Bell. Bell, however, really divided the nerve-roots throughout the central nervous system into three sets, the third being a lateral or respiratory set, and containing nerves which excite motions which depend on or are related to the act of respiration. Gaskell* says that physiologists have failed to follow this because the triple arrangement of these nerve-roots was not immediately evident, like the separation of the anterior and posterior roots. This lateral tract of nerve-roots is the same as the non-ganglionated part of the splanchnic root.

These lateral roots do not concern us much with our present lights in determining questions of localization. When, however, visceral and vascular localization have been more perfected, the question of the existence and involvement of these lateral groups will receive greater attention. Every segment of the spinal cord, according to Gaskell, gives origin to two roots, a somatic and a splanchnic,—the latter being the roots of the nerves supplied to the viscera and blood-vessels ; the former to the muscles, skin, and tissues other than those which are vascular and visceral. The somatic roots are, in other words, the motor and sensory, or the anterior and posterior roots, and in the main are connected respectively with the cells of the anterior and posterior cornua. The lateral roots arise from two columns of nerve-cells,—namely, the column of Clarke and a column in the lateral horn. The splanchnic root arising from the column of Clarke is ganglionated, while that arising from the lateral horn is

* *Jour. Physiol.*, vii. 1–80. London, 1886.

non-ganglionated; and the nerves corresponding to the latter go to muscular tissues chiefly. Lesions of these nerve-roots, or of the cell-groups or columns of the cord, with which they are connected, give rise chiefly to vaso-motor, trophic, secretory, and visceral phenomena, distributed in accordance with the segment of the cord or the special pair of nerve-roots implicated. These lateral roots are found in greatest number in the region from the second thoracic or dorsal segment to the first lumbar.

SEGMENTAL CHARACTER OF THE SPINAL CORD.

One of the first steps towards an understanding of spinal localization is the full recognition of the segmental character of the spinal cord. It is to be regarded not as a single organ, but as a series or succession of organs vertically linked together,—a chain of segments one placed above another. In many of the lower animals, as in fishes and snakes, this arrangement is obvious, the spinal cord being made up of a series of alternating swellings and constrictions. "Each segment," says Bramwell,* "may be viewed as a distinct spinal unit, or, to speak somewhat figuratively, as a distinct spinal cord for a definite area of the body,—viz., that portion of muscle (muscular area) to which its anterior roots proceed, and that portion of skin, tendon, muscle, mucous membrane, viscus, etc. (sensitive area), to which the fibres of its posterior nerve-roots are distributed. Now, the essence of the clinical examination of the spinal cord consists in the separate and systematic examination of each spinal segment by observing the motor, sensory, reflex, vaso-motor, and trophic conditions of the body area." According to Professor Hill,† of Cam-

* "Diseases of the Spinal Cord," p. 1. Edinburgh, 1884.
† *Brain*, January, 1888.

bridge, the recognition of the segmental succession in the arrangement of the nuclei of the nerves of the spinal cord is due to the late Professor Aeby. In the Hunterian Lectures, in 1885, Hill applied this principle of segmentation to the brain, or, at least, to the cranial nerves, attempting to fix the position in the cerebral axis of the nuclei of these nerves by a consideration of their segmental distribution in the head.

LOCALIZING PHENOMENA REFERABLE TO THE CONDUCTING TRACTS IN THE SPINAL CORD.

The spinal cord of man, besides being a great centre, or collection of centres, includes also a conducting medium, through which the great tracts (motor, sensory, etc.) pass, to connect the brain with all the segments situated below, and these segments with each other. In man, the conducting tracts may be regarded as developmental additions to the original spinal segment. It is in its capacity as a centre—in the capacity of each segment as a special centre—that we are particularly concerned in studying spinal localization, but the symptoms produced by irritation, destruction, or compression of the conducting tracts will often greatly modify an opinion as to the exact height, and particularly the extent of a lesion. I cannot, of course, go into a lengthy consideration of the functions of the white substance of the cord. This can be found in the treatise of Gowers, or any good work on nervous diseases. For completeness, however, I will summarize the subdivisions of the cord and their functions, and make some practical applications to the subject of localization, in the main following Gowers in giving the functions of the different subdivisions. A transverse section of the cord at different heights would give a somewhat but not greatly differing appearance. In a portion of the cervical region the greatest

number of subdivisions are present. Starting here with the posterior median portion of the cord,—the part which would be first exposed by the surgeon's knife and saw,—the posterior column is divided into two parts,—the *column of Goll*, or *postero-median column*, and the *column of Burdach*, also known as the *postero-external column*, or *posterior root-zone*. The column of Goll in all probability conducts tactile impressions. The column of Burdach, containing, as it does, the posterior root-fibres, has for its function the conveyance of sensory impressions inward through these root-fibres, and probably also for short distances it carries these sensory impressions vertically. Just external to the periphery of the posterior horn begins a most important conducting tract,—*the pyramidal tract*, or, as it is usually called, *the lateral or crossed pyramidal tract*,—which occupies the posterior half of the lateral column outside of the posterior horn. Its function is unquestionably chiefly motor conduction, particularly from the brain to the parts below the arm. A column, shallow from without inward, but wide circumferentially, is the *direct cerebellar tract*, which lies chiefly between the lateral pyramidal tract and the periphery of the cord. Flechsig believes that this column conducts impressions from the muscles of the trunk. When diseased it degenerates upward, and therefore its function must be to conduct upward. It is probably connected with Clarke's vesicular column, and therefore with visceral and vascular sensibility and control. Between the pyramidal tract and the concavity formed by the junction externally of the anterior and posterior horns is a comparatively narrow space called *the lateral limiting layer*, whose fibres may connect the gray matter at the different levels. What Gowers has termed the *antero-lateral ascending tract* occupies about the same position in the antero-lateral columns as the direct

cerebellar tract does in the postero-lateral column. Its function is believed by Gowers to be the conduction of the sensations of pain and temperature. In disease it certainly degenerates upward, and therefore must in health conduct upward. Journeying still towards the front of the cord and the median line, we find that the antero-lateral white substance is very largely occupied by what is now called the *column of the anterior ground-fibres*, a name given by Flechsig. The anterior root-fibres traverse it, and its function is probably also to connect the anterior horns at different levels, and through the anterior commissure the anterior horns of the two sides of the cord at the same or at different levels. Like the posterior root-zone, the ground-fibres of this column do not degenerate through any considerable vertical length of the cord. Lastly, the *anterior pyramidal tract, or column of Türck*, is a narrow tract lying close to the anterior median fissure, sometimes varying in form and size, and having for its function the conduction of motor impressions from the brain to the arm. If this is its function its fibres must cross in the cord.

It is evident from this brief statement of the subdivisions of the white substance of the cord that, by closely bearing them in mind, the diagnostician will be greatly helped in locating the horizontal as well as the vertical position of a focal lesion, compressive or destructive, or both. Such a consideration may enable us to say, for instance, whether such a lesion is anterior, posterior, or lateral, or how much it occupies any one or more of these positions ; or, finally,—a matter of great importance, as pointed out by Horsley, —the mode of extension of a unilateral lesion, by a tumor which eventually becomes bilateral.

One of the oldest, and, at the same time, one of the best, illustrations of the importance

of bearing in mind the functions of the various columns or tracts of the cord is afforded by the affections known as spinal hemiplegia and spinal hemi-paraplegia, which are due to focal lesions confined to a lateral half of the spinal cord. In the first place, such a lateral lesion is modified by its height in the cord. If comparatively high up in the cervical region and completely unilateral, the patient would exhibit motor paralysis of both the arm and the leg on the side of the body corresponding to the seat of the lesion, because of the implication of the column of Türck or direct cerebral tract, and the lateral or crossed pyramidal tract, which both decussate above the lesion. Sensation would be affected chiefly on the side of the body opposite to the seat of the lesion, because of the involvement of the antero-lateral ascending tract and the column of Goll, and because also the sensory tracts decussate in the cord itself. From involvement of the direct cerebellar column, vaso-motor and visceral symptoms would be present, chiefly on the same side as the lesion. The temperature is sometimes higher, for instance, on this side. The pupil may be affected through the cilio-spinal centres. This is simply the gross, sketchy picture of a lesion high up in the cord. The phenomena would vary considerably according as the lesion was chiefly compressive, irritative, or destructive.

A unilateral focal lesion of the dorsal cord would give hemi-paraplegia. In such a case the arm, the pupil, and the neck escape. Motor paralysis occurs in the leg of the same side, and anæsthesia in the trunk and leg of the opposite side. Sometimes a band of hyperæsthesia, or even wide-spread hyperæsthesia, is present on the side of the lesion, due doubtless usually to irritation of nerve-roots.

Instead of this somewhat complete symptom-picture, we may have a set of symptoms which are dependent upon involvement of

only one or two of the subdivisions of the white substance of the cord, although such exceedingly circumscribed lesions are comparatively rare. By the close study of history and symptoms we can sometimes trace the progress of a lesion from one tract to another.

Some peculiarities in sensory symptoms may occasionally give the clue to the possible extent of a lesion. The loss of the tactile sense, with the preservation of the senses of pain and temperature, for example, mentioned in one of the cases in the first part of this paper, was possibly of some value in connection with the question of the localization of the spinal paths for the transmission of the sensations of touch, pain, and temperature. Schiff's and Gowers's view, as stated, is that the lateral columns contain the tracts for sensibility to pain and probably also to temperature, and the posterior columns the paths for the conduction of tactile sensibility. In a case of focal lesion with compression, like the one reported, the existence of tactile anæsthesia, without loss of sensation of pain and temperature, would seem to be most easily explained on the supposition that the paths for these different sensations are not in exactly the same position in the cord. The columns for touch being the most posterior, the tactile anæsthesia may have been due to counter-pressure from jamming backward of the cord by the vertebral displacement.

Let me say a few words about the vertical extension of the spinal conducting tracts. The column of Goll, or postero-median column, extends throughout the whole length of the cord. The postero-external column, or column of Burdach, posterior root-zone of Charcot, continues throughout the entire cord, but degenerates only a short distance above a lesion. The direct cerebellar tract only reaches from the level of the first lumbar nerve upward, chiefly

from the region of the vesicular column of Clarke ; it does not, for example, degenerate from a lesion of the lumbar enlargement. The lateral limiting layer, between the lateral pyramidal tract and posterior horn, is throughout the cord. The fibres of the antero-lateral ascending tract of Gowers, often confounded with the cerebellar tract, pass upward through the whole length of the cord. The anterior ground-fibres also extend throughout the whole length of the cord. The column of Türck is present only in the cervical and upper dorsal cord. The lateral pyramidal tract extends through the whole length of the cord.

The importance of knowing the vertical extension as well as the position of the spinal columns for practical purposes is at once evident, and in diagnosis comes out with great distinctness in localizing focal lesions of very limited extent. Such a lesion might, for example, involve only or chiefly the column of Türck, or anterior pyramidal tract, and would, therefore, give us motor-conducting symptoms, largely confined to the arms. If the lesion was lateral or peripheral, it might only or chiefly involve the ascending antero-lateral tracts, and give us for symptoms interference, with sensibility to pain and temperature, on one or both sides. The same reasoning can be readily extended to other isolated or neighboring tracts.

In considering a lesion below the upper dorsal region the practical diagnostician would not take into account the column of Türck, which has no existence below this level. The direct cerebellar column would also be out of consideration in large measure in lesions of the lumbo-sacral cord and cauda equina. On the other hand, the columns of Goll and of Burdach, the crossed pyramidal tract, the antero-lateral ascending tract, and the region of the anterior ground-fibres would be considered at any and all heights.

SEGMENTAL LOCALIZATION.

Theoretically, it is possible to localize a lesion in any segment of the cord. It is practically possible to do this in many segments of the cord, and for some the process with our present light is by no means difficult. For any vertical subdivision of the cord containing two or three segments we should be able to localize lesions with sufficient accuracy for grave operations.

In the detailed study of segmental localization, the order in which the subject will be considered is (1) sensory localization, (2) motor localization, (3) reflex localization, (4) the localization of vaso-motor and trophic phenomena. Regarding the cord as made up of a series of reflex systems, or reflex nerve-roots or arcs, as they are commonly called, sensory impressions are conveyed inward from the peripheral end organs to sensory roots and centres ; thence are transferred to motor centres, and thence outward again reflexly to roots, nerves, and muscles themselves ; so that the study of a single segment of the cord will include sensory, motor, and reflex phenomena. When the brain takes part the sensory impression enters the cord by the posterior nerve-roots, and, if not reflected at once, passes up the opposite side of the cord to the cerebrum, and eventually calls forth a motor impulse, which is sent down the cord to the anterior motor cells, and thence to the muscles.

SEGMENTAL SENSORY LOCALIZATION.

The sensory mechanism of the cerebro-spinal system consists, then, of peripheral end organs which receive impressions, incoming nerve-fibres which carry them by way of the posterior roots into the spinal cord, commissural fibres which immediately or almost immediately transfer them to the opposite half of the cord, and ascending conducting tracts by which they are conveyed up the cord to

the oblongata, pons, crus, internal capsule, corona radiata, and brain cortex. Various interrupting ganglia are interposed along this sensory highway ; but these we shall not here consider. Dividing one-half of the spinal cord will cause anæsthesia in the opposite half of the body below the place of hemisection, and at the same time a line or belt of hyperæsthesia or anæsthesia may be produced on the same side as the lesion in the distribution of some particular nerve-root or roots injured. This has already been referred to in considering spinal hemiplegia and hemiparaplegia. This anæsthesia is in the main caused by severance of the ascending conducting paths for sensation, which are in the side of the cord opposite to the parts of the body supplied. It is necessary, however, to consider more particularly the sensory effects in certain territories and their special subdivisions, because of the implication of one or more segments, or limited vertical extensions, of the spinal cord.

Segmental sensory localization has not been much elucidated by the contributions of physiologists, who have done so much for motor localization. The contributions to segmental sensory localization are, indeed, few and of quite recent date. The most valuable—some of them already referred to in a general way—have been furnished by Ross* and Thorburn ;† and by Herringham,‡ Paterson,§ and Goodsir,‖ who are quoted by Ross. The limits

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* *Brain*, April, 1884; ibid., January, 1885; ibid., January, 1888.

† *Brain*, January, 1887; ibid., January, 1888; ibid., October, 1888; *Brit. Med. Jour.*, December 22, 1888; and *Med. Chron.*, April, 1889.

‡ *Proc. Roy. Soc.*, vol. xli., 1887.

§ *Jour. Anat. and Phys.*, April, 1887; ibid., July, 1887; and *Quart. Jour. Micr. Sci.*, vol. xxviii., 1887.

‖ *Edin. New Phil. Jour.*, N. S., vol. v., January, 1887; and *Anat. Mem.*, vol. ii. Quoted from Paterson by Ross.

of this paper will allow me only to refer briefly to this subject, chiefly giving the facts determined by Ross and Thorburn. Heiberg's* "Atlas" was of value in my studies of the various sensory areas of the skin. The papers from which the most useful data have been obtained have been clinico-pathological and embryological,—studies of cases of injury to the spinal cord, intraspinal nerve-trunks, and extraspinal nerve-trunks and plexuses; and investigations chiefly developmental and morphological of the limb plexuses of the lower animals and of man.

A spinal nerve, as Ross shows, is derived from the cord by a posterior (superior) gangliated and an anterior (inferior) non-gangliated root. Where the two roots come together the nerve divides into a superior primary division supplying the skin of the back over the neural canal; and an inferior primary division, which subdivides into a dorsal trunk, supplying the lateral, and a ventral supplying the ventral or anterior surfaces. A branch is also given off the viscera.

The entire surface of the body may be divided into three great nerve-territories,—one posterior, one lateral, and one anterior or ventral.

The sensory nerve-supply to the posterior territory is comparatively easy to understand. It is from the superior primary divisions of the spinal nerves. It occupies a region which may be roughly described as consisting of the back of the head and the back,—the region over the neural canal. Heiberg says that its lower limit may be taken to be roughly indicated by the crest of the ilium inside of the sacrum. The back of the head is supplied from the upper cervical nerves, because in

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* "Atlas of the Cutaneous Nerve-Supply of the Human Body." By Jacob Heiberg, M.D. Illustrated by Alfred Fosterud. Translated and edited by W. W. Wagstaffe, B.A., F.R.C.S.

the process of evolution the skin of the back of the neck has been dragged upward in order to cover the greatly-enlarged brain and skull of the man. This posterior nerve-territory in the head reaches forward over the top of the head and forehead to the root of the nose, its anterior portion being supplied from the superior primary or first division of the trigeminal or fifth nerve. Ross compares the distribution of the sensory nerves in the dorsal or posterior region of the cord with that which is seen in the lowest vertebratæ,—the amphioxus, for example. In the amphioxus, section of the posterior roots of a pair of nerves will cause anæsthesia of exactly corresponding sections of the body. In man, section of the dorsal posterior roots, superior primary division, likewise produces anæsthesia of the skin of corresponding segments of the body, the only differences being that in man some anastomoses of different dorsal nerves occur, and the nerve areas of distribution tend to be placed successively lower with reference to the levels at which the nerves originate.

The anterior nerve-territory of the trunk in a manner corresponds to the posterior territory,—that is, it occupies a position on the front of the trunk and face comparable in extent to that of the posterior territory on the back and head. The anterior and lateral cutaneous nerves of the thorax are branches from the same nerves. The lateral territory is the largest and in many respects the most important sensory surface. Heiberg says that the line of demarcation between the lateral and anterior territories generally divides the breast into two equal halves, the distribution of the anterior branches, however, extending on to the front of the thighs. The extremities are largely supplied by the lateral branches. In the neck the branches are all anterior and posterior; in the thorax the anterior and lateral cutaneous branches are from the same nerves.

The limb plexuses are formed from the union of the branches of the inferior primary divisions of the nerves. The sensory nerves from the two branches of this inferior primary division are very irregularly distributed in the limbs,—that is, looking at the matter from a gross topographical point of view. This is due to the fact that, in the process of development of the limbs in the evolution of higher from lower animal forms, the sensory nerves, both dorsal and ventral, have been irregularly arrested in the extremities. In the formation of the plexuses the dorsal branches nearly always unite with the dorsal, and the ventral with the ventral branches only ; although Paterson, according to Ross, admits exceptions in the case of the small sciatic nerve, which is formed by a union of dorsal and ventral fibres, and in that of the external and short saphenous nerves which are undoubtedly so constituted.

In the limbs, then, the sensory disturbances which are due to involvement either of the cervical or lumbo-sacral segments of the spinal cord, or of the nerve-roots or nerves springing from these segments, are distributed more or less longitudinally in accordance with the particular segment or nerve affected. The hyperæsthesia, for example, which in a dorsal lesion extends around the trunk in the form of a belt or girdle, in a cervical or lumbo-sacral lesion has a longitudinal distribution more or less regular in the limbs. In like manner, the anæsthesia and paræsthesia present in the limbs have distributions in accordance with the shape of the cutaneous sensory areas. A knowledge of sensory segmental localization is therefore necessary that the diagnostician shall quickly be able to decide on the spinal or other localization of a lesion by the zone of disturbance in the limb.

Sensory segmental localization follows certain laws. If the upper extremity, according to Ross, be placed in the embryological posi-

tion,—that is, with the thumb directed outward and upward, the palm forward,—the preaxial border from the tip of the shoulder down to the metacarpo-phalangeal articulations of the index-finger and thumb is supplied by the fifth cervical root, and the postaxial border, from the axilla to the finger-tips inclusive, is supplied by the humeral branch of the second, the first dorsal, and the eighth cervical nerves. Observations made in cases of disease of the cauda equina have also convinced Dr. Ross that, if the lower extremities be also placed in the embryological or tailor position, the preaxial border is supplied by the cutaneous nerves of the four upper lumbar nerves, and the postaxial by the coccygeal and sacral sensory nerves. He has concluded also that the most distal parts of the preaxial border were supplied from the lower of the four lumbar nerves, and of the postaxial border by the higher sacral nerves.

The chief law of sensory distribution, as worked out by Herringham and adopted by Ross, is as follows:

"A. Of two spots on the skin, that which is nearer the preaxial border tends to be supplied by the higher nerve.

"B. Of two spots in the preaxial area, the lower tends to be supplied by the lower nerve; and of two spots in the postaxial area, the lower tends to be supplied by the higher nerve."

While many cases of disease limited to the anterior horns or to particular cell-groups of these horns have been recorded, I do not know of any reports of lesions circumscribed in the posterior horns of particular segments of the cord. Most of our practical knowledge of sensory segmental localization is therefore to be derived from cases which have been reported by Ross, Thorburn, and others, in which both the cord and nerve-roots have been injured extensively. To the papers of these observers I will refer those interested

for details, giving here only some of the summarized conclusions.

Thorburn,* for example, in one of his papers on spinal localization as indicated by spinal injuries, speaks as follows with reference to the distribution of anæsthesia in the upper limbs : " Dr. Ross has fully demonstrated the distribution of the sensory nerves in the upper limb. Regarding the limb in its embryological position, we find that it is projected as a bud from the trunk, the hand being supine and the radius upward, so that the palmar surface is anterior. In this position the bud carries out with it branches of the anterior primary divisions of the spinal segmental nerves from the fifth cervical to the first dorsal, inclusive ; and as these nerves maintain in the adult their embryological relations, we have the several roots supplying the limbs in numerical order from the radial to the ulnar side. Hence, then, the higher the paralysis extends in the cord through the brachial region, the farther will the anæsthesia extend from the ulnar towards the radial side. This arrangement is fully demonstrated by our cases. It is necessary to refer again to the distribution which will be obvious to any one who takes the trouble to read the reports, and of which I have already spoken in my previous paper. It will be found that the fifth root supplies the region overlying the deltoid muscle, and the outer aspect of the arm and forearm as far as the styloid process of the radius or base of the thumb, and that the eighth cervical and first dorsal supply the little finger and inner side of the hand, forearm, and arm, the remaining roots providing for the central parts of the limb on both anterior and posterior aspects. It will, however, be found that these central roots supply relatively a much less extensive area than those above and below them."

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* *Brain*, October, 1888.

In his paper on injuries to the lumbo-sacral region,* Thorburn likewise summarizes the sensory distribution of the various lumbo-sacral nerve-roots. This summary is as follows :

" First lumbar nerve : ilio-hypogastric and ilio-inguinal.

" Second lumbar nerve : outer (?) and upper part of thigh.

" Third lumbar nerve : anterior aspect of thigh below second lumbar.

" Fourth lumbar nerve : anterior part of leg.

" Fifth lumbar nerve : Back of thigh, except in distribution of first, second, and third sacral.

" First sacral nerve : a narrow strip on back of thigh ; back of leg and ankle ; sole ; part of dorsum of foot.

" Second and third sacral nerves : perineum, external genitals, ' saddle-shaped' area of back of thigh."

MOTOR LOCALIZATION.

Methods, anatomical, physiological, microscopical, clinico-pathological, and clinical, have all contributed their share to our knowledge of motor localization ; and to some of the results obtained in each of these ways I will briefly refer.

ANATOMICAL RESEARCHES.

Herringham carefully and patiently dissected the nerves of the brachial plexus on many subjects, fœtal and older, in order to trace these nerves to their final destinations in the muscles and the skin. He gives a tabular statement showing the usual nerve-supply of muscles of the upper limbs, as follows :

" Third, fourth, and fifth cervical : levator anguli scapulæ.

" Fifth cervical : rhomboids.

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* *Med. Chronicle*, April, 1889.

"Fifth, or fifth and sixth cervical: supra spinatus, infraspinatus, teres minor.

"Fifth and sixth cervical: subscapularis, deltoid, biceps, brachialis anticus.

"Sixth cervical: teres major, pronator teres, flexor carpi radialis, supinator longus and brevis, superficial thenar muscles.

"Fifth, sixth, and seventh cervical: serratus magnus.

"Sixth and seventh cervical: extensor carpi radialis.

"Seventh cervical: coraco-brachialis, latissimus dorsi, extensors at back of forearm, outer head of triceps.

"Seventh and eighth cervical: inner head of triceps.

"Seventh, eighth, and ninth cervical: flexor sublimis, flexor profundis, carpi ulnaris, longus pollicis, and pronator quadratus.

"Eighth cervical: long head of triceps, hypothenar muscles, interossei, deep thenar muscles."

PHYSIOLOGICAL RESEARCHES.

Few experimental investigations with reference to the exact functions of the nerve-roots and horns of the spinal cord have been made. One of the most important and earliest of these researches was that of Ferrier and Yeo in 1880.* These investigations were as to the effects of irritation of the motor roots of the brachial and crural plexuses of monkeys. The faradic current was employed. It was found that each motor root represented a distinct functional combination. The muscles set in action were so related as to bring about a definite action of an adapted nature. The muscles affected by electricity exciting any one root were related in function. The relation was not simply one of contact or nearness.

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* *Proc. Roy. Soc.*, London, No. 212.

The actions produced by the different roots were summarized by Ferrier as follows :

" First dorsal : action of the intrinsic muscles of the hand, muscles of the ball of the thumb, interossei, etc.

" Eighth cervical : closure of the fist, with pronation and ulnar flexion of the wrist, retraction of the arm, with extension of the forearm.

" Seventh cervical : the *scalptor ani* action, —viz., adduction with rotation inward and retraction of upper arm, extension of forearm and flexion of wrist and fingers, so as to bring the tips against the flank.

" Sixth cervical : the movement of 'attention,'—viz., adduction and retraction of upper arm, extension of forearm, pronation and flexion of wrist, the palm of the hand being brought towards pubes.

" Fifth cervical : movement of the hand towards the mouth,—viz., raising the upper arm inward, flexion of the forearm with supination, and extension of the wrist and fingers.

" Fourth cervical : a similar movement of forearm and hand,—viz., the upper arm is raised upward and backward."

The actions of the *lower extremity* were respectively :

" Second sacral : action of the intrinsic muscles of the foot,—viz., adduction and flexion of the hallux, with flexion of the proximal phalanges and extension of the distal.

" First sacral : flexion of the leg, plantar flexion of the foot, flexion of all the toes at the proximal phalanges, and also of the distal phalanx of the hallux.

" Fifth lumbar : outward rotation of the thigh, flexion and inward rotation of the leg, plantar flexion of the foot, and flexion of the distal phalanges.

" Fourth lumbar : extension of the thigh, extension of the leg, and pointing of the great toe.

"Third lumbar: flexion of the thigh and extension of the leg."

Paul Bert and Marcacci,* in 1881, studied the distribution of the motor roots of the lumbar plexus, their researches being made on dogs and cats. The nerves were very carefully cut close to their origin, and stimulated electrically with the utmost care. "It was found that the first root of the lumbar plexus determines the contraction of the sartorius, the rectus, and the psoas muscles, which are closely connected in the dog and cat, and all of which flex the hip upon the trunk. The second root of the plexus excites contraction in the anterior portion of the vastus externus, a part of the tensor of the fascia lata, and the vastus internus,—*i.e.*, in the muscles which extend the leg on the thigh. The function of the third root is similar to that of the second, with some differences in detail. It excites part of the vastus internus, the anterior part only of the biceps, which is an extensor, while the posterior portion is a flexor. The fourth root causes movements in the posterior part of the biceps, the semi-tendinosus and the semi-membranosus (flexors of the leg and thigh), and the second and third adductors of the thigh, and the extensors of the thigh. It thus innervates three kinds of movements, which are in no respect opposed or contradictory. The fifth root presides over the movements of the tail. From these results the experimenters conclude that there is evidently a systematic arrangement of the innervation of the limb at the origin of the nerves of the spinal cord; it is a functional systematization,—*i.e.*, the motor filaments arising at a given level are distributed to muscular masses, which act together, and concur to produce an associated movement. In the second place (as Duchenne

* The results obtained by Bert and Marcacci were summarized in the *Lancet* for October 1, 1881.

long ago demonstrated and as is now generally recognized), the anatomical unity of a muscle has no physiological correspondence. A single muscular mass may be in one part a flexor, in another an extensor. But the nerve distribution corresponds to the function, in so far as the different roots, innervating the several functions, are concerned."

Other researches, which are referred to by Ferrier and Yeo, have been made on the lower animals with a view to determine the situations of the roots of the plexuses of the limbs. Peyer and Krouse found that most of the muscles of the limbs were supplied by more than one root of the plexus. It was determined, among other things, that the muscles nearer the shoulder were supplied by the higher roots and those of the hand by the lower roots of the plexus; also that the sensory roots have a corresponding distribution to the distribution of the anterior roots.

During the operation by Dr. Abbe* on Dr. Dana's case of intractable brachial neuralgia, several interesting experiments were made with the faradic battery, so far as I know the first of the kind on man. With a sponge electrode on the back, a metal-point electrode was applied at various points. When applied to the sixth nerve, just external to the dura, it caused contraction of the supra- and infra-spinati, rhomboid, latissimus dorsi, pectoralis major, teres, and deltoid. Application to the seventh nerve caused contraction of the pectoral, latissimus dorsi, and abductors of the arm; to the eighth nerve, similar contraction and pain.

MICROSCOPICAL INVESTIGATIONS: CELL-GROUPS WHICH REPRESENT PHYSIOLOGICAL UNITS IN THE HORNS OF THE SPINAL CORD.

Microscopical investigations show that in cross sections of the spinal cord a distinct

* *Medical Record*, February 9, 1889.

transverse localization of cell-groups which are related to certain definite muscles or muscular groups concerned in particular movements may be readily determined. The cord is not only an organ composed of a series of segments placed one above the other, but these segments differ in size both vertically and horizontally. The gray matter particularly shows marked differences in shape and size in various transverse sections. These differences depend largely upon the number and grouping of the cells.

Starr, drawing from various investigations, details at length the manner in which in the anterior horns of the cord the cells are arranged in distinct groups which can be determined and enumerated from within outward. He gives a series of diagrams which show the manner not only in which these cells are arranged in clusters at certain levels of the cord, but also how these continue or fail to continue in a vertical direction from one level to another.

These cell-clusters differ in number and in relative position at various heights in the cord. In the second cervical segment, for example, are three, two continuing downward into the third segment of the cord, the third even into the fourth segment. In the fourth cervical segment the number of groups is five. Again, in the fourth lumbar segment and below this as far as the sacral three groups are present. These groupings in the cord are enumerated and described by Starr and also by Gowers and Ross. The only point for us to remember in connection with practical localization is that each of these groups is probably a physiological unit. They are spinal centres of function comparable to the cerebral centres of motor function. Spitzka, in 1880, first advanced this opinion :

" The nearer the muscle is to the ventral aspect of an animal," he said, " the nearer will its nucleus be to the median line of the

cord ; and the nearer the muscle is to the dorsal aspect of the animal, the nearer will its nucleus be to the lateral cornu of the cord. Flexor nuclei are therefore in internal, extensor nuclei in external and posterior cell-groups. Thus the cell-group in the apex of the anterior horn as well as in the lateral cornu is fairly developed in the dorsal region, while the portion of the gray substance situated internally is deficient in cells, in evident relation to the deficiency of prevertebral muscles. To the extremities of the body, lateral extension of the cornua correspond, and in these the same relative position of flexor and extensor nuclei is probable. As is shown in the enlargements, there is much more differentiation of the cell-groups in such extensions in the multidigitate animals than in the solipeds, as is shown by comparing the cord of man with that of the horse. Whether groups of muscles be flexor or extensor, it will be found that the nearer they are to the animal axis, the nearer will their nuclei be to the central canal. This is especially true of the flexor nuclei. The increasing development of a cell-group in the upper cervical region near the central canal in animals with powerful head and neck flexors seems to support this statement. Extensors remote from the axis, such as the trapezius, have their nuclei quite remote from the central canal." (Quoted by Starr.)

The diagnosis of a spinal lesion in the horns or columns of the cord may sometimes be at least of negative importance in deciding the question of operation. This was illustrated in the case of Lloyd and Deaver, of a cyst or extravasation of the cervical region of the cord. The lesion in this case was found to be a hemorrhagic extravasation in the anterior horns of the cord, practically out of reach of the knife. In this case, however, an external swelling which made operation important was present. If the external indi-

cations had not been present, the close diagnosis of the exact location of the lesion in a transverse section of the cord would have been of great importance. We have now data on hand for such localization at different levels. We can even make a fairly exact diagnosis of the position of a lesion at various positions from within outward in the anterior horns of the cord.

It may even be found that our knowledge of the exact position from without inward of these groups of cells which represent physiological units—which are, in other words, related to certain definite movements—may occasionally prove of practical value. Decision as to operating, for example, may depend not only upon our knowledge of the exact level and vertical limitations of a lesion, but also to some extent upon the amount of compression and of destruction by invasion of the spinal cord. As has just been stated, the nuclei of extensor muscles are situated in external and posterior cell-groups, while those of flexor nuclei are more internal ; therefore, the predominance of extensor or of flexor paralysis may help sometimes to a conclusion as to the amount of transverse damage of the cord.

CLINICO-PATHOLOGICAL AND CLINICAL INVESTIGATIONS.

As already stated, Thorburn has published certain clinical and pathological observations upon injuries to the cervical region of the spinal cord, and has endeavored to draw therefrom an accurate picture of the functions of each of the nerve-roots entering into the formation of the brachial plexus. His usual method consisted in noting accurately the distribution of the paralysis and anæsthesia which ensued immediately upon a lesion of the cord, of which the exact site had been determined by post-mortem examination ; also

of pursuing the changes which resulted from the consequent ascending myelitis. He has also applied the same methods to the lumbo-sacral or crural plexus.

He has tabulated provisionally, as follows, what he is led, from the evidences of his cases, to believe to be the arrangement, from above downward, of the muscle nuclei in the cervical cord :

" Fourth cervical nerve : supraspinatus and infraspinatus, teres minor (?).

" Fifth cervical : biceps, brachialis anticus, deltoid, supinator longus, supinator brevis (?).

" Sixth cervical : subscapularis, pronators, teres major, latissimus dorsi, pectoralis major, triceps, serratus magnus.

" Seventh cervical : extensors of the wrist.

" Eighth cervical : flexors of the wrist.

" First dorsal nerve : interossei, other intrinsic muscles of the hand."

He has likewise summarized for the lumbo-sacral region of the spinal cord the arrangement of both the motor and sensory fibres of the nerve-roots. His summary, he states, is to be distinctly understood as only that for which his own cases gave evidence,—that is, they are positive evidence for the facts reported, but do not negative the existence of other facts the existence of which was not proved by the cases. Thus, for example, although no doubt the second lumbar root affords muscular branches, these are ignored because his cases yield no evidence of the same. His summary of the sensory arrangement has already been given. The following is the motor distribution as determined by him:

" First lumbar nerve : none.

" Second lumbar : none.

" Third lumbar : sartorius, adductors of thigh, flexors of thigh.

" Fourth lumbar : extensors of knee, abductors of thigh.

" Fifth lumbar : hamstring muscles.

" First and second sacral : calf muscles,

glutei, peronei, extensors of ankles, intrinsic muscles of foot.

"Third sacral: perineal muscles (erector penis, transversalis perinei, accelerator urinæ, etc.).

"Fourth sacral: bladder and rectum."

Ferrier,* in a paper on atrophic spinal paralyses, has applied the facts determined by his and other experiments, and made a provisional enumeration, as a guide to further more minute clinical research, of the muscles which are likely to be affected in poliomyelitis limited to each segment, the muscles being placed in the order in which they would probably suffer.

"First dorsal type: the intrinsic muscles of the hand,—viz., muscles of the thenar and hypothenar eminences and interossei.

"Eighth cervical type: long flexors, ulnar flexors of wrist, intrinsic muscles of hand, extensors of wrist and phalanges, long head of triceps (pectoralis major?).

"Seventh cervical type: teres major, latissimus dorsi, subscapularis, pectoralis major, flexors of wrist and fingers (median), triceps.

"Sixth cervical type: latissimus dorsi, pectoralis major, serratus magnus, pronators (flexor of wrist?) triceps.

"Fifth cervical type: deltoid (clavicular portion), biceps, brachialis anticus, serratus magnus, supinator longus, extensors of wrist and fingers.

"Fourth cervical type: deltoid, rhomboid, supra- and infraspinatus (teres major), biceps, brachialis anticus, supinator longus, extensors of wrist and fingers, diaphragm."

In the lower extremity:

"Second sacral type: intrinsic muscles of the foot, strictly parallel to the first dorsal type.

"First sacral type: muscles of calf (plantar flexors), hamstrings, long flexor of big toe, intrinsic muscles of foot."

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* *Brain*, vol. iv. p. 226, 1881–1882.

"Fifth lumbar type: flexors and extensors of toes, tibial muscles, sural muscles, peroneal muscles, outward rotators of the thigh, hamstrings.

"Fourth lumbar type: extensors of thigh, extensor cruris, peroneus longus, adductors.

"Third lumbar type: ilio-psoas, sartorius, adductors, extensor cruris."

On the principles of localization certain types of spinal paralysis have also been determined and named by others, as by Remak and Erb. Remak speaks of an upper-arm type of atrophic spinal paralysis, in which are jointly affected the supinator longus with the brachialis anticus and the biceps and the deltoid. The same author describes the forearm type in which the extensors of the wrist and fingers, and the muscles of the hand are paralyzed. He has also described three types of paralysis of the lower extremities,—"The first in which the extensors and adductors of the leg are affected together, the sartorius muscle, however, escaping, although it is supplied by the anterior crural nerve, which also supplies the extensors; a second type, in which the ilio-psoas, sartorius, and flexors of the leg are affected together; and a third type, in which the muscles upon the leg and foot are involved with the exception of the tibialis anticus. He notices that the last-named muscle is rarely paralyzed with the other muscles, and that when it is affected, the other muscles moving the foot frequently escape." (Starr.)

SEGMENTAL LOCALIZATION OF REFLEX CENTRES.

Certain cells in the gray matter of the spinal cord preside over reflex action. These reflex actions, and the areas in which they are capable of being excited, are described in the more recent text-books. These centres are

cutaneous or superficial, and muscular (or musculo-tendinous) or deep. The centres for these various reflexes are enumerated by Gowers as follows :

" Superficial reflex action : plantar, second sacral ; gluteal, fourth lumbar ; cremaster, second lumbar ; abdominal, sixth to eleventh dorsal (epigastric, sixth dorsal) ; scapular, fifth cervical to first dorsal.

" Muscle reflex action : calf muscles (foot clonus), fifth lumbar and first sacral ; knee-jerk, third and fourth lumbar ; flexor digitorum, triceps, seventh cervical ; biceps, supinator longus, sixth cervical."

An important set of reflex centres for localization purposes are those which preside over various actions more or less complex and involuntary. These include such as the following : Cilio-spinal, cremasteric, sexual, parturitional, vesical, and anal. Starr enumerates the position of these centres as follows : Cremasteric, first to third lumbar ; vesical, third lumbar ; rectal or anal, fourth lumbar. By other observers the cilio-spinal centre is placed in the lower cervical cord and dorsal, as far as the second or third dorsal vertebra. The sexual, or erection centre, has been placed without close localization in the lumbar cord, probably about the second or third lumbar segment. Korner has located what he terms the parturition centre at the level of the first or second lumbar vertebra. Those centres for complex reflexes which are really valuable for purposes of practical localization are the cilio-spinal, cremasteric, vesical, and anal centres ; their position will be indicated in table at the conclusion of this article. The latest investigations of Thorburn and others indicate that the centres for the bladder and the anus or rectum are in the extreme lower part of the cord ; in fact, in the terminal portion of the cord, the region of the third, fourth, and fifth sacral and the coccygeal nerve-roots.

VASO-MOTOR AND TROPHIC LOCALIZATION.

Vaso-motor and trophic centres are distributed throughout the entire vertical extent of the spinal cord. Those connected with the muscles are identical with, or situated close by, the corresponding motor nuclei in the anterior horns. Vaso-motor and trophic centres for the skin, nails, and joints also have a local habitation in the spinal cord. Pathological facts, as those, for instance, which have been obtained in studying syringomyelia, point to the central gray matter of the cord as the probable seat of these centres. By the central gray matter I mean here that which is situated around or near the central canal. In the posterior portion of this central gray matter it is probable that the vaso-motor and trophic centres for the skin, nails, and joints are situated, while more anteriorly are probably placed the trophic centres for the bones. A special collection of large bipolar cells is found in the postero-internal gray matter, and extends in the form of a continuous column from about the seventh cervical to the second or third lumbar segment. This collection of cells, first described by Lockhart Clarke, is usually designated as Clarke's vesicular column, and has been referred to several times in this paper. It is now almost established by various pathological reports, and the careful anatomical and microscopical investigations of Gaskell, that this column represents a series of vaso-motor and visceral centres. Communicating nerves have been traced to and from this vesicular column to the ganglia and fibres of the so-called sympathetic nervous system. When, therefore, vaso-motor and trophic phenomena are present these regions of the central gray matter must be considered.

In 1884,* and again in 1888,† Starr summarized in tabular form the functions of the

* *Am. Jour. Neurol. and Psych.*

† *Am. Jour. Med. Sci.*, May, 1888.

various segments of the spinal cord. His last table represents fairly well the status of spinal localization. In concluding this article I will give this table of Starr, with modifications based upon the results obtained by Herringham, Ross, Thorburn, and others. A study of this table, and of the various summarized statements which have already been given, will enable the diagnostician with approximate accuracy to localize the height of a lesion affecting the segments of the cord or the spinal nerve-roots. In the column for sensation the cutaneous nerves are given in parentheses. This list is not complete, but it is hoped that it will be of assistance in sensory localization.

LOCALIZATION OF THE FUNCTIONS OF THE SEGMENTS OF THE SPINAL CORD.

Segment.	Muscles.	Reflex.	Sensation.
Second and third cervical.	Sterno-mastoid. Trapezius. Scaleni and neck. Diaphragm.	*Hypochondrium*(?) Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Back of head to vertex and neck. (Occipitalis major, occipitalis minor, auricularis magnus, superficialis colli, and supraclavicular.)
Fourth cervical.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator longus. Rhomboid. Supra- and infraspinatus.	*Pupillary* (fourth cervical to second dorsal). Dilatation of the pupil produced by irritation of neck.	Neck. Shoulder, anterior surface. Outer arm. (Supraclavicular, circumflex, external (musculo-cutaneous) cutaneous.)
Fifth cervical.	Deltoid. Biceps. Coraco-brachialis. Brachialis anticus. Supinator longus. Supinator brevis. Deep muscles of shoulder-blade. Rhomboid. Teres minor. Pectoralis (clavicular part). Serratus magnus.	*Scapular* (fifth cervical to first dorsal). Irritation of skin over the scapula produces contraction of scapular muscles. *Supinator longus.* Tapping the tendon of the supinator longus produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm to the wrist. (Supraclavicular, circumflex, external cutaneous, internal cutaneous, posterior spinal branches.)

Segment.	Muscles.	Reflex.	Sensation.
Sixth cervical.	Biceps. Brachialis anticus. Subscapular. Pectoralis (clavicular part). Serratus magnus. Triceps. Extensors of wrist and fingers. Pronators.	*Triceps* (fifth to sixth cervical). Tapping elbow tendon produces extension of forearm. *Posterior wrist* (sixth to eighth cervical). Tapping tendons causes extension of hand.	Outer side and front of forearm. Back of hand, radial distribution. (Chiefly external cutaneous, internal cutaneous, radial.)
Seventh cervical.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis (costal part). Serratus magnus. Latissimus dorsi. Teres major.	*Anterior wrist* (seventh to eighth cervical). Tapping anterior tendons causes flexion of wrist. *Palmar* (seventh cervical to first dorsal). Stroking palm causes closure of fingers.	Radial distribution in the hand. Median distribution in the palm, thumb, index-, and one-half middle finger. (External cutaneous, internal cutaneous, radial, median, posterior spinal branches.)
Eighth cervical.	Triceps (long head). Flexors of wrist and fingers. Intrinsic hand muscles.	.....	Ulnar area of hand, back, and palm, inner border of forearm. (Internal cutaneous, ulnar.)
First dorsal.	Extensors of thumb. Intrinsic hand muscles. Thenar and hypothenar muscles.	.....	Chiefly inner side of forearm and arm to near the axilla. (Chiefly internal cutaneous and nerve of Wrisberg or lesser internal cutaneous.)
Second dorsal.	.....	.....	Inner side of arm near and in axilla. (Intercosto-humeral.)
Second to twelfth dorsal.	Muscles of back and abdomen. Erectores spinæ.	*Epigastric* (fourth to seventh dorsal). Tickling mammary region causes retraction of the epigastrium. *Abdominal* (seventh to eleventh dorsal). Stroking side of abdomen causes retraction of belly.	Skin of chest and abdomen, in bands running around and downward, corresponding to spinal nerves. Upper gluteal region. (Intercostals and dorsal posterior nerves.)

Segment.	Muscles.	Reflex.	Sensation.
First lumbar.	Ilio-psoas. Rectus. Sartorius.	*Cremasteric* (first to third lumbar). Stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum. (Ilio-hypogastric, ilio-inguinal.)
Second lumbar.	Ilio-psoas. Sartorius. Quadriceps femoris.	*Patellar*. Striking patellar tendon causes extension of leg.	Outer side of thigh. (Genito-crural, external cutaneous.)
Third lumbar.	Quadriceps femoris. Anterior part of biceps. Inner rotators of thigh. Abductors of thigh.	.....	Front of thigh. (Middle cutaneous, internal cutaneous, long saphenous, obturator.)
Fourth lumbar.	Abductors of thigh. Adductors of thigh. Flexors of knee. Tibialis anticus. Peroneus longus.	*Gluteal* (fourth to fifth lumbar). Stroking buttock causes dimpling in fold of buttock.	Inner side of thigh, leg, and foot. (Internal cutaneous, long saphenous, obturator.)
Fifth lumbar.	Outward rotators. Flexors of knee. Flexors of ankle Peronei. Extensors of toes.	*Achilles tendon*. Over-extension causes rapid flexion of ankle, called ankle clonus.	Back and outer side of leg and ankle; sole; dorsum of foot. (External popliteal, external saphenous, musculo-cutaneous, plantar.)
First and second sacral.	Flexors of ankle Extensors of ankle. Long flexor of toes. Intrinsic foot muscles.	*Plantar* (fifth lumbar to second sacral). Tickling sole of foot causes flexion of toes and retraction of leg.	Back and outer side of leg and ankle; sole; dorsum of foot. (Same as fifth lumbar.)
Third, fourth, and fifth sacral.	Perineal. Muscles of bladder, rectum, and external genitals.	Vesical centres. Anal centres.	Back of thigh, anus, perineum, external genitals. (Small sciatic, pudic, inferior hemorrhoidal, inferior pudendal.)
Fifth sacral and coccygeal.	Coccygeus muscle.	.....	Skin about the anus and coccyx. (Coccygeal.)

LESIONS OF THE
SACRAL AND LUMBAR PLEXUSES.

With Remarks on their Importance, and their Diagnosis, General and Localizing.[1]

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THE purpose of the present paper is to show the importance and comparative frequency of lesions of the limb plexuses, to give some suggestions or rules for their recognition, and briefly to refer to questions connected with their surgical treatment. Gross lesions, not degenerations, will be considered; and so-called functional affections, such as neuralgias proper, will be treated of only as necessitated by a discussion of diagnosis. Some of the lesions of the limb plexuses or their cords or branches are common to all, as, for example, neuritis, neuromata, non-neural growths implicating nerves, aneurisms, abscesses, gunshot and other injuries. Other gross lesions have a special character owing to location: as rectal, ovarian or uterine lesions involving the sacral; vertebral caries and certain abscesses the lumbar, and dislocations of the shoulder or fracture of the clavicle, the brachial plexus. It will be impossible in the space to which this communication must be restricted to dwell at length upon

[1] Read before the Neurological Section of the New York Academy of Medicine, May 10, 1889.

fine points of localization; but specifically in a few instances and incidentally in others, localizing facts and rules will be given. Complicated as is the sensory and motor arrangement and distribution of these plexuses, thanks to the careful work of Mitchell, Paterson, Goodsir, Ferrier, Bert and Marcacci, Forgue and Lanagrace, Walsh, Putnam, Herringham, Ross, Thorburn, and a few others, we are now able to approach the subject of the localization of lesions of even particular cords of these plexuses and their nerve roots with increasing confidence.

LESIONS OF THE SACRAL PLEXUS.

The sacral plexus formed by the lumbo-sacral cord, the anterior divisions of the three superior sacral nerves, and a part of the fourth, rests largely upon the surface of the pyriformis muscle in the true pelvis; and a single glance shows how thoroughly, in accordance with its great function, it is protected both by its position and coverings.

It may be well to recall one or two well-known points in pelvic neural anatomy which may have some localizing value. The superior gluteal nerve, for instance, arises from the back part of the lumbo-sacral cord, and is the only nerve that springs from that important link between the lumbar and sacral plexuses; so that a lesion strictly limited to this cord would give sensory and motor impairment in the distribution of this nerve, as well as the symptoms resulting from interference with the functions of the lumbo-sacral cord itself. The muscular distribution of the superior gluteal is to the gluteus minimus and medius and tensor vagina femoris. The lowest of the intra-pelvic nerves are not connected with the sacral plexus at all. These are the branches of the fourth and fifth sacral nerve roots to the levator ani, sphincter ani, coccyx, and skin

of these parts. A lesion may be so isolated as to affect only these extra-plexal pelvic nerves, or the plexus may occasionally be affected and these nerves escape.

The neurologist should know the landmarks for examining and palpating the sacral plexus and intra-pelvic nerves by the rectum, the chief of which are the great and lesser sacro-sciatic ligaments, the spine of the ischium, and the pyriformis muscle.

A few general statements with reference to lesions of the sacral plexus may serve to present the subject more clearly and comprehensively. A lesion of the sacral plexus in its entirety strongly counterfeit a unilateral affection of the lumbo-sacral portion of the spinal cord from which the nerves of the plexus arise, or of the lower part of the cauda equina within the spinal canal. Bilateral lesions of the sacral plexuses so closely counterfeit lesions of the lumbo-sacral region of the spinal cord, or of the cauda equina, as to make the diagnosis sometimes exceedingly difficult; and in some cases of this kind the differentiation can be best made by proper local manipulation and examination by the rectum or vagina, or both. Lesions of single or several cords of this plexus, or of its nerve branches within the pelvic cavity, may counterfeit neuralgias of the nerves in either their intra- or extra-pelvic distribution; sciatica is a frequent diagnosis in these cases. Lesions of the sacral plexus or intra-pelvic nerves are not infrequently supposed to be disease of the uterus or ovaries, or their appendages and surroundings; and, on the other hand, disease and enlargements of these parts often involve the plexus.

Beside growths or other diseases of the uterus, ovaries, and their connected parts, the gross lesions which may affect the sacral plexus, or in which it may be implicated indirectly, are neuritis or nerve degeneration from pressure or bruising during labor,

rectal disease, pelvic cellulitis and abscesses, pelvic or intra-pelvic tumors, particularly neuromata, osteomata, and osteo-sarcomata, gunshot wounds, and neuritis, unilateral or bilateral, of unknown cause.

Psoas abscess may follow such a course as to involve the sacral plexus, and, before taking up the special lesions of the plexus, I may dismiss this point with a few words. Psoas abscess tends often to follow an irregular or multiple course. The pus may pass down the sacrum; it may even leave the pelvis by the sciatic notch. Brodie[1] records a case in which the abscess descended from the loins and presented as a tumor in the groin. Suddenly the tumor disappeared, but later a large collection of matter was found in the posterior part of the limb, behind the little trochanter of the femur. Post-mortem examination showed that the abscess had taken the course of the common tendon of the psoas magnus and iliacus internus muscles to their insertion into the bone, afterward extending further backward below the inferior edge of the quadratus femoris. A number of cases have been recorded in which this complication has occurred. Thompson[2] has, for example, reported a case of psoas abscess which was supposed for months to be one of sciatica. Pain, terrific but remittent, followed the course of the great sciatic nerve of the right leg. It sometimes extended to the ankle and even to the dorsum of the foot and great toe. Tenderness on pressure, hyperæsthesia in some parts and anæsthesia in others were among the symptoms.

One form of puerperal paralysis is probably the result of pressure upon the sacral plexus, or upon the lumbo-sacral cord where it passes over the brim of the pelvis; more probably the former, as the

[1] Diseases of the Joints.

[2] Lecture on Sciatica, in a Series of American Lectures, edited by E. C. Seguin, M.D.

lumbo-sacral cord is particularly well protected in its descent. Few such cases, however, have been recorded. Imbert-Gourbèyre,[1] in his monograph on *Puerperal Paralyses*, speaks of several cases in which, from prolonged labor, and probably also from imprudent obstetrical manipulations, paralysis resulted. One of these cases, cited from Rademacher, was an incomplete and painful paraplegia coming on at the end of a long and difficult labor, and cured in eight days chiefly by friction. Another patient, thirty-six years old, during her third labor was paralyzed in both extremities, and recovered in a few months. Salvat is cited as reporting the case of a woman treated by him for vesico-vaginal fissure, with paraplegia, produced by the long stay of the head of the fœtus in the inferior strait. Another patient, thirty-two years old, during her fourth labor, which was prolonged and the delivery by forceps, suffered great pain in the loins, accompanied by feebleness and swelling of the legs. The feebleness increased to paraplegia, and she had lancinating pain, paræsthesia, and cramps in the limbs. These cases are imperfectly reported, and, as the neural symptoms were bilateral, it is possible that some of them may have been spinal. It is, however, more likely that they were due to direct traumatism of the lumbo-sacral plexuses. Dr. Dercum, of Philadelphia, has reported to me, verbally, some particulars about a case of unilateral paralysis, atrophy, and anæsthesia of the leg following delivery, and apparently due to pressure on the sacral plexus in the floor of the pelvis.

Dr. Howard A. Kelly, Associate Professor of Obstetrics in the University of Pennsylvania, has kindly furnished me with brief notes of two interesting cases supposed for a long time to have been

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[1] Mém. de l'Académie roy., vol. xxv.

purely gynecological. The first case, Mrs. X., had passed through a difficult instrumental confinement some twelve years before coming under observation. She visited many prominent gynecologists, and underwent a number of operations, the last being the removal of two large tubes and the ovaries. She was relieved of menstrual exacerbations, but still suffered great pain in the pelvis, for which she was receiving galvanism, message, and anti-lithic remedies. Her suffering continued unabated; and Prof. H. C. Wood, in whose charge she was, called Dr. Kelly in consultation. A careful examination showed that the uterus and its surroundings were perfectly free from disease. On making a careful rectal examination, however, outlining the sacro-sciatic ligament and pyriform muscle, and carefully palpating the roots of the great sciatic nerve, upon touching one cord she gave a sudden scream, at the same time doubling up her leg and jerking her body in bed. Here, directly over the roots of the left sciatic nerve—the left sacral plexus—was the only diseased area which could be detected in the pelvis. All subsequent treatment was directed to this condition.

In a second case the patient had constant pelvic pain, which she described more as a soreness, and located "back of her womb." She had been for several years since the birth of her last child under the care of gynecologists, who had not been able to give her any relief whatever. It was found by exploration that the only point of tenderness in the pelvis was at the roots of the sciatic nerve, and here she at once located all her pain, when the doctor introduced his finger into the rectum and made pressure on the nerve trunks. The case was one of neuritis. As Dr. Kelly remarks in the communication sending the notes of these cases, they teach the

value of exploring the pelvis outside of the uterus and its annexes.

In one interesting case of railroad injury, supposed to be an example of railroad brain and spine, careful examination showed exquisite tenderness over both sacral plexuses, but particularly marked on one side. The plexuses were palpated through the rectum. This patient suffered great pain and tenderness in the lumbar and sacral and sacro-ischiatic regions and also complained of general weakness and pains in the lower extremities. She was pregnant at the time the accident occurred, and suffered greatly later at the time of her labor, the child dying during parturition. She had other cerebral and general nervous symptoms.

In rare cases of extensive disease of the rectum or of immense fecal accumulations, the sacral plexus or some of the intra-pelvic nerves may be compressed or irritated, or both, and thus give rise to great suffering. Pelvic cellulitis may occur even in the male, and also give symptoms of irritation and of compression of the sacral plexus. Skjeldrup[1] describes such a case in a man fifty years old, in which examination per rectum showed a hard tumor. Muir, quoted by Skjeldrup, has published a similar case. Mitchell[2] speaks of a case in which the patient suffered from numbness in the left foot and leg, followed by increasing loss of power to flex the foot. The peroneal muscles were found to have lost electrical response. On vaginal examination she was discovered to have a large growth behind and to the left of the womb. Innumerable cases of pelvic cellulitis in the female are of course on record, but in few have the sensory, motor, and other neural symptoms been reported with sufficient care to

[1] Tidsskrift f. Prakt. Med., quoted in Medical Register, vol. iv., September 15, 1888.

[2] Injuries of Nerves.

allow of anything but a general diagnosis. If gynecologists paid strict attention to the exact distribution of the pain in their cases of pelvic inflammation, abscess and growths, they would have a surer hold on early diagnosis. The character of the pain in these cases usually receives fuller attention than its distribution as would be naturally expected.

It is well known that neuromata occur somewhat frequently in connection with the cords or branches of the brachial plexus. Doubtless these growths now and then are present also in both the sacral and lumbar plexuses, and are overlooked. I have not been able to find reports of such cases, but it is probable that occasional rare forms of intractable sciatica, or other forms of neuralgia of the lower extremity in the distribution of some of the sacral nerves, are due to growths of this character.

Putnam[1] has reported an instructive case of sarcoma involving the intra-pelvic nerves, notes of the autopsy being given. The first and a recurring symptom was pain in the middle toe of the left foot. In six months the sacral region became extremely painful, and also the posterior and outer surface of the thigh, and the outer side of the leg and foot. The pain was accompanied by some paræsthesia, especially in the outer half of the left foot.

The right leg was attacked with severe pain, confined to the posterior and outer side of the thigh, nearly six months after the left. The pain abated, and renewed itself in the left leg. Walking became difficult. The muscular weakness affected not only the muscles of the sciatic distribution, but also the quadriceps extensor, and the psoas and iliacus. Slight tactile insensibility was present in the vicinity of the anus. Applications of galvanism were very painful. Knee-jerk was absent on both sides.

[1] Journ. Nerv. and Ment. Dis., vol. xiv., Sept. and Oct. 1887, p. 601.

No tumor could be discovered by rectal examination.

I have seen several cases of intra-pelvic or pelvic growths, causing pressure upon the sacral plexus or involving some of its cords or branches. In one an autopsy confirmed the diagnosis.[1] A man who had been treated for many months for sciatica, had pain chiefly down the back of the thigh, in, but not strictly confined to, the line of the sciatic nerve. The pain was altogether in the right leg; it was increased by pressure and movement, and he could not bring the foot firmly on the floor. A marked symptom was swelling of the leg, the left thigh measuring thirteen and a quarter inches; middle of leg fifteen inches; ankle eight inches; the right thigh twenty-one and a half inches; middle of leg fourteen inches; ankle nine and a half inches. The thigh presented a solid œdematous condition, with distinct pitting on pressure. A tendency to outward rotation was present. Pressure at the sciatic notch seemed to produce more pain than pressure elsewhere in the leg. Some of the abdominal muscles of the right side were cramped and tense, although those of the left side were relaxed. Movement of the limb caused pain. Examination by the rectum showed distinctly a solid resisting growth on the right side. In another case the diagnosis of sciatica had been made and persisted in for many months, but examination by the rectum revealed a tumor. The nerves of nearly the entire sacral distribution were involved, but very irregularly, in pain, anæsthesia, paralysis, and atrophy. In still another case, at the Philadelphia Hospital, the patient was also treated for a long time for sciatica, but close examination demonstrated disease of the acetabulum, and after death a large osteo-sarcomatous growth was

[1] Three of these cases were briefly referred to at the meeting of the American Neurological Association, 1887.

found. Miles,[1] of Baltimore, has contributed a case of malignant disease of the pelvis which was supposed to be one of sciatica. The patient developed a malignant tumor about the hip, of which he died. Examination both externally and by the rectum revealed nothing. The one great symptom was pain in the sciatic distribution. One important practical point is that all of these cases had been treated for other affections, chiefly for sciatica or spinal disease.

A few interesting gunshot wounds of the sacral plexus have been reported, and many other cases equally interesting of rectal and pelvic gunshot injuries in which the nerve cords escaped. Habgood[2] has recently reported a bullet wound of the pelvis involving the rectum, in which, under antiseptic treatment, the patient rapidly recovered. This patient suffered great pain, not closely described as to its particular distribution. The bullet passed inward through the great sacro-sciatic notch. During the American war 103 cases of gunshot wounds are reported, of which 44, or 42.7 per cent., proved fatal. One case of gunshot wound of the sacral plexus is reported in vol. ii., *Medical and Surgical History of the War of the Rebellion*, p. 341. The patient, a private, aged twenty-three years, was wounded at Gettysburg. He was treated on the field until August 21st, and then admitted to Camp Letterman Hospital. Acting Assistant Surgeon H. H. Sutton reported that a ball from a Sharp's carbine entered the left side of the sacrum at the third segment, passed into the pelvis and there lodged. The ball in its passage injured the sacral plexus, consequently the leg of the corresponding side became paralyzed,

[1] Journ. Nerv. and Ment. Dis., vol. xiv., Sept. and Oct. 1887, p. 666.

[2] Manitoba, Northwest and British Columbia Lancet, 1887-8. Also Brit. Med. Journ.

but the natural feeling and movements of the limb were gradually returning when the patient was admitted. Unfortunately, a study of this case as to sensation and motion was not made or not recorded.

On general principles I can see no reason why neuritis of the sacral plexus, either spontaneous, rheumatic, infectious, or of unknown cause, may not occur with moderate frequency. Neuritis of the brachial plexus, either in its entirety or in some of its subdivisions, is a well-known and often investigated disease; but the sacral and lumbar plexuses seem to have rarely attracted the attention of physicians with the idea that they were the subjects of neuritis. The surroundings of the sacral plexus are such as to render it more liable than the brachial to inflammation, either by contiguity or by causes acting through its environment. It is, perhaps, less liable to rheumatic inflammation. In every obscure case of unilateral, or even bilateral, pain in the extremities, whether associated with anæsthesia and paræsthesia or not, the sacral plexuses should be carefully examined by palpation through the rectum. A word of caution is perhaps here necessary. In some individuals, particularly females, of a highly nervous temperament, it is possible that pressure upon the comparatively exposed intra-pelvic nerves, reached by the finger in the rectum, may give rise to pain, which by psychical influence will be exaggerated so as to mislead the physician into supposing that the nerves are in a state of inflammation when such is not the case.

The following cases described by Dr. L. C. Gray[1] are cases in point where a rectal examination might have been of service for diagnosis.

The first patient felt tingling pains through both buttocks, the perineum, the scrotum, the tip of the

[1] Journ. Nerv. and Ment. Dis., vol. xiii., 1886, p. 743.

penis, and down the back of both thighs, with some smarting in urination. As the result of the Turkish and Russian baths these symptoms were violently augmented and high fever ensued. Sexual desire was entirely lost, as were also the skin and cremaster reflexes. The patient was sent to the country and had a violent attack of supra-orbital neuralgia, after which he recovered.

The other case was a woman, thirty-five years old, who was suddenly attacked with sharp pain in the buttocks, perineum, labia, and down the back of the thighs to the knees, with simultaneous retention of urine. Vesical anæsthesia came on later, but no motor impairment. Over the area of pain was also impairment of the senses of touch, temperature, and pain, but slightly less near the knee than above.

These cases are sufficient to show the importance of the subject and the frequent actual occurrence of the forms of lesions involving the sacral plexus or other intra-pelvic nerves.

Let us briefly consider the main points in the diagnosis of such cases. In the first place, the great value of close examination by the rectum, which has already been dwelt upon, cannot be over-rated. Another important general point is as to the unilateral or bilateral character of these affections. They are commonly unilateral, or begin on one side, and, in exceptional cases, become bilateral. In some cases, however, of spontaneous neuritis, or pressure neuritis, or palsy, as in those reported by Imbert Gourbèyre, the symptoms may be nearly uniformly bilateral.

Large compressing and destructive lesions of the sacral plexus give nearly the same syndrome, only usually unilateral, as is presented by lesions of the lower part of the cauda equina. These symptoms, of course, vary somewhat according to the amount

of nerve involvement. They are pain variously distributed in the domain of the sacral nerves—in the buttock, backs of the thighs and legs, soles of the feet and outer sides of the feet; anæsthesia, analgesia, and paræsthesia in the distribution of the nerves, from the second sacral nerve down to and including the coccygeal; paralysis and wasting particularly in the muscles below the knee, nearly all of which are in the sacral distribution; and in those muscles above the knee which occupy the distribution of the great sciatic and gluteal, small sciatic and superior gluteal nerves; trophic disorders, such as bedsores, perforating ulcers, abscesses, joint and bone changes, etc.; and vasomotor affections, such as changes in surface temperature, flushing, pallor, sweating, œdema, priapism, and coldness of the extremities. The bladder and rectum will be involved when the lowest sacral nerves are implicated—the nerves which do not strictly belong to the plexus. Reactions of degenerations will be present in most of the paralyzed muscles. Knee-jerk, ankle clonus, and the plantar reflex are likely to be absent on both sides; the cremasteric reflex as likely to be retained. The symptoms enumerated for lesions of the cauda equina are, in nineteen cases out of twenty, bilateral, although often not absolutely symmetrical. Consider now the same symptom-picture in one limb, and we have the phenomena produced by a large irritating, compressing, and destroying lesion of the sacral plexus.

Thorburn's[1] commentary on one of his cases of cauda equina lesion might, if the symptoms were not bilateral, be almost equally applicable to a pelvic plexus lesion. He says of this case that there was sensory paralysis of all the nerves of the sacral plexus, and possibly of the obturator, but not of the

[1] Brain, January, 1888.

anterior crural or other lumbar nerves; the perineum, penis, scrotum, and urethra, being supplied by branches of the pudic, were anæsthetic, but the root of the scrotum retained sensation, owing to the presence of twigs of the ilio-inguinal nerves, which, however, only descend to a very short distance. Paralysis with the reaction of degeneration was complete in the muscles supplied by the nerves of the sacral plexus. Those supplied by the anterior crural, although presenting the reaction of degeneration, were only weakened, and the adductors, supplied by the obturator, appeared also to retain some power. Cremasteric reflex remained, but below this point reflex action was lost.

Unilateral lesions of the sacral network must be occasionally differentiated from unilateral affections of the cauda equina. Usually, of course, cauda equinal symptoms are bilateral; but if the lesions are hemorrhagic, inflammatory, or from injury, they often are not symmetrically bilateral, and rarely they may be unilateral as in a case described by Erichsen, and quoted by Thorburn. It is, indeed, doubtful whether this case, which recovered under four months' treatment, was not, after all, one of rheumatic or traumatic neuritis of the sacral plexus. The patient could not stand but could move his legs in bed; he had not complete paralysis except in the peronei and extensors of the left ankle; rapid wasting of the left leg came on; numbness and tingling were present on the outer side of the left thigh, and partial loss of sensation below the left knee. The right limb was normal, he had occasional loss of control over the sphincters, and coldness of the extremities, especially of the left foot. The case was due to injury and came on gradually during ten days. Tenderness over the third lumbar vertebra was present after the tenth day, and symptoms of cervical injury were also present.

Weir Mitchell has given some diagnostic rules for the separation of spinal and cerebral from nerve lesions. Evidently, however, these are applicable only to the separation of true spinal cord lesions from lesions of the spinal nerves whether within or outside of the spinal canal. In other words, he has not made any distinction between lesions of the cauda equina within the canal and those of the plexuses in the abdominal and pelvic cavities. With our present lights we have little difficulty in differentiating lesions which involve the spinal cord itself from peripheral lesions. A much greater difficulty, as just indicated, is that which arises in separating irregular lesions of the extreme lower part of the cauda equina from lesions, particularly bilateral lesions, of the plexuses outside of the spine.

In cases of extensive bilateral lesions of the abdominal or pelvic plexuses, supposed to be cases of traumatic myelitis, the points mentioned by Mitchell might be of some service. He speaks, for instance, of a plan proposed by Stich.

"When cutaneous anæsthesia exists," he says "it is often easy to learn whether its cause lies in the nerves or in the central organs by following this plan. If the insensible region can be made the point of departure of reflex movements, the anæsthesia is of central birth, because to have reflex motion an excitation must have reached the spine, by which we infer healthy nerves, while the mere presence of anæsthesia will in this case indicate the existence of disease in the spine above the point which is the seat of the reflex power exhibited. If the proof be negative, and if the excitation cause no movement, we can arrive at no definite conclusion until, following the same sensitive nerve up the limb, and by seeking to excite it through its reflex acts, by touching the skin with a hot sponge or ice at successive points, we learn if at any upper portion of the tegument we can produce this result. Should we get an affirmative reply, we may presume that the anæsthesia is of peripheral origin. When, finally, the answer is negative up to the spine itself we learn nothing by this method, the total absence of all reflex move-

ment being equally compatible with either loss of conducting power in the peripheral trunks and branches or with extensive alteration of spinal centres. The value of this means must be necessarily limited by the difficulty of exciting reflex acts from all regions of the skin." (*Injuries of Nerves.*)

Of another point which has, I think, but limited application, or is of doubtful value, Mitchell writes as follows:

"There is another peculiarity which separates all extra-central nerve lesions from cerebral, and also from spinal disease; but I do not feel that as yet it is available to any large extent. I noticed some years ago that in even the gravest lesions of nerve trunks, if a touch were felt at all, it was felt with no remarkable delay; while in many central palsies, if severe, and especially in such as result from extensive spinal malady, the time required for transmission to the sensorium was, as Cruveilhier pointed out, very largely increased—so much so, indeed, as to be readily estimated in a rough way by the hand of a watch beating quarter seconds, or still better by a metronome. The cause of this difference is still obscure to me, nor is it easy to see why diffuse sclerosis, for example, should so retard a sensory impression, while injured nerve fibres have no such effect."

Certain intra-pelvic neuralgias must be distinguished from neuritis or other lesions of the sacral plexus and intra-pelvic nerves. It is a question whether some of the cases reported as examples of neuralgia have not been rather forms of neuritis. Mitchell,[1] under the name of "Anal and Perineal Neuralgia," reported several interesting cases of this kind. This disorder, as described by him, is a painful affection of the anal and perineal regions, accompanied or not, as the case may be, with spasmodic contraction of the anal muscles and of those of the perineum. It is met with in locomotor ataxia, and also as an isolated affection usually following masturbation or sexual intercourse. It is likely that in these cases examination by the rectum

[1] Philada. Med. Times, vol. iii., July 19, 1883, p. 659.

would show tenderness over the anal and perineal nerves. Treatment by rest and anodyne suppositories is most useful, and points also to the probable neuritic character of the affection.

LESIONS OF THE LUMBAR PLEXUS.

One of the commonest and most misleading affections involving the lumbar plexus is that compound lesion to which I have already referred, usually described under the comprehensive term psoas abscess. Eleven years ago I called attention to the frequent simulation by it of certain special forms of nervous affection, painful, paralytic, and spasmodic;[1] and, of course, the subject has been frequently written about, both before and since. Still, mistakes are constantly being made, and the subject is of considerable importance to neurologists. In one of my cases the diagnosis of crural neuralgia had been made, and no abscess was suspected. Pain most severe distally extended from Poupart's ligament to the inner side of the knee, along the course of the anterior crural nerve and its internal saphenous branch. This pain was relieved by sitting, or lying, or flexing the thigh on the pelvis, at the same time keeping the knee bent. A partial paralysis, or pseudo-paralysis, of the ilio-psoas muscles was present, the patient being compelled to lift the leg with his hands in order to cross the knee.

In another case on which I made an autopsy, the patient died three years after his initial symptoms, having been, in the meantime, the victim of numerous and diverse diagnoses, such as lumbago, sciatica, rheumatism, and paralysis from spinal exhaustion. His first symptom was severe pain in the lumbar region, which later extended down the right hip, and still later down the inner aspect of the

[1] Trans. Med. Soc. Pennsylvania, 1878.

thigh. He became unable to walk without support in eighteen months. It is not necessary to describe his progressive symptoms in detail. Later, he carried his body flexed upon the thigh, and both legs were wasted, the right more than the left. A significant fact was the absence of implication of the bladder and rectum, showing that the ano-vesical nerves, which arise from the conus and are distributed to the sacral rather than to the lumbar plexus, escaped. An accurate localizing study of this case, in its initial period, would probably have indicated its true nature; and operation performed early, after the method recommended by Treves and others, might have saved the life of the patient.

In one historical surgical case,[1] that of President Garfield, a quick appreciation of the symptoms produced by irritative lesions of separate cords of the lumbar plexus, and of the lumbo-sacral roots, was of considerable service in reaching a conclusion as to the exact course taken by the bullet. This case is also probably a study in intra-spinal nerve-root localization and worthy in this connection of our passing attention. Studying its abundant literature I find somewhat differing statements as to the nervous symptoms presented. The question whether the subjective symptoms presented sufficient facts for diagnosis was argued pro and con. with much warmth in some of the medical journals. One report states that the President suffered pains of a symmetrical character in both feet; another that hyperæsthesia was always more severe in the right than in the left foot and extended up the leg to some distance, and it was far more intense upon the dorsal

[1] Among the contributions discussing this case are the following: Official Report—Amer. Journ. Med. Sci., Oct. 1888; White—MED. NEWS, vol. xl., No. 25, June 24, 1882; Bliss—Med. Rec., Oct. 8, 1881; Walsh—Walsh's Retrospect, Oct. 1888; Baker—MED. NEWS, vol. xli., No. 5, July 29, 1882.

than upon the plantar surface. The different contributors to the controversy all, however, agreed that hyperæsthesia was present on the right side of the scrotum, differing only as to whether it also extended over the lower abdominal region. The pointing was clearly to injury of the right ilio-inguinal, and probably also of the right ilio-hypogastric nerve, or their common trunk, as these nerves are both derived from the first lumbar root, and as a frequent position for this root is almost exactly where the ball entered the lumbar vertebra.

The ground has been taken that the cause of the symmetrical, or at least bilateral, pains in the feet was central, and therefore due to a lesion by jarring or concussion of the spinal centres opposite or near the vertebra perforated by the bullet. The terrific jarring of the spinal column by the concussion of the bullet was amply sufficient to have caused minute extravasations at the origins of the last lumbar and upper sacral nerves, which may have so irritated the roots of these nerves as to have caused bilateral pains in the distal portions of the lower extremities. The sensory nerves for the feet on both the posterior and anterior surfaces are derived from the sacral plexus; with the exception of the long or internal saphenous nerve, a branch of the anterior crural derived from the third and fourth lumbar roots. Within the spinal canal the nerves which go to form both the lumbar and sacral plexuses arise in a vertical extent of the cord which is very short, and is mainly opposite to the last dorsal and first lumbar vertebræ. It is significant that the roots of the first, second, and third sacral nerves are almost exactly opposite the upper part of the first lumbar vertebra where it was perforated by the bullet. The phenomena presented seem to admit of no other reasonable explanation. A crushing or destructive lesion of the lumbo-sacral region of the spinal cord

was out of the question, as such a condition of paralysis, atrophy, anæsthesia, vaso-motor and trophic disturbances, and interference with the functions of the bladder and rectum, would have been present as could not have deceived.

True lumbo-abdominal neuralgia is comparatively rare, and, therefore, in cases of lumbo-abdominal pain, whether joined with crural pain or not, we should carefully inquire for local lesions of the lumbar plexus, as aneurisms, abscesses, neuritis, etc. A close study of such a case will often reveal its true nature. Lumbo-abdominal neuralgia, as stated by Erb, is commonly associated with intercostal. The presence of marked motor and anæsthetic disturbances with certain vesical, rectal, and sexual symptoms will help to decide against true neuralgia and in favor of lumbar plexus disease, if the affection is unilateral. Many of the cases of so-called lumbar, sacral, abdominal, lumbo-abdominal, and other forms of plexic neuralgias are in reality cases of neuritis of these plexuses.

Certain affections which involve some of the cords or branches of the lumbar plexus, particularly abscesses—lumbar, iliac, psoas, and perinephritic—have not infrequently been wrongly supposed to be other diseases. Lydston[1] has reported a case in which acute lumbar abscess simulated at its beginning nephritic colic. A young man, aged nineteen, who had been previously healthy, awoke suddenly one morning with a severe pain in his right iliolumbar region, doubtless due to implication of the upper lumbar nerves. This pain had persisted for the entire time prior to his visit, and had been but imperfectly relieved by morphia. In view of the history and physical condition of this case, a diagnosis of renal calculus—probably impacted in the

[1] Medical Register, vol. iv., September 8, 1888.

ureter—was made, as, according to the patient's statements, the location of the pain had gradually descended toward the right iliac fossa. The usual line of treatment for the condition which was supposed to exist was ordered, but later a large abscess pointed in the back to the right of the spine, between the ribs and the crest of the ilium.

An available knowledge of the segmental distribution of sensory nerves in the trunk and extremities may be of considerable service in diagnosticating lesions of the sacral and lumbar plexuses, even at an early period. Pain, anæsthesia, and paræsthesia are often experienced in these cases in special peripheral areas, as illustrated in the case of President Garfield, and in the pelvic tumor reported by Dr. Putnam, to which reference has been made. In Putnam's case the first and a recurring symptom was pain in the middle toe of the left foot, and subsequently, other symptoms were paræsthesia of the dorsum of the foot, and later of the outer side of the leg and the posterior aspect of the leg and thigh. These symptoms definitely indicated involvement of sacral nerve cords. The only portion of the foot which escaped was the inner side and great toe supplied by the long saphenous nerve, a branch of the lumbar plexus. The sensory symptoms and the particular order of their occurrence fairly indicated the particular method of the progressive involvement of the nerve cords and branches of the sacral plexus. Pain in cases of lesion of the nerve roots, either in their intra-spinal course or in the plexuses, is often projected to distal portions of the sensory nerves, and a close study of the distribution and progress of this pain will lead to a diagnosis of the location of the lesion producing it.

The question of operation for certain lesions of both the sacral and lumbar plexuses is one of some practical interest. It is probable that trephining the

pelvis could be performed successfully in a few cases for lesions of the sacral plexus, such as abscess and removal of intra-pelvic growths, or excision of parts or the whole of the plexus. This could, probably, be best done by entering the pelvis from the median line above and behind; but I leave the question of the best place and method of operation for the consideration of surgical anatomists, simply suggesting its performance for some of the lesions discussed in this paper. In gynecological surgery several intra-pelvic operations by the sacro-coccygeal route have recently been reported; and this plan of operation has received the indorsement of distinguished German surgeons. It has been employed for pelvic abscess, for disease of the ovaries and Fallopian tubes, and for extirpation of the uterus. The lumbar plexus, in part of its course, can be reached by operation through the back.