

NOTES ON A CASE OF PURE APOPLECTIC BULBAR PARALYSIS.

BY J. DIXON MANN, M.D., M.R.C.P.,

Physician to the Salford Royal Hospital.

APOPLECTIC bulbar paralysis, when associated with hemiplegia, is not of unfrequent occurrence; but when the bulbar symptoms alone exist, the case, from its rarity, is of exceptional interest. The reason is obvious: the anatomical relationship of many of the motor nuclei in the medulla is so close, that an extremely localised lesion is required in order to limit the mischief to two or three motor nerves. Apoplectic bulbar paralysis is usually associated with alternate hemiplegia, or with paralysis of the upper, lower, or of all four extremities. The disease, as a rule, is initiated by an apoplectic seizure, varying from a momentary loss of consciousness to a profound attack, which in some cases is instantaneously fatal. The region attacked is perilous, and very little deviation of the lethal force is sufficient to convert a circumscribed lesion, capable only of producing a local paralysis of a limited extent, into an occasion of immediate death. In looking over the literature of the subject I have been able to find only three cases comparable with the one about to be described, as regards the limited area attacked, and the uncomplicated nature of the onset of the disease.

In 'Guy's Hospital Reports for 1870,' Dr. Wilks mentions a case in which the patient, whilst at dinner, fell off her chair without losing consciousness, and at the same moment lost the power of speech. She could not swallow without great difficulty, and had constant dribbling of saliva from the mouth; the mental faculties and powers of locomotion were undisturbed. The patient lived for two or three years after the attack, but never spoke again. Hérard, in the 'Union Médicale,' 1868, reports the case of a man aged 68 who was healthy up to the time of the attack. He awoke one night and found himself unable to speak; both tongue and lips were paralysed. The left corner of the mouth was lower than the right; food fell out of the mouth, and saliva constantly ran

over the lower lip. The act of swallowing was not greatly interfered with, but the movements of the vocal cords were imperfectly performed. The intelligence was unaffected, and the limbs retained their normal power. The patient gradually improved, and was able to resume his work, although nine months after the attack a trace of the injury still remained.

A third case by Charcot is reported by Huret in his '*Tribut à l'histoire de l'Embolie des artères vertébrales, 1873.*' It is that of a woman aged 68, who, three years before the attack in question, had an attack of left hemiplegia from which she had quite recovered. One day, without any apoplectic seizure, she lost her speech; she understood all questions quite well, but could not answer them. The right corner of the mouth hung down and could not be moved; the movements of the tongue were partially lost. The patient had considerable difficulty in swallowing, the food returning partly through the nose. There was no paralysis of the extremities. The right pupil was smaller than the left; the heart-sounds were normal. The symptoms gradually improved, and in three months the patient could almost be said to have recovered; the right corner of the mouth, however, remained somewhat lower than the left. In addition to the above cases, Duret quotes one of Voisin's in which bulbar paralysis occurred suddenly, but I have not been able to meet with a detailed account of the symptoms. The following case came under my own observation.

M. D., a married woman, 42 years of age, rather above the average build, of pallid complexion, active in habits, and free from any syphilitic taint, eleven years ago had an attack of rheumatic fever. Since the fever, she has, on two occasions at least, suffered from general dropsy of cardiac origin; at the time of the attack about to be described, she was in her ordinary health. She had not previously suffered from any cerebral symptoms. The patient attributed the attack to a state of excitement (induced by some domestic trouble), into which she fell shortly before the seizure.

On the 27th of October, 1883, whilst walking in the street with another woman, the patient suddenly became conscious of having lost the power of speech. This occurred without any apoplectic seizure whatever; she did not fall, nor had she any pain or dizziness in the head, nor did she even momentarily lose consciousness. Within an hour after the occurrence of the symptoms, the patient walked without difficulty to the Salford Royal Hospital, a distance of about half-a-mile, and there saw Dr. Haslam, who has since informed me that her mental faculties were then quite clear, and that her bodily movements, except those of the tongue and mouth, were perfect. She went home, and remained there for a few days, when, on account of the difficulty experienced in

administering nourishment, she was admitted into the hospital, and came under my care. On November 1st, 1883, her condition was as follows:—Mental faculties quite clear; she responded intelligently to questions either by signs or by writing on the slate. The tongue, somewhat coated, lay flaccid on the floor of the mouth, and could not be moved in the slightest degree, by any effort of the patient. The soft palate was flabby, and hung loosely down, bringing the pillars of the fauces somewhat forwards. The pterygoids were completely paralysed, no lateral movement of the lower jaw being possible. The masseters and buccinators did not appear to have suffered so much; the jaws could be brought together, but with less than the usual power. The muscles attached to the lower jaw and the hyoid bone were not affected, the elevation of the larynx in the act of deglutition being normal in amount. The orbicularis oris was completely paralysed; the patient was totally unable to purse the lips as in the act of whistling, nor could she kiss the back of her hand, nor blow her nose; the latter disability, of course, was partly due to the condition of the tongue and soft palate, as well as to that of the orbicularis oris. The levator anguli oris, levator labii superioris, and zygomatici of the right side were completely paralysed; the corresponding muscles on the left side were not fully paralysed, but their motility was very slight; the patient was only able to impart the least flicker of movement to the left corner of the mouth, and none whatever to the right. The corners of the mouth were equal in height. The ocular and upper facial muscles were altogether unaffected; the patient was able to close the eyelids, and to resist any attempt to open them by force. The trapezius, sterno-mastoid, and all the other muscles of the trunk and extremities were altogether unaffected. The nerves of special sensation were severally tested, and found to be in their normal condition. There was no ringing in the ears, nor was there any sensory paralysis. All the facial muscles responded normally to both constant and interrupted currents; the reflex of the soft palate was not materially, if at all, lessened. The only sound the patient could utter was a nasal "a," being the primitive sound produced by the vocal cords without glossal or labial modification. A laryngoscopic examination revealed nothing abnormal about the action of the vocal cords. Saliva ran continuously out of the mouth, but there did not appear to be any excess in amount; the overflow was simply the result of inability to swallow, and to close the lips. On being asked to swallow, the patient took some water into the mouth, and after a momentary pause, accompanied by a peculiar, expectant look, she slowly turned back her head so as to allow the water to flow towards the fauces. The result was an explosive cough, which forcibly ejected the fluid both from mouth and nostrils. Occasionally the attempt was more successful, and the fluid wholly or in part reached the œsophagus; the act of swallowing, however, was always followed by more or less cough. It was observed that in the early stage of the disease the patient could swallow aerated fluids with greater chance of success than

still fluids. The temperature was normal, the pulse 72; there was no increase in arterial tension. A slight mitral systolic murmur was heard over a limited area. The urine contained neither albumen nor sugar.

For the first day or two after admission the patient was nourished by nutrient enemata, afterwards she was fed with the œsophageal tube. On the eighth day after admission the tongue had recovered a slight amount of motility, and as the patient strongly objected to the use of the tube, she was allowed to try to swallow small quantities of finely divided meat. When she concentrated her attention on the act of swallowing, and performed it in a deliberate manner, as a rule she was successful; but if she attempted to swallow quickly, the food was promptly ejected from the mouth by an explosive cough. Fourteen days after the attack she could protrude the tongue about a quarter of an inch beyond the teeth, could slightly purse the lips, and swallow prepared food with tolerable certainty; she was, however, quite unable to masticate. She has continued to improve, and her present condition, six months after the commencement of the attack, is as follows:—The tongue can be protruded about half an inch beyond the teeth, but she cannot lift it to the roof of the mouth, nor can she curl it up at the sides. She can purse the lips pretty well, but without much power; she cannot smack them, nor can she blow her nose. She can swallow soft pulpy food better than fluids, but cannot masticate even bread. There is now a trace of lateral movement in the lower jaw, and the muscles on the left side of the face have considerably improved; the corresponding muscles on the right side have also recovered a limited amount of motility. The patient can make some attempt at articulation, and occasionally one can make out the meaning of what she endeavours to say, but there is no real speech. She complained a few weeks ago of giddiness when walking, but has improved in this respect. She also complains of weakness of vision. In an early stage of the case I examined the discs without finding anything abnormal, at that period the patient made no complaint about her vision. Later on, my friend Dr. Glascoth kindly examined them and found the retinal veins engorged, especially those of the right side, the margins of the discs were ill-defined, but there was no retinitis. The deep reflexes of the arms are now slightly more marked than in the normal state; in the early stage nothing abnormal in this respect was observed. The compressive power of the hands tested by the dynamometer equals 25 kilogrammes for the right, and 23 for the left; the difference is too slight for unilateral paresis, and is more probably due to the usual predominance in muscular power of the right hand over the left. The excess of reflex just mentioned would probably be caused by slight inflammatory processes set up in the immediate neighbourhood of the focus of injury; the disturbance of the retinal circulation might proceed from the same cause.

There is not much difficulty in localising a lesion such as

this, the symptoms at once point to the posterior part of the rhomboid sinus—to a region embracing the nuclei of the hypoglossal, facial, and motor branch of the fifth nerves. The facial nucleus proper is removed from that of the hypoglossal; but the inferior facial nucleus is about on a level with, and closely external to it. It is supposed that the accessory facial nucleus exclusively controls those branches of the facial nerve which supply the lower facial muscles: it is therefore probable that, as regards the facial nuclei, the accessory nucleus only was attacked, the supply to the upper facial muscles on this account not being interfered with.

As regards the nature of the lesion, acute myelitis may be omitted, as this disease, although it may occur very suddenly, requires at least several hours to develop, and usually runs a rapidly fatal course. The present case, on the contrary, was instantaneous in onset, and the tendency from the first was to improvement. There is then left the choice between blood-extravasation, thrombosis, and embolism. Against extravasation is the early improvement, and the improbability of the occurrence of such a very limited effusion. Whatever was the cause of the lesion, it was of a peculiarly circumscribed and selective character; it attacked a series of nuclei in the same order that they are attacked in progressive bulbar paralysis, the course of which follows a certain definite sequence. It is difficult to imagine that extravasation—the amount and localisation of which would be determined by contingency—could produce such a methodic arrangement of symptoms. It would rather be expected that other motor centres would be attacked, and that an anomalous chain of symptoms would result; still the rupture of a miliary aneurism might produce a sufficiently localised extravasation. Thrombosis presupposes, more or less, the existence of preliminary symptoms: the gradual closure of a vessel would reduce the blood-supply of the region nourished by the vessel, and would probably produce prodromata as dizziness or paræsthesiæ, both of which were absent in this case. Here again the elimination cannot be absolute, for thrombosis does at times develop symptoms suddenly and without previous indications. Embolism seems to be the most likely causation: the attack of rheumatic fever and the subsequent heart mischief give a certain colour to this supposition. It is probable that an excited action of the heart, induced by the domestic trouble before mentioned, led to detachment of a minute vegetation from the left side of the heart, which was carried with the blood stream along the vertebral and basilar arteries and finally lodged in one of the small branches. Almost all the arteries of the medulla are terminal arteries, and therefore such a

clot would give rise to one or more of those well-known wedged-shaped infarcts, with their basis directed towards the floor of the fourth ventricle, and including in their sphere of action the nuclei before mentioned.

There is one point worthy of note—the two groups of muscles most profoundly attacked in this case, the same muscles which are first attacked in progressive bulbar paralysis, viz. the extrinsic lingual muscles and the orbicularis oris, were the first to show signs of returning motility, and have relatively undergone the most marked improvement. The nuclei concerned are the accessory facial and the hypoglossal: the hypoglossal nucleus receives its blood-supply from terminal branches of the anterior root-artery and of the anterior lateral artery. Both the hypoglossal and the inferior facial nuclei are supplied by branches from the artery of the median raphé. Assuming embolism to have been the cause of the paralysis, it is possible that the main blood-supply of the nuclei affected was cut off by the embolus, producing for a time total cessation of function, which was afterwards restored within certain limits by collateral supply.