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SHORT COMMUNICATION

"FUNCTIONAL MOTOR AMNESIA" IN STROKE (1904) AND "LEARNED NON-USE PHENOMENON" (1966)

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The "learned non-use phenomenon" described by Taub, one of the most original recent contributions to rehabilitation medicine probably corresponds to what Henry Meige (1866–1940), who studied under J.-M. Charcot, described in hemiplegics in 1904 using the expression "functional motor amnesia". He specified in 1914 at the time of the Babinski description of anosognosia, that: "Even with educated subjects who are still relatively young we are sometimes confronted with strange incapacities that are not due to impotence, negligence, or lack of confidence in the results. […] With the transitory halting of the motility all memory of the function appears to have disappeared". Meige describes motor disorders that are: (i) distinct from lesional paralyses; (ii) secondary to the absence of activity; (iii) linked to a learning process; (iv) linked to a phenomenon of functional memory loss; (v) reversible; and (vi) motor re-education focusing on extended and repeated practice of the lost function: the same characteristics as the "phenomenon of learned non-use" described by Taub in monkeys then in man.

Key words: learning, rehabilitation medicine, stroke, hemiplegia, history of medicine.

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INTRODUCTION

Following on from what had recently been learned concerning neuroplasticity, 2 major advances have marked neurological rehabilitation over the past 2 decades: (i) demonstration of the pernicious nature of inactivity that generates tenacious learned non-use underpinned by cortical reorganization ("learned non-use"); and (ii) demonstration of neural reorganization after a cerebral lesion under the effect of use ("use-dependant") and, consequently, the possibility of "driving plasticity".

The phenomenon referred to as "learned non-use", described by Taub et al. (1, 2) probably corresponds to what Henry Meige described in hemiplegics in 1904 using the expression "functional motor amnesia" (3).

Henry Meige (1866–1940) (4) was one of the last pupils of Jean-Martin Charcot (1825–93) at Salpêtrière Hospital in Paris, where he exercised his clinical activity and worked closely with Edouard Brissaud (1852–1909). He was the editor of the *Revue Neurologique (Paris)* for 25 years, General Secretary of the French Society of Neurology and held the chair of Anatomy at the École des Beaux Arts art school in Paris. His name is associated with the median spasm of the face: "A clinical form of bilateral and median convulsion" (5), (Meige Syndrome II, hemifacial spasm, orofaciomandibular and lingual dystonia, idiopathic orofacial dystonia) and Nonne-Milroy-Meige syndrome (Meige Syndrome I, chronic familial lymphoedema of the limbs). He published several works, in particular on tics and Charcot (6). J. M. Charcot had equipped the clinic for patients suffering from nervous system disorders at Salpêtrière Hospital with "ancillary departments", and in particular a department equipped with "all the instruments necessary for electrodiagnosis and electrotherapy" (7) under the influence of Duchenne (of Boulogne); another department for exploration of pathological gait was set up by A. Londe and P. Richer and by Gilles de la Tourette (8). Various physical treatments were used, such as suspension by the Motchoutkowsky method (9). It is in this context that Meige benefited from a culture of rehabilitation.

EDWARD TAUBS' "LEARNED NON-USE PHENOMENON"

As of 1968, Taub et al. demonstrated in primates (1), then in man (2), the role of non-use in the genesis of certain motor disorders: non-use induced by a paralysis generates a "learned non-use" phenomenon that prevents or limits the expression of the motor recovery, thus compromising the possibilities of recovery itself.

> When a single forelimb is deafferented by dorsal rhizotomy in a monkey, the animal does not make use of it in the free situation. However, the monkey can be induced to use the deafferented extremity by either (i) restraint of the intact limb or (ii) application of training techniques such as operant conditioning. A useless limb is thereby converted into a limb capable of extensive movement. Restraint of an unaffected

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limb also improves use of the affected limb following unilateral cortical area 4 ablation and unilateral pyramidotomy in monkeys.

Several converging lines of evidence suggested that the non-use of a single deafferented limb is a learning phenomenon, termed "learned non-use," involving a suppression of movement. The restraint and training techniques appeared to be effective because they successfully overcame the learned non-use. It was hypothesized that the non-use or limited use of an affected upper extremity in humans after stroke could, in some cases, be due to a similar learned suppression phenomenon.

The central premise of this view is that immediately after somatosensory deafferentation a monkey cannot use a single deafferented limb because of the presence of a shock-like condition that follows substantial neurological injury, whether at the level of the spinal cord (spinal shock) or brain (diaschisis). In monkeys, recovery from this shock-like phenomenon requires weeks or months. An animal with one deafferented limb tries to use that extremity in the immediate postoperative situation, but finds that it cannot. It gets along quite well in the laboratory environment on three limbs, and this pattern of behavior is therefore strengthened. Moreover, continued attempts to use the deafferented limb often lead to aversive consequences, such as loss of balance and falling during ambulation or climbing, loss of food objects, and indeed failure of almost any attempted use of the limb. This has the effect of suppressing all behavior with that limb; the monkey thus learns not to try to use it. This tendency persists, becoming stronger with time, and consequently the monkey never learns that, several months after surgery, the spinal shock has passed and the limb has become potentially useful.

The consideration that led to the conduct of the present research with human stroke patients is that, according to this formulation, learned non-use could develop after any neurological injury resulting in central nervous system (CNS) shock and an initial inability to use an extremity. The operation of the mechanism, as proposed, should be independent of the nature of the lesion that gives rise to the CNS shock and limb non-use. If there is then a recovery from the initial CNS shock state and if sufficient neural substrate remains intact to provide a basis for movement, then the techniques used for overcoming learned non-use following somatosensory deafferentation in monkeys should be equally applicable following other types of neurological injury, including stroke in humans, in restoring the ability to use the limb. This would be the case even though entirely different lesions are involved, and though stroke in man involves different physical deficits and cognitive defects beyond those produced by somatosensory deafferentation in monkeys (2).

Wolf et al. (10) were the first to show, in a man who had suffered from hemiplegia for several years, that forced use of the partially paralysed upper limb, by exercises carried out several hours a day for 2 weeks, could durably improve the strength, speed and function of the upper limb. They thus showed that at least part of the motor deficiency was not linked to the paralysis but to learned non-use, reversible by relearning the activity. Numerous works have confirmed this concept and have provided the basis for "constraint-induced movement therapy" or "CI therapy", then, more generally, induced or forced use (11). Different protocols were proposed, calling on devices capable of generating repeated movements: treadmills and suspension to relieve the weight of the body, robot and electric stimulation. The phenomenon of learned non-use and its therapeutic consequences are not limited to hemiplegia but concern all ailments leading to non-use of the locomotor apparatus.

HENRY MEIGE'S "FUNCTIONAL MOTOR AMNESIA" DESCRIPTION

Meige spoke twice on the loss of motor functions in hemiplegics.

The first time he presented "functional motor amnesias" at the Seventh Congress on Internal Medicine which was held in Paris between 24 and 27 October 1904: the report, published in the *Revue Neurologique (Paris)* in 1905 (3), merits being cited *in extenso*.

> When hemiplegia occurs following an ictus, the affected limbs are incapable of any movement for several hours and often several days. Then, in the majority of cases, some movements emerge and gradually become more marked, with *ad integrum* restitution possible though rare. Generally, progress appears limited and after a while it can be thought that the hemiplegic is not capable of making any further progress: his infirmity appears definitive.
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> However, a careful examination of motility reveals a difference, often considerable, between motor acts that a hemplegic, left to his fate, spontaneously executes and those that he would be capable of executing. Muscles, primitively inert, gradually recover all or part of their contractility, but the subject does not make use of them. Numerous movements that were impossible in the initial stages of the illness subsequently become possible but are not carried out. The hemiplegic does not know them. He has forgotten them.
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> These are motor amnesias and these motor amnesias are above all functional. The patient sometimes makes the muscles in question move, but without a goal. He no longer knows how to use them for the purpose of a determined functional act. Having been, for a while, really incapable, of executing a familiar act, then, later, not managing to execute it correctly the first time, the patient generally concludes that he will no longer be able to do it. Not knowing how to go about it, he stops trying to do it. He ceases to improve. Motor aboulia is added to motor amnesia.
>
> The situation of hemiplegics is worsened by these motor amnesias and aboulias; their infirmity appears greater to them than it really is.
>
> Without claiming to restore all motor acts, we can, at least

in a number of cases of hemiplegia, develop the numerous movements that are forgotten and unused by the patients. We can teach them to direct them to achieving various functional goals: walking, standing, getting up, sitting down, going up and down stairs, etc., and also for the upper limbs, the gestures involved in getting dressed, eating, writing, etc. The psychomotor discipline, whose good results we have underlined with Mr Brissaud on more than one occasion, has already given us noticeable results in this respect. By greatly increasing psychomotor interventions using exercises adapted to defined goals and repeating these sufficiently, we manage to create these habits of motor associations that constitute our usual acts and of which hemiplegics have often only lost the memory. These functional restorations, by virtue of a well-known law, have also positive repercussions on both the motor apparatus (muscles develop, contractures and retractures are reduced) and the nerve centres and conductors; the organ benefits from exercising the function; finally the patient's moral is improved.

Meige goes back to the subject of functional motor amnesias in the discussion that followed J. Babinski's description of anosognosia (12) during the meeting on 11 June 1914 at the French Society of Neurology.

I was often surprised by the rapidity with which hemiplegics appeared to have forgotten the functioning of their paralysed limbs. Attempts at motor re-education clearly showed this. Even with educated and relatively young subjects, who have perfectly understood the therapeutic goal we are sometimes confronted with strange incapacities, which are not impotence, negligence or lack of confidence in the results. […] With the transitory halting of motility all memory of the function appears to have disappeared.

DISCUSSION

The first description by Meige clearly differentiates the motor disorder that he describes in princeps paralysis, and the process of recovery that follows. He considered the initial absence "of motor acts" to be the cause of the deficiency and suggests it is caused by a learning mechanism based on unsuccessful attempts, errors and finally abandonment ("learned helplessness" (13)). The expression "forgotten movement" or "functional motor amnesia" makes it possible to differentiate the mechanisms of two distinct processes, one lesional and the other functional appearing due to "learned non-use". We had to wait until functional magnetic resonance imaging and magnetic cortical stimulation became possible before we could show that the inactive cortical areas, reorganized for other uses, were deviated from their original functions and that these initial functions had been forgotten.

The 1914 discussion is particularly pertinent since it very briefly summarizes the different mechanisms of motor expression disorders encountered in vascular accidents and at the origin of hemiplegia or associated with it. Meige clearly distinguishes between paralysis, neglect, anosognosia and even negative attitude, thus marking *a contrario* the special nature of such motor function memory losses. Finally, he underlines the possible, though difficult and unpredictable, reversibility resulting from rehabilitation.

In his conclusion Meige, by affirming the reciprocal repercussions between the locomotor apparatus and the nerve centres, "the organ benefiting from the function", is even announcing plasticity and its orientation (14).

Meige therefore indeed describes a disorder with the same characteristics as the phenomenon of learned non-use described by Taub in monkeys then in man.

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