CASES OF SUPPOSED PERIPHERAL NEURITIS OF LARYNGEAL NERVES.

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WHETHER peripheral neuritis of laryngeal nerves ever occurs is at present not definitely proved. The cases which appear to be of this nature do not afford the opportunity of confirming the diagnosis by *post-mortem* examination, as the disease does not tend to cause a fatal result. The following cases seem, however, to point, as far as clinical evidence goes, to the existence of a peripheral neuritis of the motor laryngeal nerves, possibly similar to what occurs in cases of paralysis of the portio dura (Bell's paralysis), and due to the same causes, that is, exposure to cold and "rheumatism."

(For permission to publish the following case I am indebted to the kindness of my colleague, Dr. Wolfenden, under whose care at the Throat Hospital I had the opportunity of examining the patient.)

CASE I. Complete Paralysis of Right Vocal Cord: Rapid Onset.—C. C., aged 25, in good general health, came for hoarseness and loss of tone in the voice. The history was of rapid onset after a severe cold, due to getting wet; she had rheumatism in the shoulder at the same time. There was subsequent dysphagia, due to swelling of the tonsils, but there was no pain in the throat at the time of onset of the aphonia; the condition has remained the same since. On laryngoscopic examination, the right cord was seen to be paralysed, both for adduction and abduction. The right ventricular band moved on adduction so as to somewhat cover the cord. The left vocal cord moved across the middle line on phonation, compensating for its fellow. The voice was slightly hoarse and deficient in tone. During the several months this patient was observed no change took place in the larynx.

Fig. 1.—Showing the position of the cords on phonation.

(The subject of the following case came to see my father on February 4th, 1891, by whom I was afforded the opportunity of examining the patient.)

CASE II. Complete Paralysis of the Left Vocal Cord; Rapid Onset.—Mr. N. came on account of hoarseness of four months' duration. There was a history of former syphilis, several years previously. He had also had rheumatism, and had lost the sight of the right eye from it.

The history of his aphonia was that he had lost the voice suddenly in November, 1890, while out for a walk in a cold wind. There was no pain in the throat at the time of onset or subsequently. The voice improved again slightly, but subsequently became worse some months later. On laryngoscopic examination, complete paralysis of the left vocal cord was found, both for adduction and abduction. The left arytenoid moved slightly on phonation; there was no swelling of the latter, but it stood out more rigidly than the opposite side. There was no sign of syphilitic disease in the throat. The voice was hoarse and somewhat forced, but was quite loud.

CASE III. Complete Paralysis of the Left Vocal Cord; Rapid Onset.—Miss J., a girl, aged 15, from Edinburgh, came to me in July, 1890, on account of alteration in the tone of the voice. She was otherwise in very good health. She had formerly suffered from enlarged tonsils and post-nasal growths, which had both been removed. The history was that the voice became rapidly affected after exposure to cold winds in the north of Scotland. No pain in the throat was experienced at the time. On laryngoscopic examination the left vocal cord

was seen to be completely paralysed, both for abduction and adduction. The right vocal cord moved across the middle line on phonation to compensate for the other side. The larynx was in every other respect healthy. Her voice was a little "whistling" in character, and she stated that she took breath less easily when reading aloud. It was on account of the deficient tone, and the fact of the voice easily tiring, that the mother brought the child.

Fig. 2.—Showing the position of the cords on phonation.

(For permission to publish the two following cases I am indebted to the kindness of my colleague, Mr. Mark Hovell, under whose care at the Throat Hospital I had the opportunity of observing the patients.)

CASE IV. Complete Paralysis of Right Vocal Cord: Sudden Onset.—W. P., aged 45 (?), came August 1st, 1889, for weakness of voice. The history was that the voice went suddenly in March while travelling by train from Oxford to Cheltenham, and being exposed to a draught from the window. The voice remained quite absent for three hours. On laryngoscopic examination the right vocal cord was seen to be completely paralysed, there being no manifest abduction or adduction. The left cord adducted, but failed to compensate fully for the opposite side, and the ventricular band moved beyond it on phonation. His voice was, at the time of his visit, clear, but

Fig. 3.—Showing the position of the cords on phonation. The dotted line indicates the position assumed by the ventricular band after the cord had adducted as far as possible.

weak and rather forced. In this case it was difficult to be certain how much the left vocal cord was affected as well as the right. The latter was completely paralysed, but the left adductor failed (unlike the other cases) to compensate fully for its fellow's want of movement, and the ventricular band came into play on phonation. The left vocal cord also appeared to abduct feebly.

CASE V. Complete Paralysis of Left Vocal Cord: Sudden Onset of Aphonia.—J. B., aged 58, an apparently healthy man, came May 5th, 1890, on account of inability to sing. He had formerly been a singer, bass voice, but now "could not sing a note." There was a doubtful history of syphilis thirty-five years previously. He had suffered from an almost constant

Fig. 4.—Showing the position of the cords on quiet inspiration. The left arytenoid was situated more anteriorly than the right, but this difference was lost during phonation.

pain in the back for some time previous to his throat trouble. The history of his aphonia was that the voice went suddenly while singing. On laryngoscopic examination the left vocal cord was seen to be completely paralysed, both for abduction and adduction. On phonation the right cord moved across

the middle line to compensate for its fellow. His voice was now slightly husky. Nothing was found in the chest to account for the paralysis or for the pain in the back.

Summary.—All these five cases present the same features—of rapid onset, of complete paralysis of one cord (in one case probably partially of both), of absence of pain or other symptoms in the throat beyond the aphonia, and failure of the condition to recover itself after several months. In no case was the patient seen till some time after the onset of the aphonia, so that it has to be assumed that paralysis was present from the first. The suddenness and completeness of the condition, the absence of pain or signs of inflammation, seem to point to the nerve origin of the disease. No explanation such as fixation of the arytenoid joint seemed probable, as there was no sign of implication of that region, and pain would have been sure to accompany the onset. There would probably besides have been some subsequent improvement. In three cases the paralysis was on the left side, in the other two on the right; in none of them was any chest sign present to suggest pressure on the nerve trunk. Enlarged thoracic glands might exist without being detected by physical examination, but the rapidity of the onset and want of chest symptoms seemed to negative this supposition. The absence of pain would be accounted for by the fact that the nerve involved is a motor one, unaccompanied by sensory fibres. A history of rheumatism was present in the first two cases, and in the last the pain in the back may have been of a rheumatic nature. Whether it is a rheumatic inflammation of the fibrous sheath of the nerve must remain at present undecided. Why laryngeal paralyses, other than of a functional nature, fail to recover, is so far unexplained; but such seems to be almost invariably the fact, except perhaps in cases of pressure on the nerve in which occasionally some slight improvement may take place with amelioration of the cause. Cases of bilateral paralysis of abductors with rheumatic history, present the same permanent character, and a tracheotomy tube can rarely be dispensed with after having been once inserted.

As regards other possibly allied forms of paralysis occurring elsewhere in the body, paralysis of the facial nerve would seem to present the most similar features. Dr. Gowers states:¹ "Neuritis is the cause of the common facial paralysis due to cold, and often termed 'rheumatic.' The majority of cases of facial paralysis seem to be of this nature, but very often no special exposure to cold can be traced.....There is little doubt that this condition is a neuritis within the Fallopian canal. The inflammation probably affects the sheath of the nerve, which swells and compresses the fibres, expansion outwards being prevented by the rigid bone.....In more than half the cases a special and considerable exposure to cold can be traced; generally a draught of cold air has blown on the side of the face and head, as in sitting in a railway carriage opposite an open window." Pain about the ear is very common at the onset. Dr. Bristowe also states:² "Inflammatory or so-called 'rheumatic' paralysis, is generally of rapid development, and is not necessarily attended with any pain or constitutional disturbance. Yet not infrequently the draught which caused the paralysis causes also earache or some neuralgic symptoms referable to the fifth nerve." It has been pointed out that the facial nerve in the aqueduct of Fallopius is exposed more than any nerve in the body to the influence of cold air blowing directly in the ear.³ No such analogy exists as regards the trunk of the laryngeal nerves, though the terminations of the latter are specially exposed to the influence of cold in respiration. In both the facial and the recurrent laryngeal the analogy exists of their being motor nerves, unaccompanied by sensory fibres. As regards prognosis, facial paralysis usually recovers, whereas laryngeal paralysis seldom if ever does so; in the former, however, recovery is very often incomplete, and, in rare cases, permanent.⁴

Cases of motor paralysis in other parts of the body have also been met with, such as the adductors and extensors of the thigh, after chilling of that region. In the eye, though paralysis of individual muscles not infrequently occurs from

syphilis, cases, it would seem, are never met with of sudden onset which could be properly classed as rheumatic or catarrhal.

As regards comparative pathology it appears that the horse is also subject to laryngeal paralysis of rapid onset after exposure to cold or great exertion. Mr. J. Penberthy, of the Royal Veterinary College, has kindly furnished me with the following facts concerning it. He writes: "I have known roaring follow very rapidly after exposure to cold; by very rapidly I mean to be observed in a week. Also horses pulling up roarsers after extreme exertion, that were never regarded as such previously. I am not, however, in a position to state that immediately before being so exerted the breathing was normal. There is an often quoted instance of Brigantine, a race horse. I have had two cases of extreme roaring occurring in an animal manifesting itself without exertion and passing off again after a few hours, to become observable again in a short time. This has been described as 'laryngismus stridulus.' My experience of *post-mortem* examination of roarsers is that I have most rarely been able to discover any pressure cause. The fact of horses which have been roarsers for any considerable time in the vast majority of cases showing trophic changes on the left side of the larynx only has naturally caused pathologists to regard of greatest importance the anatomical differences of the left and right recurrent laryngeal nerve, and consequently to locate the mischief in that part of the left nerve (recurrent) below the situation corresponding to the giving off of the common root of the posterior cervical and the anterior dorsal arteries. Here, I am bound to say, I have looked, most often in vain, for any pressure cause."

The horse thus seems to furnish the pathological evidence, which is difficult to obtain in man, that paralysis of one of the recurrent laryngeal nerves may occur without any pressure cause being found to account for it. In chronic cases the nerve presents a shrunken appearance.

ABSTRACT OF AN ADDRESS ON

THE SUPPOSED CURATIVE EFFECT OF OPERATION PER SE.¹

Delivered before the Surgical Section of the New York Academy of Medicine.

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Dr. White stated that his attention was first directed to this subject by his experience with the operation of trephining for so-called traumatic epilepsy.

During the past five years—with Dr. D. Hayes Agnew—he had trephined in fifteen cases of supposed traumatic epilepsy. All but one recovered from the operation. The patient who perished was an imbecile, and a confirmed drunkard as well as an epileptic. Death occurred from suppression of urine, probably secondary to etherisation.

In one case a bullet was found imbedded in the brain substance, in another an irregular portion of the internal table was dissected out from beneath the dura mater, to which it was attached by cicatricial adhesions. In another there were projecting spicules of bone on the internal surface of the button removed and the adjacent portions of the skull. In two marked sclerosis and thickening of the cranium were observed about the field of operation. In the remaining cases nothing abnormal was seen. Although this was the case, they were, without exception, markedly improved by trephining; in two instances even to the point of apparent cure, no return of symptoms having been observed for eighteen months and for two years after the operation. In the other seven the results were strikingly favourable, convulsions disappearing for

¹ *Diseases of the Nervous System.*

² *The Theory and Practice of Medicine.*

³ *Hutchinson's Archives of Surgery*, vol. iii.

⁴ Gowers, *loc. cit.*

¹ The full text of the address will be published in the *Annals of Surgery* for August, 1891.