ON A POSSIBLE SIGNIFICANCE OF THE BABINSKI AND OTHER PATHOLOGIC REFLEXES¹

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The Babinski toe phenomenon is looked upon as a pathognomonic sign of organic lesion of the pyramidal tract, or its point of origin. In order, however, to be diagnostic it must be demonstrated in undoubted fashion; *i.e.*, on stroking the sole of the foot there must be a slow and isolated dorsal flexion of the great toe. The stimulus applied must not be too great in order to eliminate complicating defense or withdrawal movements, and is best applied to the outer side of the plantar aspect of the foot. There are normal individuals who do not respond to stroking of the sole of the foot as the great majority of people do with plantar flexion of the toes, but show a more or less definite dorsal flexion of the great toe either alone or in conjunction with a similar movement of the other toes. But this response is usually much more swift and inconstant; it signifies infantilism in the sense that the cerebral flexion reflex has not the normal predominance over the spinal extension reflex. This pseudo Babinski is especially common in the neuropathic, in ductless gland disease and other degenerative conditions.

Simultaneously with the dorsal movement of the great toe there is a contraction of the tensor fasciæ femoris (Brissaud, Marie Foix), and of the hamstring muscles (Walshe). There is also frequently a fan-shaped spreading of the toes (*phenomene d'éventail*) seen especially in the infantile hemiplegias.

The Babinski phenomenon also occurs in the first stage of narcosis, in post-epileptic states, and in uremia. It disappears during deep anesthesia and comes on at once after the insult to the cortico-spinal system. It is absent in transverse lesion of the cord, although this has been called in question recently. For example, Kausch has seen a positive Babinski in complete transverse lesion of the cord.

¹ Paper presented at a joint meeting of the New York Neurological Society and the Section in Neurology and Psychiatry of the New York Academy of Medicine, November 11, 1919.

He thought that dorsal flexion of the great toe originated in the cord, and that plantar flexion was due to the influence of the brain.

The Babinski reflex has no pathological significance in children under one year of age. Engstler always found extension in infants up to eight weeks, and in the second year 5 per cent. still showed extension.

The zone for reflex excitation of the Babinski sign is very wide, and there have sprung up a great number of modifications in the method of eliciting this reflex. I shall only mention the Oppenheim, Chaddock, Gordon and Crafts methods of obtaining the same end result (dorsal extension of the great toe).

There are other reflex manifestations frequently associated with positive Babinski, and apparently of similar significance. Rossolimo found that in pyramidal tract disease, striking the plantar aspect of the little toes produced plantar flexion and spreading of the toes. The Mendel-Bechterew reflex is exemplified in the same way but elicited through tapping the dorsal aspect of the fourth and fifth tarso metatarsal joints. One finds frequently also in pyramidal tract disease, the so-called Hoffman (sometimes called Klippe-Weil) reflex in the upper extremity. This consists in a grasping movement of the hand when the terminal phalanx of one of the fingers is pinched. Recently Mayer has described the loss of function of the opponens pollicis in hemiplegias. Whereas, in the normal, passive flexion of the metacarpo-phalangeal joint of the second or third finger produces opposition of the thumb and flexion of its metacarpo-phalangeal joint, these movements are absent in hemiplegia and in the newborn.

Viewing all that has been said in the light of adaptation to function, might it not be possible that all of these pathologic reflexes are an expression of an atavism? The human cortico-spinal system is, from the phylogenetic standpoint, one of the latest to appear; as a matter of fact, it is not yet myelinated at birth, but becomes so sometime afterward. May we not, therefore, say that lesion of the cortico-spinal system causes man to revert to the stage of the tree climbing monkey in whom there seems to be a dissociation between the great toe and the little toes, and in whom there is also very little opponens pollicis function? In climbing the monkey stems himself with his great toe in extension against the branch of a tree while clutching it with the remaining four toes in flexion; his hand is used very much after the manner of the Hoffman reflex in holding on with his upper extremity.

The presence of a positive Babinski sign in epilepsy and uremia

would be explained by the injury (perhaps only functional, if not anatomic) to the cortico-spinal system. The other phenomena associated with the Babinski sign—contraction of the hamstring muscles, of the tensor fasciæ femoris, the Rossolimo and Mendel signs—would seem to suggest a purposeful mechanism. The wide reflexogenic zone for eliciting these pathologic reflexes could most readily be explained on the assumption of adaptation to function.

In the adult reciprocal innervation² leads to the transformation of the extensor response to a flexor type. In strychnine poisoning where, according to Sherrington, reciprocal innervation³ is abolished, the more powerful extensors assume the upper hand, and Babinski has found in such cases dorsal extension of the great toe. In pyramidal tract disease, we may, therefore, assume also a loss of reciprocal innervation characteristic of the adult human. The fact, too, that the Babinski sign disappears in the child coincidentally with its learning to walk, is suggestive in this regard. The child, so to speak, moves forward phylogenetically at this time, advancing to the stage of locomotion in the erect posture. By means of reciprocal innervation he converts the extensor response now to a teleologically better suited flexor response. One can readily see how serious a handicap dorsal extension of the great toe would be under such conditions and how ill adapted it would be to the carrying out of this, evolutionally speaking, lately acquired function. The hypertonia of the newborn child too is readily explained by the fact that the tonus-accelerating cerebellum still has the upper hand. The cortico-spinal system, which is tonus-inhibiting, is not yet myelinated, and hence does not yet function fully. Whether the flexor flexion reflex of the English school of neurologists is concerned in the reflex mechanism discussed, I am not prepared to say, although the fact that the Babinski sign is an extensor phenomenon would seem to point away from this hypothesis.

There has recently appeared a paper by Brouwer⁴ in which he attempts to explain the phenomena of multiple sclerosis on a phylogenetic basis. His assumption is that those tracts and systems which

² The fact that an interference with reciprocal innervation might play a rôle was first suggested to me by Dr. H. C. Jackson, professor of physiology of New York University Medical School.

³ Reciprocal innervation, according to Sherrington, leads to coördinated movement through motor discharge in the agonists and simultaneous inhibition of the antagonists.

⁴ Brouwer, significance of phylogenetic studies for the neuropathologist, *Psychiatrische en neurologische Bladen*, Feestbundel Winkler, September 20, 1918. See this number of the *JOURNAL*.

(I have had access to Brouwer's paper through the courtesy of Dr. S. E. Jelliffe.)

are phylogenetically most recently acquired are the most vulnerable. In this way he explains the loss of abdominal wall reflexes, the speech disturbances, the nystagmus, the contrast between marked motor signs, and scant sensory changes, and the temporal pallor of the optic discs. He also found that in lesions of the median nerve the abductor pollicis brevis and the opponens showed the reaction of degeneration when it could not be demonstrated in the other muscles. In lower monkeys these muscles are poorly developed; hence their greater vulnerability. He apparently also is seeking a biologic explanation for observed phenomena, and, that, to my mind, is a desirable method of approach. Therein lies the value too of comparative neuro-anatomy.

It has been our notion that teleology plays a great part in medicine just as there is evidence of design in nature, and it is with this idea in mind that I present this hypothesis on an obscure mechanism in medicine. I recognize fully the inadequacy of this theory in places, and I should be very loath to consider it in any other light than that of an invitation to fruitful discussion.

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