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HISTORICAL NOTES

Hysteria to conversion disorders: Babinski's contributions

Histeria aos transtornos de conversão: contribuições de Babinski

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ABSTRACT

The main objective of this paper is to present the importance of hysteria on Babinski's oeuvre, and the conceptions of pithiatism from Babinski until the one of conversion disorder. Babinski gave a mental basis for hysteria in the place of Charcot's encephalopatic one, and several important semiotic tools to differentiate organic from hysterical manifestations based on studies from 1893-1917/8. His teachings were spread worldwide, and in Brazil they were also appreciated in the work on hysteria by Antonio Austregesilo, the first Brazilian neurology chairman. The neurobiological basis of hysteria conceived by Charcot is nowadays reappraised, and Babinski's neurosemiological contribution is everlasting. The patients believed to be hysterical, and the two outstanding neurologists, Charcot and Babinski, gave support for the development of the modern neurology.

Keywords: hysteria, Babinski, Charcot, somatoform disorder, conversion disorder, history of Neurology.

RESUMO

O objetivo principal deste trabalho é apresentar a importância da histeria na obra de Babinski e a concepção de pitiatismo de Babinski até a de transtorno de conversão. Babinski deu uma base mental para histeria no lugar da encefalopática de Charcot, e várias ferramentas semiológicas importantes para diferenciar manifestações orgânicas de histéricas, com base em estudos de 1893-1917/8. Seus ensinamentos foram disseminados em todo o mundo, e no Brasil eles também foram apreciados no trabalho sobre a histeria por Antonio Austregésilo, o primeiro catedrático da neurologia brasileira. A base neurobiológica da histeria concebida por Charcot é reavaliada hoje em dia, e a contribuição neurosemiológica de Babinski é perene. Os pacientes considerados histéricos e os dois grandes neurologistas, Charcot e Babinski, deram suporte para o desenvolvimento da neurologia moderna.

Palavras-chave: histeria, Babinski, Charcot, transtorno somatoforme, transtorno de conversão, história da Neurologia.

Joseph Jules François Félix Babinski (1857-1932) had at the beginning of his career the influence of Jean-Martin Charcot (1825-1893) on hysteria conception. Nevertheless, from 1901 on, he presented his own theory about the issue, as well as several approaches to differentiate organic from hysterical symptomatology what he expressed in several publications (1893-1917/8)[1-3]. Babinski's work on hysteria is the subject of this paper. Additionally, the thoughts about hysteria stated by Antônio Austregésilo Rodrigues de Lima (1876-1960)[4,5], the first Brazilian professor of Neurology, are pointed out. Furthermore, the nowadays neurobiological conception and classification of what was once called hysteria, mainly that with pseudo-physical-neurological symptoms, are also considered, as well as an appraisal of Charcot's and Babinski's contribution on hysteria *vs.* neurology.

BABINSKI'S AIM TO UNDERSTAND HYSTERIA AND DIFFERENTIATE IT FROM ORGANIC DISORDERS

Babinski graduated in Medicine at the University of Paris (1884) with a thesis on multiple sclerosis. He was chosen to become Charcot's *chefe de clinique* at La Salpêtrière (1885-1887), a starting point in his interest on hysteria[2,3]. He later became a great diagnostician, relying considerably on clinical findings, and head of the neurological clinic at the Hospice de la Pitié.

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Box 1. Babinski and Froment explanation about differential diagnosis of hysteria and organic hemiplegia in their book *Hystérie-pithiatisme et troubles nerveux d'ordre réflexe* en neurologie de guerre (1917) (Hysteria-Pithiatism and Reflex Nervous Disorders in the Neurology of War)[6].

Organic Hemiplegia	Hysterical Hemiplegia
1. The paralysis is limited to one side of the body.	1. The paralysis is not always limited to one side of the body. This particularly applies to paralysis of the face, in which the symptoms are generally bilateral
2. The paralysis is not systematic, e. g., if unilateral movements of the face are very much weakened, this weakness appears quite as distinctly on the hemiplegic side during the execution of synergic bilateral movements.	2. The paralysis is sometimes systematic. It is almost so in the face, e. g., unilateral movement of the face may be completely abolished, while the muscles on the hemiplegic side act normally during the execution of synergic bilateral movements.
3. The paralysis affects the conscious voluntary movements as much as the unconscious or subconscious voluntary movements, giving rise to the platysma sign (more energetic contraction of the platysma on the sound side in the act of opening the mouth or bending the head in opposition to the resistance which the observer makes to this movement), combined flexion of the thigh and trunk, and in walking the absence of active swinging of the arm, as contrasted with the exaggeration of passive swinging (the limb oscillating as an inert corp when the patient suddenly turns around).	3. The unconscious or subconscious voluntary movements are not affected; with the result that there is an absence of platysma sign, and of combined flexion of the thigh and trunk; active swinging of the arm may be absent, but there is no exaggeration of the passive movement.
4.The tongue is usually slightly deviated to the side of the paralysis.	4. The tongue is sometimes slightly deviated to the side of the paralysis, but the deviation may be very pronounced or be directed to the opposite side of the paralysis.
5. There is, chiefly at the onset, muscular hypotonus, which may be shown in the face by obliteration of the naso-labial fold and the lowering of the eyebrow, and in the upper limb by exaggerated passive flexion of the forearm, and by the sign of pronation (the hand when left to itself assumes a position of pronation).	5. There is no muscular hypotonus. When there is facial asymmetry, it will be found to be due to muscular hypotonus, but to spasm; the sign of exaggerated flexion of the forearm and the sign of pronation are absent.
6. The tendon and bone reflexes are frequently affected at the onset, when they may be lost, diminished or exaggerated. Later, they are usually exaggerated and in many cases there is clonus foot.	6. The tendon and bone reflexes show no change; there is no clonus foot.
7. The cutaneous reflexes are generally affected. The abdominal reflex and cremasteric reflex are usually diminished or lost, especially at first. The character of the reflex movement of the toes following stimulation of the sole usually undergoes inversion; the toes, and especially the great toe, instead of being flexed, become extended on the metatarsus (toe phenomenon). Extension of the great toe is often associated with abduction of the other toes (fan sign). Exaggeration of the reflexes of defense may sometimes be noted.	7. The cutaneous reflexes do not appear to be affected. The abdominal and cremasteric reflexes are normal. The reflex movement following stimulation of the sole does not undergo inversion. The toe phenomenon and fan sign are absent. The reflexes of defense are not exaggerated.
8. The form of contracture has particular characteristics and cannot be reproduced by a voluntary contraction of the muscles. There is a "clawing" of the hand, which gives the sensation of an elastic resistance automatically increased during passive movements of extension of the fingers.	8. The form of contracture may be reproduced by a voluntary contraction of the muscles.
9. The course is regular, contracture succeeding flaccidity. The disappearance of the motor disturbance, when it does take place, is progressive. The paralysis shows no tendency to become better and worse alternately (permanence of motor troubles).	9. The course is capricious; the paralysis may remain flaccid indefinitely, or it may be spastic from the first; spastic phenomena are sometimes associated with paralysis, especially in the face. The symptoms are frequently liable to subside and to get worse alternately, to become rapidly modified in their intensity as well as in their form, and to present transitory remissions which may last only a few moments (variety of motor troubles).
Other signs of organic hemiplegia	
10. Raimiste's sign, which is observed during the period of flaccidity. It is obtained as follows: place the paralysed forearm and hand in a vertical position, with the elbow resting on the table. It will be found that if the hand be left to itself, it will become rapidly flexed and at the same time pronated.	
11. The interossei phenomenon described by Souques: movement of extension and abduction of the fingers whenever the patient raises the affected arm.	
12. Klippel-Weil's sign: involuntary flexion of the thumb accompanying passive straightening of the flexed fingers (in the period of contracture).	
13. The tibialis anterior phenomenon (Strümpell): an associated movement of dorsal flexion and adduction of the foot caused by voluntary flexion of the affected limb.	
14. Associated adduction and abduction of the paralyzed lower limb (Raimiste) observed in the patient lying on his back when he makes an energetic effort to adduct and abduct the sound limb against resistance.	
15. Various associated movements which according to P. Marie and Foix may be divided into the three following classes: global synkinesis (general contraction of all muscles of the hemiplegic side on the occurrence of any effort), imitation synkinesis (involuntary movements of the hemiplegic sided tending to reproduce the movement carried out voluntarily by the sound side), and co-ordination synkinesis (voluntary contraction of certain muscular groups in the paralysed limb giving rise to involuntary contraction of the functionally synergic muscles).	
16. Neri's sign: flexion of the knee, accompanying flexion of the trunk on the paralyzed side.	
17. The dorsal reflex of Mendel-Bechterew, or dorso-cuboid reflex: flexion of the toes of the paralyzed subject produced by percussion of the latero-dorsal surface of the cuboid, an opposite movement to that which occurs normally. This interesting phenomenon is associated with exaggeration of the tendon reflexes.	
18. Reflex hyperkinesis (Claude). Painful stimulation by pricking, pinching, or pressure of the muscles sometimes causes reflex movements in the paralyzed upper limb.	
19. The reflex of adduction of the foot (Raïchline, P. Marie and H. Meige) obtained by stimulation of the skin on the inner border of the foot.	

Note. This work was a direct consequence of the World War I, 1914-1918, at the time with more simulation preoccupation[1]. Enrolled also in this book the motor symptoms, distinguishing organic hemiplegia from hysterical one (also mentioned other semiological maneuver by other authors). Babinski gave much contribution to the neurosemiology (in addition to the previously mentioned maneuvers) such as cutaneous and osteo-tendinous reflexes, precise localization of spinal cord compression, cerebellar (asynergy, adiadochokinesia, hypermetria, cerebellar catalepsy) plus vestibular signs. In 1899, Babinski introduced the conception of asynergy as the cardinal symptom of cerebellar deficit[3].

Babinski's academic trajectory may be examined through *Oeuvre scientifique*, published by his pupils, two years after his death (1934), that recollected 288 publications[2,3]. Babinski's first work on hysteria was about *L'atrophie musculaire dans Ies paralysies hysteriques* (1886). However, over a period of 25 years, he had a long lasting preoccupation to develop criteria for differentiating hysterical symptoms from signs produced by organic lesions of the nervous system. This series commences in 1893 with *Contractions organique* until 1917-8 (with his book on *Hystérie-pithiatisme et troubles nerveux d'ordre réflexe en neurologie de guerre*)[3]. In 1896, three years after Charcot's death, he published the toe phenomenon description, later known as the Babinski reflex or Babinski sign, in a communication of merely 28 lines[2]. He made also several contributions to neurological semiology. In 1901, he launched his *Définition de hysterie*, unfolding later his concepts on hysteria. This approach guided him to advocate that hysteria was a psychical state in which the patient had a predisposition to self-suggestion, and consequently he recommended the term "pithiatism" (from the Greek: created by suggestion and curable by persuasion)[1,6]. This theory of the preeminence of suggestion, in spite of being subsequently rejected by many neurologists such as Dejerine and Raymond, reached large acceptance worldwide[1,4]. Additionally, during World War I, there was a new neurological charge on "traumatic hysteria", as first proposed by Charcot. Furthermore, several Charcot's students became actively involved in medical military care, including Babinski, who worked with Jules Froment (1878-1946), from the University of Lyon[2]. They had to distinguish patients with nervous organic lesions from those with "pithiatism" and malingering. In 1917, they published an important book on hysteria (Box 1, Figure)[2]. Clovis Vincent incentivized by Babinski to be a neurosurgeon, developed a treatment called torpillage (literally, torpedoing) for war hysteria, associating painful galvanic current discharges with "persuasion", put into practice also to distinguish between the recalcitrant simulator and the pithiatic[1]. The last work of Babinski's career was about hysteria: *Reponse à Radovici. Sur l'Hysterie* (1930)[2,3]. From the 1920s on arose not only a growing criticism on the theories of Charcot, but also a change in the conceptualization of hysteria, henceforth understood by the neurologists according primarily to Babinski's theories.

HYSTERIA IN BRAZIL

In Brazil, Antônio Austregesilo at the time physician at the National Hospice for the Insane (1904-1910), considered hysteria as the major diagnosis in women admitted over there[7]. He viewed it as a "diagnosis of the facility, above all when dealing with females"[4]. He believed that "difficult and unusual cases of nervous affections, particularly in women, received a label of hysteria"[4]. He defended a division of true hysteria (hysterical syndrome or pithiatism) and a pseudo-hysteria (histeroid syndrome or false hysteria due to other physical or mental disorders)[5]. In his "New concepts on hysteria", his first work on the subject (1908), he stated that the phenomenon was produced by suggestion, following the already Babinski's respected ideas[1]. Austregesilo was also aligned to the currents that placed hysteria in the field of Psychiatry, and in the same way he admitted that hysteria was a psychoneurosis that developed from a large diathesis: "nervousness", in harmony with Charcot's thoughts[4].

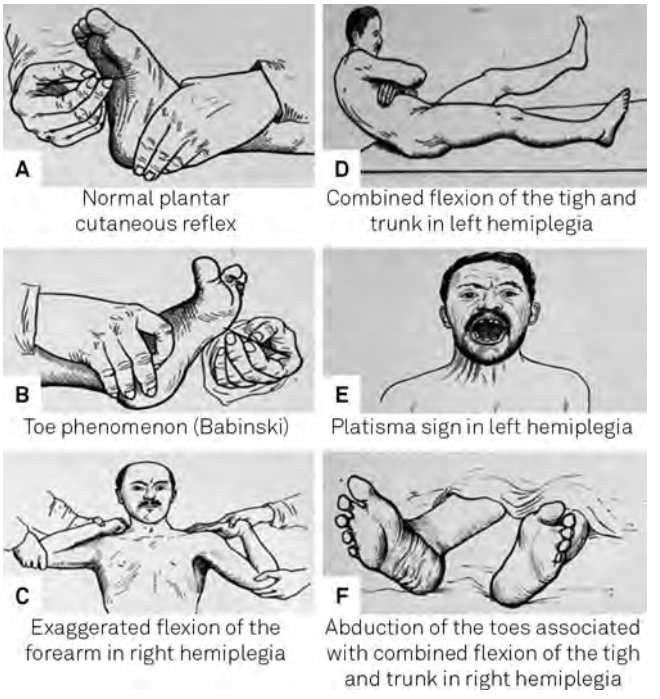

Figure. Babinski facilitated the distinction between functional and organic neurological symptoms. Babinski's description of the cutaneous-plantar response (1896) - now called Babinki's sign, figure B (and its association with pyramidal tract lesions). He described also other differential signs besides that presented in the figure, several others, such as: Sign of contracture of the hand (1893), the first one described by Babinski, where in hysterical hemiplegia, unlike organic, spasticity is such that the examiner cannot introduce his/her fingers between the patient's fingers whose hand is tightly flexed against the palm (figures reproduced from the book by Babinski and Froment[6]).

RENEWING OLD PARADIGMS WITH NEW TERMINOLOGY, BUT STILL CONFUSING

The complex construct of "hysteria", from 1980 until the present time, was split in several diagnostic categories. The term was no longer included in the DSM-III and following editions, and it was mainly replaced by a disorder group under "Somatoform Disorders" and "Dissociative Disorders", this last one with psychological manifestations. One of the aspects of the hysteria spectrum was called

Box 2. Summing up the hysteria-neurology complex.

Jean-Martin Charcot (1825-1893)

Joseph Jules François Félix Babinski (1857-1932)

The changing paradigm of hysteria includes somatic (uterine, humoral, nervous and then "functional"), psychological (demonic possession, psychogenetic) or psychosomatic basis. This somatic (encephalopathic) and psychosomatic basis comprise two great leaders, Charcot, and his dissident pupil on hysteria conception, Babinski, the great neurosemiologist. Both have hysteria as the intellectual ferment of their career. Charcot's theories now re-emerge as a theoretical explanation for somatoform disorders. This revival is in part due to the enrichment of neurosciences and women social position to whom hysteria has been pejoratively attached. Moreover, functional neuroimaging development in the last decade justifies the longevity of the neurological approach once persecuted by Charcot. However, this anatomo-functional-clinical renewal needs further experimental investigation and replication studies.

"somatization disorder" and Briquet's syndrome, but this eponymous, in this only edition[8]. The term somatization comes from an English translation (1925) of *Organsprache* by a Viennese psychoanalyst Wilhelm Stekel, former Freud's pupil[8]. In short, the old Freud's concept of conversion neurosis (unconscious conflicts become converted to bodily manifestations) is today mainly called somatoform (such as conversion and somatization). This somatization concept became characterized by an ample assortment of somatic symptoms affecting different organ systems, and it was enrolled among the five somatoform disorders, the others being conversion, pain, hypochondria, and dysmorphophobic disorders[8]. Regarding conversion disorder (DSM-IV), it involves one or more symptoms affecting voluntary motor or sensory function related to psychological factors, unintentional and unfeigned, resembling neurological or medical ailments[9]. However, its nomenclature suffered changes over the time: in 1952 (DSM-I), the used term was conversion reaction; in 1968 (DSM-II), hysterical neurosis (conversion type); in 1980 (DSM-III), conversion disorder[9]. From this time forth, the label 'dissociation' and 'conversion' disorders began to be used[9]. Concerning the ICD-10 (1992), contradictorily, it includes conversion disorder under the category of dissociative (conversion) disorders, together with dissociative amnesia and fugue states[9].

In conclusion, "Hysteria" was the main leitmotif of Charcot's and Babinski's work. This favored studies on brain-mind link, brain functioning conceptions, nervous (dys)functions, neurological examination and differentiation of several disorders (Box 2).

References

1. Allilaire JF. Babinski and hysteria. Bull Acad Natl Med 2007;191:1329-1339.
2. Clarac M, Massion J, Smith AM. History of Neuroscience: Joseph Babinski (1857-1932), IBRO History of Neuroscience 2008. [http://www.ibro.info/Pub/Pub_Main_Display.asp?LC_Docs_ID=2990]
3. National Institute of Neurological diseases and blindness, National Institutes of Health, Bethesda, Maryland. Great names in neurology. Bibliography of Writings by Joseph Babinski, Sir Victor Horsley, Sir Charles Sherrington and Arthur Van Gehuchten. Prepared for the International Congress of Neurological Sciences, Brussels, Belgium, July 21-20,1957.
4. Austregésilo A. Novas concepções sobre a hysteria. Arch Bras Psychiatr, Neurol Med Legal (Rio de Janeiro) 1908;4:52-66.
5. Austregésilo A. Hysteria e syndromo hysteroide. Comunicação à Sociedade de psiquiatria e neurologia. Arch Bras Psychiat Neurol Med Legal (Rio de Janeiro) 1909;5:59-77.
6. Babinski JFF, Froment J. Hystérie-pithiatisme et troubles nerveux d'ordre réflexe en neurologie de guerre. Paris: Masson et Cie, 1917.
7. Gomes MM, Cavalcanti MT. National hospice for the insane and the Brazilian Neurology in the beginning of the 20th century. Arq Neuropsiquiatr 2012;70:823-825.
8. Mai F. Somatization disorder: a practical review. Can J Psychiatry 2004;49:652-662.
9. Owens C, Dein S. Conversion disorder: the modern hysteria. Advances in Psychiatric Treatment 2006;12:152-157.