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Further Investigations as to the Existence of a Cortical Motor Center for the Human Larynx.

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

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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).

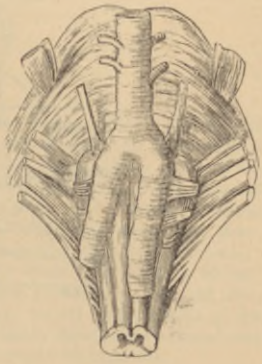

Fig. 1.

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

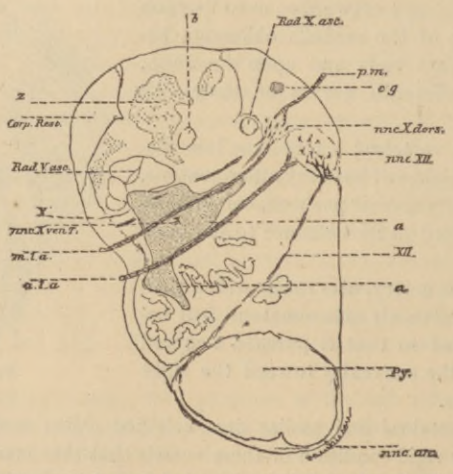

FIG. 2.—Section of the medulla near the lower level of the wedge of softening. *a*, triangular softened area; *nuc. arc.*, nucleus arciformis; *py.*, left pyramid; *xii*, hypoglossal nerve; *nuc. xii*, hypoglossal nucleus; *c. g.*, column of gelatinous substance of Rolando; *rad. x asc.*, ascending root 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; *ε*, common sensory nucleus of ix and x nerves; 8, 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 Giesen, 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.

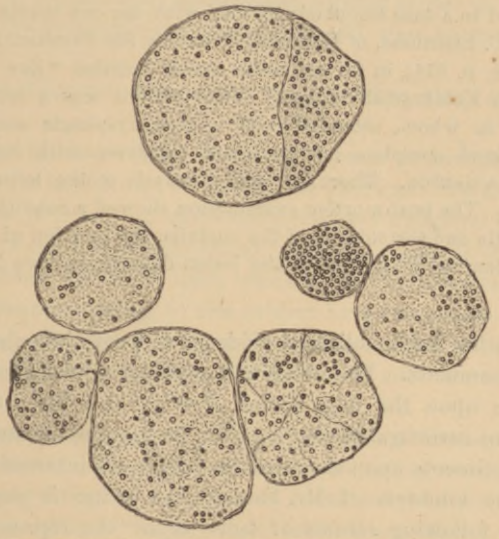

FIG. 4.—Transections of left vagus root-bundles, showing the degenerated areas.

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.