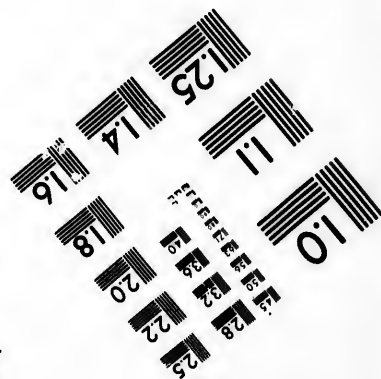
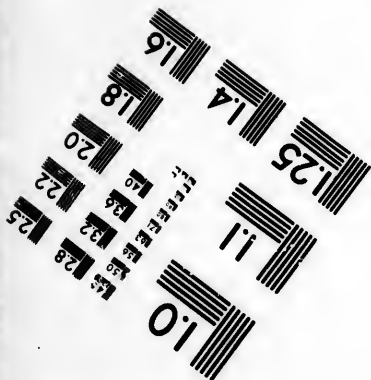
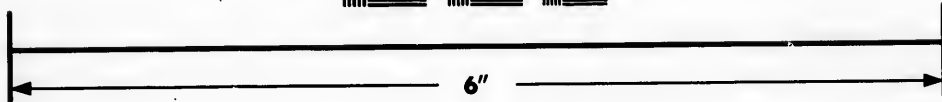
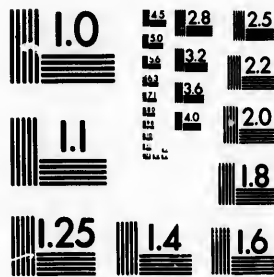


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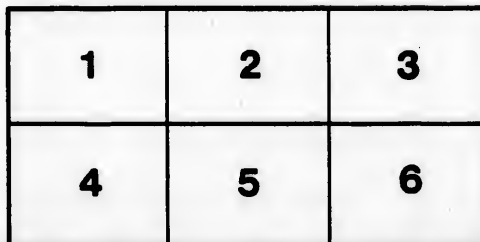
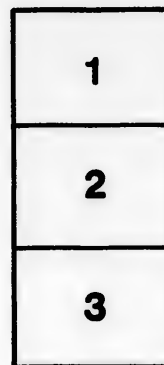
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CLINICAL REMARKS ON THREE CASES OF DIFFERENT VARIETIES OF NUCLEAR PARALYSIS—BEING THE SUBSTANCE OF TWO CLINICAL LECTURES DELIVERED IN THE ROYAL VICTORIA HOSPITAL, MONTREAL, DURING DECEMBER, 1898.

BY

JAMES STEWART, M.D.,

Professor of Medicine and Clinical Medicine, McGill University ; Physician to the Royal Victoria Hospital.

(Reprinted from the Montreal Medical Journal, January, 1899.)

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CASE I.

Chronic Progressive Ophthalmoplegia Externa.

The patient before you entered the Ophthalmological Department some time ago, and through Dr. Buller's kindness he was transferred to the Medical Department.

He came complaining of double vision, which he says has troubled him at intervals for nearly three years. The intervals of freedom varied much in duration, from days to months. For some time he was annoyed with drooping of the right upper eyelid. He is a fairly well-developed, healthy looking man, 36 years of age, and is able to carry on his work, which is that of a machinist. Sixteen years ago he had a chancre which was followed by a rash over his body. He has used both alcohol and tobacco in excess. He makes no complaint of any pain in the head, nor does percussion over the scalp elicit any pain or tenderness.

With the exception of the ocular paralysis nothing abnormal is noticeable.

Dr. Buller found, when he first examined him some weeks ago, a nearly complete ophthalmoplegia externa. The inferior recti still retained some power, especially that of the right eyeball.

Vision of right eye $\frac{6}{6}$ —L. $\frac{3}{6}$.

At present it will be noticed the paralytic state of the ocular muscles remains the same.

All the structures supplied by the 3rd nerve are paralyzed, except the inferior recti, the levator palpebræ and the pupillary fibres. There is an appreciable difference between the right and left upper lids, the former presenting a slight droop, while there is complete dower over the left lid. He is able to accommodate, but there is

diminution of the normal contraction to light. The external recti and the superior oblique are almost completely paralyzed.

The distribution of the paralysis renders it almost certain that the morbid process is situated in the nuclei of origin and not in the root fibres or in the nerves in their course. The involvement of certain parts of the 3rd, while others have escaped, cannot be readily explained in any other way.

The third nerve arises from a series of nuclei, the most anterior of which presides over the mechanism of accommodation, while behind this we have the nucleus which has to do with the contraction to light. The two nuclei are near and form what is called the anterior group of third nerve nuclei.

Whether there are separate nuclei for each external muscle supplied by the 3rd, is a question. Very exact descriptions of such are usually described in the text-books, since Kahler and Pick published their observations. We, however, have no anatomical proof that each muscle has its own definite nucleus. All that can be said is that there is a posterior group of nuclei belonging to the 3rd nerve, but in our present state of knowledge these cannot be arranged in a definite order. In close proximity to the posterior group of the 3rd nerve nuclei, we have the nucleus for the fourth nerve, and still further back the nuclei for the 6th nerve, both of which are here involved.

We have bilateral disease of the nuclei of the fourth, sixth, and those nuclei of the third which supply the external muscles of the eyeball, except the inferior recti and levator palpebræ of one side.

There are no symptoms pointing to any further cerebral changes. The bulbar nuclei are normal as far as can be made out. There are no symptoms of tabes or any other spinal affection. Nuclear ophthalmoplegia is sometimes a part of tabes and sometimes of general paralysis, but here it appears to be the only evidence of disease in the central nervous system. It is quite possible that tabes may eventually develop, but at present there is no evidence of a morbid process other than that situated in the third, fourth and sixth nerve nuclei.

It is instructive to observe how the degeneration has involved structures which have similar functions, although separated, while more contiguous tissues of a similar nature, but different in function, have escaped. The fifth and seventh nuclei, for instance are intact, while lying nearer the third and fourth nuclei than do the sixth nuclei.

What has been the exciting cause of this progressive degeneration

in the nerve nuclei? We have a history of both syphilis and alcoholism. It is recognized that alcoholism is an exciting cause of an acute form of nuclear-ophthalmoplegia, a very rare disease, which runs a rapid and usually a lethal course, but I am not aware of any evidence to show that it gives rise to a slowly progressive form of ophthalmoplegia. The after effects of the syphilitic poison is the greatest of all factors in bringing about chronic degeneration of nerve nuclei, an eminent example being the degeneration of tabes. It is reasonable to conclude that here we have to do with post-syphilitic degeneration.

Seeing that the condition has been in existence for three years the prospects of even a stay in the progressive character of the downward tendency are not bright. It is not likely that an anti-syphilitic course will help to check the advance of the degeneration, judging from the effect of similar treatment in tabetic degeneration.

For several weeks iodide of potassium has been given and it is the intention to continue its use for some time.

CASE II.

Chronic Bulbar Paralysis.

Miss E. B., aged 44, a seamstress, has had good health, with the exception of an attack of diphtheria at 15 and of acute rheumatism at 20. The latter did not involve the heart, otherwise there is nothing to note in her past or in her family history.

She dates the onset of her present troubles to Feby. 1895, when a few minutes after exposure to a cold wind, she felt a chilly sensation at the back of the head, which was at once followed by some difficulty in speaking.

The following day, she had less trouble in articulating, but a few days after there was return of the difficulty in speech and she now noticed for the first time a difficulty in swallowing both solids and liquids. Since the onset of the above symptoms, both the patient and her friends have noticed great variability in their intensity, at times she appeared to articulate well and have but little trouble in swallowing, but on the whole there appears to be progressive tendency, although very irregular in its progression, to a more marked disability in both speaking and in swallowing. For upwards of two years she has been annoyed with saliva drooling over the lips.

Her speech has a nasal twang. The chief difficulty appears to be with the labials.

Her tongue is atrophied, and she is unable to protrude it but very slightly beyond the lips. It is the seat of fibrillary twitchings. She has not complete control over the muscles of the lips. She is for

instance unable to press them firmly together. The palatal muscles are weak, fluids are often returned through the nose, and there is a very marked difficulty in not only swallowing liquids, but also solids.

There is a bilateral paresis of the laryngeal abductors, not sufficient, however, to give rise to stridor or other symptoms of laryngeal stenosis.

We have then in this case the following changes :

1. A marked paresis of the tongue with wasting of its muscular fibres shown by inability to protrude it, and the imperfect articulation of the lingual consonants.

2. A paresis and wasting of the orbicularis oris muscles, shown by the imperfect closure of the mouth, and the inability to correctly pronounce letters requiring the action of this muscle.

3. Paresis of the muscles of the palate resulting in regurgitation of fluids through the nose and nasal twang to speech.

4. Paresis of the muscles of the pharynx, causing a great difficulty in swallowing both liquids and solids.

5. A paresis of the abductors of the larynx, which has not as yet caused any distressing symptoms of laryngeal stenosis.

The following nerves are involved :

1. Part of the 7th.

2. The whole of the 12th.

3. Part of the 11th.

4. Parts of the 9th and the 10th.

That part of the 7th nerve which innervates the orbicularis is involved. Some observers contend that this muscle receives its supply from a nucleus immediately in the neighbourhood of the 12th nucleus, or even from the latter itself, but recent observations tend to show that the lower facial muscles receive their motor supply from the main facial nucleus, while the upper facial muscles are innervated from a nucleus situated higher up.

The laryngeal and palatal paralysis arise from an involvement of the bulbar part of the 11th nerve. While the pharyngeal difficulty is due to involvement of either the 9th or 10th, or both. It is not definitely known from which nerve the pharyngeal muscles receive their motor nerve supply.

We have to do here with a bulbar paralysis. No doubt symptoms of a more or less similar character may arise from a morbid process at the base of the brain, damaging the nerve roots as they emerge from the pons and medulla. The nerve roots of the 10th, 11th and 12th are very close together as they emerge from the brain, but a growth

in such a situation would almost certainly damage also other structures, thus giving rise to symptoms distinctive of the situation.

The accompanying diagram shows the chief nerve nuclei situated in the medulla and pons. It will be seen that there is a common nucleus for the 9th, 10th and 11th nerves, the nucleus for the latter being situated at the lowest part of this common centre. The nucleus for the 12th is close to and parallel with the 11th nucleus, while the 7th nucleus is only a little above the upper end of the 12th nucleus.

The symptoms are dependent on loss of function in parts of the 7th, 9th, 10th, 11th and in the whole of the 12th.

What has brought about this loss of function, in the bulbar nuclei?

I. By far the most common cause of chronic bulbar paralysis is a slowly progressive degeneration of the nuclei.

This is a well recognized disease met with most frequently after the age of forty. Little is known about the causes that give rise to it. It is not infrequently a part of a more diffuse but similar morbid process in the ventral cells of the spinal cord.

II. Bulbar paralysis may arise suddenly from plugging of the vessels that supply this part of the brain with blood. In this form we, however, rarely meet with softening confined wholly to definite nuclei. Other parts are generally robbed of their blood supply, so that additional symptoms to bulbar paralysis are found to be present.

III. A rare form of bulbar paralysis from acute inflammation of the bulbar nuclei.

IV. Cases of bulbar paralysis have been described where no lesion or change of any kind has been found after death. The disease has an intermittent character and is at first chiefly characterized by a marked weakness of certain muscles after exertion, but which disappears after rest. This disease known by the name of asthenic bulbar paralysis, soon involves other nuclei than those innervated from the pons and medulla.

V. We may have a disseminated sclerosis only involving the bulbar nuclei.

VI. Finally, pseudo-bulbar paralysis from bilateral lesions of the tracts between the cortex and nuclei.

It is difficult to arrange the case before us in any one of the above classes. We have to do with a condition setting in suddenly, irregularly, but still slowly progressive and with the intensity of the symptoms varying much from day to day. Were it not for the fact that there are no symptoms other than bulbar it would conform more closely to the fifth variety (a limited disseminated sclerosis) than to any of the other forms.

We may exclude without discussion, the 2nd (hæmorrhagic, embolic or thrombotic bulbar paralysis); the 3rd (acute bulbar myelitis); the 4th (asthenic bulbar paralysis); and also the 6th (pseudo-bulbar paralysis).

The history of a sudden onset with very slow progress are against its being of the first mentioned or progressive degenerative variety. It is just possible that the patient is mistaken as to the suddenness of onset. How frequently we meet with histories of sudden onset of disease, when we know that there must have been symptoms in existence for some time! It is the effects of the *last straw* that makes the impression on the patient's mind. She may have had slight difficulty in articulation without its being noticed by either herself or friends. The fact that the trouble has been slowly progressive certainly points to a degenerative lesion, but whether this is a pure atrophy of the nuclei or a sclerotic process, limited to the nuclei or their immediate neighbourhood I don't think we have sufficient evidence to determine. The diagnosis as to the nature of the bulbar paresis rests I believe between these two varieties of lesion. The course in the future will determine the nature of the morbid process.

CASE III.

Chronic Progressive Spinal Muscular Atrophy involving parts of both the Cervical and Lumbar Cords.

The man whose case we will now consider is 36 years of age and a weaver by occupation. Six years ago his left forearm was injured in machinery, and an abscess developed on the ulnar side of the forearm, about midway between the wrist and elbow joints, which was opened. In the course of two months the wound had healed, but it was noticed that there was wasting of the muscles of the hand. The wasting steadily increased for two years, but he thinks there has been but little increase during the past four years. Five years ago he found that the muscles of the left leg were weak, his toes catching any object above the usual level. A few months ago he first noticed the weakness and wasting of the small muscles of the right hand. Although there is marked wasting of the muscles of the right thigh, the patient was not aware of this until his attention was directed to it after his admission into hospital.

At 21 years of age, and again at 27 years, he had attacks of pulmonary hæmorrhage. One brother and four sisters died from consumption. There is no history of either syphilis or alcoholism. With the exception of the muscular wasting described, he does not present any evidence of disease.

Left Upper Extremity.—There is extreme wasting of the thenar and hypothenar eminences, of the interossei and lumbricales. The hand is a typical example of the "Claw Hand." The muscles on both surfaces of the forearm are much wasted as compared with those of the right forearm, the circumference of the left forearm, a few inches above the wrist, being an inch less than the corresponding part of the right forearm. There is no disturbance of any form of sensibility in the left hand and forearm. The reactions to the faradic and galvanic currents are nil in the wasted muscles. There is no wasting in the upper arm or shoulder muscles.

Right Upper Extremity.—There is wasting of the thenar eminences and of the first and second interossei. This was first noticed a few months ago. The wasting is progressive in character, but has not involved to any appreciable extent the muscles of the forearm. There is some wasting, however, in the upper arm, the difference in circumference between it and the corresponding part in the left upper arm being half an inch. The difference between the two upper arms is also distinctly to be made out on testing the power of the muscles. The faradic reaction of the muscles is lessened but very slightly. There is no disturbance of sensibility.

Left Lower Extremity.—The patient makes the complaint that when walking he is unable to lift the toes of the left foot as well as those of the right. This difficulty is very apparent when he is asked to walk across the room. There is slight wasting of the anterior group of muscles of the leg. There is no disturbance of sensibility.

Right Lower Extremity.—There is marked wasting of the muscles of the right thigh, the circumference at the middle being an inch and a quarter less than the circumference at the same part on the left side. There is a distinct diminution of both the faradic and galvanic reactions, but no objective or subjective sensory disturbance.

Reflexes.—The knee jerk cannot be brought out on the right side. It is, however, normal on the left. Ankle clonus is not noticeable in either limb, but a rectus clonus is present in the left limb. The superficial and organic reflexes are normal.

Fibrillary twitchings are to be seen in all the atrophied muscles, and also in those of the left thigh. There is no disturbance of co-ordination or special senses.

The condition present in this patient may be summarised as follows :

- I. A progressive wasting of the muscles of the left hand and forearm extending over six years.
- II. A slight wasting of the interossei and of the thenar and hypothenar groups of the right hand extending over a period of 6 months.

III. Wasting of the muscles of the anterior surface of the right thigh.

IV. Wasting of the muscles of the anterior surface of the left leg.

The state here differs considerably from the usual course met with in cases of progressive muscular atrophy.

1. In the prolonged course. The atrophy was marked in the left hand for some years, before there was any evidence of wasting elsewhere.

2. In the involvement of a limited but different set of muscles in each lower extremity. It is not usual for progressive muscular atrophy to involve the lumbar enlargement as is here present.

3. The different groups of muscles of the lower limbs which are the seat of the atrophy is likewise an unusual feature. The atrophy is generally symmetrical.

From the course and distribution of the atrophy and the entire absence of sensory disturbance we must conclude that the disease is spinal and not peripheral in origin. We have to do with a wasting of the anterior cornual cells in the lower cervical enlargement and at two different levels in the lumbar cord.

There is nothing that we can find in this man's past history that gives any clue as to a probable exciting cause, except the presence of an abscess in the ulnar border of the left forearm: shortly after its discharge, atrophy of the small muscles of the hand were noticed. Whether this is an accidental coincidence or not, it is impossible to say. A number of cases of spinal muscular atrophy have been reported following injury to a limb, the atrophy starting in the injured member and afterwards becoming general. It is not rare to meet with cases of other degenerative diseases of the central nervous system, where the starting point appears to have been a traumatism either general or local.

It would seem as if the nerve elements when injured suffer in their nutrition, and when once started, the degenerative process may extend far beyond the original influence exerted by the traumatism. There is no history of either alcoholism or syphilis. It is rare, I think, to meet with cases of spinal atrophy due to the syphilitic poison. Such cases do occur, but they are very rare as compared with the frequency with which the syphilitic poison attacks the sensory side (tabes) of the cord; rarer also than cases of ophthalmoplegia from syphilis.

We cannot tell why the poison of syphilis should select, as it were, more readily the nuclei of the ocular nerves, and rarely the bulbar and anterior cornual cells.

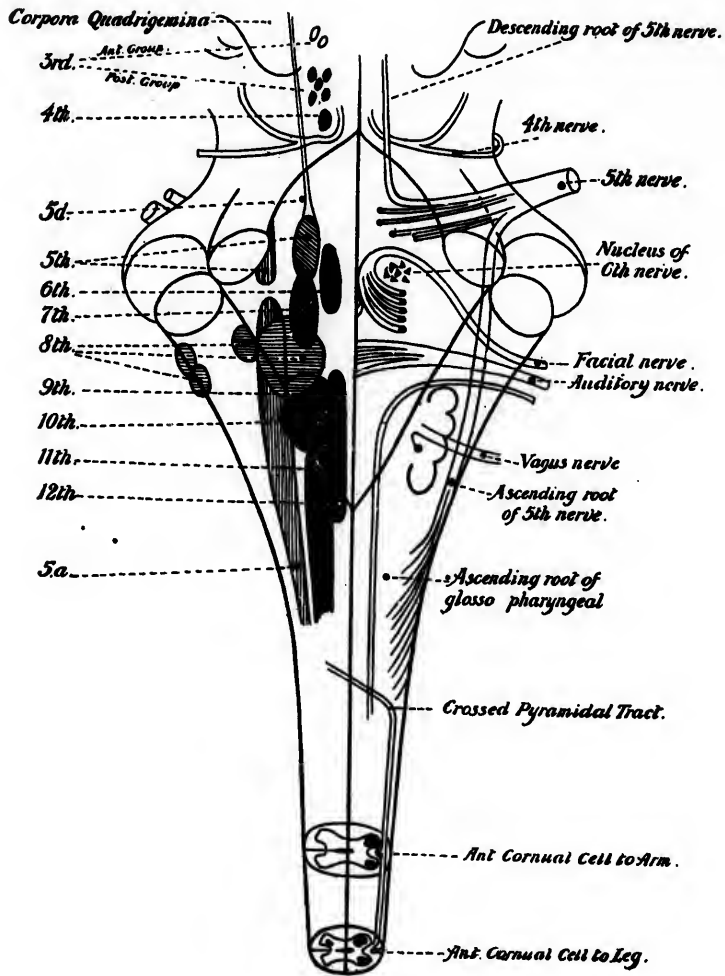


Diagram to illustrate :—

I. The relative positions of the 3rd, 4th and 6th nerve nuclei, the nuclei involved in the first case. The two groups of third nerve nuclei are shown, the posterior are deeply shaded to indicate their degeneration, while the anterior are shown in outline. The 4th and 6th nuclei are also shaded.

II. The various pontine and bulbar nuclei. The 7th, 9th, 10th, 11th and 12th are deeply shaded, to represent the supposed condition present in the second case (chronic bulbar paralysis). The whole of the main nucleus of the 7th nerve is shaded, which is not strictly correct. The same holds true with the 9th and 10th nuclei, which, like the 7th, can only be partially degenerated.

III. The course of the crossed pyramidal tract in the spinal cord and its relations to the anterior cornual cells in the cervical and lumbar enlargements. These cells are shaded to represent their degeneration, the condition of things as they are supposed to be in case No. III.

The nuclei are represented as if seen through transparent material.

