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THE CORTICAL MOTOR LARYNGEAL CENTRE,
AND THE INTRA-CEREBRAL FIBRES WHICH
PROCEED FROM IT.

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IN May, 1886, we communicated to the French Otological and Laryngological Society¹ a case, unique of its kind, of unilateral paralysis of the larynx, diagnosed during life as of cerebral origin. Autopsy confirmed the diagnosis, and we were able to assign to the motor cortical laryngeal centre a pretty well-defined situation. This case supported the ideas held as to the existence and situation of this centre. Since that time we have in vain looked for any new case which would allow the confirmation of our preceding conclusions. In fact, the publication of the first memoir aroused objections. Semon and Horsley,² basing their opinions upon their experiments, clearly declared that our conclusions were erroneous, and that we had certainly overlooked lesions situated deeper. Gottstein³ contented himself with reproducing this criticism of our case. Until now we have not thought proper to answer such an objection. We were, however, confident of the minute care with which we had performed so important an autopsy. On the other hand, the refutation of clinical observation by experiment has only a restricted value. A long time ago, Charcot remarked that in the matter of cerebral localization it is necessary to keep to the facts furnished by clinical observation and pathological anatomy. The numerous contradictions of various experimenters is sufficient proof of the truth of this remark. In the present case it will be easy to demonstrate the exact value of the former

¹ Garel: "Annales des Maladies du Larynx," etc., May, 1886.

² Semon and Horsley: "Brit. Med. Journ.," August, 1886.

³ Gottstein: "Die in Zusammenhange mit den Organischen Erkrankungen des Centralnervensystems stehenden Kehlkopfaffectionen," Wien., 1888.

and recent experiments of Seimon and Horsley. Other experimenters, equally distinguished, have obtained quite different results. In any case, experimentation may be criticised—a clinical fact, on the contrary, is stubborn and decisive. We are now in possession of a fresh clinical fact, which, if not similar to the first one published by us, is nevertheless a fresh confirmation of our former statement. It is a case of unilateral paralysis of the larynx, resulting from a cerebral embolism of small importance, situated at the level of the lenticular nucleus, and having ruptured the internal capsule at the level of the knee of the capsule. We have thought it best to compare this case with our first one, each case completing the other, and permitting the establishment of two important landmarks in the cerebral topography of the laryngeal motor fibres.

CASE I.

Apoplexy. Right hemiplegia and aphasia from atheroma of the cerebral arteries, determining multiple foci in the left hemisphere. Complete paralysis of the left vocal cord, due to a lesion of the right hemisphere, localised in the probable seat of the cortical centre of the larynx.

Anne Tutel, aged seventy-two, came under observation on January 9, without any information as to her antecedents. Two days before she was attacked with apoplexy, with loss of consciousness, right hemiplegia, facial paralysis of the same side, and aphasia. The right hemiplegia affected also the upper limb. Sensibility intact. Facial paralysis of the same side. Feebleness of heart sounds, but no murmur. Some scattered râles in the lungs. Very marked elevation of temperature. On January 10, complete paralysis of the right arm, with epileptiform convulsion of the thumb and fingers. Incomplete paralysis of the right leg. Three or four erosions or phlyctenulæ existed on the right buttock. The patient understood what was said to her, but could only answer by a grunt somewhat resembling a “oui.” On January 12, five days after the attack, laryngoscopic examination was practised, in order to determine whether the right vocal cord participated in the hemiplegia of the same side. To my great astonishment, I found that it was the *left vocal cord*, which was paralysed in the cadaveric position. The larynx was intact, *i.e.*, presented no special organic alteration. There was no cause assignable for compression of the left recurrent. The state of the patient improved little by little, and the aphasia tended to very slightly disappear. On January 23, the left vocal cord was still paralysed. A second attack supervened during the night of the 25th to 26th Jan. The patient passed into absolute coma, the head being directed to the right side. Death occurred on January 27, at five o'clock in the morning.

Autopsy.—In my first memoir, I have given all details concerning the viscera. There were no important lesions, at least as regards the question which now interests us.

Brain.—Moderate hyperæmia of the meninges at the base and laterally. The basilar artery showed marked signs of endo-arteritis, consisting of almost complete rings, or yellow hard plates of atheromatous and calcareous nature. The same lesions were observed in the posterior, middle, and anterior cerebral arteries. The middle cerebrals or sylvian arteries presented nodules of atheroma in their whole length.

Left hemisphere.—The atheromatous plates ceased at the entrance of the sylvian artery into the fissure of Sylvius. At the first bifurcation of the artery there was a small recent red clot, situated *à cheval* on the spur of the

division. The fissure showed no lesion. A first small nucleus of softening was found between the ascending frontal and the base of the third left frontal. A second focus was found in the fissure of Rolando, situated up to the level of the second frontal. A little higher, on the ascending frontal, two or three very small spots of red softening were discovered. Further away, a focus of a reddish tint. This focus, which was the most extensive, was situated at the level of the second and third frontals, towards the middle third of the ascending parietal. This nucleus was separated and enucleated with the greatest facility, leaving a base slightly altered. It was without connection with the other foci. The foci of the ascending frontal and ascending parietals alone bruised the subcortical white substance a little. Nothing in the central ganglia.

Right hemisphere.—Atheroma extending into the fissure of Sylvius. The inferior part of the ascending frontal, slightly adherent to the meninges. These removed, this part presents a rough surface, sharply differentiated from the smooth aspect of the rest of the cortex; this surface is also slightly yellow-coloured (C). In front, at the foot of the third frontal convolution, two points (A and B) of red

FIG. 1.

softening were found near together, in appearance like those of the opposite hemisphere. No lesions in the anterior parts of the third left frontal. On section these lesions are only little in depth, and only affect the cerebral cortex; in the upper one only there exists a single yellowish lesion involving the white substance. The lesion of the ascending frontal penetrates a little into the white substance, in the fissure which separates it from the third frontal convolution and reunites with the superior nucleus B, forming thus one and the same lesion. No lesion of the bulb, or cerebellum was found.

There was nothing in the neck which could cause compression or thus explain a laryngeal paralysis of peripheral origin. The larynx was nowhere abnormal, and the left crico-arytenoid articulation was not ankylosed.

We will remark that this was not a case of an anatomical condition interpreted *post-mortem* in a fantastic manner, but the exact verification of a diagnosis made during life. From the presence of *left laryngeal hemiplegia* in a patient affected with right complete hemiplegia, and without the least sign of alternating hemiplegia, we had supposed that

there would be a lesion of both cerebral hemispheres. We expected that at the autopsy we should find extensive lesions in the left cerebral hemisphere, explaining the complete right hemiplegia, and in the right hemisphere, a less extensive lesion answering to the very localised paralysis of the left half of the larynx. The facts confirmed our hypothesis. We insist strongly upon the importance of this observation, for this is the only case known up to now of *pure laryngeal monoplegia without any other paralysis of the same side*. It is the only case verified by laryngoscopic diagnosis as of cerebral origin, and confirmed at the autopsy. The value of this observation from the point of view of cerebral localization of the laryngeal centre has been recognised by Massei,⁴ by Lubet-Barbon,⁵ by Jonathan Wright,⁶ etc. We do not understand how Masini,⁷ in his excellent experimental study, accords more importance to the case published by Rébillard, in 1885 (Thèse, Paris). This was the case of a patient in the clinic of Luys, who presented right hemiplegia with paralysis of the right vocal cord. The coincidence of vocal paralysis, with paralysis of the limbs, is an untrustworthy point in regard to localisation, for it involves the existence of extensive lesions. But what makes the case less valuable yet, is the existence of very numerous lesions at the autopsy. We cannot do less than enumerate them in detail, quoting from the author's text :—

"At the *autopsy* the cerebral matter was generally softened, but tolerably consistent. Some adhesions were found. The right frontal lobe contained in its substance a tumour, which adhered to the meninges, and was of opalescent tint."

"*Left frontal lobe.*—Fissure of Rolando, tortuous and irregular. Both frontal convolutions of rosy tint without notable hyperæmia; no adhesion with meninges. The third frontal presented at its foot a very large hæmorrhage, which had destroyed all the grey matter of the convolution. The cortical substance of the left hemisphere was pale, yellowish, with projecting granulations, probably of syphilitic nature. The corpus striatum was invaded by a yellowish mass. The tissue of the optic layers was very indurated, but without appreciable granulation. The cerebellum was small and atrophied, and presented projecting granulations, sensible to touch. The fourth ventricle was dilated and slightly hyperæmic. The pons was a little indurated."

"*Right hemisphere.*—The second and third frontals were destroyed by a tumour of the size of a nut, incorporated with the meninges. A small tumour was also seen on the ascending parietal at the level of the paracentral lobule. This hemisphere appeared to have suffered most. The same granulations were found as on the other hemisphere. The centrum ovale was softened."

Such is the case which Masini considers the most favourable for precise localisation of the cortical motor centre of the larynx. The reader may form his own opinion of the value of this assertion. In our opinion the case of Rébillard proves only one thing, viz., that there really exist cases of hemiplegia of the larynx along with a hemiplegia of the limbs, and that

⁴ Massei: "Sopra un Caso di Paralisi Laringea per Lesione Corticale." "Archiv. Ital. di Laring.," 1887.

⁵ Lubet-Barbon: "Etude sur les Paralysies des Muscles du Larynx." "Th. Inaug.," Paris, 1887.

⁶ Jonathan Wright: "Two Cases of Laryngeal Paralysis." "New York Med. Jour.," Sept., 1889.

⁷ Masini: "Sui Centri Motori Corticali Della Laringe." "Archiv. Ital. di Laring.," 1888.

this hemiplegia may depend upon lesions of the brain. It proves also that concomitant laryngeal hemiplegia may occur from a special cerebral lesion, since numerous cases of hemiplegia occur in which laryngeal paralysis is absent. This case and ours are also the only ones in which laryngeal paralysis has been stated with the laryngoscope during life, and in which an autopsy has been made. Masini in the clinical part of his memoir passes in review all other observations known till then, but only having a secondary value. In a first category, he cites the observations of vocal affection from cerebral lesions without laryngoscopic examination, but with autopsy. He found five such observations :—

1. The two well-known cases of d'Ange Duval (traumatic origin).
2. The first case of Luys. ("Annales des Mal. du Larynx," etc., 1875.)
3. The case of Livio Ronci (aphonia after apoplexy). A new case well detailed.
4. The case of Seguin.

In all these cases the absence of laryngoscopic examination is a capital defect. No conclusion is possible.

More interesting is the second category of hemiplegias from cerebral lesions without autopsy, in which has been found laryngoscopically a unilateral laryngeal paralysis. Masini has only collected five observations.³

1. Lewin : "Case of Paralysis of the Left Vocal Cord of Central Origin. Left Hemiplegia" ("Berl. Klin. Woch.," 1874).
2. Bryson Delavan : "Note on the Localisation of the Cortical Motor Centre of the Larynx" ("Med. Record," Feb., 1885).
3. Cartaz : "Observation from the Clinic of Landouzy at La Charité" ("France Med.," 1885). Right hemiparesis and paralysis of the right cord.
4. Garel : "Right Hemiplegia. Aphasia. Paralysis of the Right Vocal Cord" ("Ann. des Mal. du Larynx," etc., 1886).
5. Garel : "Left Hemiplegia, Paralysis of the Left Vocal Cord" (*loc. cit.*).

Since this time our distinguished confrère, Dr. Massei, has published a new observation of paralysis of the left vocal cord, with left facial paralysis and deviation of the tongue to the same side. The case was not completed by an autopsy. We hesitate greatly to class it with those of the second category which we have just cited, since the diagnosis may be disputable between a bulbar lesion and a cortical-lesion. The case of Bryson Delavan, which made much stir at its announcement in 1885, and which had the merit of bringing the question to the front, ought really to be displaced from the important rank which Masini has given it. Bryson Delavan, whose work was the signal for a series of others (Cartaz, Lannois, Rébillard, etc.), published in June, 1889 ("New York Med. Jour.") a second note. From this note it results that the unilateral laryngeal paralysis attributed by him to a cortical lesion of the laryngeal motor centre, came from a bulbar lesion. Here is, therefore, a case which re-enters into the well-known category of cases of laryngeal paralysis from bulbar origin. In Delavan's observation, the case was one of a patient attacked with total hemiplegia, facial paralysis, and

³ These cases ought certainly to be more frequent. They will be multiplied when laryngoscopy is practised in a general manner.

unilateral laryngeal paralysis. The hemiplegia of the limbs disappeared, the vocal paralysis remained permanent. It was precisely upon the persistence of this latter that Bryson Delavan rested the affirmation of its cortical origin. Autopsy unhappily did not confirm the diagnosis.

We have thought it best to recall our first observation in order to answer to the criticisms which it raised. It was moreover indispensable for this study, wherein we desire to treat the question in its entirety.

This case in connection with our second more recent case will be the indispensable complement of the latter.

CASE II. (*Original and unpublished.*)

Ulcerative endocarditis and pericarditis. Pulmonary infarcts. Paralysis of the left vocal cord from a small cerebral embolism localised in the corpus striatum, and involving the internal capsule.

Case reported by M. L. DOR, interne.

Henri F., thirty-five years of age, a labourer, entered the "Hôpital de la Croix-Roussi," Salle Saint-Nizier, No. 14, on October 8, 1889. His father is still living and enjoys perfect health. His mother died in 1875, after an illness lasting three days. He has two brothers and a sister in good health. A brother died from pleurisy at twenty-eight. In 1874 the patient had an attack of acute articular rheumatism of great severity. All the articulations were successively involved with the exception of the coxo-femoral, temporo-maxillary and articulations of the vertebral column. He, however, experienced great pain in the cervical spine at the level of the fourth articulation. This rheumatic attack was followed by working in water for nine months day and night. The patient has never had rheumatic symptoms since this date. He has been marvellously well, and never has had dyspnoea, palpitation, or oedema of the lower limbs. On December 12, 1888, he received an accident. An iron point having penetrated his finger, occasioned phlegmonous inflammation of his right arm. Incisions and counter openings were made, of which the traces yet remain. He was treated at "l'Hôtel-Dieu," of Lyons, under Prof. L. Trippier, who proposed to amputate the finger, but the patient refused, and left the hospital at the end of twelve days. From January to June, 1889, it supplicated pretty abundantly, which was treated by some quack by an ointment. Suppuration ceased towards the month of June; but up to then the finger constantly discharged a small quantity of pus. He then had quarrels with his employers, who refused to pay him what they had promised, and engaged in legal proceedings, which were only just commenced at the time of his death. The trouble which was caused to him, by finding himself without the means of existence, prevented him from sleeping and from taking nourishment. He succumbed then rapidly. On Sunday, October 6, he had a slight rigor with headache, and went to lie down. He coughed all night. Next day he went to a physician, who advised him to enter a hospital. On the day of his admission, *i.e.*, October 8, he appeared to be thin and weak. He had a brown tint and red cheeks. He coughed, and had great oppression. Respiration was short, and 48 to the minute. For two days he had coughed rusty or rather reddish expectoration. On examination of the chest dulness of the right apex was found before and behind. In front, behind, and in the axilla, respiration was very rough; and in front, at a very limited spot, a little *souffle* and bronchophony existed. Vibrations were slightly exaggerated. The rest of the lungs presented nothing abnormal. Temperature on the evening of admission did not exceed 38°. The urine contained a very large quantity of albumen. The heart's beat was

under the sixth rib, outside the nipple. The heart could be heard to beat at a distance, with the ear more than two centimètres from the thorax. On auscultation, rubbings and gratings of considerable intensity were heard. On palpation very sensible tremors were felt. At the apex, a "bruit de gallop," which, along with the pericardial frictions, produced a veritable rhythm like a weaving shuttle. On placing the ear well to the right, outside the pericardium, the existence of a systolic souffle was recognised, and of a diastolic souffle at the base. There was no cardiac irregularity.

On Oct. 9.—Slight cedema of the ankles and of both pulmonary bases. Champagne, diuretic wine, and two litres of milk were prescribed.

Oct. 10.—Slight raucity of voice was noticed, to which no attention had been paid yet, and the laryngoscopic examination was remitted to the morrow.

Oct. 11.—The temperature, which during the preceding two days had oscillated between 37.2 and 37.5, fell this morning to 36.8, and rose in the evening to 37.4. Laryngoscopic examination was performed, and we were astonished to find *complete paralysis of the left vocal cord*. The cord was in the cadaveric position, a little separated from the mid-line; it was not tense, its free edge being slightly concave. Greatly puzzled by this paralysis, the patient was asked when he developed any affection of the voice. He then replied that during the night of October 6-7, feeling ill in the middle of the night, he wished to call one of his parents who slept in the neighbouring room. He could not emit any sound, and had to strike upon the partition dividing the rooms as a signal for assistance. We practised a more thorough laryngoscopic examination, and proved that the right vocal cord was absolutely healthy; its movements were normal. During phonation it came to the mid-line without crossing over it, and left an open glottis. The glottic chink thus imperfectly closed, presented a rectilinear line on the right side and a slightly elliptical outline on the left. We examined with the greatest care the mobility of the arch of the palate and the tongue, and found nothing abnormal. Nothing abnormal was found on the part of the face or eyes. No paralysis and no anæsthesia of the upper or lower limbs. We therefore had to deal with a case of purely laryngeal hemiplegia. We made a diagnosis of cerebral embolism, involving the cortical motor centre of the larynx, in face of the pulmonary affection which we had regarded as of embolic origin, the presence of ulcerative endocarditis, and the absence of any cause of compression of the recurrent in the neck. We eliminated bulbar embolism, not seeing how an embolism, even when localised in this region, could produce a laryngeal hemiplegia without involving at the same time the nuclei of origin of other cranial nerves.

Oct. 12.—Temp. 36° 8 in the morning, and 37° 4 in the evening. We found the patient with a face not only terrified but cyanosed. There was very marked cooling of the upper and lower extremities; the index and middle fingers were violet and completely cold, in a symmetrical fashion; the two great toes presented an asphyxiative tint, and on their lower surface was symmetrical ulceration surrounded with violet areola. There was, however, no anæsthesia of the extremities. Since yesterday the patient had abundant diarrhœa, which, however, diminished a little this morning.

Oct. 13.—Morning and evening temperature did not go beyond 36° 8. The cyanosis of the extremities had disappeared; the patient's hands and feet were very cold, but the violet appearance no longer existed. The diarrhœa had also stopped.

Oct. 14.—Reappearance of a slight degree of cyanosis of the extremities, localised to the same fingers as before. Resp. 48, pulse 88, hypothermia persisted at 36° 8. The red sputa had disappeared, also the souffle and dulness. There

was no longer any oedema of the pulmonary bases. To the pericardial frictions were added pleural frictions, and when the cardiac apex was ausculted the intermixture of these sounds was heard, some coinciding with respiration and stopping with it, others coinciding with the cardiac pulsations. The urine always contained a considerable quantity of albumen, was greenish in reflection, clear and frothy. There was a deposit which, microscopically examined, enclosed many casts of all dimensions, granular, hyaline, and epithelial. Tincture of iodine coloured them only yellow. A certain number of large casts were easily coloured brown mahogany by iodated tincture of iodine. Some spermatozoa and white corpuscles were present. Laryngoscopic examination gave the same results as before, and no new paralysis was added to the other symptoms. Upon ophthalmoscopic examination it was found that the papillæ were normal. They, however, presented a white colouration, recalling that of atrophy. The rest of the fundus showed nothing particular. No peri-papillary oedema.

The patient appeared very feeble this morning, and his aspect became more and more cachectic. In the evening, he had moments of obfuscation, and when spoken to did not answer. Respiration was panting, and the *alæ nasi* expanded with each inspiration. Respiration : 48 a minute.

On ausculting the heart, the souffle was heard better at the apex than at the base. There was no femoral double souffle. At ten o'clock at night, the patient fell into a comatose condition, and died at eleven.

Autopsy.—Thirty-six hours after death. We must remark at first that examination of the brain was commenced with, from the certainty we had of finding a lesion in this organ confirming our diagnosis, *i.e.*, explaining the laryngeal hemiplegia noticed during life. We shall see the details of the lesions of this organ at the end of the description.

The heart was very hypertrophied, being many pounds weight heavier than normal. The pericardium contained about 100 grammes of yellow liquid, and a deposit of white and thick false membrane. Similar membrane, rugose, were dispersed on the whole visceral and parietal surfaces of the serous membrane. A number of small bud-like granulations of vegetating endocarditis existed on the mitral valves. The valve was rendered incompetent only in consequence of the irregular nature of its edges in consequence of the vegetations above mentioned. The chordæ and papillary muscles were of normal aspect, the neo-formations existing only at the base of the valves.

The tricuspid valve was absolutely normal. The sigmoid valves of the aorta presented a notable thinning of their free extremity, which rendered them very transparent. One was completely fenestrated, the free edge representing a thin ribbon. Below this was a reticulated and very friable fenestrated tissue. This all contributed to render the aortic valve insufficient, and it was also incapable of keeping the water turned in above its orifice. The lungs inclosed no great infarcts, but, principally in the right lung, spots were found of more dense non-aerated greyish substance, and some fragments cut out fell directly to the bottom of the water. The liver and spleen presented nothing special, except a slight augmentation of volume. The kidneys had a very remarkable marbled aspect, not permitting the distinction of the cortical zone especially from the rest of the kidney. Section presented a uniformly whitish aspect, and an innumerable series of deep red points were seen, almost black, forming a kind of depression in the middle of the white tissue. They were in fact small numerous infarcts. The white substance was affected with amyloid degeneration, as was proved by the iodine reaction. No tumour was found on the left side of the neck which could cause compression of the recurrent. The larynx itself appeared perfectly healthy.

The two cords were of course in cadaveric position, and nothing could account for the paralysis noticed during life. No ankylosis of the left arytenoid existed. The two vocal cords presented the same dimensions. We dissected the recurrent nerve with care, and placed it immediately in osmic acid. Microscopic examination was negative. The nerve presented no sign of degeneration.

We pass now to the examination of the brain. The encephalon presented nothing remarkable externally. The bulb was separated at the level of the peduncles. The floor of the fourth ventricle was absolutely normal. Different bulbar sections proved that not the smallest lesion of these parts existed. The peduncles were also healthy. The cerebellum presented nothing particular. We exposed the cerebral cortex of both hemispheres, and to our great disappointment found not the least alteration of the frontal convolutions, either right or left. We then believed ourselves to have made faulty diagnosis. We, however, cut the left hemisphere first in slices which presented no lesion. We expected this since the laryngeal lesion was on the left. We then cut the right hemisphere, keeping the sections closer together, without following the classical oblique

FIG. 2.

sections of Pitres. Our sections were all made nearly perpendicularly. We then discovered a small lesion starting above, from the foot of the first frontal convolution, traversing the middle of the ascending frontal and the inferior part of the ascending parietal. Figure 2 gives an idea of this section. We have represented the left hemisphere although we were dealing with the right hemisphere, but it is to compare more exactly with the classical sections of Pitres figured on the right hemisphere. The reader in comparing Fig. 2 with the plate of Pitres will remember that our section commenced above at the level of the superior portion of second pediculo-frontal section of Pitres, and terminated below at the level of the inferior point of the section called parietal, of the same author. In the section thus determined we found a small area of red softening, of recent date, as large as a pea at its upper part, and terminating in a point below. The total length of this was estimated at about 15 millimètres from top to bottom. Its greatest upper extremity was only about 8 millimètres. In this area the nervous substance was completely disorganised, and had the appearance of red pulp. This area of softening was situated in the superior internal part of the lenticular nucleus (Fig. 3). It encroached slightly from one to two

millimètres upon the external part of the internal capsule, but only in the upper half of the lesion. We desired then to follow exactly the nature of the lesion by a horizontal section analogous to that of Flechsig. We had some trouble at first to establish the bearings, the anterior part of the brain being reduced into multiple sections, but as the posterior part of the hemisphere remained entire, we had all that was necessary to indicate the seat of the lesion by horizontal section. Fig. 2 indicates the direction we gave to our horizontal section. We have, moreover, by many trials upon other brains, verified the exactitude of our section, vertical and horizontal.⁹

Fig. 4 shows the nucleus of softening in the lenticular nucleus up to the level of the knee of the internal capsule, involving thus even the most external fibres of the internal capsule.

We had, therefore, a fact of extreme rarity, from which it was possible to draw important conclusions as to the intra-cerebral path of the motor laryngeal fibres. We will repeat here what we have already said upon the subject of the first observation, viz., that we will insist upon the existence of *pure laryngeal hemiplegia* without any other kind of paralysis, either of limbs, face, or arch of the palate. Circumstances occurred, so that by accident the motor fibres of the larynx only became involved. As we have before remarked, this case has not been only an anatomical work, but the verification of a diagnosis made during life, thanks to the laryngoscopic examination. Without doubt, we committed an error in thinking of a cortical lesion, but we were right in affirming a lesion of the opposite hemisphere, and absolutely rejecting that of a bulbar lesion. We do not regret this, since it discloses a new horizon, and permits us to open up an entirely new chapter upon the path of the intra-cerebral motor laryngeal fibres. Indeed, just as, theoretically, it has been thought that the laryngeal motor centre ought to exist in the neighbourhood of the third frontal convolution near the other centres of

FIG. 3.

FIG. 4.

⁹ We are indebted for the drawing of the plates to M. Bonnet, interne des hôpitaux.

language and speech, so it is right to conclude that the motor fibres of the larynx ought to occupy in the path through the internal capsule a situation analogous to the fibres which emanate from the before-mentioned centres. It is necessary here to review shortly the knowledge acquired as to the anatomical structure of the knee of the internal capsule. M. Grasset in his last edition (1886) has set forth these anatomical data in a clear and precise manner in his chapter on the semiological value of paralyses of the tongue. He avails himself of the work of Raymond and Artaud,¹⁰ relative to the intra-cerebral path of the hypoglossal nerve, and of the memoir of my excellent and learned teacher, M. Lépine, treating of a labio-glosso laryngeal syndroma of cerebral origin.¹¹ We will follow from the cerebral cortex the two fasciculi actually known up to the protuberance. In the cortex, according to their ten observations of glossoplegia of cerebral origin, Raymond and Artaud localise the hypoglossal centre in the foot of the ascending frontal, whilst the aphasic centre occupies the foot of the third frontal convolution. In the centrum ovale the path of the fibres furnished from these two centres is well known. It results from the case of pseudo bulbar glosso-labio-laryngeal paralysis of M. Lépine, and the cases of MM. Raymond and Artaud, that the fibres of the hypoglossal, of the inferior facial, and of the motor branch of the trigeminal, arise in the inferior frontal fasciculus of Pitres, whilst the conductors of speech are situated in the inferior pediculo-frontal fasciculus. At the internal capsule, in the horizontal section of Flechsig, we leave aside the posterior and anterior segments, occupying ourselves only with the knee of the capsule—that is, the geniculate fasciculus. According to the earlier work of Brissaud, this fasciculus contains the fibres for speech, the motor nerves of the tongue, of the face, and the palatine vault. Raymond and Artaud have gone a step further, and divided the geniculate fasciculus into two parts—the anterior, or fasciculus of aphasia, and the posterior, or hypoglossal fasciculus.

In the cerebral peduncle Brissaud assigns to the geniculate fasciculus a place upon the lower surface, between the middle and internal thirds. Raymond and Artaud differentiate here, the geniculate fasciculus into two distinct parts, making the fasciculus of aphasia pass in contact with the internal third of the peduncle, and the hypoglossal fasciculus with the middle third. But this peduncular localisation is not based upon any clinical proof. The pathway is unknown through the protuberance. Raymond and Artaud think that the fasciculi of the hypoglossal are directed towards the posterior and internal part of the pyramids. From thence they cross and reach the bulbar nuclei. With these data we may examine the indications resulting from the autopsy of our second case. Doubtless we cannot deduce from this complete data upon the total intra-cerebral pathway of the motor laryngeal fasciculus, but we have precise particulars of the most important part, viz., the path at the level of the

¹⁰ Raymond and Artaud: "Archives de Neurologie," tom. vii., 1884.

¹¹ Lépine: "Note sur la Paralyse Glosso-Labiale Cérébrale à forme Pseudo-Bulbaire." "Rév. Mens. de Méd. et de Chir.," 1877.

knee of the internal capsule. If we refer to the description of the lesion found at the autopsy we see that the most external capsular fibres of the geniculate fasciculus were damaged by the pathological focus. The belief may then be permitted that besides the fasciculus of aphasia and the hypoglossal fasciculus, there exists in the most external part of the knee of the internal capsule a definite motor laryngeal fasciculus ; absolutely distinct and having its special place in the constitution of the geniculate fasciculus. Whether the fibres of the motor laryngeal fasciculus may have been involved and damaged by the focus or whether they yielded to a neighbouring influence, the complete autonomy of the laryngeal motor fasciculus remains an acquired fact in the present case. It is difficult to determine the question of a lesion of the lenticular nucleus. According to Grasset it is probable that the fibres for speech below the inferior pediculo-frontal fasciculus pass through the corpus striatum. Is it the same for the laryngeal motor fasciculus? We are not in a position to answer this question. We are persuaded that this observation will arouse objections and receive various interpretations. But one point is indisputable, viz., that this limited lesion determined a *pure laryngeal hemiplegia* without the least paralysis of any other muscular group. On this account alone, the case is of capital importance in the history of localisations.

In connection with the history of my first observation, it helps to throw a certain light on the complete study of the cortical motor laryngeal centre and the pathway of its fibres. We are contented with having assisted in bringing this question into important prominence. Indeed, the case of Rébillard and our two original observations are the three single facts of laryngeal hemiplegia determined during life with verification upon the cadaver. We have already indicated the little value of Rébillard's case. Bryson Delavan has had occasion to perform an autopsy upon a patient, in whom he had found laryngeal hemiplegia, but he found a bulbar lesion. We reproach him only with one thing—that is, his too hasty conclusion formulated in these terms :—"It would thus result—that the theory sustained by Gottstein (that laryngeal paralysis of central origin is due to a lesion of the bulb, and not the cortex) receives direct confirmation from this fact, and that the localisation of the cortical motor centre for the human larynx remains up to now uncertain." Bryson Delavan quotes as his support the analogous cases of bulbar paralysis published by Eisenlohr, of Hamburg.¹² But this proves nothing, except that there exist laryngeal paralyses, originating oftener from the bulb than the cortex. Jonathan Wright (*loc. cit.*) *à propos* of the case published by himself supports the conclusions of Delavan and Gottstein. As to the observation of cerebral lesions without autopsy, in which laryngeal hemiplegia has been recorded along with total hemiplegia, four only have been precisely studied ; of this number two are the cases of ourselves. In our former work we had mentioned another case of hemiplegia of the larynx, appearing to have a cerebral origin, but this, from the absence of total hemiplegia, had only the restricted value of an hypothesis. We may say as much of the recent case of Massci. There exist other cases cited

¹² Eisenlohr : "Arch. für Psych., Berlin," Vol. XIX., 1888.

by Gerhardt, Mackenzie, etc., but none of them are precise enough to enter into the same category. Our task is not yet finished. If at the commencement of this memoir we have done justice to the objections raised by experimenters, we should be ungrateful not to cite the results of their patient researches. If there are divergences between them it is well to signalize them. We can prove that the results obtained, compared with one another, are favourable to the existence of a cortical motor centre for the larynx. The only question in doubt, moreover, is less the existence of a centre than whether the centre has a unilateral or bilateral action.

Before 1877 all physiologists agreed in placing the centre for phonation in the bulb. It is evident that when the movements should produce not merely a cry but articulated speech, a higher centre must be in action. At least, this is the opinion of Bristowe.¹³ As Rébillard remarks (*loc. cit.*), when a singer varies of his own will the tension of his vocal cords, or passes from the chest voice to falsetto, not only is the bulb in action, but a superior centre commands these difficult movements. Duret (Soc. de Biol., 1879) removed in a dog the region which Ferrier regarded as the centre for movements of the tongue and lips, and found that the dog lost the faculty of barking. In his thesis, in 1878, he cites other facts of the same kind. Soltmann,¹⁴ destroying with hot iron the pre- and post-frontal convolutions in a young dog, found that the result was that the animal could not bark, but emit only cries like a new-born animal. Hermann Krause¹⁵ first combined laryngoscopic examination with direct experiment. My colleague and friend, Lannois,¹⁶ has admirably reviewed the experiments of Krause, who concluded in favour of the existence of a cortical region—the *gyrus præfrontalis*—presiding over the movements of the larynx and pharynx. Semon and Horsley,¹⁷ in 1886, objecting to my first observation, made known the results of their experiments upon various animals. They confirm the conclusion of Krause as to the situation of the laryngeal centre in the *gyrus præfrontalis*. In the monkey this centre corresponds to the centre for language in man, *but it is bilateral and symmetrical*.

According to them, every excitation of one centre alone, determines bilateral action of the vocal cords, and they obtained the same result with either right or left hemisphere. The absence of aphonia in cases of aphasia would prove, according to them, that in man, the phonatory centre has a similar bilateral representation. From whence they conclude that my first case proves nothing. Without wishing to criticise rigorous conclusions drawn from the monkey or rabbit to man, we will reply that the clinical facts are there to refute such an assertion. According to such a theory, there ought never to be a single case of total hemiplegia

¹³ Bristowe: "Lectures on the Pathological Relations of the Voice and Speech." "Brit. Med. Jour.," 1879.

¹⁴ Soltmann: "Experimentelle Studien über die Functionen des Grosshirns der Neugeborenen." (Jahrb. für Kinderh., 1876.)

¹⁵ H. Krause: "Ueber die Beziehungen der Grosshirnrinde zu Kehlkopf und Rachen." (Du Bois Reymond's Archiv., 1884.)

¹⁶ Lannois: "Y-a-t-il un centre cortical du Larynx?" "Rev. de Méd.," 1885.

¹⁷ Semon and Horsley: "Paralysis of Laryngeal Muscles and Cortical Centre for Phonation," "Lancet," 1886.

(limbs and larynx) ; for if the lesion were unilateral, laryngeal hemiplegia would be impossible, if the lesion were bilateral, the larynx would be totally paralysed. But clinical facts prove the contrary. Quite recently the same authors¹⁸ have given under a provisional title the details of more extensive experiments upon the same subject. The *résumé* of their experiments is as follows :—

1. There exists in each hemisphere a centre for the bilateral representation of the adductor movements of the vocal cords. Excitation of parts more or less near to this point produces movements of adduction of the vocal cords less and less complete as the distance from the centre is increased.

We note, *en passant*, that the cat does not yield the same results as the rabbit.

2. It has not been possible to determine in the cortex any centre for abduction of the cords.

3. If one of the two cortical adductor centres be completely destroyed, so that excitation of the neighbouring parts produces no effects upon the larynx, no paralysis of the vocal cords is then observed. If afterwards, the opposite centre is excited there results bilateral adduction of the cords as complete as if the opposite centre was intact. Semon and Horsley conclude from these facts—

(A.) Unilateral excitation produces bilateral action. Then clinically an irritative unilateral affection of the laryngeal centre would produce spasm of the glottis, *i.e.*, bilateral adduction of the cords (*e.g.*, stridulous laryngitis).

(B.) Unilateral destruction of the centre produces no effect. Unilateral paralysis of the larynx from unilateral lesion of one hemisphere is absolutely impossible.

We shall not further follow the authors in quoting other experiments less directly connected with our subject. We only think it proper to quote their too absolute conclusions in order to put them in parallel with the earlier contradictory experiments of Masini. These experiments have been passed over in silence by Semon and Horsley ; they deserve, however, to be known. All the experiments were made under the control of Professor Luciani. According to Masini there is no doubt of the existence of a laryngeal centre ; but in opposition to Krause, who admits a precise and limited centre, he thinks that the centre is essentially diffused. According to him the larynx responds not only to excitations of the circumscribed zone of Krause, but to excitation of the same intensity applied to other motor centres. The fact is so true, according to Masini, that it is impossible to produce the phenomena of glottic paralysis without finding associated, paralysis of other important muscular groups (tongue, arch of palate, lips, limbs).

He has, however, observed isolated paralysis of a cord without affection of other muscles. He moreover admits that every portion of the motor zone of the glottis described by him cannot produce vocal paralysis with the same intensity, and he concedes that the area of

¹⁸ Semon and Horsley : "On the Centre for Motor Innervation of the Larynx." "Internat. Centralb. für Laryngol.," Feb., 1890. "Brit. Med. Journ.," Dec., 1889.

Krause represents the principal nucleus of this centre. Extirpation gave the same results as electric excitation. If only the nucleus of Krause is destroyed, glottic paralysis is less durable than when all the part situated in front of the *sulcus cruciatus* is destroyed. In order to obtain definite paralysis, the *post crucial* region has had to be destroyed, *i.e.*, nearly the whole of the so-called motor region. He adds, besides, that even after total bilateral destruction of the whole of the cortical motor zone, the loss of phonation is not permanent. In order to explain this phenomenon, he admits with Luciani, the existence of phonatory *sub-cortical* nuclei, capable of supplying the loss of the cortical centres. He then studies the question of the crossed or direct unilateral or bilateral relation of the laryngeal centre with the peripheral vocal organ. He bases his opinions on the hypothesis of Luciani and Goltz, who maintain that each half of the brain has bilateral relations with peripheral organs, but always recognises the predominating action of the crossed fibre. His experiments prove that in exciting the laryngeal centre with a very feeble current (scarcely sensible on the tip of the tongue) adduction of the vocal cord of the opposite side is produced. Paralysis of the cord of the opposite side is produced when the centre is destroyed. In order to produce bilateral action of the cords it is necessary to employ stronger currents. Then the vocal cord of the side opposite to the centre excited is put into complete adduction to the middle line, whilst the cord situated on the side corresponding to the centre excited has only an imperfect tendency to approach the mid-line, and this is simply because the direct fibres are less numerous than the crossed fibres.

From his experiments Masini draws the following conclusions :

1. The existence in the dog of a motor glottic centre in the anterior part of the hemispheres.
2. This centre extends to almost all the motor zone, although its focus of greatest intensity is limited to the laryngeal centre of Krause (base of the pre-crucial convolution).
3. The centre is neither isolated nor distinct, but is confounded with the other motor centres ; more intimately with those of the pharynx, tongue, velum, and less intimately with the others.
4. Unilateral lesion originates motor laryngeal affections predominating on the opposite side, and affections of sensibility of the mucous membrane.
5. A bilateral lesion produces a persistent paralysis of movement and sensibility without attaining the degree of absolute paralysis.
6. Besides the laryngeal cortical centres it is necessary to recognise the existence of sub-cortical laryngeal centres, if one desires to account for the perfect compensation of disorders resulting from unilateral ablation, and the absence of complete paralysis after bilateral extirpation.

We may now compare these conclusions with those of Semon and Horsley. It is seen that Masini has arrived at results which permit the comprehension of clinical facts, and explaining at the same time the facts resulting from the experiments of the English authors.

The whole question was to know if a cerebral cortical lesion could give rise to paralysis of one vocal cord of the opposite side. Masini

has proved experimentally that this fact was indisputable. In other experiments in which he has modified the intensity of the stimulus he has obtained the same results as Semon and Horsley—*i.e.*, bilateral action—on excitation or destruction of one centre only. The English authors have, therefore, given an interpretation to their experiments, which, though not erroneous, is incomplete, seeing only one part of the question. They have been entirely wrong in endeavouring on such proof to demolish the data furnished by clinical observation. All the cases of laryngeal hemiplegia of cerebral origin published up to now preserve their value. As to the question of the precise delimitation of the centre to Krause's area, or to a zone more or less extensive (Masini), this is perfectly disputable. It has already been proved that it is not always possible to guard against phenomena of diffusion of electric currents used to produce excitation. There is a vast difference from the precision of a lesion circumscribed accidentally and precise localisation from experimental excitation. It is, moreover, not well to push too far the comparison between animals and man, especially when dealing with an organ calling into action functions quite different in the two species.

Semon and Horsley themselves admit, as we have previously remarked, that they have obtained results quite different upon the cat, monkey, and rabbit. We have thought it right to enter into these details in order to present the complete state of the question. We desired also to demonstrate that experimentation may come to the assistance of clinical observation, but that it ought never to be allowed to supplant it.

We conclude from this work :—

1. There exists a cortical motor centre for the larynx in each cerebral hemisphere.
2. This centre is located at the foot of the third frontal convolution of the fissure which separates it from the ascending frontal.
3. The fibres originating from this centre pass to the level of the external part of the knee of the internal capsule, forming in the geniculate fasciculus, a motor laryngeal fasciculus, independent of the fasciculus for aphasia, and the hypoglossal fasciculus.
4. The laryngeal centre has a crossed action. Its destruction determines total paralysis of the vocal cord of the opposite side (cadaveric position).

A STUDY OF THE DIAGNOSIS AND TREATMENT OF MALIGNANT TUMOURS OF THE NASAL FOSSÆ.¹

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MALIGNANT tumours of the nasal fossæ, under clinical aspects, are so different as to render their treatment singularly variable. Regarding them from a purely operative point of view, they may be divided arti-

¹ Translated from the Memoir in the "Annales des Mal. du Larynx," etc., March, 1890.