

POST-DIPHTHERITIC CHRONIC BULBAR PARALYSIS, AND ITS DISTINCTION FROM MYASTHENIA.

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CHRONIC bulbar paralysis occurring as a sequel of diphtheria is a rare event, and I can find only four instances in the literature. Two cases are described by Gowers (18), in both of which primary optic atrophy and external and internal ophthalmoplegia developed slowly some time after the diphtheria, in one of them six months afterwards. In his other case there was additional weakness of the lips, tongue, and pharynx. Stadthagen (41) in 1884 described a case of a boy, aged 11, who at the age of 4 had suffered with diphtheria, followed four weeks later by paralysis of the palate, nasal speech, and regurgitation of food, and later by gradual weakness of the arms and legs. When aged 11 he still presented the symptoms of chronic bulbar palsy, paralysis of the lips, tongue, and palate, with slight weakness of the lower face. Collins (10) in commenting on this case considered it to be a case of associated bulbar neuritis, rather than a chronic nuclear lesion, but Gowers (19), when referring to Stadthagen's case, says: "In extremely rare cases some part, as the palate, has not recovered, and other paralysees have developed and persisted, apparently due to chronic disseminated myelitis." Dundas Grant (12), in 1892, published a case of persistent paralysis of the muscles of mastication after diphtheria.

In the two cases which I am now publishing, one of which I showed at the Harveian Society a year ago, and also at the Clinical Society in January, 1904, the similarity of the

mode of onset of the symptoms and the distribution of the paralysis have led me to regard them as a distinct type as yet unrecorded. In both the cases, after an attack of ulcerated sore throat, there developed a few weeks later paralysis of the palate, followed soon afterwards by weakness of the tongue and lips and inability to close the eyes. In each case there was profound lasting paralysis of the sphincters of the eyes and lips, with complete escape of the remaining facial muscles, with the exception of the frontalis, which appeared to be slightly weak in both. In one case the symptoms lasted six years, followed by complete recovery; in the other, a more severe case, with more marked atrophy of the tongue and facial muscles, the symptoms remain without any improvement after the lapse of four years.

Several cases have been published of chronic bulbar palsy, with involvement of the upper facial muscles, by Wachsmuth (43), Remak (36), Eisenlohr (13), Oppenheim (33), Brissaud and Marie (6), and others.

A progressive juvenile form of bulbar palsy was described by Fazio in 1892 (14) in a girl aged $4\frac{1}{2}$, whose mother was also suffering from a most complete form of bulbar paralysis, with involvement of the upper facial muscles. The child had almost complete paralysis of both the upper and lower facial muscles, with defective articulation, difficulty in breathing and in swallowing, with atrophy of the tongue. There was paralysis of the left vocal cord, less marked of the right. The limbs were normal. Complete reaction of degeneration was present in the facial muscles. An interesting point, probably bearing on the cause of the child's condition, was the fact that it was born five months before the development of symptoms in the mother. Bernhardt had previously (2) published a case of chronic bulbar palsy in a child, but without involvement of the upper facial muscles, and a similar case was also published by Hoffman (22) of chronic progressive bulbar palsy in a boy, aged 11, which commenced with salivation, followed five weeks later by difficulty in swallowing, with nasal regurgitation. Paralysis of the lips followed, and of the middle and lower facial muscles, but with escape of the upper facial muscles. In addition

there was paralysis of the abductors of the vocal cords, and of the left half of the soft palate. There was also atrophy of the tongue and some of the trunk muscles. Reaction of degeneration was present in the lips. Hoffmann insists that diphtheria can be excluded as the cause, but Londe (28), in referring to the case, draws attention to the commencement of the paralysis with palatal palsy.

Charcot (9) in 1893 described two cases of the progressive juvenile type of chronic bulbar palsy in two brothers, aged 5 and 9. Londe, who reported the cases for Charcot, published them at greater length (28), and Brissaud (5) had published the case of the elder brother as a case of probable bulbar tumour. Londe states that the symptoms commenced in the elder boy, when aged $8\frac{1}{2}$, with inability to close the eyes completely. This was followed by defect in articulation and paralysis of the tongue and of the lower facial muscles, including the lips. The forehead was immobile, with paralysis of the frontales and corrugators. Nasal regurgitation, some abductor paralysis of the vocal cords, and slight left ptosis were present. There was no true reaction of degeneration, but diminution to both forms of current was found in the paralysed muscles. The knee jerks were brisk, and there was no weakness of the limbs or sphincters. No variability in the symptoms was noticed. The paralysis in the younger brother was limited to the upper face, there being paralysis of the orbicularis palpebrarum and of the frontalis and corrugator, but there was no difficulty in swallowing or of articulation, and scarcely any weakness of the tongue or lips. Charcot considered the cases to be progressive bulbar nuclear degeneration, and drew attention to the resemblance to the myopathic facies, due to the symmetrical involvement of the sphincters of the eyes and lips. Myasthenia can, I think, be excluded in these two cases of Londe's, on account of the progressive weakness without variability of the symptoms, and the alteration in electrical reactions and the fact of its occurrence in two brothers, suggesting a familial disease. It is, however, apparent that Londe was unacquainted with myasthenia, as he refers as a similar case to a case of Remak's (37),

which is a clear case of myasthenia. This was the case of a girl, aged 12, in whom the symptoms followed otitis media after influenza, and ran a fatal course in four months. The symptoms commenced in this case with inability to close the eyes, and defect of articulation. Later difficulty in swallowing, paresis of the facial muscles, of the tongue and palate, and lips. Frequent attacks of dyspnoea from weakness of the respiratory muscles, ending in death from respiratory failure.

Although my two cases bear a certain superficial resemblance to myasthenia, it is evident on studying them carefully that they must be considered as belonging to a separate class. I shall return to the differential diagnosis from myasthenia after detailing the cases in full :—

Case 1.—M. L., a young woman, aged 25. Four years ago, in November, 1899, she suffered from a slight attack of ulcerated sore throat, which was treated, but no suggestion of diphtheria was made. Six weeks later she noticed difficulty in swallowing, and nasal regurgitation, for which she went to see Dr. Ferris, of Uxbridge, who found that the soft palate was paralysed, and told her that she must have passed through an attack of diphtheria. Dr. Ferris in August, 1902, sent her to St. Mary's Hospital, where I saw her, and he has kindly given me some notes on the early history of the case. Her voice was altered from the first, and soon afterwards her lips became affected, so that she could no longer purse up her mouth, or whistle. It was not until January, 1901, that much difficulty in articulation was noticed, and about this time she found that her eyelids were getting weak, and that she could no longer close her eyes properly. There was never any ptosis or weakness of the jaws or limbs, nor numbness, or any affection of vision. From that time onwards she became slowly slightly worse, the weakness of the eyelids and lips increasing, and the articulation becoming more indistinct, but with occasional slight temporary improvement of the articulation and of swallowing. When I first saw her on August 5, 1902, her condition was much as it is now, with the exception that weakness of the abductors and internal tensors of the vocal cords has since developed, and the voice is more nasal, owing to the paralysis of the soft palate having become complete, there having been slight movement of the velum on phonation a year ago. She has quite recently married.



FIG. 1.

Case 1.—M. L. This photograph shows the absence of ptosis, and the normal power of the muscles at the angle of the mouth, in the act of showing the teeth.



FIG. 2.

Case 1.—M. L. This photograph, taken immediately after the former, shows the complete paralysis of the sphincters of the eyes and lips, in the endeavour to close the eyes and to purse up the lips.

Present state.—There is almost complete paralysis of the orbicularis oris and of the orbicularis palpebrarum on each side, with inability to purse up the lips, or to close the eyes completely. The facies resembles that seen in the myopathy of Landouzy-Dejerine. The remainder of the facial muscles are normal, with the exception of slight weakness of the frontalis, only the sphincters of the eyes and lips being involved (see figs. 1 and 2). The articulation is nasal, and there is complete paralysis of the soft palate, which hangs motionless on phonation, with loss also of the reflex. There is also considerable wasting and paralysis of the tongue, which cannot be protruded properly. There is also weakness of the constrictors of the pharynx, with bilateral weakness of the abductors and internal tensors of the vocal cords, which during phonation do not lie properly adjacent, but leave an elliptical space between. Occasionally she has been unable to open the eyes on waking, but there is never any ptosis. There is no weakness in chewing, or of the arms or legs. She says she tires rather easily, but no loss of power is to be detected in the muscles of the limbs after repeated exertion more than is to be expected in a slightly built woman. All the deep reflexes are brisk, but not excessively so. The plantar reflex is flexor. Before I saw her she had been treated in another hospital by injections of strychnine, which, she thinks, made her worse. For about a year I have treated the palate and pharynx, and facial muscles with galvanism, at first twice a week, afterwards once a week, but without improvement.

Electrical reactions.—Very diminished and sluggish contractions to faradism in the sphincters of the eyes and lips, with stronger and markedly sluggish contractions to galvanism, KCC > ACC. The soft palate reacts only to galvanism, with a sluggish contraction, KCC > ACC. The tongue reacts more briskly both to faradism and to galvanism, but much less so than normal. There is no exhaustion to faradism of the facial or jaw muscles or biceps after tetanisation for two minutes, therefore the myasthenic reaction is absent, and the reaction of degeneration is present, best marked in the soft palate and in the orbicularis palpebrarum. Probably at the commencement there was even better marked R.D. in the palate, perhaps with the polar change ACC > KCC, but, as usually happens in a case of long-standing paralysis, the kathodal contraction has regained its superiority. The sensation is perfectly good on the face and tongue, but there appears to be considerable diminution of sensation on the pharynx and palate.

NUCLEAR SUPPLY OF THE UPPER FACIAL MUSCLES.

The difficulty in opening the eyes in the morning, which she has noticed on several occasions, having to push up her upper eyelids three or four times before she can keep her eyes open, is not to be confounded with real weakness of the levator palpebræ, which, if present, would certainly be most manifest at the close of the day instead of at the commencement, especially were it a case of myasthenia. It is, no doubt, to be explained, as Brissaud has stated, as being due, not to any weakness of the levator palpebræ, but to the paralysis of its antagonist, the orbicularis palpebrarum. The difficulty then of raising the eyelid may be looked on as comparable to an ataxy; the normal muscular tension of the orbicularis not being opposed to the action of the levator, and its appearance only in the morning in this patient on waking, is thus explicable by the fact of the levator palpebræ having been disused for several hours. This point is of some importance, partly because ptosis is such a common symptom in myasthenia, and also for the reason that if the temporary difficulty in raising the eyelids were ascribed to weakness of the levator palpebræ on each side, it would indicate that the third nucleus was partially involved, and on account of the upper facial weakness present in this case, would thus be of some support to the view originated by Mendel (30) in 1887, after extirpating the orbicularis and frontalis in dogs, that the upper facial muscles were represented in the posterior portion of the third nucleus, and not in the facial nucleus proper. That this view is probably incorrect has been shown by Schwabe (38), who found by the use of Nissl's method after section of the facial nerve that extensive cell changes took place in the facial nucleus, but not a single cell appeared altered in the third nucleus. Marinesco (29) has also performed a similar experiment in dogs, dividing the branch of the facial nerve to the three muscles, orbicularis palpebrarum, frontalis and corrugator, the facial *supérieur*, and examining the nuclei by Nissl's method fifteen days later. He divides the facial nucleus into three portions, upper, middle,

and lower, and found the *réaction à distance*, or chromatolysis, in the lower cell-group and lower portion of the middle group. He concludes from this that this portion of the facial nucleus is the nucleus for the upper facial muscles, as he found all the cells of the third nucleus normal. Van Gehuchten (47) has confirmed Marinesco's work in the rabbit, and he divides the facial nucleus into four cell-groups, three anterior, parallel to each other, and one posterior. The latter he concludes to be the nucleus of the upper facial muscles, as he found marked chromatolysis in the cells of this group alone, after dividing the facial *supérieur*, the cells of the third nucleus showing no changes by Nissl's method. On the other hand, a considerable number of cases have been published of ophthalmoplegia with weakness of the orbicularis palpebrarum or frontalis, or of both, by Hughlings-Jackson (23), Smith (40), Hanke (21), Birdsall (3), and others. With regard to the weakness of the orbicularis palpebrarum in these cases a possible source of fallacy has, I think, been overlooked, namely, an apparent weakness only of the orbicularis due to the paralysis of its antagonist, the levator palpebræ, in the cases of ophthalmoplegia with ptosis. This apparent weakness is exactly comparable to the apparent weakness of the levator palpebræ in cases of palsy of the orbicularis, its antagonist already referred to above as explained by Brissaud. An instance illustrating this point has lately come under my own observation—a woman with well-marked signs of tabes dorsalis, with complete external ophthalmoplegia and marked ptosis. When told to screw up her eyes tight she closed them perfectly, but the eyes could be easily opened by the fingers without using any force. At first sight, therefore, the orbicularis appeared to be weak, but, on telling the woman to screw the eyes up again, and urging her several times to screw them as tightly as possible, it was found that after three or four trials she could screw up the eyes as tightly as a normal person, but she always required considerable urging before this was accomplished. Since she was a tabetic, there was an additional reason for apparent weakness of the muscle, due to

ataxy, from sensory paralysis within the muscle, in addition to the paralysis of its antagonist.

Tooth and Turner (42) describe a case of bulbar paralysis with involvement of the lower facial muscles, in which, at the microscopical examination, they found complete degeneration of the facial nucleus, arguing therefrom that the upper facial muscles derive their nervous supply from the third nucleus through the posterior longitudinal bundle. There is, however, no anatomical basis for this view, beyond Mendel's original results, in which he stated that after extirpation of the orbicularis palpebrarum and frontalis muscles in dogs, he found the nerve-cells in the posterior portion of the third nucleus on the side operated on smaller and less numerous than on the other side, while the facial nucleus appeared normal. This was before the days of Nissl's method, and similar experiments done by this method have failed to substantiate Mendel's contention. Cassirer and Schiff (8), moreover, describe a case of complete degeneration of the oculo-motor nucleus, including its posterior portion, in which the upper facial muscles were normal. Siemerling and Boedeker (39) also state that they have found no evidence in support of the view that the upper facial muscles are supplied from the third nucleus.

Wilbrand and Saenger (45), who give a good abstract of most of the literature on the subject, declare their adherence to the view of Cassirer and Schiff, and of Siemerling and Boedeker, that the third nucleus has no connection with the upper facial muscles, and they further cite as evidence against Mendel's view the common overaction of the frontalis in nuclear ophthalmoplegia with ptosis. The question is thus still somewhat unsettled, and must be decided in the future by instances of third nuclear palsy (with autopsy) with uninjured facial nucleus, or by Nissl-stained sections of the nuclei in cases in which the upper facial nerve has been damaged about a fortnight before death. The numerous instances of pontine tumour or hæmorrhage, with paralysis of the whole face, upper and lower, are of no value, since the descending fibres from the third nucleus, if present, would be damaged on their way to join the facial nerve. Similarly

cases of acute degeneration of the seventh nucleus in polio-encephalitis inferior are of little help, for the same reason of possible destruction of the descending fibres.

Of similar interest is the question of the nuclear origin of the fibres for the orbicularis oris, which it has been suggested indeed, with no want of dogmatic assertion on the part of some teachers, derive their nuclear supply from the hypoglossal nucleus. Gowers (20) leaves the question quite open, while showing the possibility of some of the facial fibres descending to, and perhaps arising from, the hypoglossal nucleus. Experiments of extirpation of the orbicularis oris with subsequent examination of the nuclei by Nissl's method may perhaps clear up this point, but nothing positive is yet known.

The following case of bulbar palsy following diphtheria is of great interest owing to its close resemblance to Case 1 in the distribution of the paralysis, and also to the fact that after the symptoms had persisted for six years, rapid recovery took place. The girl was ten years ago in the Queen Square Hospital under the care of Dr. Hughlings Jackson, for whom I was then clerking, and who has kindly given me permission to make use of the notes of the case.

Case 2.—L. C., aged 19 years, an unmarried girl, had diphtheria in the Summer of 1892. Three weeks later she had a good deal of difficulty in swallowing, with nasal regurgitation of liquids. Shortly after this, she noticed her tongue was clumsy and useless in eating. This was followed by diplopia, and soon by inability to close the eyes. There was slight trouble with the fingers, being unable to button her frocks, but there was no weakness of the legs. No family history of nervous disease. The diplopia soon improved, but the condition of the lips, eyelids, tongue, and palate remained unaltered, and she was admitted under the care of Dr. Hughlings Jackson on November 29, 1893, eighteen months after the attack of diphtheria. She still complained of slight diplopia, but there was no apparent ocular weakness. Pupils normal, and no ptosis. There was considerable weakness of the orbicularis palpebrarum, so that she could only just close her eyes, but very inefficiently. There was marked weakness of the orbicularis oris, so that she could not purse up her lips or whistle, or suck things. She could wrinkle the forehead,

and frown, but only badly, while all the remainder of the facial muscles escaped completely. The soft palate was completely paralysed, and the voice nasal. The reflex of the palate was also lost, but its sensation was good. The tongue was protruded straight, but she still complained it was clumsy. There was no wasting of the tongue. The laryngeal muscles were normal. No weakness of the jaw. After five months she had improved somewhat. The diplopia had disappeared, and there was no longer any nasal regurgitation, and she was now able to curl her tongue well, but the paralysis of the sphincters of the eyes and lips remained as before, and the palate also remained paralysed, and deglutition was performed with an obvious effort.

The electrical reactions on admission showed diminution to faradism in the orbicularis oris and orbicularis palpebrarum as well as in the frontalis and corrugator supercilii, while the remainder of the facial muscles reacted normally. On April 7, 1894, the reactions are noted as follows: "The orbicularis palpebrarum reacts readily to a very weak current, as readily as the levator anguli oris, though the weakness of the eyelid continues as before. The orbicularis oris reacts to the same current, but more sluggishly. All the lingual muscles react smartly on faradic stimulation of the nerve at the angle of the jaw, a very weak current was enough for the frontalis, while a strong, painful current was necessary before the sphincters of the eyes or lips would contract." After leaving the hospital in August, she attended the hospital for about three years as an out-patient for electrical treatment, and married in September, 1897, but there was little or no alteration until a year after her marriage, when she noticed rapid improvement soon after her first child was born. She has remained perfectly well for the last five years, after suffering with the bulbar symptoms for six years. I saw her in April, 1903, and found that she had recovered perfectly. All the movements of the eyes and face were normal, and the electrical reactions of all the facial muscles were normal and brisk. The palate had also completely recovered.

The course of this case, following diphtheria and recovering after pregnancy, recalls one of Saenger's cases of myasthenia (48). His was the case of a young married woman, aged 22, who suffered from ulcerated sore throat, and four weeks later developed difficulty in swallowing, diplopia, ptosis, ophthalmoplegia, paralysis of the palate and of the lower face, with weakness of the extremities and neck

muscles. There was no myasthenic reaction or reaction of degeneration. Campbell and Bramwell mention in their abstract that she began to improve in a fortnight, and subsequently, during her first pregnancy, recovered completely, but I can find no mention of the sequel of the case in Saenger's own description of the case (48).

The resemblance in type of my two cases is remarkable. In both there was complete paralysis of the palate, almost complete paralysis of the sphincters of the eyes and lips, with slight weakness of the frontalis, and complete escape of the muscles of expression round the mouth. In each the tongue was partially paralysed, and there was considerable difficulty in swallowing. In both the symptoms commenced with paralysis of the palate, in one three weeks, in the other six weeks, after an illness with ulcerated sore throat, which was recognised as diphtheria in one of them. There is no bacteriological evidence of diphtheria in either, even if that were considered conclusive, but the fact that paralysis of the palate, with nasal regurgitation, supervened a few weeks after an attack of ulcerated sore throat would be considered at the time, at all events, as conclusive for diphtheria, I imagine, by anyone. Because the after sequence of events was unlike the usual course of post-diphtherial paralysis is insufficient reason, in my opinion, for disregarding the previous diagnosis of diphtheria. In some respects the cases resemble myasthenia, or asthenic bulbar paralysis. The paralysis of the sphincters of the eyes and lips, with weakness of the frontalis and escape of the other facial muscles, is not uncommon in myasthenia, as is also weakness of the tongue and palsy of the palate, with difficulty in swallowing. Some of the most constant and typical symptoms of myasthenia are, however, conspicuous by their absence in both of these cases. Such are ptosis, weakness of the jaw muscles and of mastication, ophthalmoplegia, weakness of the limb muscles and of the neck, attacks of dyspnœa, and especially the variability in the symptoms, being worse after exertion and in the evening, with the myasthenic reaction in some of the affected muscles. All these signs are absent in my two cases, the symptoms being

remarkably constant, with no more than the slight variations that may be met with in any case of chronic nervous disease.

Of the 114 cases of myasthenia tabulated from the literature by Hun (24), weakness of the extremities was a symptom in every case but one, and that a doubtful case. Ocular paralysis was found in all but four cases, two of which were doubtful. Ptosis was present in 85 per cent. of the cases. Of the fifty-six definite cases of myasthenia abstracted by Campbell and Bramwell (7), ptosis and weakness of the jaw muscles were present in 80 per cent. and 82 per cent. respectively. Of the eleven cases of this series in which ptosis was not present, seven had weakness of the jaw muscles, and five suffered from attacks of dyspnoea. In every case, except Case 47, there was present one of the three symptoms—ptosis, weakness of the jaws, or attacks of dyspnoea—while in Case 47 there was weakness of the arms and legs, with the myasthenic reaction. Even the so-called myasthenic reaction, or rapid exhaustion of reaction to faradism of the muscle, has been shown to occur in numerous conditions besides myasthenia, such as hemiplegia, cerebellar tumour, neurasthenia and muscular dystrophies. It was first described as the *Reaction der Erschöpfbarkeit* by Benedikt in 1868 (46), twenty-seven years before Jolly (25) described it in myasthenia, and it has been similarly described in other forms of nervous diseases by Mosso (32), Kollarits (26), Flora (16) and Feinberg (15). On the other hand, this form of electrical reaction has been found absent in undoubted cases of myasthenia by Saenger (48), Sinkler (49), Punton (50) and Feinberg (51).

Of my two cases the second, perhaps, resembles myasthenia more than the other, in the absence of muscular atrophy, and of distinct reaction of degeneration, and in the presence of diplopia, with eventual complete recovery. Since, however, diplopia is such a common transient event in post-diphtheritic paralysis, it is not strange that it should form part of the symptom-complex of the more permanent type of bulbar lesion following that disease. In my first case the distinction from myasthenia may be summarised as follows: Little or no variability of the symptoms; absence of

any weakness of the jaw muscles, or of the neck or limbs; absence of the myasthenic reaction in any of the facial or arm muscles; absence of any ptosis or ocular palsy or diplopia; absence of any dyspnoëic or choking attacks, or attacks of palpitation; the presence of atrophy of the tongue and lips; reaction of degeneration in the palate, orbicularis palpebrarum and orbicularis oris.

The two cases must be classed together, and it is evident that the first case is a more severe one than the second, though the lesion must be considered to be of the same type in both. What then is the lesion? In myasthenia, the bulbar nuclei, nerves and muscles have been found many times to be perfectly normal, and Campbell and Bramwell suggest that the pathological process is a diminution of functional activity of the peripheral nerve-fibres of the bulbar nerves, due to some circulating microbic toxin. More recently, however, Weigert (44) has found in a case of myasthenia lymphoid infiltration of the muscles, secondary to a lympho-sarcoma of the thymus gland, and this pathological finding has been exactly confirmed in Hun's case. Hun also refers to a case of Link's (27), in which there was present lymphoid infiltration of the muscles, with a persistent thymus, and in Oppenheim's case (34) there was a sarcoma of the anterior mediastinum. In Goldflam's case (17) there was a lympho-sarcoma of the whole upper lobe of one lung, with lymphoid infiltration of the muscles, which had also been found in a piece of the deltoid muscle excised twelve months before death. Hun's view of the paresis in myasthenia is that it is due to some alteration in the end plate in the muscle fibre, due to a chemical change in the lymph bathing the muscle fibre, and dependent in some way on the lymphoid infiltration of the muscle. Now in diphtheritic neuritis the lesion is proved by microscopical examination to be a structural alteration or degeneration of the nerve-fibres from the periphery up to the intramedullary portion of the anterior roots, close to their origin from the anterior horn cells, as shown by Katz and Luce, Bruns, Batten (1), and others. Occasionally, however, nerve-cell changes have been found, as by Bolton (4), in the

nucleus ambiguus in a case dying from heart failure, by Preisz (35), and by Michell Clarke (31), who showed gross changes by Nissl's method in the anterior horn-cells and nuclei after diphtheria. It is, then, not difficult to picture to oneself cases of increasingly severe lesion of the nerve-fibres and anterior horn cells and bulbar nuclei, the slighter forms of which, in which the lesion is no doubt limited to the nerve-fibres entirely, recover entirely, as in the ordinary non-fatal form of post-diphtheritic neuritis. A more severe lesion would be associated with more or less gross changes in the anterior horn cells or bulbar nuclei, which on the one hand might recover after many months or years, according to the severity of the cell damage, as in Case 2, or on the other hand might remain permanent, or might even become slowly progressive, as in Case 1. This is the lesion I consider to have occurred in my two cases, the greater cell destruction being in Case 1, perhaps irrecoverable, but certainly more severe than in Case 2, on account of the greater weakness, atrophy, and reaction of degeneration. Since, however, the second case recovered after the paralysis had persisted for six years, and, curiously enough, recovered after the birth of her first child, it is, perhaps, not impossible that Case 1 may also improve, as the symptoms have been present only four years. She married recently, in December, 1903, and it will be very interesting to observe the effect on the paralysis, should she become pregnant and give birth to a child. If I have an opportunity, I hope to publish the sequel of this case in *BRAIN* at a later date.

In conclusion, it seems clear that, very occasionally, permanent palsy may result following diphtheritic paralysis. That when such permanent paralysis remains, it is usually of muscles supplied by one or more of the bulbar nuclei. That a distinct type of bulbar paralysis may ensue, closely resembling in its distribution that which is familiar in myasthenia, or asthenic bulbar palsy, but which is to be distinguished from the latter by the non-variability of the symptoms, the absence of the myasthenic reaction, the absence of ptosis, or weakness of the jaw muscles or of the neck or limbs. It is further distinguished by the absence

of the attacks of dyspnoea so characteristic of many cases of myasthenia, and by the presence of muscular atrophy and the reaction of degeneration.

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