AN ADDRESS ON DIPHTHERITIC AND RELATED FORMS OF PARALYSIS.

Read before the Thames Valley Branch.

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DIPHTHERITIC paralysis is so interesting in itself, and has such interesting clinical relations with other paralytic affections, that I need make no apology for introducing it for discussion, or for plunging at once *in medias res* by giving brief details of a very instructive case of it which was under my care some years ago.

CASE I.—H. A., a clergyman, aged 36, came into St. Thomas's Hospital on November 6th, 1879. He had had in the previous June a bad sore-throat, attended with enlargement of the glands behind the left angle of the jaw. But, although he had felt feverish and ill, he had not consulted any medical man, and could therefore give no trustworthy information as to the nature of his attack. Doubtless he had then suffered from diphtheria. The first evidences of paralysis had shown themselves early in October, and were impairment in the quality of his voice and a tendency for fluids when being swallowed to return by the nose. Towards the end of the month he had further observed that he was getting weak in the legs, that he had numbness in the tips of the fingers and in both feet, and that there was loss of tactile sensation in the tongue and lips, and impairment of taste.

On admission he was a healthy-looking man, complaining of various paralytic symptoms. The tips of his fingers were numb, but he had no obvious loss of power in the hands. His feet were numb, especially the left, so that he could scarcely feel the ground; and he walked with a feeble uncertain gait and with a tendency to fall over to one side. His eyesight would fail (words appearing misty) after reading for a minute or two; and there was some degree of loss of accommodation, but the pupils were equal and contracted to light, and there was no squint. He had numbness of the lips, tip of the tongue, and point of the chin, and was unable to distinguish between pepper, mustard, and salt placed on the tongue. The soft palate was pendulous, and did not contract when irritated; moreover, when he swallowed, the food was apt to pass into the nose, and also to lag about the level of the thyroid cartilage. His voice was nasal. Smell was much impaired. In all other respects he seemed fairly well.

During the next ensuing ten days several changes in his condition were recorded; and careful special examinations were made. The anæsthesia in the fingers gradually disappeared, first on one side and then on the other; but while sensation was returning weakness of the fingers and hands came on. The numbness became more profound in the feet, and extended up the legs, and the lower extremities grew weaker. At the same time he complained of pain and soreness in the feet. The numbness of the chin, lips, and tongue diminished, and he recovered to some extent taste and smell. His eyesight also improved, but his defective voice and deglutition continued. It was about this time also that he complained of numbness of the tip of the nose, of the glans penis and scrotum, and about the anus, and also that he was unable to strain at stool, owing to the fact that during the effort the air imprisoned in the chest escaped explosively through the rima glottidis. Mr. Nettleship's examination confirmed the fact that there was weakness of accommodation in the eyes, but that they were otherwise healthy. Dr. Kilner ascertained that there was some impairment of electric sensibility in both forearms and both hands, increasing from above downwards; but that the only arm-muscles whose electric contractility was impaired were the extensors of the first and second joints of the thumbs; that there was increasing loss of electric sensibility from the knees to the feet, where the strongest current could scarcely be felt; and that, while none of the muscles of the left leg below the knee responded to the induced current, the only

ones that responded on the right side were the tibialis anticus, the peronei, and the extensor brevis. They were all unduly responsive to the continuous current. Dr. Greenfield's laryngoscopic examination gave the following results: There was partial paralysis of the muscles moving the vocal cords, mainly of the abductors, and slightly more on the right than on the left side, and considerable (though not absolute) anæsthesia of the epiglottis and of the soft parts around the vocal cords, but not of the cords themselves. In deep inspiration the cords separated to about one-third of the normal amount, and tended constantly to approximate; in phonation there was complete closure; and the voice (excepting that it was slightly nasal and a little hoarse, owing to slight catarrhal swelling of the parts) was unimpaired.

Subsequently to November 17th the changes were very gradual. The hands and legs became more and more numb, and the patient progressively lost power in them, so that, about the middle of December, he could scarcely use them for any purpose, and his grasp was extremely feeble. Towards the latter end of the month, also, it was noted that all the fingers except the first were flexed, more or less, into the palms, and could not be straightened. The numbness still extended up the legs, and became so profound in the feet and ankles that he could not there feel the prick of a pin. The loss of power also increased, so that by December 9th or 10th he was unable to walk, or even to stand, without assistance. He still also complained of pain and tenderness in the feet. On December 20th he first manifested some difficulty of breathing, and had to sit up in bed. It was thought that the respiratory muscles were weak; but there was no absolute paralysis of either the intercostal muscles or the diaphragm. This difficulty lasted a few days, and then subsided. The impairment of accommodation, and the loss of taste and smell, together with the anæsthesia of the tip of the nose, lips, tongue, and chin subsided gradually shortly after admission. The nasal quality of voice and the dysphagia, as also the difficulty in straining at stool, were not observed after the first week in December. The improvement in the state of the hands and legs took place later, the hands recovering first. He left the hospital well on March 10th.

The case was obviously a typical and an interesting example of the paralysis which supervenes after diphtheria. The nervous symptoms tended to be symmetrical, and spread from region to region, affecting the hands and legs in larger proportion than other parts. The paralysis of the extremities was attended with the so-called "reactions of degeneration," and the anæsthesia of the feet with a sense of soreness and pain. In addition, also, to defective accommodation and to weakness of the soft palate; there was anæsthesia of certain circumscribed tracts occupying the middle line of the body, including among others the epiglottis and parts about the rima glottidis; and some paralytic failure of the muscles of the larynx, which contributed to render straining difficult, and of the muscles of respiration causing dyspnea.

CASE II.—In 1880 I was not aware that any interest attached to the condition of the tendon reflexes in diphtheritic paralysis. My personal attention was first drawn to the fact that the knee-jerks tend to disappear in this affection, by being called into the country to see in consultation a lady who was partially paralysed, and who was supposed, on account of the absence of knee-jerk, to be suffering from locomotor ataxy. The patient was a middle-aged widow, who had been accustomed to somewhat excessive indulgence in alcohol, but on the whole had had very good health.

On close inquiry I ascertained that just two months previously (under the care of another practitioner) she had been attacked with sore-throat, which had laid her up for two or three weeks, and left her somewhat enfeebled in health. The affection had been declared not to be diphtheritic. A fortnight before my visit she had noticed a little numbness across the upper lip, which disappeared in the course of a day or two. Four days later she had been attacked with numbness in the fingers and hands, not confined to the region of the ulnar nerves, but of general distribution. The anæsthesia was not absolute, for she could still feel, and was unattended with loss of power. Three days later she had noticed impairment of feeling and of muscular power in the feet, and this had since then reached the knees.

She was, when I saw her, a healthy-looking woman. Her hands were still somewhat numb, but they had much improved in this respect, and were not weak. The numbness in her lower extremities reached the thighs, and the weakness in her legs was so great that she could not stand or walk without assistance. The affection of the legs was still increasing. The knee-jerks were wholly wanting.

It was clear (even though a diphtheritic origin had been denied) that the case was really one of diphtheritic paralysis, and, in confirmation of the view that it could not be due to ataxia, I may add that the eyes were healthy in all respects, and that there had never been any gastric, rectal, or urinary trouble, or lightning pains. She recovered.

About the time at which this patient came under my notice, the abolition of the tendon reflexes in diphtheritic paralysis was recognised also by other physicians; and since then it has been generally observed in this affection, and has come to be regarded as one of its distinctive features.

Lately, however, Dr. Herringham first, and subsequently Drs. Money and Barlow, have shown that, although the tendon reflexes do disappear in these cases, their actual disappearance is generally, if not always, preceded by a period of several days' duration, in which they presented excessive briskness. The fact had probably not been earlier recognised, because such patients are apt first to come within medical observation only after the symptoms have been in existence for a few days, and after the stage of exaggerated reflexes has passed.

The case I shall next narrate is interesting, because, not only was there this excess of tendon reflex, but it was maintained practically throughout the whole of the patient's illness, and was only replaced by its abolition after paralysis and numbness had disappeared, and the patient seemed to be in good health.

CASE III.—M. O., a married woman, aged 29, came under my care on February 18th, 1887. She had had what was supposed to be quinsy in the previous December. Her illness had begun with feverishness and shivering; she had had lumps in the neck; and her throat had been "covered with a sort of skin." She had recovered by Christmas, but had continued weak ever since. Her present illness had been coming on for two or three weeks. She had first observed some difficulty in swallowing; that, if she were not careful, fluids would return through her nose; and that often two or three efforts had to be made before even solids could pass from the pharynx into the œsophagus. Soon afterwards she had observed numbness and pins and needles in the fingers, and at the same time similar sensations across the upper lip. According to her recollection, weakness of her eyes had come on a little later. This had comprised loss of distinct vision for near objects, with inability to read, and some degree of diplopia. A week or two before admission numbness had come on in the feet, with a feeling as if of treading on cotton wool; and the hands had become weak, but had since improved. The legs, which had been similarly attacked somewhat later, had continued to lose power. Her friends had noticed some change in the quality of her voice ever since the beginning of convalescence.

She was a weakly-looking person, complaining mainly of defective sight for short distances, numbness in the toes and finger tips, and inability to walk without assistance. Her eyes were in most respects normal; but the power of accommodation for near objects was impaired, and there was slight weakness of both external recti, with double vision on looking to the extreme right or left, or at distant objects in front of her. The facial muscles, the tongue, and the fauces appeared natural, and she swallowed without difficulty, but her voice was still slightly nasal. There was no absolute anesthesia, but the tips of the fingers and the toes and the front of the left leg were obviously numb. The grasp of the hands was feeble, and the movements of the left arm generally were relatively weak. She could move her legs freely; but in standing or walking (which she could only do with assistance) her knees were bent and inclined to give way, and she tottered on them. The abdominal and thoracic organs were all healthy. The knee-jerks were much exaggerated, the plantar reflexes were normal.

On February 21st it was noted that she complained of stiffness and a sense of twitching in the left arm; that the hand trembled when held out, and especially when carrying a glass of water to her lips; and that the hand and arm of this side remained weaker than their fellows; that there was slight horizontal nystagmus, and still slight diplopia as at the time of admission. Also the left pupil was a little larger than the right. During the next few days she regained the power of accommodation, and lost her diplopia and nystagmus; moreover, the numbness disappeared from her fingers, and there was some improvement in the sensibility and strength of her lower extremities. But the knee-jerks continued unusually brisk.

On March 1st, she was able to stand for the first time without

assistance, and with her eyes shut; but the feet were still numb; the knee-jerks were about normal. She continued to improve, and on the 10th was discharged, apparently well. At this time she had no anesthesia or obvious loss of muscular power, but the knee-jerks had become much less active than on admission, and were, if anything, less brisk than natural.

I assumed that the case would prove an exception to the rule with respect to the abolition of reflexes in diphtheritic paralysis. But the patient, who still continued in somewhat weak general health, came up periodically to see me at the hospital for some few weeks after her dismissal, and to my surprise, the knee-jerks (although apparently she had completely recovered from her sensory and motor paralysis) disappeared absolutely at the end of a week or two, and had not returned when she finally ceased attendance.

Although as a general rule the paralytic phenomena of diphtheria are remarkable for their tendency to symmetry of distribution, as is illustrated in the foregoing cases, not merely by the condition of the extremities, but also by the patches of anesthesia distributed along the middle line of the body; the symmetry is not absolute; and already in two of the cases hints are afforded that local paralyses due to implication of single nerves are likely to be met with. The next case illustrates this fact.

CASE IV.—Mr. S., a gentleman, about 32 years of age, had had a sore-throat about a month before I saw him. He had not been sufficiently ill to lie up, and did not know he had had diphtheria, but had looked down his throat and seen a white patch on each tonsil. Within the previous week or two paralysis had been coming on, first in the left leg, then in the right leg, and later in the arms.

I saw him in consultation with Dr. Mennell, in April, 1884. At that time he was confined to bed. Feeling was impaired in his legs, from the knees downwards; he had very little motor power at the knee-joints, and could scarcely move the feet or toes at all; the muscles below the knees were tender, and the tendon reflexes were absent. He had numbness of the hands and forearms, and much impairment of muscular power in them. He also had numbness of the upper lip, tip of the tongue, and in a circumscribed area at the back of the neck. He complained of slight difficulty of swallowing, and he presented well-marked general paralysis of the left portio dura, which had only come on within the previous two or three days. There was no obvious affection of the eyes, no dyspnoea, no implication of the rectum or bladder; and in all other respects he seemed healthy. I did not see him again, but learnt that he recovered perfectly in the course of a few weeks.

I add the following case, because it is typical, and because it contains an exhaustive account of the electrical condition of the paralysed muscles by Dr. Kilner.

CASE V.—R. O., a school girl, aged 12, came under my care on March 12th, 1883. In the middle of December last she had diphtheria and was in bed for several days. This was followed by some difficulty of swallowing (fluids especially returning through the nose); by loss of visual accommodation, so that she could not see near objects distinctly; and, somewhat later, by loss of power and numbness, first in the arms, and then in the legs. The muscles of the trunk also became feeble, and before long her weakness grew so extreme that she not only was confined to bed, but could not even turn in bed. During the last month her symptoms had greatly improved; she had recovered the power of swallowing and the use of her eyes, and the paralysis in her limbs had diminished.

On admission she was a healthy-looking girl. She was unable to stand, and only able to flex the legs at the knee-joints. She could move her arms pretty freely, but these limbs were obviously much weaker than natural; and the hands, when extended, assumed a claw-like form, the wrists dropping, the first phalanges being extended and the last two phalanges flexed. The muscles were not noticeably wasted, but they were limp and flabby; and there was a total absence of tendon reflexes in the arms and legs. But all the muscles were irritable, and contracted on being sharply struck. No loss of feeling. No loss of control over the emunctories. There was no paralysis of the facial or ocular muscles, or of the palate, and the eyes now acted to accommodation. Thoracic and abdominal organs healthy, excepting that the urine presented a trace of albumen.

A few days after admission Dr. Kilner tested electrically the muscles of the arms and legs, and found them generally

to present the reactions of degeneration.² The patient gradually improved, and left the hospital at her own wish, before she was well, on April 8th; but she could at that time walk with tottering gait, and the patellar tendon reflex was just showing itself in the left leg.

The foregoing cases are all of them typical, and afford between them excellent illustrations of most of the more interesting incidents of diphtheritic paralysis. They show how the affection comes on some little time, usually a few weeks after, the acute illness has subsided; how the paralysis (which involves sensory and motor nerves alike) tends to be of symmetrical distribution, to creep from part to part, and to subside in one region as it invades another; how the sensory disturbances are not limited to the extremities, but are apt to involve districts situated in the middle line of the body, including the tongue and mouth, and senses of taste and smell; how, also, the paralysis is liable to affect not only arms, legs, respiratory muscles and muscles of the trunk, muscles of accommodation, and of swallowing, but also the vocal cords, and even single nerves; how the tendon reflexes in the legs are first exaggerated, then disappear, and the affected muscles acquire the characteristic reactions of degeneration; and how, finally, while many of the phenomena are such as would seem to be best explained on the assumption that they are due to spreading neuritis, others (such as the sensory affections along the middle line, the early undue briskness of the knee-jerk, and nystagmus) seem rather due to some spreading central lesion. I am inclined to believe that, in diphtheritic paralysis, a wave, so to speak, of slight inflammatory mischief, spreads not only through the medulla oblongata and cord, but along the nerve trunks also.

This brief recapitulation leads me on to speak of some of the diseases of the nervous system which present a resemblance to diphtheritic paralysis, and may even, under certain circumstances, be confounded with it.

I have already shown how the association of numbness in the feet, loss of voluntary power over the legs, and abolition of tendon reflexes (in a case which occurred when the condition of the tendon reflexes in diphtheritic paralysis had scarcely begun to be investigated, and was certainly not as yet generally known), led to the fear that symptoms, which were undoubtedly diphtheritic, might be due to tabes. I do not think such mistakes can often be made now. Yet it is well to recollect that paralysis in diphtheria often follows so mild an attack of the acute disorder, that the patient forgets to volunteer any remark as to the previous occurrence of sore throat; and, on the other hand, that occasionally, in tabes, the paralytic symptoms are, or seem to be, of almost sudden onset.

But the cases most likely, I think, to be thus misinterpreted are those of multiple neuritis of other, and especially, perhaps, alcoholic, origin. It will be recollected that the lady whose case was thought to be one of tabes had been what is called a free liver.

CASE VI.—On November 19th, 1886, a policeman, aged 39, came under my care. He had been a thoroughly healthy man, and had never had syphilis; but, though he was not a drunkard, or even

in the ordinary sense of the term intemperate, it was ascertained, and admitted by himself, that for a long time he had been in the habit of taking a very large quantity of beer in the course of each day. Twenty-six days before (being at the time quite well) he got repeatedly wet through, and was in wet clothes nearly the whole of the day. On the morrow he went to his work, but complained of sore-throat, aching pain in his limbs, vomiting, diarrhoea, and general sense of illness. These symptoms seem to have been the beginning of a severe attack of quinsy, which, first attacking one side and then the other, kept him continuously ill, but without any special change in his symptoms until June 13th. On that day he first complained of tingling in the arms and legs, attended with some loss of power; and, in fact, on trying to walk his knees yielded under him, and he fell down. The muscular weakness increased upon him day by day, until, on the sixth day of his paralytic symptoms, he entered the hospital.

He was a spare, but well-built, and not unhealthy looking man, complaining of sore-throat and weakness in the arms and legs. The tonsils, especially the right, the soft palate, and uvula were still swollen and congested. He complained of a sense of numbness and tingling in the hands, but could feel. The grasp on both sides, and more particularly on the right, was extremely feeble, and although he could move his arms at the elbow and shoulder joints, their motions could be arrested by the mere pressure of the finger. There was no numbness in the legs, but he complained of pain in them, and their movements were extremely defective. He could not raise them from the bed, though he could bend his knees; his feet were extended, and he could not flex them. The muscles of the upper and lower extremities, and chiefly those of the forearms and hands, legs, and feet were exceedingly tender and irritable; the tendon reflexes were wholly absent.

The case was a very long one, and the patient remained continuously under my care for the next nine months, and has lately again been a tenant of one of my hospital beds. The history, however, can be summed up in a few sentences. The throat affection rapidly got well. The muscles of the upper and lower extremities, but mainly those of the forearms and legs, hands, and feet, rapidly lost power, and wasted to an extraordinary degree, and remained exceedingly tender. These conditions continued for the first few months of treatment, at the end of which time almost every trace of muscle had disappeared from the parts above specified: the forearms and legs apparently at length consisting of little more than the bones with the skin stretched tightly over them; the thenar and hypothenar eminences and the interossei being completely wasted, and the fingers bent so as to give the hands a clawlike form; and the feet being in essentially the same condition, but extended at the ankles, while the toes were flexed into the soles. About the end of this time amendment began to take place. The patient, whose bodily health had only been moderately good, began to recover the aspect of health, and flesh and strength. The muscular tenderness slowly disappeared, and the muscles of the upper arms and thighs began to regain bulk and power. Unfortunately, however, no beneficial restoration of the muscles of the forearm, legs, hands, and feet followed; and (with the exception that a few fibres of the muscles of the hands, sufficient only to cause almost imperceptible flickering of some of the fingers, have been spared) the distal parts of the limbs remain to the present time as much emaciated, as absolutely void of muscular tissue, and as useless as they were at the end of the acute stage of disease.

It may be added that, shortly after admission, the muscles of the trunk became weak, so that he could not turn in bed, but recovered comparatively early; that the affected muscles soon showed the reactions of degeneration; and that the patient presented no other indications of nervous disorder besides those above detailed, and no evidence of the presence of any visceral disease.

I suppose it is arguable that the case just cited was really one of diphtheritic paralysis. That view is one which I considered and discarded. The following are the reasons which influenced me. I came to the conclusion from due inquiry that the patient's primary attack had been one of tonsillitis and not of diphtheria; the paralysis came on while the primary disease was still acute, instead of a few weeks afterwards; and the general symptoms and progress of the disease corresponded with what one generally observes in cases of that form of paralysis which his habits had fairly earned for him. I have never known of a case of diphtheritic paralysis in which (if the patient do not die of it) ultimate recovery was not attained.

² (a) With induced current a much more powerful current was required to cause contraction than in health; (b) with constant current.

	Right Arm.		Left Arm.	
Deltoid	2.375—	1.200+	1.725—	1.625+
Triceps	2.800—	2.800+	1.525—	2.150+
Biceps	2.250—	2.275+	2.500—	1.000+
Extensors F.	1.950—	2.025+	1.980—	.800+
Flexors F.	2.200—	2.000+	2.125—	2.150+

	Right Leg.		Left Leg.	
Quadriceps	2.800—	3.000+	2.200—	2.000+
Adductors	3.000—	3.000+	2.250—	2.250+
Extensors of leg ...	1.800—	2.200+	1.225—	1.825+
Flexors of leg	1.900—	2.250+	1.225—	1.500+

Normal reaction with constant current,.....1.500— 3.500+

I quote one other case of so-called "alcoholic" paralysis, because it presented two features which bear some relation to phenomena, also observed in diphtheritic paralysis—namely, loss of power in the respiratory muscles, and a curious kind of difficulty in swallowing.

CASE VII.—The patient was a married woman, aged 2, who came under my care on September 20th, 1886, and was discharged convalescent on August 19th of the following year. She had indulged excessively in drink, especially of ardent spirits, for some years, and had been suffering from loss of appetite, sickness, and increasing weakness in the arms and legs for three months. She was fairly well nourished. There was some degree of numbness in both arms and legs; the arms were weak, and the weakness increased towards the fingers, which could not be straightened; the legs were so feeble that she could not stand on them, or even raise them from the bed. Her ankles were extended, the toes flexed towards the soles, and she had no power of moving them. The muscles of the forearms and legs were exceedingly tender, emaciated, and irritable, and the tendon reflexes were absent. There was also slight nystagmus. The symptoms were in progress at the date of admission, and within a few days it was found out that she had difficulty of breathing, liable to paroxysmal aggravation, and difficulty in the swallowing of fluids. As regards the dyspnoea, it was observed that there was a rapid expenditure of breath, so that she could only utter a few words at a time, that she had complete paralysis of the diaphragm, so that her breathing was wholly intercostal, and that she was liable to attacks of suffocative cough (induced mainly by swallowing fluids), in which she became livid in the face, and seemed in serious danger of dying. She had no paralysis of the mouth or soft palate, her voice was not nasal, and, as before stated, she could swallow solids without difficulty; but whenever she took even a mouthful of fluid, it was ejected within a few seconds with the symptoms of choking. She had no difficulty in carrying the fluid to the back of the mouth, and performing the act of deglutition; and it was not until a second or two had elapsed after the performance of the latter act, until the fluid had presumably passed some distance along the oesophagus, that the choking came on, that she coughed, and brought up with coughing the fluid she had swallowed. I confess I was puzzled to explain these attacks, and seeing that they did not occur until the swallowed fluid had had ample time to reach the lower end of the oesophagus, I was inclined to associate them with the diaphragmatic paralysis. The dangers which attended her paroxysmal attacks of choking and cough were manifestly due to the mechanical difficulty in the way of effective coughing, occasioned by the paralysis of the diaphragm.

The affection of the respiratory muscles and of swallowing, which had only come on recently, subsided gradually and disappeared in the course of a few weeks. The numbness, wasting, and tenderness of the muscles of the limbs increased progressively for some months; and then, after a period of apparent quiescence, convalescence slowly took place. The arms improved, even while the disease was still in progress in the legs. When she left the hospital her general health was re-established; she had complete use of her upper extremities; and, although the movements at the ankles and knee joints remained much enfeebled, she could walk in clumsy fashion, but without assistance.

I will trouble you with yet one other case, not so much because it has any direct bearing on the subject of my paper, as because it throws suggestive light on the difficulty of swallowing which was observed in the last case, and shows how a similar difficulty might have arisen in my first case, in which it will be recollected there was loss of power in the laryngeal muscles and impairment of sensation in the laryngeal mucous membrane.

CASE VIII.—J. H. A., a clerk, aged 44, was sent to me by my colleague, Dr. Semon, on December 7th, 1886. He had had syphilis ten years previously, followed by secondary symptoms. But otherwise he had had good health up to March last, when he was attacked with sore-throat, the nature of which remains somewhat uncertain. However, during its continuance, he experienced some impairment of voice, and some difficulty in swallowing both fluids and solids. When the soreness of throat had subsided, his voice was left impaired, and he found that, although he could now swallow solids with perfect ease, fluids invariably caused choking. After a time he placed himself under Dr. Semon's care, who recognised some small growths below the vocal cords, and that there was paralysis of the arytenoid muscle, in consequence of which the arytenoid cartilages were not approximated when the vocal cords

were brought into apposition, and a triangular chink remained unclosed at the posterior extremity of the rima glottidis. This fact, however, though it explained the huskiness of voice, did not seem fully to explain the difficulty in swallowing fluids; for in recorded cases of this form of paralysis such choking had seemingly not been observed. The manner of choking was very peculiar. There was no doubt that the patient could swallow solids with perfect freedom. He could also, when drinking, perform the act of deglutition without any hitch. But invariably, within a second or two after a mouthful of fluid had been swallowed, and at a time, therefore, when it had presumably passed beyond the larynx and reached the lower part of the oesophagus, he began to choke, and presently coughed up some of the swallowed fluid. The phenomena were just what might be expected to happen, and what I have witnessed, when there is a communication between the oesophagus and trachea. And I at first thought that such a communication existed in this case. That, however, was disproved; for Mr. C. Evans, my house-physician, in my presence tested the suggestion, by feeding the patient with milk through an oesophagus tube, first passed into the stomach, and then gradually withdrawn, and thus found that no choking occurred until the lower orifice of the tube reached the level of the larynx. He ascertained, also, on further examination, that there was distinct impairment of sensibility, and of reflex excitability in the laryngeal mucous membrane, more especially in the inter-arytenoid fold—an observation which was confirmed by Dr. Semon. The explanation of the choking was now clear; at least so it seemed to me. I assumed that, in consequence of the non-approximation of the arytenoid cartilages during deglutition, the portion of the laryngeal cavity situated above the vocal cords remained incompletely cut off from the tube along which food was passing; that, in consequence of their ready diffusibility, fluids, while being urged onwards into the oesophagus, were also driven through the inter-arytenoid chink into the space above the vocal cords; and that actual choking was induced by this fluid remaining there up to the moment when the inspiration which naturally follows the act of swallowing sucked it into the windpipe. The obtuse sensibility of the mucous membrane of course explained the absence of irritation due to the presence of the foreign matter in this unwonted locality. I tested the truth of this hypothesis by making the patient drink, and hold his breath as long as he could after swallowing. And although he was apt to choke after a time, even before he inspired, there was no doubt that, by holding his breath, choking was retarded, and that choking always came on, or was aggravated with the first inspiration. The most interesting confirmation of the truth of my hypothesis, however, was furnished later. After he had left the hospital and passed from under my care, he continued to attend as an out-patient of Dr. Semon's; who one day sent him up to me to show me how, though his paralysis remained, he had learnt to circumvent the difficulty in swallowing fluids. He had discovered that, when standing, and at the same time stooping so as to put his head between his legs, he could drink without difficulty; whereas still, if he drank in the ordinary way, choking followed. I expressed my surprise at his having thought of this manoeuvre; when I was reminded that just prior to his leaving the hospital I had, in talking to him and to the students, remarked that, if his affection should not be cured, he would have to swallow (if he wanted to enjoy drink) while standing on his head; and thus learnt that he had simply put into practice the suggestion I had half-jokingly made. My reason for making the remark was that it seemed to me that if the patient's choking were due to the inhalation of fluid accumulated above the vocal cords, it would be obviated if by any means this fluid could be made to escape thence before the patient could draw a breath, and that if he stood on his head while drinking, it would, from the effects of gravity, at once trickle back into his mouth.

I do not know to what my alcoholic patient's difficulty in swallowing fluid was due, but it was so exactly like that presented by the case just given, that I cannot doubt that she also suffered from a combination of anaesthesia with some paralytic affection of the muscles which cut off the communication between the oesophagus and larynx during the act of swallowing. I am sorry that his larynx was not examined at the time.

DR. ROMOLO GRIFFINI died at Varese on January 9th, aged 63. He was for some years editor of the *Annali Universali di Medicina*, and took a leading part in the management of several medical charities at Milan.