A note on excessive patellar reflex of functional nervous origin, and especially the 'trepidation' or 'spinal epilepsy' form / by F. Parkes Weber.

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A NOTE ON EXCESSIVE PATELLAR REFLEX OF FUNCTIONAL NERVOUS ORIGIN, AND ESPECIALLY THE "TREPIDATION" OR "SPINAL EPILEPSY" FORM.

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I THINK that too much value is sometimes attributed to the phenomenon of excessive knee-jerks as suggestive of the presence of organic nervous disease. It has been stated that in patients who have had syphilis the presence of excessive knee-jerks should lead one to suspect the commencement of syphilitic disease of the central nervous system; but in examinations for life assurance and in ordinary medical examinations one so frequently finds excessive knee-jerks* apart from any nervous disease whatever that I now regard the mere presence of excessive knee-jerks apart from other symptoms as practically without any pathological significance. I quite admit, however, that this "functional" excess of patellar reflex is especially likely to be found in individuals with a neurasthenic tendency and in persons of a rather weak habit of body who are easily thrown into a state of nervous excitement, and that it is more frequently found in persons of Jewish than of Northern European descent. The class of so-called "hysterical malingerers" will doubtless also be found often to show excess of patellar reflex.

Extreme exaggeration of patellar reflex, especially what I would term the "trepidation" or "spinal epilepsy" form† (when in addition to a violent knee-jerk there is

* Incidentally I would remark that examination of knee-jerks in ordinary routine cases teaches one also not to rely too much on the absence of knee-jerks as a sign of there being organic nervous disease (especially tabes dorsalis) present. Whatever may be said to the contrary, there are a certain number of healthy persons in whom knee-jerks cannot be obtained even by the help of devices such as making the person who is being examined lock his hands together and pull apart. Of course, it can always be objected that the absence of knee-jerks is only temporary in such cases; but it must be admitted that there are apparently healthy persons in whom the knee-jerks are either persistently (congenitally?) or temporarily absent.

† In some of these cases a gentle tap on the bone (that is, periosteum) anywhere near the patellar tendon is sufficient to produce a violent knee-jerk with "trepidation."

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also a kind of "knee clonus"), is, I believe, far more often due to functional than to organic nervous disease. In these extreme forms there is generally, likewise, ankle clonus present, and I would here state that I cannot recognize the existence of any infallible distinctions between "functional" ankle clonus and ankle clonus due to organic disease. In some functional cases the associated absence of plantar reflex might help one in diagnosing functional nervous disease, whilst the presence of Babinski's phenomenon would of course practically negative the disease being merely functional. I have heard, however, of a plantar reflex of the extensor type (Babinski's phenomenon) accompanying attacks of hystero-epilepsy, and I have myself met with this sign present on one side shortly after the occurrence of cerebral embolism (of cardiac origin) on the other side, so that the original idea that Babinski's phenomenon is a really absolute sign of the presence of "organic" change in the descending motor tracts cannot be maintained.

In a young man whose case I described elsewhere[1] the slightest tap over the patellar tendon caused the leg to be violently jerked forward. Similarly, if the patient lay on his side a tap anywhere on the Achilles tendon caused great contraction of the calf muscle, but the movement of the foot was somewhat less sudden than that of the leg on tapping the patellar tendon. The triceps jerks and supinator jerks were exaggerated in the same way. There was sometimes great ankle clonus to be obtained, but this varied considerably on different occasions, like the clonus often met with in functional nervous affections. On one occasion, when the patient was on his side in bed and temporarily supporting the upper part of his body with one hand on the bed, I noticed a clonus of the upper extremity (from the flexed elbow-joint) resembling the ankle clonus in the lower extremity. The symptoms in this case appeared to be of uraemic (or toxaemic) origin, connected with double hydronephrosis and calculus in the urinary bladder. Probably increase of knee-jerks (though without the extraordinary exaggeration of all the deep reflexes just noted) is not rare at the commencement of uraemia, and it is quite natural that nervous symptoms of the "functional" class should be sometimes induced by general toxic or toxaemic conditions, just as mental symptoms are (for instance, the various abnormal mental states induced by alcohol, cannabis indica, septic pyrexial conditions, etc.).

The "trepidation form" of patellar reflex is of course closely allied to the trepidation of an extremity induced in some functional nervous cases by handling the limb. Even where there is obviously "organic" disease the presence of such trepidation probably signifies that one has likewise to deal with a functional nervous element, and this supposition is supported by the fact that the trepidation may pass off, whilst the obviously organic disease remains. In a man aged 25 years, with a remarkable and severe trophic disorder of the left foot, whose case I described in 1901,[2] the left leg was affected not only with

persistent spasticity, but at times also with violent rhythmical clonic movements. This trepidation of the extremity was very well marked on the patient's admittance to the German Hospital, and was easily excited by handling the foot. He seemed, however, to be able to some extent to stop it at will, and it disappeared whilst the patient was in the hospital. The plantar reflexes were absent in that patient.

With such "trepidation cases" some cases of persistent hysterical tremor of an extremity may be compared. The functional tremor (or trepidation) is sometimes influenced by the state of the patient's emotions, and an occasional curious feature of it is its tendency to "spread" when the joints involved in the movement are kept motionless by force. Thus, in a woman under my care at the German Hospital, the tremor in the left lower limb could be stopped by holding the foot firmly in a position of extreme dorsal flexion, but then the right lower extremity would commence to tremble. If some one else prevented this by grasping the right foot in the same way, the trembling tendency in the left limb became so great that the foot could no longer be kept still.‡

I now come to the question, What part of the nervous system is chiefly responsible for excessive knee-jerks, ankle clonus, and the trepidation (sometimes misleadingly called "spinal trepidation") of functional nervous origin? The not uncommon connexion of these phenomena§ (at all events in their extreme form) with well-known symptoms of functional nervous disease, such as hysterical hemianaesthesia, hysterical monoplegias and hysterical spasms suggests that the nervous action or functional change on which the phenomena in question depend is in the brain, probably in the cerebral cortex.‖ This suggestion, moreover, derives support from the decided influence of temporary emotional states on the phenomena in question.

My view is that the excess in the patellar reflex in the class of cases under consideration is due to a peculiar functional condition of the cerebral cortex, which when present in greater degree gives rise to the "trepidation" form of patellar reflex, and to the so-called "spinal trepidation," and to "functional ankle clonus"; that this functional condition of the cerebral cortex may be excited or modified by temporary emotional factors, and that a

‡ I showed this patient at the Medical Society of London when the tremor of the left foot had lasted on and off for ten years. See *Transactions of the Medical Society of London*, 1905, vol. xxviii, p. 293.

§ Kernig's sign is probably intimately allied to these phenomena, and the probable cerebral (as opposed to the formerly generally supposed spinal) origin of this sign has lately been discussed by Paul Sainton and Roger Voisin at the French Medical Congress held at Paris in October, 1907. (*Presse Médicale*, October 23rd, 1907, p. 692.)

‖ S. J. Sharkey (*Brain*, Spring, 1904, p. 4) has summed up the main reasons why the cerebral cortex is to be considered the part chiefly at fault in hysteria. He suggests in particular that the reason why patients with hysterical hemianaesthesia are not usually conscious of their loss of sensation is that the cortical centres are the parts affected, whereas in hemianaesthesia due to gross brain disease the sensory fibres in the internal capsule are generally diseased and the cortical centres are sound, so that the patient perceives his anaesthesia.

similar state may be induced by toxaemias, such as uraemia; that the signs (exaggerated knee-jerks, etc.) of the functional cerebral condition in question may be imitated by organic nervous disease¶ (cerebral haemorrhage, sclerosis in the lateral columns of the spinal cord, disseminated sclerosis, etc.), but that it is just possible that in some rare cases of organic nervous disease (for instance, some cases of early disseminated sclerosis) the organic changes may give rise to the phenomena of increased tendon reflexes, etc., merely by inducing the above-mentioned (hypothetical) functional cerebral change.

To illustrate my meaning further, I shall now allude to the analogous question of the nature of functional nervous vomiting. In an article contributed to *Brain* in 1904[3] I endeavoured to show that functional nervous vomiting, like the hemianaesthesia, palsies, and spasms of hysteria, must be regarded as due to an abnormal state of the brain (cerebral cortex), and is just as much a symptom of functional brain disease as the vomiting in cases of cerebral tumour is of organic brain disease. We know that violent vomiting is frequently a result of organic disease (tumours, etc.) and injuries (involving concussion) of the brain, and that vomiting of cerebral origin often occurs in the absence of any gross change in the brain (for instance, in simple concussion and in ordinary sea-sickness, and from toxic causes acting on the cerebral cortex, such as uraemia, and from the inhalation of chloroform and ether), and may even be due to purely psychical causes—such as ideas connected with disagreeable smells and sights, the view of blood and surgical operations, and various mental emotions. Now, patients with functional nervous vomiting mostly exhibit characteristic signs of hysteria, and it is generally recognized, I believe, that the main symptoms of hysteria, such as hysterical hemianaesthesia, hysterical monoplegias, and hysterical spasms, owing to their characteristic distributions, etc., are of cerebral cortical origin, just as much so as the mental symptoms of hysteria are. I believe, therefore, that in vomiting of functional nervous origin the main site of the disturbance in the nervous system is most probably likewise in the brain (cerebral cortex), and, practically speaking, I regard the vomiting of organic brain disease as an imitation of that due to functional brain disease (hysteria). The vomiting due to functional disease may in rare cases—true faecal vomiting of functional nervous origin—be more violent than any vomiting caused by organic disease. With regard to the violence of the symptoms in such exceptional cases, I wrote in the paper above referred to:

"Why the vomiting in functional brain disease should be sometimes actually more violent and severe (faecal vomiting)

¶ I believe in this regard it is better to speak of the symptoms due to the functional state of the brain being imitated by organic changes in the nervous system than (conversely) to speak of the functional disease imitating the symptoms of organic disease, as is usually done.

than it is in organic cerebral disease (faecal vomiting practically never occurs in organic brain disease) I cannot understand, unless it be that disturbance of function can show itself in a more pronounced form in a brain without any gross organic disease than it can in a brain the finer mechanism of which has already been impaired by severe organic disease, such as cerebral tumour. In connexion with such a supposition one may remember that a delusion is apt to be less stable (and not so well organized) in a general paralytic, whose brain is the site of grave organic disease, than in a patient with chronic paranoia, whose brain, could it be examined, would probably show no obvious organic changes."

In conclusion, I maintain that in functional nervous vomiting we probably have to deal with a peculiar functional change in the brain (cortex), analogous to the cerebral condition that produces the exaggerated tendon reflexes and "trepidation" of functional nervous origin, and which, like it, may be excited or modified by emotional factors, and is analogous to certain states induced by toxaemias—such as uraemia. The symptoms produced by these abnormal functional states may equal or even exceed those produced by organic disease.

REFERENCES.

[1] Spontaneous Symmetrical Ecchymoses of the Eyelids and Conjunctivae, with Extraordinary Exaggeration of all the Tendon Reflexes preceding Fatal Uraemic Coma, *British Journal of Dermatology*, September, 1906, p. 323. [2] Trophic Disorder of the Feet, *British Journal of Dermatology*, February, 1901, p. 41. [3] Faecal Vomiting and Reversed Peristalsis in Functional Nervous Disease, *Brain*, Summer, 1904, p. 170.