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A CASE OF CROSSED HEMIDYSERGIA AND TREMOR, WITH ASYNERGIA OF BINOCULAR MOVEMENTS: COMMENTS.

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THE following case seems worthy of publication, as it may in the future afford material with which to answer the problem of the anatomical distribution of the cerebello-fugal systems comprised in the rubro-spinal and vestibulo-spinal tracts.

In some respects the patient conforms to the syndrome of Foville (1858) as described by Grasset before the Neurological Society of Paris in 1900. This type consists of a crossed paralysis of the limbs on one side of the body and of the face on the opposite side, along with the loss of power to rotate the eyes to that side. My case, however, differs from the Foville type in that he presents no paralysis, but a most marked dysergia, which, however, in its distribution, is the same as that of the paralysis in the syndrome of Foville.

J. T., colored, aged thirty-two, a laborer, was seen at the Emergency Hospital Clinic of Dr. Dudley Morgan in April, 1908. He complained of inability to walk steadily, and clumsiness of the left hand. While walking in the streets, "silly thoughts," chiefly about wealth, occupied his mind.

The previous and family history is negative so far as can be ascertained in his present slow-witted state.

Present illness. — About six months ago, he began

to feel weak and dizzy, and had severe pains in the loins and back of the head and neck, intense enough to keep him awake at night. In this state, he dragged through his work for about three weeks, until he was sent to bed under medical orders, and there he stayed about two months in a lethargic state. After this he was able to sit about the house and to walk a little, but could not return to work for some little time. He had complained of a " misery in the head " after taking his medicine, or when alcohol was given.

Later he returned to work, and states that he could walk quite well and did not tremble. About two months ago, the left hand became clumsy, swallowing was difficult, and the foolish thoughts, generally vague, though about money, began to occupy his mind. About a month ago he began to tremble, and walking became difficult.

Physical examination. — Motility: Face — at rest, there is slight asymmetry of the face, but no drawing of the mouth nor smoothness of the brow, such as occurs in facial paralysis. When movements are made, however, a violent intentional tremor occurs in the right lip, cheek, jaw and tongue, and there is poor control over the mouth. This tremor is at first very extensive and violent; but when the position is maintained for some seconds, the excursion of movement becomes less extensive and more regular, its frequency being about eight to ten per second. The left side of the face does not tremble, but is, of course, moved by the communicated oscillations from the right side.

The speech is slow, slurring, indistinct and thick.

The eye movements are unsteady and difficult to control; but there is no nystagmus. The pupils react well to both the light and accommodation. The movements of the left arm are slow, clumsy and inaccurate, and there is marked adiadocokinesis. Intentional movements are accompanied by a tremor similar in character to that of the face. It ceases when a position has been maintained over ten seconds. There is no muscular weakness. The movements of the left leg are still more incoordinate, and it also drags upon walking He walks " *aux petits pas.*"

The movements of the trunk are slow, especially at commencement, and poorly maintained, and there is a rigidity which seems to prevent ready bending movement of the right side. The eyes are also unsteady when turned to the right. On protrusion of the tongue, its right side is agitated by a similar tremor, which almost ceases in about ten seconds, after which interval there is also more control over the facial trembling.

In the left arm there is slight asthenia, considerable impairment of the attitude sense, decided clumsiness and marked impairment of the diadocokinesis. There is also an intention tremor, finer than that of the face, and not maintained more than ten seconds. The general sensibility is very acute and there is no true muscular weakness.

Rotation tests show no anomaly of vestibular sense; the pupils react well to light and accommodation.

The movements do not tend to continue when the resistence is removed, and there is recoil. There is no atonia.

The sensibility is not impaired, with the exception of the notion of attitude in the left arm. There is no anomaly of discernment of rotation.

Reflexes are exaggerated on the right side; but there is no flexion of the great toe on stroking the sole.

Psychic examination was not systematic; and, beyond his dull and confused state, I could find no definite gaps in the memory, attention, perception or judgment. There had been causeless crying, and he had been generally miserable. He stated that there had been no automatic laughter, rage nor emotionalism in general. As to the will, he had felt useless and incapable, but his condition justified this attitude.

Progress.— On April 23, after two weeks of potassium iodide, he was in every way worse and had developed dissociation of the ocular movements. On protrusion, the whole tongue jerked violently forth and back. The control over the facial movements was much impaired. The arms shook violently, and the walk was very difficult. All the movements can be performed, however, when he is emphatically urged.

The speech is very uncertain, and toneless.

When binocular fixation is attempted, asynergic dissociation occurs. The kaleidoscopic series of squints which ensues produces a most weird effect. When either eye is closed, however, the other can perform all movements, although the right eye only does so with much difficulty and wide tremor. On looking widely to the right with both eyes open, the left eye moves across normally, while the right fails to follow sufficiently. The same phenomenon occurs with the left eye when the gaze is directed towards the left. In each case, vehement urging overcomes the motor incapacity, and the eyes for the moment fix the same spot, the laggard, however, quickly relapsing. When he endeavors to fix with his gaze a point 18 inches distant, he sees double, and the optic axes are noticed to converge.

April 30 he had been taking hydrargyri iodidum gr. $\frac{1}{3}$ t.i.d., and can now converge and externally rotate the right eye in binocular vision with less difficulty and urging than before, though he cannot maintain the position completely and though diplopia still exists. The diadocokinesis and incoordination of the left leg and arm have improved, and he can better control the protrusion of his tongue. The face trembles less, but his attempts at grimaces are accompanied by associated movements, especially in the left finger. There is no apparent asynergia revealed by the leaning back or chair-mounting tests.

May 4 the ocular movements are better performed; there is less ataxia. The speech is clearer, but the diadocokinesis is still much impaired.

Nov. 15. He failed to return to the hospital during the summer, and the mental symptoms and trembling became much worse, but two months ago a local practitioner accidently treated him, and caused a remission of the *debacle*. When examined, he presented much less tremor of the right cheek, though now the whole tongue trembled coarsely, somewhat in the same manner as, though less extensively than, on April 23. The movement is more of a dysmetric than of a trembling kind. The control over his facial muscles

in general is poor, though he can succeed after some effort to elevate and depress the eyebrows. He is unable to whistle. There is no longer dissociation during binocular vision, and all the movements can be effected, although with slowness and difficulty.

The control of the limbs, more especially the left, is slow, hesitating and trembling. The right hand when at rest on his knee exhibits an oscillatory tremor like that in paralysis agitans. This he can inhibit quite easily. When he extends the arms, they move up and down rhythmically through a space of about an inch, and at a rate of about six per second. With the left hand and arm he is particularly clumsy, failing to button his coat; and when he endeavors to touch any distant object, moves it with a series of jerks. The speech varies, sometimes being thick and indistinguishable, and sometimes quite clear. The tendon reflexes are much exaggerated. The examination was very difficult on account of his suspiciousness and unwillingness to make the movements requested. His parents state that he can do light work in the house, but that he loses himself in the streets and sometimes laughs without cause, though he never cries unless beaten, as for disobedience and gluttony, for he has become very voracious.

The outstanding features of the case are hemi-asynergia and intentional tremor of the left arm and leg, intention tremor and possible asynergia of the right face, and tremor of the right tongue. Along with these occurred a dysarthria and exaggeration of the reflexes, a fugitive diminution of the attitude sense of the left arm and a temporary dissociation of the ocular movements and apparent enfeeblement of the movements of the right eye, more especially in levo-gyration.

It is unlikely that the pyramidal tract is seriously if at all affected, for the sign of Babinski is not present and there is no true spasticity nor inability to perform any movement, the

incapacity merely extending to the *co-ordination* of several muscular contractions into a determined act. This attempt on the part of the left hand and right face and tongue always provokes tremor.

Now lesions in the neighborhood of the nucleus ruber [1] are known to provoke tremors without in any way implicating the pyramidal tract. They do so by interrupting the impulses which descend in the cerebello-rubro-spinal tract. The tremors are usually, however, mono- or bilateral, and this is the first case of which I am aware which presents a syndrome comprising a crossed tremor due apparently to a single lesion.

I venture to explain the occurrence by the implication of another system of fibers, viz., the ponto-olivary, which traverse the median raphé on their way from the nuclei of the pons and which are thought to receive their afflux through the cerebello-fugal fibers of the middle peduncle. If those of the left system were implicated on the left side while en route to mediate the kinetism of the facial, trigeminal and hypoglossal nuclei, the lesion which interrupts them would necessarily be upon or near the course of the rubro-spinal tract, which mediates the kinetisms of the limbs and trunk of the crossed side of the body. It crosses, however, in the decussation of Forel almost immediately below the red nucleus, and composes the ventral of the two decussations of that region. Hence, in the aforesaid situation in the brachium pontis, it has already crossed and is on its way to mediate the muscular acts of the side of the body upon which it is now situated. Thus the lesion, if in the left medullary-pontine region, would implicate at the same time the left rubro-spinal

tract en route to the left limbs and trunk at the same time as it interrupted the impulses as they passed across from the nuclei of the pons to govern the movements of the opposite right side of the face, jaw and tongue.

There may be much uncertainty in the interpretation of symptoms in any particular case where the cerebellar apparatus is implicated. In the case before us the tremors are very significant of an impairment in the course of the cerebello-rubrospinal efferent system, as has been shown by the cases reported by Gordon Holmes.[1] In some of his cases, however, the lesion was shown to be more widespread by the additional symptoms, such as a definite oculomotor palsy, outbreaks of crying or laughter without external cause, a rigidity of half the face much resembling that in paralysis agitans, and the hesitating steps of the *marche aux petits pas*.[1]

The emotionalism arises when the ventral thalamic nucleus is affected. This is just a little forward and to the middle of the nucleus ruber, the part of the cerebellar system implicated in many of Holmes' cases. The oculomotor palsy is, of course, due to interruption of the fibers of the third nerve, which run just internal to the nucleus ruber and through it. The short steps are due to the timidity which comes from the disturbance of the patient's control over synergic movements; for though he can make volitional simple movements as easily as before, yet he has lost the power of combining the movements of different joints into harmonious act, except by the exercise of much volition. This requires close attention, which in turn rapidly induces fatigue, one of the prominent symptoms of defect in any part of the cerebellar apparatus.

Rigidity of the face is, on the other hand, due to an exaggeration of cerebellar influences, either by the cutting off from the cerebrum, or by a lesion which stimulates the efferent fibers of the cerebellar system.

The monotony and tonelessness of the speech again are derived from diminished cerebellar influx. In these cases, speech, like the other movements, is often begun with great slowness and difficulty, and it also quickly tires.

The muscle rigidity may affect the trunk, and may take the form either of the bowed forward attitude seen in Parkinson's disease or, on the contrary, the forced extension and even complete opisthotonos shown by some of Hughlings-Jackson's early cases and one of the author's. We do not yet know what determines these attitudes respectively, though we do know that section of the brain stem between the colliculi produces the state of decerebrate rigidity,[2] of which opisthotonos is a feature. The cause of this is the unrestrained exercise of cerebellar afflux, through interruption of the afflux from the neopallium. The tonic nature of the influence exerted by the metencephalon is shown by the character of the fits caused by intravenous injection of absinthe [3] before and after removal of one cerebral hemisphere. While the pallium is intact, the whole convulsion is a clonic one similar to that in a convulsive phase of an epileptic seizure. After one hemisphere has been removed, the opposite side of the body manifests a rigidity similar to that in the tonic phase of the epileptic fit, while the other side of the body is convulsed, as before, clonically.

Very different is the state caused by *destruction* of the cerebellar apparatus; here muscular flaccidity dominates. It is often so severe that

the patient cannot sit up, although individual movements are performed quite well, through the agency of will. Its distribution is homolateral, and it has no relation with the state of the deep reflexes. A useful clinical test in the limb is for the observer, after resisting the patient's movements, to suddenly cease to do so. If cerebellar atonia is present, the patient's limb continues its movements to the extreme point, and there is no recoil.

The tremor in these cases is purely kinetic, but does not always increase towards the end of the movement.

As regards attitude, a very characteristic one is that where the occiput is rotated towards and pulled down to the homolateral shoulder, the face thus necessarily looking upwards and to the side away from the cerebellar lesion. The eyes, however, look in the reverse direction back towards the lesion, or in some cases present skew deviation. This consists of the homolateral eye turning inwards and downwards, while its fellow looks outwards and slightly upwards. Horsley has found [4] this experimentally producible by stimulation of a spot in the neighborhood of Deiter's nucleus, which is known to send decussating fibers into the posterior longitudinal fasciculus, the coordinating path of the ocular movements.

The functions of the cerebellum are both static and dynamic. It is a center of energy and of reinforcement for all movements. It maintains tonus, and is necessary for the harmonious co-ordination of separate movements. Interference with its functions, therefore, leads to asthenia, atonia and astasia.

These perturbations are more manifest when the subject is deprived of ocular and vestibular sensorial aids.

The cerebellar syndrome may be summarized in its clinical aspect by calling it a dysbasia with relative integrity of isolated movements. The walk is not exactly ataxic, for the person does not throw out his legs as does the tabetic; it is that of the drunkard. The upper limbs show incoordination; and clumsiness is often shown in the writing. The speech is generally slow and scanning. This is due to the loss of the tonus-regulation, which normally takes up the slack of the muscles (so to speak) so that they respond quickly to nerve impulses, having little inertia to overcome.

This normal diadocokinesis cannot be accomplished when the cerebellum or the tracts which emanate from or lead to it are interfered with. The lack of tonus, too, is the cause of the asthenia, and the fatigability is due to the need of constant cortical intervention for the regulation of movements. This connotes volition, which is the most quickly tired of all functions. This atony may be so extreme as to prevent walking, the patient falling in a heap when placed upright.

A valuable clinical law is that the fall is towards the side of the lesion. When the head is drawn to one side, it is also generally but not always towards the lesion; but the face will look to the opposite side, as the head is rotated by the sternomastoid of the affected side, and hence must look in the opposite direction. The eyes, however, when turned, look back towards the lesion. An apparent exception was shown by Gordon's four cases.[5]

Asynergia [6] is shown firstly in walking by the trunk not following the limbs. Secondly, in standing, if the patient is pushed backwards, the lower limbs remain fixed even without the

bending of the knees and ankles, which naturally automatically tends to maintain equilibrium in the normal person. Thirdly, on endeavoring to sit up, the thighs flex and the heels fly up in the air, and, fourthly, in touching with his foot an object above and in front of him, the patient does so by the movement of the thigh succeeding that of the leg, and jerking like a mechanical doll. Fifthly, by intentional tremor of the hand and arm,[9] and of the ocular movements (nystagmus). All this is generally combined with an exaggeration of the power of static equilibrium.

The deep reflexes may be exaggerated on the affected side.

It is very important diagnostically to determine the complete integrity both of the sensibility and the intelligence, for lesions of the frontal lobe sometimes give rise to a dysbasia not very different from cerebellar titubation.[7] When a neoplasm is in question, the situation of the headache and tenderness on percussion should not receive too much stress, though they are valuable if they do not conflict with the physical signs. When it occurs, the implication of cranial nerves and the sensory tract is usually upon the side of the lesion, though in one extraordinary case of Guldenarm [8] there was left facial palsy, neuralgia, mydriasis and tendency to fall to the left, due to a fibrous sarcoma below the right tentorium. It was the size of an apple and crowded the pons against the left side of the basi-occiput, thus causing the contra-lateral symptoms.

Tumors of the vermis are apt to cause falls backwards, stiffness and extension of the neck, and opisthotonos, along with the foregoing symptoms.

But there was hardly a question of a tumor in our case, for it was most unlikely that so much retrogression could have occurred in symptoms affecting so important a region as the bulb-pons had a new growth been present. Moreover, mercury seemed to be the beneficial agent. Although no lumbar puncture was permitted and the Wassermann test was not then available, yet syphilis was a very probable cause of the symptoms. A basal meningitis causing the abundant exudation followed by absorption so characteristic of the Frascatorian lues could have interfered with both abducens nerves and prevented external movement, but such a lesion could not account for the loss of binocular movements, while those of the eyes singly were retained. Hence, even though meningitis was probably present, it is certain that central lesions also existed in very close proximity to the fasciculi which mediate the activity of the nuclei of the ocular nerves. Indeed, the ocular dysergia was merely a part of the general dysergia syndrome, and, like the latter, was not complete, but was mainly homolateral.

The permanence of the symptoms make it likely that the destruction or interruption of tracts in the mid-brain and isthmus were due to gummatous or arteritic ischemia, a common enough phenomenon, and the interest of the case lies in the rarety (for I have found none other like it) of an attack of the efferent co-ordinating mechanism of the cerebellar apparatus, the distribution of which attack resembles that of the syndrome of Benedict (type Foville) where the cerebrospinal system is concerned.

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