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LABIO-GLOSSO-PHARYNGEAL PARALYSIS OF CEREBRAL ORIGIN

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THE first case which I find recorded in medical literature in which a lesion in the hemisphere of the brain gave rise to facial paralysis, and difficulties of articulation and deglutition, closely simulating the symptoms of progressive labio-glosso-pharyngeal paralysis, is the well-known one reported by Magnus,¹ and subsequently quoted by Romberg.²

"The subject was a widow, aged 50 years, who had already passed through two apoplectic attacks, causing loss of speech and paralysis of the left side; the first had supervened during a confinement, after violent vexation and the cessation of the lochia, the second in consequence of her menses being suppressed by a cold. After the latter the paralysis of the extremities was removed, but speech was not restored, as had been the case after the last seizure. The face of the patient was perfectly smooth, without either a furrow or an expression. All the muscles of the face were deprived of voluntary movement. She was neither able to contract her forehead nor her eyebrows, to raise the nostrils, nor to move her cheek and chin.

¹ Müller's 'Archiv,' Jahrgang 1837, p. 258.

² Romberg's 'Manual of the Nervous Diseases of Man.' Sydenh. Soc. Trans., vol. ii. p. 278, 1853.

She was incapable of closing the eyelids voluntarily. When required to do it she used her finger, or looked on the ground, by which the eyeball was directed downwards, the levator tarsi relaxed in its contraction, and the upper eyelids also moved downwards. On the other hand, the eyelids closed perfectly as soon as a sudden movement was made towards the eye of the patient, if she was suddenly directed into a bright light, or when she sneezed. During sleep the eyes were also perfectly closed." The mouth was constantly slightly open, and the saliva ran out, the movements of the jaw were limited and feeble, the tongue was completely paralysed, deglutition was effected with great difficulty, and articulation was so much interfered with that the patient was only able to utter grunting and inarticulate sounds.

The patient died from an attack of cholera, and at the post-mortem examination, conducted by Froriep, a hæmorrhagic cyst, about the size of a small walnut, was found situated "at the external edge of the right hemisphere of the cerebrum, where the anterior lobe adjoins the middle one." The inner surface of the cavity was invested with a yellowish membrane, and two gyri were destroyed by it.

In 1871 Dr. Friedrich Jolly reported a case of multiple sclerosis which "presented during life all the symptoms of progressive bulbar paralysis without any disease being found after death in the nuclei of origin of the nerves of the medulla oblongata and pons."¹ Extensive sclerotic patches were found in the corpus callosum and walls of the lateral ventricles; a small patch was observed in each crusta, while a limited degeneration, evidently descending in its character, could be traced through the pons, medulla oblongata, and lateral columns of the spinal cord; but the pons, with the exceptions just mentioned, and the bulbar nuclei were completely free from disease.

In more recent years Dr. Barlow² has directed particular attention to the fact that the main symptoms of labio-glossopharyngeal paralysis may be caused by lesions situated in the

¹ 'Archiv für Psychiatrie,' Band iii., 1872, p. 772.

² "On a case of Double Hemiplegia, with Cerebral Symmetrical Lesions, by Thomas Barlow, M.D., B.S." 'The British Medical Journal,' vol. ii. p. 103, 1877.

cerebral hemispheres, and the case reported by him possesses all the more significance inasmuch as the lesions discovered were very definite, and localised in the cortex of the brain. The subject was a boy, aged 10 years, suffering from aortic regurgitation, who had had an attack of right hemiplegia with aphasia, from which he made a good recovery. Four months afterwards he had an attack of left hemiplegia with aphasia, and in addition, the muscles of mastication and articulation, and those concerned in the first act of deglutition, were paralysed. The patient died from the results of the aortic disease, and at the autopsy evidence of an embolus was found in both sylvian arteries. A focus of softening, about the size of a shilling, was found in the cortex of each hemisphere, involving the inferior extremity of the ascending frontal, and the posterior extremities of the second and third frontal convolutions.

About the same time, or very soon after the publication of Dr. Barlow's case, M. Lépine¹ wrote an important paper on the subject, and described several cases illustrative of the affection. The first of these was that of a woman, aged 51 years, who entered the Salpêtrière in 1876 under the care of Prof. Charcot, and whose case was reported by M. Oulmont. The patient had in 1871 a slight attack of right hemiplegia associated with some difficulty of speech, from which she made a rapid recovery. Two years afterwards she had an attack of left hemiplegia, accompanied by difficulty of deglutition and salivation, but without loss of consciousness. The prominent symptoms in 1877, when a full report of the case was taken, were great difficulty of deglutition, paresis of the muscles supplied by the inferior branches of the facial nerves on both sides, paresis, with fibrillary contractions, of the muscles of the tongue, an excessive flow of viscid saliva, and feebleness of the muscles of mastication, while the speech was "slow, drawling, difficult, and badly articulated." The patient, however, was able to pronounce the separate vowels and consonants fairly well. The patient had several epileptiform seizures, accompanied by

¹ "Note sur la Paralyse Glosso-Labiale Cérébrale en forme Pseudo-Bulbaire, par R. Lépine." 'Revue Mensuelle de Médecine et de Chirurgie,' tome i. 1877, p. 909.

coma, from which she recovered. She ultimately had a severe apoplectic attack, and died two days subsequently in profound coma. At the autopsy a recent hæmorrhagic focus was found in the left optic thalamus, which communicated with the lateral ventricles, these cavities being filled with blood. An old hæmorrhagic focus (*foyer ocreux*) was found in the right hemisphere occupying the third or external segment, and a smaller focus situated in the second segment of the lenticular nucleus. Similar foci were found in the third and second segments of the lenticular nucleus of the left hemisphere. The internal capsule was not involved in either hemisphere. The medulla oblongata and pons were free from disease.

The second case, also reported by Oulmont, was that of a woman, aged 58 years, who entered the Lariboisière under the care of M. Raynaud. The main symptoms were double facial paralysis limited to the inferior branches of the nerve, paralysis of the tongue, extreme difficulty of deglutition, anarthria, salivation, and great diminution of the reflex of the palate. At the autopsy an old hæmorrhagic focus was found in the posterior extremity of the lenticular nucleus in each hemisphere. Another focus was situated in the white substance of the occipital lobe, and one about the size of a pea, containing milky fluid, was observed in the white substance of the anterior lobe of the left hemisphere near its base and anterior extremity. The third case mentioned by Lépine is the one recorded by Magnus, which has already been quoted; while his fourth case, although in all probability an example of this affection, need not detain us, inasmuch as the diagnosis was not verified by post-mortem examination.

In 1878 an instance of this affection was described by Dr. C. Eisenlohr.¹ The subject was a man, aged 73 years, who for the three years before his death had suffered from gradually increasing weakness of the lower extremities, and progressive difficulty of speech. The patient on admission presented paralysis of the inferior branches of both facial nerves, the articulation of the consonants was indistinct, and the speech had an explosive and monotonous character, but the general movements of the tongue were retained, and deglutition was

¹ 'Archiv für Psychiatric,' Band ix., 1878, p. 43.

unaffected. Motor power was diminished in the upper extremities, while there was great feebleness of the lower extremities, which were seized with tremor on any attempt at walking, or on passive movements of them. At the autopsy a cystic cavity was found in the anterior part of each corpus striatum (lenticular nucleus?) situated immediately beneath the surface of the brain, this portion being depressed and discoloured. A small cyst was also found in the optic thalamus on each side. The pons, medulla oblongata, and spinal cord appeared normal to the naked eye, and no secondary degeneration was found in the spinal cord on microscopic examination. Dr. Broadbent¹ published also in 1878, under the title of "Spurious Aphasia," four cases; and two, if not three, of these appear to have belonged to this category, the fourth being probably an example of apoplectic bulbar paralysis. The affection of speech in one of these cases (left hemiplegia) is thus described: "She answered questions correctly, but very slowly; laryngeal phonation was imperfect, and the voice was a hoarse half-whisper, each syllable requiring a separate effort; the articulation also was indistinct." The absence of a post-mortem examination in Dr. Broadbent's cases renders it unnecessary to enter upon a further analysis of them. Another case of bulbar paralysis of cerebral origin was reported in 1881 by Dr. Kirchhoff.² The subject was a joiner, aged 24 years. The disease was ushered in by an attack of dizziness but without loss of consciousness, during which he suffered from clonic spasms of the extremities, difficulty of speech and of swallowing, salivation, and deviation of the face to the left. At the end of four days these symptoms had completely disappeared. Eight days later he fell on the stairs during an attack of dizziness, but he was able to drag himself towards the bed. He was unable to swallow during the whole of that night, but next morning he regained the power of deglutition. Speech was drawling, the lower lip was pendulous, the saliva flowed over it, and the tongue felt heavy to him. On his admission into the hospital in July 1877, four weeks from the onset of the disease, he complained principally of difficulty of speech.

¹ 'Transactions of the Clinical Society,' vol. xi., 1878, p. 37.

² 'Archiv für Psychiatric,' Band xi., 1881, p. 132.

The naso-labial folds on both sides were not well marked, the patient could not whistle, and the angles of the mouth could only be slightly retracted by a voluntary effort. The tongue could only be protruded 1 cm. beyond the mouth, the speech was drawling, the labial and guttural consonants were pronounced with difficulty, while the lingual consonants were pronounced with comparative freedom. The lips moved but little during articulation. Both eyes could be closed, but during closure there were fibrillary contractions of the muscles of the lids, and the right lid offered less resistance to opening than the left. The patient suffered from mitral stenosis. On the 25th of December the patient had an apoplectic attack with left hemiplegia, and died ten days afterwards, and six months from the onset of the disease. At the autopsy the right sylvian artery was found blocked up by an embolus. The right corpus striatum (caudate nucleus) in its posterior two-thirds was of a yellowish-white colour, turbid, and depressed, so that the grey substance was reduced to a thin and softened layer, which was covered by a thickened ependyma. The subjacent internal capsule was of a grey colour, and semi-translucent. The external third of the lenticular nucleus, the claustrum, external capsule, and island of Reil were softened and porous, but not discoloured. A careful microscopic examination of the medulla oblongata and pons, undertaken by Professor Quinke, gave entirely negative results.

A typical case of bulbar paralysis of cerebral origin has been described by myself.¹ The subject was a man, aged 49 years, admitted to the Manchester Royal Infirmary, November 15th, 1880, under the care of Dr. Leech, who kindly transferred the case to me. Eleven months before admission the patient complained of headache, and it was observed that his speech was thick; six months subsequently he had some kind of seizure, which apparently was not attended by loss of consciousness, but from that time up to the date of his admission his speech became more and more unintelligible. On admission there was loss of facial expression, and the patient could not compress his lips or whistle. He could protrude his tongue, but could not curl the tip towards the nose, or roll it up laterally

¹ 'The Diseases of the Nervous System,' vol. ii. p. 626.

so as to render it tubular. He could pronounce the separate consonants with tolerable distinctness, but his articulation, even of words of two syllables, was so imperfect, that his speech was almost unintelligible. Food collected between the teeth, the power of deglutition was greatly impaired, and the viscid saliva which flowed from the mouth had to be constantly wiped away. The uvula was pendulous, but not distorted, its reflex excitability was greatly diminished, and the fauces and epiglottis could be examined with the point of the finger without provoking a cough. The patient complained of great general weakness, but there was no distinct paralysis of any of the extremities. Death took place on the 10th of December from exhaustion, caused by intractable diarrhoea; the lenticular nucleus of each hemisphere was found post mortem replaced by a well-defined cystic cavity, containing a clear straw-coloured fluid, the internal capsules being apparently uninjured. Microscopic examination failed to detect any evidence of disease in the nerve nuclei of the medulla oblongata, or descending changes in the pyramidal tracts in any part of their course.

As the following two examples of this affection, recently under my observation, have not yet been published, and as they present some special features worthy of notice, I shall take leave to report them in detail. I am indebted to Mr. Harris, one of the house physicians of the Manchester Royal Infirmary, for the notes of the following case.

Henry H., æt. 40 years, was admitted into the Royal Infirmary on May 4, 1881, under the care of Dr. Ross. The patient, although much addicted to alcoholic excesses, enjoyed good health until fifteen months previous to his admission. He then had a "stroke" accompanied by partial unconsciousness, and followed by paralysis of the right upper extremity and loss of speech, the latter of which lasted for several days. The right arm gradually regained strength, and at the end of three weeks from the onset he could express himself without difficulty, although his speech remained "thick" and indistinct. He has recently suffered from dizziness, general debility, palpitation and shortness of breath, and it is for these symptoms that he has sought advice.

Present Condition.—The grasp of the right hand is feebler than that of the left, and the tendon reflexes are slightly increased in the right arm and hand. The right naso-labial fold is obliterated, and there is a decided loss of expression on the right side of the face, the difference between the two sides becoming more marked when the patient smiles. There is no deviation of the tongue on protrusion. The patient never appears to be at a loss for the names of objects, or for the proper word whereby to express his meaning, but his utterance is thick and indistinct. He can pronounce the separate vowels and consonants with tolerable distinctness, but his articulation of words of two or more syllables is so wanting in clearness that the patient has some difficulty in making himself understood. There is also some difficulty of deglutition, and fluids find their way into the glottis and cause distressing cough. The action of the heart is forcible, the left ventricle is hypertrophied, and the urine contains a small quantity of albumen. The patient was discharged on May 12, but was readmitted on June 16, on account of a gradually increasing paralysis of the lower extremities. Every form of cutaneous sensibility is diminished in the lower half of the body as far up as the area of distribution of the 7th or 8th pair of dorsal nerves, the sense of temperature being entirely abolished. The lower extremities are extended, the feet are in the position of equinovarus, the patellar tendon reflex is increased, and ankle clonus can be readily elicited in both the lower extremities. There is no deformity or marked tenderness of the vertebral column. The patient has slight dribbling of urine, and the skin over the sacrum is erythematous, but not yet ulcerated. The cerebral symptoms have not undergone any perceptible alteration since last report.

August 7.—Yesterday morning the patient had several distinct rigors and his temperature rose, and now stands at 102.6 F. He complains of pain across the lower part of the chest. About an hour ago severe dyspnoea supervened; the patient's lips are now livid, the breathing is accompanied by a guttural stertor, and although the patient is quite conscious his articulation is so indistinct that his speech is nearly unintelligible. The breathing is entirely abdominal, and loud

bubbling râles are heard over both sides of the chest, but the patient is too ill and helpless to permit of a careful examination of the chest posteriorly.

August 15.—The symptoms noticed in the last report continued with slight fluctuations, and the patient died somewhat suddenly to-day, apparently from asphyxia.

Post mortem.—The post-mortem examination was conducted by Mr. Harris ten hours after death. The brain presented nothing unusual externally. The anterior part of the lenticular nucleus of the left hemisphere presented two small cyst-like cavities, containing clear fluid. These cavities occupied the second and third segments of the nucleus, and one of them appeared to encroach to some extent on the knee of the internal capsule. The right hemisphere was preserved in spirit, and was not examined until a fortnight later, when a small cavity was found in the lenticular nucleus of this hemisphere also. This cavity occupied the anterior part of the nucleus, but did not appear to encroach upon the internal capsule.

On opening the vertebral canal a small sequestrum of bone was found opposite to the bodies of the sixth and seventh dorsal vertebræ, but there was no displacement of the vertebræ. A layer of pus lay external to the dura mater, opposite to the diseased vertebræ. The dura mater was thickened at this spot and the spinal cord itself was softened, although not much compressed by the thickened membranes. The cord appeared normal to the naked eye above and below the lesion in the dorsal region.

The left ventricle of the heart was hypertrophied, and the aorta was atheromatous, but the valves were healthy. The greater portion of the right lung was dense and airless. The capsules of the kidneys were adherent, and on being stripped off the surfaces of the organs were granular; on section the cortex was seen to be diminished in size. The liver and spleen were normal.

A microscopical examination of the crura, pons, and medulla oblongata revealed a streak of descending degeneration, which occupied the positions respectively indicated in the accompanying diagrams (Figs. 1, 2, 3 and 4 *g*), kindly drawn for me by Mr. A. H. Young, Pathological Registrar to the Infirmary. The

patch of sclerosis in the anterior pyramid of the medulla oblongata is considerably less than that in the crusta. A large number of the degenerated fibres must, therefore, have been lost in the pons. The diseased fibres in the anterior pyramid appeared to bend back in the median raphe to reach the nerve nuclei of the opposite side. The bulbar nuclei were quite healthy. The spinal cord exhibited the usual evidences of transverse myelitis, with its ascending and descending degenerations.

FIG. 1.—CRURA CEREBRI.
(g. Degeneration of portion of the geniculate fasciculus.)

FIG. 2.—TRANSVERSE SECTION OF THE PONS ABOUT ITS MIDDLE.
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Family History.—The patient's father and mother are both alive and healthy, each being above 70 years of age. He has three brothers and three sisters, all enjoying good health. No family predisposition to any particular disease can be traced.

Previous History.—The patient has never suffered from any serious disease until about four years ago, when he had an

FIG. 3.—TRANSVERSE SECTION OF ANTERIOR HALF OF THE MEDULLA OBLONGATA ON A LEVEL WITH THE MIDDLE OF THE OLIVARY BODIES.
(g. Descending degeneration.)

FIG. 4.—TRANSVERSE SECTION OF THE MEDULLA OBLONGATA IMMEDIATELY BELOW THE OLIVARY BODIES.
(g. Descending degeneration.)

attack of rheumatic fever, from which he was laid up for three months. At the same time he suffered from an eruption which the medical man who attended him called chicken-pox. One morning last June the patient was writing a letter when he was suddenly seized with cramp in the fingers of his right hand, the sensation of cramp travelling up his arm to his head. He states that he did not lose consciousness during

the attack, but there was partial loss of the power of speech, the patient being only able to utter portions of sentences. Speech gradually improved after a few hours, but complete recovery did not take place until the lapse of three days. During the attack he suffered from intense headache, which lasted half an hour; his right hand and arm were numb during the seizure and for five or six hours subsequently. A few days after this attack he felt quite well and able to attend to his work as usual. Towards the end of October he observed that his water dribbled instead of coming in a full stream, but he did not think this symptom of much consequence. On November 6, 1881, he took a walk of five or six miles into the country, and as he was returning he observed that his foot kept catching the ground so that he stumbled several times. On getting up the next morning he was unable to pass water, but went to work as usual. On walking home at noon to dinner he stumbled and staggered so much that he was obliged to steady himself by catching hold of the rails on the road-side. After dinner and a few hours' rest the patient made another attempt to go to work, and actually walked to the building on which he was engaged, a distance of nearly half a mile from his house, but on arriving there he found himself so ill that he was obliged to return home. He walked back, but with great difficulty, and when he got near to his own house he fell, but his wife came to his aid and assisted him into the house. A medical man was then sent for, and on arriving he drew off his water. This operation had to be repeated night and morning for a week, but at the end of this time the patient was able to pass water himself.

The lower extremities were now more or less completely paralysed, and the patient was confined to bed until about the middle of December; he then began to get up, and was even able to walk a little when supported.

On Christmas Day as the patient was lying in bed he had another seizure. He has an indistinct recollection of feeling a curious sensation in the right arm and right side of the face, but he soon became unconscious. He states that he remained in a dazed condition for two weeks; during this time there were intervals when he knew something of what was

going on about him, but at other times he was quite wild, and then he sang, shouted, and struggled with his attendants. The patient states that the doctor told him that three days after the commencement of unconsciousness he had a fit during the night, which left his mouth drawn to the left side. When he regained his senses he was only able to speak in broken sentences, the right arm was paralysed, the mouth was drawn to the left, and he could not close his right eye. Gradual improvement took place in the symptoms occasioned by the last attack, so that at the time of the patient's admission into the Infirmary his right hand had regained a considerable degree of its former power, he could close his right eye, and the only affection of speech from which he suffered was a certain degree of indistinctness of articulation.

Present Condition.—The patient is a well-made, muscular man, of sandy complexion and healthy appearance. Both the lower extremities are completely paralysed. Some degree of muscular tension is provoked by passive movements of the legs, the patellar tendon reflexes are slightly exaggerated, but ankle clonus cannot be elicited. The muscles of both calves and the peroneal groups are somewhat wasted; they do not reach to a strong faradic current, but respond to a galvanic current from 40 Leclanché cells, the reaction to negative closure being stronger than that to positive closure. The reflex of the sole is exaggerated on both sides and the cremasteric reflex is not entirely abolished, although it is very sluggish. Every form of sensibility is diminished on both sides up to within an inch of the umbilicus. There is constant dribbling of the urine, and the patient passes his stools in bed. There is a small and superficial bed-sore over the sacrum. The grasp of the right is feebler than that of the left hand, and the right forearm is slightly jerked when the lower end of the radius is tapped. The triceps reflex is not perceptibly increased on the right side. The angle of the mouth is on a lower level on the right than on the left side; the right naso-labial fold is obliterated, and there is a decided loss of expression on the right side. The right lower eyelid is slightly depressed, while the upper lid is somewhat retracted so that the palpebral fissure is wider, and the eyeball appears to protrude more on the right than on

the left side. The patient can close both eyes by a voluntary effort, but the resistance which he can offer to the upper lid being raised by the finger of the observer is much greater on the left than on the right side. There is no deviation of the tongue on protrusion, and the patient can turn the tip laterally and towards the nose without apparent loss of power. He can also pronounce the separate consonants and vowels fairly well, but his articulation is indistinct and imperfect, especially on attempting to pronounce words of many syllables, like "Constantinople." The reflex of the palate is very sluggish when the fauces are tickled. The urine is alkaline, and contains bacteria and crystals of triple phosphate. The other organs are healthy. There is no evidence of valvular disease of the heart.

March 8.—The patient has become steadily worse since his admission. The muscles of the lower extremities are much atrophied, and the subcutaneous tissue is soft and œdematous. A large slough has formed on the sacrum, and an erythematous blush, which appeared a few days ago, has spread from the sore upwards as far as the angles of the scapulæ, and downwards below the gluteal folds. The urine is alkaline and contains a large quantity of pus, bacteria and crystals of triple phosphate. There are great variations in the daily records of the temperature, that in the evening being generally above 103° F., and in the morning down to 98° F. The articulation is much more indistinct, but this appears to be due to the dryness of the tongue. The other cerebral symptoms are unchanged.

March 15.—The patient became gradually weaker since last report, and died to-day in a state of great marasmus.

The post-mortem examination was conducted by Mr. A. H. Young fourteen hours after death. Body well developed, poorly nourished, limbs emaciated. Cutaneous hypostasis well marked dorsally. Large bed-sore over the sacrum. Rigor mortis absent. A small cystic cavity was found in the left lenticular nucleus, close to the border of the genu of the internal capsule. No secondary degeneration perceptible to the naked eye in the crura.

There was marked congestion of the meningeal rachidian

veins. The membranes of the cord were normal. The spinal cord itself, in the lower dorsal and upper lumbar regions, was abnormally soft, and on section the white matter protruded while the grey matter appeared depressed. The vessels of the grey matter were engorged. The heart weighed 13 ounces; it was hypertrophied and dilated, but there was no valvular lesion, and nowhere were there any indications of previous endocarditis. Multiple abscesses were found in the kidneys, but the other organs did not present anything of special interest.

A careful microscopical examination of successive sections of the crura, pons, and medulla oblongata failed to detect any secondary degeneration of the pyramidal tract. The bulbar nuclei were healthy. The usual evidences of a transverse myelitis in the lower dorsal and upper lumbar regions, with ascending and descending degenerations, was discovered in the spinal cord.

A case, evidently belonging to this group, has just been reported by Dr. Hobson, of Croydon;¹ but I shall only refer to it briefly, as the subject is still living, and the diagnosis is consequently more or less conjectural. A lady, aged 40 years, suffering from albuminuria, had, during a period extending over twelve months, four successive attacks of partial aphasia, with slight implication of the left hand in the third and of the right hand in fourth attack. After the fourth attack, when the patient came under Dr. Hobson's observation, there was complete facial paralysis. The patient was unable to close the eyes by a voluntary effort. "On the other hand, *blinking* occurred, and, when amused, the face would be drawn up into an expression of laughter, but never into a smile." The tongue was paralysed, deglutition was difficult, and the power of speech and articulation was almost abolished, but the jaws were clenched from spasm of the masseters. The clinical picture presented by this case forcibly reminds us of the case recorded by Magnus, although it is probable that in Hobson's case a lesion exists in each hemisphere.

The cases which have just been described are full of theoretical and practical significance. They show, in the first

¹ 'The British Medical Journal,' April 29, 1882, p. 618.

place, that speech, in its objective or expressive aspect, consists of highly special and complex movements, and that ataxic aphasia is a want of power of effecting the combinations of muscular contractions necessary for the production of complex articulate sounds; or, in other words, that ataxic aphasia is essentially paralytic in its nature. If that be so, it may be asked why are not the general movements of articulation paralysed? The reply is, that as a rule they are paralysed, inasmuch as aphasia is usually associated with some degree of oro-lingual hemiparalysis. The special movements of the organs of articulation called into activity during speech are organised in one hemisphere only, usually the left, while the general movements are organised in both hemispheres. And destruction of the centres of articulation in both hemispheres, such as occurred in Dr. Barlow's case, gives rise not only to aphasia but to complete anarthria, along with difficulty of deglutition and bilateral paralysis of the inferior branches of the facial nerves.

It is worthy of note that in Dr. Barlow's case the patient made a good recovery from his first attack of hemiplegia (right) and aphasia, although the autopsy showed that the symptoms were caused by destruction of the cortical centre of speech. The solution of this difficulty appears to be that the special movements of articulation are not so exclusively organised in one hemisphere in young people as in adults; and during youth the brain undergoes structural changes more readily than at a late period of life, so that a new organisation is more rapidly developed in the former than in the latter. The recovery of speech in Dr. Barlow's case must be supposed to be due to a rapid development of new structural arrangements taking place in the oro-lingual cortical centre of the right hemisphere, after the corresponding centre in the left hemisphere had been destroyed. And when the centre in the right hemisphere had become subsequently destroyed, not only the special movements of articulation concerned in speech, but the general movements, were arrested, and the patient suffered from anarthria, difficulty of deglutition, and partial facial paralysis.

Passing over the cases in which the lesion was situated in

the cortex, let us come to those cases in which the lesion was situated in the substance of the hemisphere. It is probable that in one or two of the cases in which the lesion was situated in the substance of the hemisphere the cortical centres were also damaged; such, for instance, as the case reported by Magnus, and the right hemisphere in Lépine's first observation. In the remaining cases, however, the lesions observed were strictly limited to the substance of the hemispheres, and the cortex was uninjured. In all these cases lesions were found in the lenticular nuclei, either in one or in both hemispheres.

The questions which naturally suggest themselves are, Is the lenticular nucleus to be regarded as an independent centre for the regulation of the movements of articulation and deglutition? Is it a ganglion of interruption, as Meynert asserts, between the cortical centres and the nuclei in the medulla? or have the lesions been situated in such positions that the direct conducting paths between the cortex and the nuclei in the medulla oblongata and pons were injured? The first of these questions may be negatived at once, inasmuch as no known anatomical or physiological fact can be adduced in favour of the hypothesis which an affirmative answer would imply. It is somewhat difficult to decide between the alternatives presented by the two remaining questions. The theory that the lenticular nucleus is a ganglion of interruption, and that a lesion of it would interrupt the channels of communication between the cortex of the brain and the pons, medulla oblongata, and spinal cord, was a few years ago generally accepted as an established doctrine. But the anatomical investigations of Wernicke render it very doubtful, to say the least, whether the lenticular nucleus is possessed of radiating fibres to connect it with the cerebral cortex; and pathological observation has proved that the nucleus may be completely destroyed without giving rise to hemiplegia or any other decided symptom.

The supposition that the direct conducting paths from the cortex to the medulla and pons have been injured by the lesions is not free from difficulty, although it is the one we are inclined to adopt. It has been found by Charcot and

Brissaud that secondary degeneration occurs in the fibres of the knee of the internal capsule in cases of long standing aphasia. And a case was reported by these observers in which, along with extensive recent softening of one hemisphere, an old focus of softening was found limited exactly to the knee of the internal capsule (Fig. 5, D).

FIG. 5.—(CHARCOT AND BRISSAUD). RECENT SOFTENING OF THE FRONTAL LOBE, THE ISLAND OF REIL, AND MIDDLE THIRD OF THE LENTICULAR NUCLEUS.—D, old focus of softening occupying the knee of the internal capsule; A, Caudate nucleus; B, Optic thalamus; C, Anterior, and E, Posterior division of the internal capsule.

A streak of degeneration was observed in this case lying between the internal and middle thirds of the crusta. It may be presumed, therefore, that the fibres which descend from the cortical centres of speech to join the nuclei in the medulla pass through the knee of the internal capsule, and through the crusta at the point of junction of its internal and middle thirds; and consequently Charcot proposed to call this bundle of fibres the *geniculate fasciculus*. Now, in my second observation, a streak of degeneration was actually observed in the crusta, occupying the position assigned to the geniculate fasciculus by Charcot. But in my first observation (a much more typical example of this affection than the second) and also in the third case the most careful microscopical examination failed to

detect any descending degeneration, although the time which elapsed from the onset of the symptoms to the fatal termination was amply sufficient for a secondary degeneration to have taken place. But in my second and third cases there was, in addition to the bulbar symptoms, a slight degree of paralysis of the right hand, accompanied by excess of the deep reflexes. But notwithstanding this, no secondary descending degeneration of cerebral origin was found in either case in the spinal cord. It must, therefore, be admitted that in a cerebral lesion either slight paralysis of the muscles of the hand, with excess of the deep reflexes, may occur in the absence of any damage to the pyramidal tract or to the cortical centre, or that it is possible for the pyramidal tract to be slightly, but more or less permanently, damaged without being followed by a recognisable descending degeneration. I incline to adopt the latter supposition; and if it be possible to have injury of the fibres of the pyramidal tract which belong to the spinal nuclei regulating the muscles of the hand without descending degeneration, it must be equally possible for the fibres of the geniculate fasciculus to be partially injured without giving rise to a recognisable degeneration. This supposition is still further strengthened by the three cases reported by Binswanger,¹ in which lesions in the motor area of the cerebral hemispheres were discovered at the autopsies, while the patients during life suffered for some months from paralytic symptoms, and yet there were no secondary degenerations of the pyramidal tracts. In my second and third cases the lesions were of very limited extent, and all of them were situated opposite and close to the knee of the internal capsule, while in my first case the fibres of the internal capsule, those of the knee included, in each hemisphere could hardly help being compressed by the distended cystic cavities which replaced the lenticular nuclei. But in Lépine's second observation the lesions were situated in the posterior and external parts of each lenticular nucleus, and consequently away from the knee of the internal capsule. It must, however, be remembered that the spot of discernible softening never represents the whole of the diseased area in the cerebrum, but that an inflammatory condition may spread

¹ 'Archiv für Psych.,' Bd. xi. p. 727.

for a variable and often very considerable distance beyond the diseased focus. It is, therefore, quite possible that the fibres of the knee might have been injured in both hemispheres in Lépine's second case. It is very probable that the cortical centres were injured in the right hemisphere in Lépine's first observation, while the knee of the internal capsule could readily be damaged in the left hemisphere. In the case reported by Magnus the cortical centres of the right hemisphere were injured, and the centres in both hemispheres were probably damaged in the case described by Eisenlohr. The lesions found in the case reported by Kirchhoff demand separate notice. The right sylvian artery was occluded, and the external third of the lenticular nucleus, the claustrum, external capsule, and island of Reil were softened, the softening being probably the result of the embolic closure of the artery. But the seizure which corresponded with the closure of the artery only took place ten days before death, and the bulbar symptoms were in existence for six months previously. It seems clear, therefore, that the lesion in the caudate nucleus was the cause, the indirect cause at least, of the bulbar symptoms. It is expressly mentioned that the portion of the internal capsule subjacent to the lesion in the corpus striatum was grey and translucent, and certainly the knee of the capsule would be subjacent to some part of the posterior two-thirds of the caudate nucleus, the portion said to have been softened. The bulbar symptoms would, therefore, in this case, be directly caused by the secondary implication of the internal capsule."

But if the affection of speech present in my third case, for example, were caused by damage to the fibres of the pyramidal tract which connect the cortical centre with the nuclei of articulation in the medulla, it may be asked why it is that the dysarthria was not associated with aphasia. The reply is that the two symptoms were associated for a time, but the aphasia was only temporarily present. In the first cerebral attack from which this patient suffered the symptoms were very transitory, but some degree of aphasia was present for a few hours. The second cerebral attack was attended by prolonged unconsciousness, but for some time after he regained consciousness the

patient, to use his own words, could only "speak in broken sentences." But why did the aphasia disappear some weeks after the second attack, while the dysarthria remained more or less permanently? The assumption that the right cortical centre had become educated for speech which we made in reference to Dr. Barlow's case is not fully adequate to explain the rapid disappearance of aphasia which takes place in all those cases where the lesion is situated in the basal ganglia. The most plausible explanation of such cases is that proposed by Dr. Broadbent. He suggested that when the usual channel of communication between the centre of speech in the left hemisphere is interrupted, the impulses from the centre make their way through the fibres of the corpus callosum to the corresponding centre in the right hemisphere, and down through the fibres of the right pyramidal tract to the nuclei in the medulla, the nuclei of the two sides being connected by commissural fibres, through which the impulses make their way. In Fig. 6, for instance, let C' and 3' 3' represent the centre and usual conducting path of speech. The conducting path joins V, the nucleus of articulation in the opposite half of the medulla, and the impulses are conveyed through the commissural fibres c'' to V', the nucleus of articulation in the medulla, on the same side as the centre. If the centre C' is completely destroyed, then the aphasia must either be permanent, or the centre C of the opposite hemisphere must undergo structural development. In adults the aphasia, after complete destruction of the centre, is, as a rule, more or less permanent, but, as we have seen, a new organisation is more readily developed in young people. But when the usual conducting path 3' 3' is ruptured in any part of its course, the centre C' is only temporarily cut off from the nuclei in the medulla. The impulses from C' make their way through the dotted line c to the opposite cortex C, and through the dotted line 33 to the nucleus V', and thence through the dotted c'' to the nucleus V.

In my third case, a somewhat exceptional symptom was observed. I allude to the fact that, although the paralysis of the right side of the face was of cerebral origin, yet the upper branches of the facial nerve, which usually escape under such circumstances, were partially paralysed. It has been observed

for a long time that in cases of hemiplegia some of the muscles on the affected side are but little or not at all paralysed; but it was first pointed out by Dr. Broadbent, that the muscles which

FIG. 6.

C C', cortex of right and left cerebral hemispheres respectively; 1, 2, 3, 4, 5, 6, fibres of the pyramidal tract uniting c, the cortex of the right hemisphere, and r', e'r, v', a', d', l', the respective nuclei of the internal rectus, and the external rectus muscles of the eye, the muscles of articulation and vocalisation, those of the upper extremity, the dorsal muscles, and those of the lower extremity, all of the left side; 1', 2', 3', 4', 5', 6', fibres of the pyramidal tract, connecting the cortex of the left hemisphere with r, e'r, v, a, d, l, the nerve nuclei of the right side corresponding to those already enumerated on the left side; c, c, fibres of the corpus callosum, uniting identical regions of the two hemispheres; c', commissural fibres, connecting the nucleus of the internal rectus muscle of one eye with that of the external rectus muscle of the opposite eye; c'', those connecting the nerve nuclei of the muscles of vocalisation and articulation of the two sides; c''', those connecting the nerve nuclei of the muscles of the trunk; c'', those connecting the nerve nuclei of the posterior extremity of one side with the anterior extremity of the opposite side. The arrows indicate the direction of conduction.

escape are those which are associated in their actions with the corresponding muscles of the opposite side. Dr. Broadbent further conjectured that the muscles which are associated with the corresponding muscles of the opposite side in their action are innervated from both cerebral hemispheres, so that severance of the connection between the spinal nuclei of the muscles of one side and the cortex of the opposite hemisphere leaves the connection with the cortex of the hemisphere on the same side still intact. In Fig. 6, for instance, d' repre-

sents the spinal nuclei of the intercostal muscles, and when the conducting path 5 5 is ruptured, impulses will still reach the nucleus *d'* from the cortex *C'* of the same side through 5' 5' *d* and *C'''*. Dr. Broadbent very happily named this principle "the bilateral association of the nerve nuclei of muscles bilaterally associated in their actions," and the association is supposed to be effected by means of commissural fibres between the nerve nuclei in the spinal cord and the medulla oblongata. In facial paralysis of central origin the special and unilateral function of winking is abolished, while the general and bilaterally associated functions of raising the eyebrows and closing the eyelids are retained on both sides. In my third case, however, there was distinct paresis of the orbicular of the eye on the affected side, which was so marked at one time as to have prevented closure of the eye by voluntary effort. It must, therefore, be assumed that in this case the commissural fibres in the medulla, between the nuclei of the upper facial branches, were deficient. But in the case recorded by Kirchhoff, both eyelids appeared to have been somewhat enfeebled, and there was distinct paralysis of the inferior branches of the facial and twelfth nerves on both sides, while the lesion was a unilateral one. A bilateral paralysis of the muscles of the face and tongue from a lesion in one hemisphere was still more markedly present in the case recorded by Magnus. It must be assumed that in these cases, not only the special, but the general facial and lingual movements were regulated exclusively from one hemisphere; and, curiously, in both cases the organisation was localised in the right hemisphere, contrary to what occurs with regard to the special movements concerned in speech, which are usually organised in the left hemisphere. In Dr. Hobson's case the upper and lower branches of the facial nerves were paralysed on both sides, but a lesion probably existed in each hemisphere.

As regards diagnosis, labio-glosso-pharyngeal paralysis of cerebral origin is likely to be mistaken for the progressive form of the disease. The chief points of distinction between them are that the progressive form begins insidiously and advances slowly and gradually, the affected muscles undergo

atrophy, and reflex excitability is abolished in the tongue, palate and pharynx at a comparatively early period of the disease, while in the cerebral disease the symptoms supervene more or less suddenly, and are ushered in, as a rule, by one or more apoplectiform attacks, often followed by aphasia and some degree of paralysis of one or both upper extremities, the reflexes of the palate, tongue, and pharynx are not abolished, although they were greatly diminished in the cases observed by me, and the paralysed muscles do not undergo atrophy.

Bulbar paralysis of cerebral origin may also be simulated by local disease, such as thrombosis, embolism, or hæmorrhage in the medulla. But in local disease the symptoms are usually more pronounced on one side than on the other, while in the more decided cases of the affection caused by cerebral disease the paralysis is symmetrically distributed on both sides. Local disease in the medulla may also give rise to polyuria, or diabetes mellitus, great disturbance of respiration and circulation, or alternate hemianæsthesia, as in the cases described by Gowers¹ and Senator,² the latter symptoms never being present in the cerebral variety. In local disease of the medulla the paralysed muscles often undergo atrophy and manifest the "reaction of degeneration," while in the cerebral variety the muscles do not undergo atrophy, and their electrical reactions remain normal.

A case related in Virchow's 'Archives'³ shows that a peripheral lesion of some of the cranial nerves may give rise to symptoms like those of bulbar paralysis. A man, aged 19 years, suffering from leucocythæmia, developed in the course of a few days, and some weeks before death, paralysis of all the branches of both facial nerves, with complete loss of faradic irritability and the reaction of degeneration in the facial muscles, and a high degree of paralysis of the tongue, soft palate, and pharynx, causing serious difficulty of articulation and deglutition. There was temporary diminution of

¹ Illustrations of the Diseases of the Pons Varolii. 'BRAIN,' Vol. II., 1880, p. 476.

² "Apoplectische Bulbärparalyse mit wechselständiger Empfindungslähmung. Von Prof. H. Senator." 'Archiv für Psychiatrie,' Bd. xi. S. 713, 1881.

³ See 'Archiv für Psychiatrie,' Band ix. S. 45, 1878.

sensibility in the territories of both facial nerves, and loss of taste. At the autopsy hæmorrhages were found in the sheaths and textures of the facial, hypoglossal, pneumogastric, glosso-pharyngeal, and lingual nerves on both sides, accompanied by a high degree of degeneration of the substance of the nerves. Such a case, however, is not likely to be mistaken for the cerebral affection just described.