Fatigue and its disturbances: conditions of possibility and the rise and fall of twentieth-century neurasthenia

ZORZANELLI, Rafaela Teixeira. Fatigue and its disturbances: conditions of possibility and the rise and fall of twentieth-century neurasthenia. *História, Ciências, Saúde – Manguinhos*, Rio de Janeiro, v.16, n.3, July-Sept. 2009. Available at: http://www.scielo.br.

Abstract

The article first analyzes some of the social and historical components underlying the conditions of possibility that allowed neurasthenia to emerge as a nosological category in the latter half of the nineteenth century and then explores the elements that influenced its demise in medical and lay circles. It offers a brief introduction to this medical category and a more detailed discussion of some supporting debates, including the idea of nervous exhaustion, twentieth-century studies and concerns on fatigue, and the malady's presumed somatogenesis. The concluding analysis of how the category met its demise highlights elements that altered its status and its diagnostic usefulness.

Keywords: neurasthenia; conditions of possibility; demise.

Rafaela Teixeira Zorzanelli
PhD in collective health from Instituto de Medicina Social/ Universidade do Estado do Rio de Janeiro
Rua Correa Dutra, 39/408
22 210-050 – Rio de Janeiro – RJ – Brazil
rtzorzanelli@yahoo.com.br

Submitted on January 2008.
Approved on November 2008.

Translation by Derrick Philips.

The trivial nature of the symptom of fatigue – the fact of being a daily complaint on which each of us can give an opinion, and of being common in both public and private healthcare environments – does not mean we can deny the level of complexity of the experience *per se*. A sign of this is the intermittent presence, since the nineteenth century, of pathological cases in medical history, which include the central complaint of exhaustion after minimal effort and fatigue without detectable organic cause. In the twentieh century environment, this complaint was known as neurasthenia. In current times, the most emblematic example is the chronic fatigue syndrome, although other functional cases exist in which fatigue is an associated symptom, such as fibromyalgia. If we broaden the spectrum, we can include burn-out syndrome which, although not considered a functional complaint, is among the contemporary disorders related to exhaustion and is to be found in the pages of weekly magazines as well as being a subject of concern in psychology and workplace medicine. Our goal in this article is to address specifically the twentieh century version of disorders related to fatigue, namely neurasthenia, examining the conditions of its emergence at the end of the nineteenth century and the elements that led to its decline in the first half of the following century.

Neurasthenia emerged as a category in mid-1869, based on the early publications of the New York neurologist, George Miller Beard (1838-1883). Prior to that date, he had been an in-depth expert on methods of electrification and had published reference works on the medical use of these methods. Beard can be attributed with the merit of isolating neurasthenia as a clinical entity from sundry other nervous complaints. The golden age of this category was mainly from the 1880s onwards, after the aforesaid author published *A practical treatise on nervous exhaustion (neurasthenia)* in 1880, followed by *American nervousness*: its causes and consequences in 1881. The decline of the category, in turn, occurred from 1920 onwards, and we will also dedicate ourselves in this article to the variables that contributed to this process. It is important to emphasize that even in the middle of the twentieh century there was a revival of this diagnosis in China, though under a different guise, as Kleinman analysis (1986).

Roughly speaking, the symptoms of neurasthenia were presented in a very varied manner and included general exhaustion, sensitivity of the spine (spinal irritation) and body (general hyperesthesia), localized and peripheral numbness, vague pains and neuralgias, temporary paralysis and gastric disorders (dyspepsia, dysphagia). They also included headaches, sleep disorders, pressure in the head, deficient mental control, difficulty concentrating, mental irritability, morbid fears (phobias in general), sexual disorders (in males, involuntary emission of semen, partial or total impotence, irritation of the urethra; and in females, organ displacement, inflammations, ovarian and uterine irritation). Neurasthenics were easily fatigued and slow to recover, with the fatigue not being alleviated by sleep or rest. Despite this wealth of symptoms – not always all appearing in one patient – neurasthenics enjoyed good health and were normally well nourished and muscularly developed.

According to a report by Wessely (1990), Beard and his colleagues justified the absence of physical symptoms of the illness on the assumption of a functional lesion[1] in the brain – supposedly, a submicroscopic pathological change in the nutrition of brain cells. Being a condition whose organic basis is unclear, it was often attributed to a combination of

cerebral overload and nervous diathesis, and the emphasis on each of these poles varied according to the different authors, although the debilitation that the demands of civilization caused on the functioning of the brain was the most emphasized point. Despite pointing to the role of nervous diathesis, Beard had no doubts as to the etiology of the condition: the cause of American nervousness was civilization and its corollaries, though it did not provoke it on its own. However, Beard and his contemporaries never doubted that neurasthenia is an organic disease and that the absence of macroscopic characteristics only reflected the limitations of the investigative techniques of the time, as Rosenberg (1962) also points out.

In general, this entity articulated an idea of medico-scientific objectivity demonstrated by the assumption of organicity, which allowed it to be accepted as a legitimate diagnosis by physicians and patients. Furthermore, it was accepted by medical ideas in vogue, such as social evolutionism and degeneration theory, as demonstrated by the initial gesture of Beard – supported by his followers – of declaring the disease as a result of highly developed societies (such as the United States) affecting people in the upper social echelons. The theory of degeneration, in turn, was the basic prerequisite for the assumption of a transmissible condition to which the nervous diathesis of neurasthenics was attributed.

This category was upheld in the moral and medical paradigms of the time and in its standards of objectivity, to the extent that its decline was linked to changes in its conditions of possibility. There was a 'democratization' of the diagnosis, which came to be given to patients of the proletarian classes, no longer being exclusive and a mark of distinction of the most refined. Concomitantly, with the vogue of the psychogenetic paradigm from the beginning of the twentieh century, especially after the World War I, neurasthenia 'exploded' in other diagnoses linked to the category of neuroses and psychoneuroses then emerging, making the assumption of organicity well nigh irrelevant. When the elements that led to its existence lost importance, the diagnosis went into frank decline. We will proceed to analyze the conditions of possibility of this medical category below.

Some bases of support for neurasthenia

The depletion of the nervous supply

It is interesting to note that, as Gay (1990) pointed out, until 1800 the word 'nervous' had connotations of energy, strength and liberation from debility. A nervous racehorse was one with an extra reserve of energy. The old meaning of the term co-existed with its connotation of lack of energy for the next one hundred years. Beard and other physicians assumed that nervous excitability and lethargy, excessive activity and lack of activity, excellent health and debilitating disease were primarily attributable to the patient's nervous reserves. They even assumed that it might be possible to deduce the nature of the moral exhaustion from one's nervous condition and declarations. Nervous energy then came to be understood based on the metaphor of a reservoir that should be saved for future usage and, for this reason, it should be spent wisely.

The hypotheses of Beard and his colleagues occur in a context in which physicians discussed the pathologies on the basis of heat and energy. According to Lutz (1989, 1991),

medical theory was preceded by and based on popular prevailing theories regarding body energy that were clearly economic in nature. It was thought that people had a certain amount of nervous energy to be expended, reinvested or wasted. Work and procreation were seen as examples of the expenditure of nervous energy. Masturbation and illicit forms of sex were seen as an expenditure of nervous energy without any parallel reinvestment. The fear of dissipation was based on the possibility of dispersal of nervous energy and irreplaceable expenditure, capable of leading to decadence, deterioration of the nerve centers of the individual and, in the worst case scenario, the decrepitude of civilization. The end result of the dissipation process or of any imprudent nervous investment could be neurasthenia.

Similarly, if the patients were sensitive and refined, they could develop the disease despite their moral probity, by simple exposure to the agitation and over-stimulation of modern life, and by nervous diathesis. Life at the end of the twentieh century, transformed by urbanized and industrialized technology, may not have had any discernible effect on the proletariat, but it was enough to completely exhaust the so-called more civilized strata of the population. The disease was a sign of sensitivity. The consequence of the theories based on the idea of nervous exhaustion was to recommend treatment by rest and forced inactivity to conserve the limited supply of energy.

Porter (2001) reminds us that the second law of thermodynamics, proposed around the middle of the nineteenth century, contends that the amount of available energy in the universe is decreasing gradually and inexorably. This formulation was incorporated and became highly visible in the conceptualization of neurasthenia, as well as in the Freudian concepts regarding the psychic apparatus and psychoneuroses. In the specific case of neurasthenia, mental or physical overload could deplete the nervous energy supply of individuals, rendering their systems deficient. The disease was therefore the price to be paid for the expenditure of energy in any area of the body that, in the final analysis, was the brain. The increased demand for energy consumed brainpower and withdrew it from the periphery, causing fatigue and other symptoms.

In addition to this, Roelke (2001) states that the work of physiologists and clinicians such as Emil Du Bois-Reymond (1818-1896) and Guillaume Duchenne (1806-1875), the studies with electrical stimulation of the brain conducted by Eduard Hitzig (1838-1907) and Gustav Fritsch (1837-1927), the collaborations of Schmiedebach (2001) and Gosling (1987), the theories of the reflex arc of Marshall Hall (1858-1927) and the studies of Brown-Séquard (1817-1894) on the inheritance of acquired characteristics were the pillars for the construction of a mechanistic model to explain the pathologies of nervous exhaustion and gave rise to a consensus around the importance of electricity for the functioning of the nervous system. Later, during the course of the nineteenth century, theories of nervous exhaustion became integrated with theories of heredity and degeneration.

It is important to remember that electricity was the main means of industrial and urban energy, and was therefore the mainspring of all types of processes associated with modern life at the time. It seemed plausible, therefore, that in the last decades of the century, the nervous system was thought of as a complex of fibers and cells activated by electrical impulses and energy flows from the center (brain) to the periphery (nerves and organs) and vice versa. The concept of energy conservation in a closed system, legitimated

by physics and physiology, possessed considerable explanatory power for everyday experiences as well as diseases.

The studies on fatigue

Studies on neurasthenia fall into the broader context of works relating to fatigue. According to Rabinbach (1990), the interest in fatigue permeated the final decades of the nineteenth century and merely served to confirm the nineteenth-century anxiety about the possibility of society being unable to withstand the increasing pressures of modernization. For physicians, physiologists and reformers of the nineteenth century, fatigue did not exist in isolation from issues such as desire and morality and the entire gamut of social forces. Being overstretched, the developed nations were given over to the vicissitudes of unregulated desire, to the emotions and to the enemies of productive order. Therefore, in addition to expressing the entropy associated with the conservation of energy, fatigue also represented a threat to modernization. For this reason, the author states that nothing was considered more disturbing than fatigue among the young. In the summer of 1887, the French Academy of Medicine was assailed by the problem of exhaustion among its students. The crisis was attributed to the exaggerated demands of the accumulation of knowledge required of young people, such as highly competitive admission examinations, Greek and Latin classes, assignments to be completed at home and recent additions to the curriculum, such as natural history lessons, for example.

Germany also suffered from these problems. A study by Schmiedebach (2001) reviewed journals published between 1880 and 1919 with issues related to neurasthenia and noted that the question of *Schulnervosität* and the intellectual demands on children became a prominent topic from 1870 onwards. This concern culminated in a series of suggestions for reforming the curriculum and the establishment of regular times for games and physical training, to prevent overwork. According to Bakker (2001), neurasthenia became very attractive as a diagnosis for children who had problems of indiscipline and lack of attention in the classroom, and useful for specialists in education and for parents and helping to promote a range of prophylactic educational measures.

It was in this context that works emerged in which the central theme was fatigue. This was either from a purely physiological standpoint, such as *La fatica* (1891)[2], Angelo Mosso (1846-1910), or not, as *La fatigue intellectuelle* of Binet and Henri (1898), which discusses the influences provoked by intellectual stress on the organism and on the different mental functions, based on the problem of academic overloading. Therefore the 1890s saw the emergence of research into exhaustion – including intellectual exhaustion, as the health of American and European youth was at a bad juncture, arising either from the new demands now being made by the education system, or the labor market. Neurasthenia appeared then as the clinical entity to deal with cases of pathological fatigue.

Gosling (1987) refers to neurasthenics as the group between the majority of mentally healthy individuals who quickly embraced the values of moderation, rationality and ambition, impregnated in the emerging urban classes in the United States at the end of the nineteenth century, and another group suffering from the torments of insanity. Neurasthenics aspired to a useful place in society, but because of a supposedly invisible

defect, they achieved neither mental harmony nor social adaptation. They lacked "the mysterious substance that gives vitality to the organism" (p.X), the absence of which weakened the central nervous system and the will as a whole.

Early in its career as a diagnosis, fatigue was only a problem for those in more socially prominent positions. An adept of social evolutionism, especially in *American nervousness*, Beard's (1881) outlook was imbued with the evolutionist terminology and viewpoint. It is not for nothing that this disease was considered the price that, in principle, the Americans – and later all developed societies – would have to pay for progress. It mainly affected the brain-workers, whose supply of nervous energy was destroyed by the impositions of industrialized urban life, and high-class girls, with their delicate nervous systems unfit for the demands of life emerging in major cities. As was pointed out by Blocq (1891) and Laurent (1897), it afflicted the so-called more advanced ethnic groups such as Jews, Slavs and Anglo-Saxons, but not the blacks and Asian immigrants.[3]

López-Piñero (1983) agrees with the idea that, from these origins, neurasthenia provided the conceptual framework and social climate which made *A practical treatise on nervous exhaustion (neurasthenia)* an immediate success when it appeared in 1880. It was followed by the works of Mitchell, in 1878 and 1881 (Mitchell, 2004, 1881), describing the female form of neurasthenia and suggesting a therapeutic program. From that time onwards, neurasthenia became one of the fashionable diseases, catching the attention and acquiring a following among neurologists of the time. The success of the term made it possible to forget that it was created to refer to a particular disease in the United States, once it crossed the Atlantic and took hold in Europe.[4]

Rabinbach (1990) also espouses these viewpoints, arguing that the elaboration of neurasthenia as a medical entity provided a scientific and lexicographical basis for the socially available ideas on the following relationships: the body with the will; the will with values, and values with the progress of civilization or the threats to its existence. The author points to an important detail, which is the fact that neurasthenic exhaustion is quintessentially intellectual: in other words, it represents the dissipation of the brain rather than the body. In this context, it is worth reiterating the fact that cerebral work was and is associated with the culture and the work of the higher classes.

This context was instrumental in giving neurasthenia a degree of scientific legitimacy, because it tallied with degeneration theory and with the changes in the society of the late nineteenth century. It also fitted in with the concepts of social evolutionism and the medical theories that demanded organicity for nervous diseases: "thus, neurasthenia gained popularity because it reinforced the attitudes of class and gender; as a result, neurasthenia and its various causes helped to 'prove' the validity of the dominant social theories" (Gosling, 1987, p.XI). It then became an umbrella diagnosis, applied to a syndrome with vague and varied symptoms, and a variety of unverifiable physical complaints.

Speculative somatization

According to Wessely (1990), hardly any relationship could be detected in the history of neurasthenia between the physical symptoms and organic findings or between the patient's recovery and the resolution of any physical abnormalities. Despite this, the disease

was considered to be organic, and also the as-yet-indemonstrable pathological alterations were considered to be its causes.

It is important for us to understand this desire for organicity more fully. One element that contributes to it, as demonstrated by Rosenberg (1989), is the fact that the second half of the nineteenth century was a crucial period for the articulation between etiological hypotheses and moral assumptions, or between emotions and their seemingly pathological results. This period is considered by the author to have been a time of expansion of the categories of disorders that combined emotional characteristics, altered mood states and behavior deviations, to be legitimated as supposedly organic diseases. There were many conditions that aspired to the status of emerging diseases from 1860 onwards, such as alcoholism, Erichsen's disease or *railroad spine*[5] (spinal cord concussion) and irritable heart or soldier's heart (Da Costa's syndrome).

Categories of disease therefore began to be used to explain a variety of forms of behavior that were either socially stigmatized or considered self-destructive. An attempt was made to justify these by material mechanisms – physiological findings that purported to be the cause of these conditions and afford them some legitimacy – which is shown by the dissemination of somatics as the preferred explanation. According to Rosenberg (2006), these categories create norms and define deviations, rationalize idiosyncrasies and explain human suffering by means of deterministic and mechanical strategies. Morally reprehensible behavior metamorphosed into pathologies then come to have medical treatment and management. For the author, Beard (1869) rationalized his discovery in material terms because he had no choice if he wanted to be taken seriously by his peers. Social and scientific legitimacy presumed a somatic identity, in other word some physiological principle or mechanism to justify the symptomatic framework. "I am sure that it [neurasthenia] will be substantially confirmed over time by microscopic chemical examinations of patients who died in a neurasthenic condition" (p.218). However, this speculative somatization, as defined by Rosenberg (2006), dated from way before Beard's time.

There are many possible examples of the somatic mechanisms to be found for diseases, as listed by Rosenberg (1989). Sympathy and irritability, and the nineteenth century fashion of heredity and social evolutionism all constituted significant factors that linked biology to behavior. The pathological anatomy, with its emphasis on localized lesions, and studies on normal and abnormal physiology pointed to the articulation of stable disease entities, which were incorporated into private individuals and explained in terms of causal mechanisms in the bodies of the sufferers.

The diseases were then equated with the idea of specificity expressed in terms of a physiopathological mechanism and, in effect, construed as existing outside the body as abstract entities. The process of diagnosis then became focused on the presupposition of this specificity, being constructed by means of laboratory tests, defining thresholds, statistically determined risk factors and certain other artifacts seemingly devoid of value. Even in a speculative manner, the somaticity of diseases came to be required for them to be considered real.

The growing prestige of the emerging biomedical sciences of the time (histology, biochemistry, physiology, pharmacology) contributed directly to the exacerbation of this

process. It would culminate in the twentieh century in the cytological and imaging procedures that claimed to generate ever more accurate guarantees for the construction of diagnoses. The process described is not exclusive to neurasthenia, but it is certainly one of its conditions of possibility. According to Rosenberg (1989), this speculative somatization is one of the rhetorical prices to be paid in order to permit the medical approach of nervous diseases at that time, which is a process in which the entity we are addressing is included.

Since then, explanations for problematic behavior and emotions take shape in various nosological entities, neurasthenia being one of them. The innovation included by Beard, as highlighted by Rosenberg (2006), was to build a specific nosological entity considering the various concerns of the time, naming it from acceptable scientific concepts at that time and reuniting the many complaints into an explanatory model for the disease.

It is necessary to highlight the crucial role of the concept of functional damage in this process, because it made it possible to approach the disease with no known organic cause, without altering the sovereignty of somatic mechanisms in its justification. This is, in fact, a starting point for entering the field of studies on neurasthenia, as common to its various propagators is the fact of considering it as arising from a functional lesion of the nervous system, as in Beard (1869, 1880, 1881), Blocq (1891), Charcot (1888b), Beevor (1898), Laurent (1897), Levillain (1891), Mitchell (1881, 2004,), Bouveret (1891), Proust and Ballet (1897) and Savill (1906).

A distinction is needed. The structural lesion was that which was detectable upon clinical examination, demonstrable in laboratory evaluation or through other medical technologies. The more uncertain functional injuries involved an implicit pathological process and their existence was speculative because there were no changes in the structure of tissues and organs. This was the case with neurasthenia, considered an organic disease, despite not being linked to changes in the tissues or organs, but only in their operation. Faulty operation of the nervous system, albeit imperceptible, was attributed to the disease. The pathological changes were supposedly submicroscopic and related to nutrition of the brain cells, therefore invisible, despite being real: “What the microscope can see we call structural; what it cannot see we call functional” (Beard, 1880, p. 95).

Ehrenberg (1998) brings to light some issues that should be discussed thoroughly. For the author, neurasthenia is the starting point for attention to social factors as the cause of diseases. This category made it insufficient to refer to heredity, and, through the concept of functional lesion, there was a switch of the model of the disease as an anatomical lesion to that of the disease as a pathological reaction to the environment, without necessarily the presence of any structural lesion. Despite being present in the etiologic hypotheses, degeneration was not sufficient explanation, and for this reason the author claimed that the social factor was in the forefront of the etiology.

For Ehrenberg (1998), the concept of functional lesion promotes the permeability of the mental aspect in medical explanations, attracting a new form of socialization of the mind. The mental aspect begins to be considered by the supposition of the influence of that which is outside it, i.e. that which is not intrinsic to it – in this case, the potentially hostile characteristics of modernity. This therefore opens up the possibility of thinking

that society can bring illness upon its participants and, especially, that it is not only nature that can cause disease, but also culture.

The interesting thing is to understand that a first reading of these statements by Ehrenberg leads us to associate neurasthenia with etiological explanations and hypotheses that are predominantly psychogenic or psychosocial. Since the causes of the disease are social, it does not only arise from ineluctable heredity, but also from the exercise of action and will. Curiously, a reading of the primary sources shows that this openness to the realm of desire is based mainly on a view of heredity as something that moral education can change. There is not, however, any lesser appeal to organic origins as being responsible for the source of the disease. On the contrary, heredity[6] and somatogenesis, taken as a starting point, are responsible for it, thereby justifying the need for biomedical care. Perhaps the emphasis on culture as a producer of disease was only accepted as an explanation for the unquestionable somaticity upon which neurasthenia was based.

The concept of functional lesion, is however no less important, as it is that which dictates the division between what is voluntary and involuntary, intentional or unintentional. Whatever the case, the way the social causes impinged on the nervous system remained vague and obscure, and a pathogenic basis was lacking for establishing the relationship between brain, mind and society, and the boundaries between the endogenous and the exogenous. Given the absence of anatomical lesions, what could the mechanism producing the symptom be? What could one do about it? The concept of functional lesion addressed these questions. It is therefore seen that a disease that seemingly had no anatomical basis was not synonymous with a disease without a cause. In the case of neurasthenia, the somatic causality is not abandoned. It is replaced instead by the assumption of somatogenesis, in which the physical agent cannot be found by the means available.

Claiming that the clash with modernity was the most plausible explanation for the diagnosis of neurasthenia at its crowning moment should not mean, in our opinion, shunning the organic grounds on which the disease was based. Indeed, the work of Rosenberg (1989, 2006) stressed the omnipresence of the somatic as the preferred explanation, at a time when such a category existed. The main operator of this speculative somatization is the concept of functional lesion, which oscillates between two poles: for almost the entire duration of the existence of the category, the words served as a euphemism for organic with no change in structure. As the psychogenic paradigm took shape, the idea of 'functional' came to include the psychogenesis of physical symptoms, making room for the emergence of a psychoanalytic interpretation of diseases *sine materia* and heralding the falling out of favor of the neurasthenic diagnosis. Only at the end of the career of the diagnosis of neurasthenia, already under the focus of the psychogenic paradigm, it also began to be possible to think in this way about the notion of functional lesions. Initially, however, and during its heyday, 'functional' was simply another way of referring to organic ailments with no known causes.[7]

The development of neurasthenia as a disease over the course of time, runs counter to, or at least stymies the assumptions for its existence: class, heredity and organicism. As Wessely (1994) informs us, although the disease was initially considered fashionable in

the most affluent segments, neurasthenia gradually filtered down to the other social classes. It is possible, however, that although physicians maintained that the symptoms of neurasthenia were restricted to the upper classes, they could also be found in the lower classes. But Beard's patients, for example, being exclusively from the upper social class, offered a very limited sample of those afflicted. What is observed is that the democratization of neurasthenia, coupled with other factors, contributed to its decline, as we shall see below.

The decline of neurasthenia

With respect to the decline of neurasthenia as a diagnosis, there are various elements to highlight as being factors that contributed to this process. Its disappearance is directly related to a change in the conditions that had made it a useful and socially accepted diagnosis. The emergence of the psychogenetic paradigm at the beginning of the twentieh century changed its status and its usefulness as a diagnosis, with other categories adapted to new conditions emerging, namely psychoneuroses. There also occurred a 'democratization' of the diagnosis, now also attributed to patients of the proletarian class, no longer being a sign of distinction of the upper classes. When the elements that justified its existence became dissipated in the rhythm of socio-medical change, the diagnosis went into decline.

In these circumstances, Wessely (1990) points out that medical skepticism related to that category grew and the sophistication of psychiatric nosology made the general nature of the diagnosis unsustainable. Pierre Janet (1859-1947) defined the characteristics of psychastenia on the basis of neurasthenia, as did Sigmund Freud with the anxiety neurosis.[8]

In order to understand this process, it is useful to turn to the painstaking analysis by Taylor (2001) of data collected from the historical records of the National Hospital Queen Square in London. Originally called The National Hospital for the Relief and Cure of the Paralysed and Epileptic, the institution started its activities geared to charitable works in 1860 with ten beds. Its remit was to provide medical care to the poor, and it was the first hospital in London to specialize in disorders of the nervous system. In forty years, the institution grew significantly, with fully two hundred beds by the turn of the twentieh century, by which time it had become a center of excellence in training, research and treatment of functional disorders and attracted patients from all over England and even from other countries.

Taylor had access to the annual reports of the hospital – which cover the period from 1860 to 1947 – and compiled data relating to the diagnosis, nature, and results of treatment from the number of neurasthenic patients discharged from treatment. Although Taylor's research only covers the records from this hospital – and not the wider panorama of the diagnosis in other European capitals or in other London hospitals – it acquires relevance by virtue of the institution having been a prominent center for the study and treatment of nervous diseases, and serves as a small illustrative sample of the situation of the diagnosis in the period under scrutiny.

On the rise and fall of neurasthenia, Taylor (2001) states that the diagnosis first appeared in 1886, followed by a linear increase, peaking in 1906, with 11% of patients discharged;

in 1908 the average fell to 7.4% of patients discharged, and in 1928 there was another peak of 10%. There follows a steep decline to 3% of patients discharged in 1930, and 1% in 1935, after which the diagnosis virtually disappeared. In its golden age, the prevalence of neurasthenia was equivalent to other important organic diagnoses, such as brain tumors, which had between 5% and 10% of patients discharged per year.[9] This was the period of its first appearance as a separate category, until it fell out of favor and became a subcategory of psychoneurosis. From 1932 to 1944 the prevalence of new categories related to psychoneuroses scored an average of 7%, falling to 3% between 1945 and 1957, when the Queen Square report ends.

The data of this sample contradict the myth outlined and upheld by many physicians of neurasthenia that this disease was a hallmark of the wealthy classes, as the Queen Square offered care and treatment to the poor. However, there is a marked discrepancy between the original description that Beard makes of the disease in 1869, and the first appearance of patients discharged from Queen Square Hospital, which one imagines to be the time necessary for the spread of the diagnosis to other continents.

In line with Taylor, Savill (1906), already at the turn of the twentieh century, drew attention to the number of poor patients in anther charity hospital, namely the Paddington Workhouse. The author compared data from private and public practice, and found no evidence of any class trend in neurasthenia. It is possible that this diagnosis had also appeared and become prevalent in the lower classes at the same time as it appeared in the upper classes, although Beard, Mitchell and some of its main proponents have samples that are highly restricted to patients from the upper classes, omitting data regarding its existence in the other population brackets.

According to the abovementioned report, the proportion between the sexes was 50%/50% when the diagnosis first appeared. The proportion of men afflicted fell between 1891 and 1905, but was never below 1/3. The male prevalence then increased gradually until there was a slight preponderance of men, between 1911 and 1915, and remained so until 1920, when a slight female preponderance began to prevail. It is important to note that the increase in male neurasthenia occurred during World War I and was linked to the return of combatants. Between 1914 and 1918, 1/3 of hospital beds available at Queen Square were occupied by the military. In 1918 the institution opened a special home for the treatment of neurasthenic officers, and many other men were also admitted. However, as the author emphasizes, the war does not fully explain the prevalence of male neurasthenia.

The sociological explanations were also changing. Doubt was being cast on whether neurasthenia was really a disease of modern life, under the justification that it was the population that was becoming more sensitive in its illnesses. Other etiologies were put forward, such as poor housing or poor dental hygiene arising from the vogue of eating ice cream, prevalent among children of the lower classes, as shown by Savill (1906). Gradually, the disease lost its characteristic of being a mark of social status and a sign of sophistication.

Another point that contributed to the decline of neurasthenia is the idea defended by Shorter (1992) that the assumption of organicity, which always indirectly supported the diagnosis, came to be challenged by the attribution of the psychic origin of symptoms. It was around 1920 that the category lost popularity, with the emergence of a new paradigm

which, among other things, stated that the origin of symptoms was in mental processes and not in any disorder of the central nervous system. The basis of the somatic model thus became discredited.

For Will (1998), the proposal of rest therapy[10] used in neurasthenia, was consequently questioned – if there was no cellular basis for exhaustion and if the psychogenic hypotheses were beginning to emerge, what was the purpose of rest? The growing awareness was that diet, massage and electricity in the healing process were merely ways of encouraging the patient to slow down their expenditure. Even the apparent efficacy of electrical treatment came to be attributed more to psychological reasons than to possible organic modifications of nerve centers. There was a decline of the rest cure, which was superseded by new speech and occupational therapies.

The organic model of neurasthenia lost ground in favor of the ever increasing status of the new schools of thought, and management of the neurasthenic patient gradually migrated from the neurologist to the psychiatrist. Since neurasthenia was not clearly organic, nor was it psychological enough to continue to be sustained as a diagnosis.

In the gradual shift to the psychogenic paradigm, neurasthenia continued initially as a diagnosis, but it was considered to be more psychological than physical. The psychological symptoms, initially considered to be a result of the disease were gaining in importance, although not supremacy, and finally came to be seen as its cause. After that, the category was dismembered and replaced by new psychiatric diagnoses, and the space set aside for it in the texts on neurology was reduced and finally disappeared altogether. Either that or the disease began to receive scant psychiatric mention. Gosling (1987) also confirms this process of transformation of the etiological concepts regarding neurasthenia, from being a somatic disease with a variety of physical and mental manifestations to being an essentially mental disorder.

The development of theories about psychogenic mechanisms of illnesses began, according to Shorter (1992), with a debt to neurasthenia. Freud (1996), for example, distinguishes the anxiety neurosis from neurasthenia and its all-encompassing characterization. The first attributes symptoms such as general irritability, expectative anxiety, respiratory disorders, episodes of sweating, trembling, cold sweats, among others. The most important change, however, was with respect to the etiological factor, since for Freud a number of disturbances and influences of the sexual life of the sufferer were at stake. Pierre Janet, in turn, introduced the term psychastenia in 1903, in *Les obsessions et la psychasténie*.[11] For Shorter (1992), Janet's psychastenia contained many features of obsessive and phobic neuroses, which would come to be better defined later. The decline in the prevalence of neurasthenia between 1920 and 1930 therefore coincides with the establishment of sub-categories of psychoneurosis. The diagnosis disappeared, but not the symptoms, which were reassigned to the new categories that had emerged.

It is possible that neurasthenia, as a cluster of complex symptoms, had never really disappeared, but came to be modified according to prevailing nosology. This argument is supported by Shorter (1992), who emphasizes the diagnostic fluctuation of certain categories, as also happened with hysteria. It is also possible that the situation equivalent to the disorder treated here was in fact less prevalent in that period, rather than just distributed

to other categories. After neurasthenia apparently disappeared, physicians continued to see cases with similar symptoms, but did not make diagnoses.

Before the emergence of the psychogenic paradigm, neurasthenia served a purpose, as explained by Sicherman (1977): at a time when physicians only felt comfortable with clearly organic disorders, a diagnosis like this made the situation medically tangible, offering essentially psychological therapy under the somatic label. With the rise of the psychogenic school, this function of the diagnosis was lost.

The term was revived in non-Western cultures, particularly in China, where it was called *shenjing shuairuo* (neurological weakness), during the 1970s and 1980s – as was indicated in the renowned analysis of Kleinman (1986). But why did a diagnosis discarded in western and European countries reappear in China, becoming the most widely used in medical, psychiatric and lay circles in that country? To answer this question, Kleinman sought to understand how suffering acquired significance and was interpreted in the Chinese cultural context, and how this process contrasted with those in the West, where, at the same time, the diagnosis of depression and somatization was increasing significantly.

Kleinman investigated the relationship between neurasthenia in China, and somatization and depression, especially in the United States, in an attempt to understand how culture contributes to the interpretation and negotiation of the experience of illness. His interest was to situate each of these entities in relation to their consequences – demoralizing or otherwise – and the ways in which they acquire legitimacy or are rejected in a given group and their power systems. One of the points emphasized in the study is the fact of neurasthenia being understood in the Chinese context, as a brain disorder involving cortical weakness. On the other hand, the psychological symptoms were understood as being from somatic sources, which gave to patients, to use the words of Ray and Gosling (1986), the right to fall ill without the stigma of mental illness.

The studies of Ware and Kleinman (1992) pointed to the fact that the political and social ramifications of China's Cultural Revolution, at the end of the 1960s, changed the perception of many people about the prospects for the future. Many people lost their homes, jobs and social status. Families were separated, and several of their members had to migrate to rural areas due to increased rates of violence. The legacy of this process was experienced during the 1970s and 1980s, in which parents were mourning their children and vice versa; the urban professionals who now lived in the countryside and still felt dissatisfied with their condition; young adults struggling to catch up with the years of education that they had missed while engaged in political struggles. Neurasthenics in China suffered from mistrust in political freedom and any improvement in the quality of life. The symptoms of the disease in the Chinese context were a metaphor for the social experience the sufferers were living through.

In Western cultures, signs of the revival of neurasthenia from the 1980s onwards were reflected in the rise of chronic fatigue syndrome as a nosological entity. A point in common between neurasthenia and the chronic fatigue syndrome is that, in spite of its causes being related to environmental pressures and demands, it is in the physiology of the body that the manifestations are predominantly to be found. It is the depletion of brain cells – in the case of neurasthenia – and in viruses, immune deficiency and brain abnormalities

– in the case of the chronic fatigue syndrome – which, in theory, produce each of the set of symptoms. What is at stake in arguments, both with respect to neurasthenia and to the chronic fatigue syndrome, is what should be considered a legitimate disease and, consequently, legitimate suffering deserving support, empathy, care, and in the final analysis, social security. It is the answers of each socio-medical moment to these questions that ensures the survival – or demise – of neurasthenia and its derivations in the present.

NOTES

[1] Jean Martin Charcot (1888a) also uses the idea of dynamic or functional lesion, initially in cases of paralysis due to traumatic hysteria. The author describes differences between cortical lesions and those caused by hysteria: the former had limited outbreaks and are distributed randomly in the motor and sensory regions of the cortex, being distinct and distant from each other; the dynamic lesions of hysteria, besides being diffuse, simultaneously and systematically affect the motor and sensory regions that are physiologically involved in the execution of the movement of a given articulation – there was, therefore, a cortical lesion that was not structural in the patients examined.

[2] I did not consult this work in the preparation of this article, but for further information on this topic, see Mosso, 1891.

[3] Some non-Anglo-Saxon and non-Protestant groups were considered moderately nervous and were situated between the strength of the barbarians and the sensitivity of the highly civilized, according to Beard (1881).

[4] According to Gijswijt-Hofstra and Porter (2001), the reception given to neurasthenia was varied as it crossed the ocean and took root in Germany, France, England and Holland, which meant that the category acquired individualized traits and interpretations. For more detailed discussions on the reception of the category in the different European nations, see the in-depth work of Gijswijt-Hofstra and Porter (2001).

[5] The discovery of Erichsen's disease is attributed to John Eric Erichsen and deals with spinal cord concussion. The disease was frequent from the middle of the nineteenth century onwards and was characterized by disquiet with respect to road travel and possible accidents in transit. The symptoms noted were irritability, inability to rest, malaise, pain in the body, and sometimes even paralysis (Hodgkiss, 2000).

[6] Schmiedebach (2001) and Slijkhuis (2001) draw attention to another point: the Lamarckian nature of heredity upon which the neurasthenic condition was based. Most authors accepted the existence of an individual nervous disposition, which was not considered a pessimistic argument; on the contrary, heredity implied the need to increase the effort of will to overcome the effects of the particular nature of the individual. Since they inherited a weak nervous constitution, neurasthenics could not be cured and, therefore, had to be educated to live with and optimize their weaknesses. The explanations for neurasthenia were therefore a combination of inherited weakness with personal effort, which implicitly sustained an important theme in the United States at the time: moderation was the only path to health and happiness. Rabinbach (1990) even claimed that neurasthenia, understood as a clash with modernity, is more widely defended by American theorists and that the emphasis on hereditary factors as the main focus, is concentrated particularly in European authors, especially the French. Moreover, according to Roelke (1997, 2001), the value of heredity as a component of neurasthenia varies as the diagnosis is developed, especially when faced with the influence of Germanic nosology.

[7] While still on the subject of the quest to understand the various somatic axes of neurasthenia, Wessely (1990) draws attention to the fact that the original descriptions of the disease did not suggest an infectious origin for it. Only in Van Deusen (1869), in Blocq (1891) and in Savill (1906) does one find mention of infections as possible triggers of neurasthenia.

[8] Here I use the expression *neurose de angústia* (*Angstneurose*; anxiety neurosis) following the translation of the text "Sobre os fundamentos para destacar da neurastenia uma síndrome específica denominada 'neurose de angústia'" (Freud, 1996; "On the grounds for detaching a particular syndrome from neurasthenia under the description 'anxiety neurosis'"), although we should not overlook the important debate on the translation to Portuguese of the word *Angst* and its derivatives in the work of Freud. In this regard, an introductory reference is to be found in Hanns, 1996.

[9] The research by Wessely (1990), for whom there was an apparent decrease in the prevalence of neurasthenia to the point of its disappearance, points to the same findings of data from Taylor (2001)

[10] The treatment of neurasthenia, well covered by Mitchell (2004, 1881), was based mainly on the rest cure, the use of hydrotherapy, massage, diet and stimulation by electricity.

[11] I did not consult this work in the preparation of this article, but for further information on this topic, see Janet, 1903.

REFERENCES

BAKKER, Nelleke.
A harmless disease: children and neurasthenia in the Netherlands. In: Gijswijt-Hofstra, Marijke; Porter, Roy (Ed.). *Cultures of neurasthenia*: from Beard to the First World War. Amsterdam: Rodopi. p.309-327. 2001.

BEARD, George.
American nervousness: its causes and consequences. New York: Putnam Sons. 1881.

BEARD, George.
A practical treatise on nervous exhaustion (neurasthenia). New York: William Wood. 1880.

BEARD, George.
Neurasthenia, or nervous exhaustion. *Boston Medical and Surgical Journal*, Boston, v.80, p.217-221. 1869.

BEEVOR, Charles Edward.
Diseases of the nervous system: a handbook for students and practitioners. London: H.K. Lewis. 1898.

BLOCQ, Paul.
Neurasthenia. *Brain*, London, v.14, p.306-334. 1891.

BINET, Alfred; HENRI, Victor.
La fatigue intellectuelle. Paris: Schleicher. 1898.

BOUVERET, Leon.
La neurasthénie : epuisement nerveux. Paris: J.B Baillière. 1891.

CHARCOT, Jean Martin.
Leçons du mardi à la Salpêtrière, septième leçon : progrès medicale. t.1. Paris: Lecrosniew & Babe. p.111-34. 1888a.

CHARCOT, Jean Martin.
Leçons du mardi à la Salpêtrière, douzième leçon : progrès medicale. t.2. Paris: Lecrosniew & Babe. p.247-69. 1888b.

EHRENBERG, Alain.
La fatigue d'être soi : depréssion et societé. Paris: Odile Jacob. 1998.

FREUD, Sigmund.
Sobre os fundamentos para destacar da neurastenia uma síndrome específica denominada 'neurose de angústia'. Rio de Janeiro: Imago. (Obras Psicológicas Completas de Sigmund Freud, v.3). 1.ed., 1985. 1996.

GAY, Peter.
A paixão terna: a experiência burguesa da rainha Vitória a Freud. Rio de Janeiro: Companhia das Letras. 1990.

GIJSWIJT-HOFSTRA, Marikje; PORTER, Roy.
Cultures of neurasthenia: from Beard to the First World War. Amsterdam: Rodopi. 2001.

GOSLING, Francis.
Before Freud: neurasthenia and the American medical community, 1870-1910. Urbana: University of Illinois Press. 1987.

GOSLING, Francis; RAY, Joyce.
The right to be sick: American physicians and nervous patients, 1885-1910. *Journal of Social History*, Fairfax, v.20, n.2, p.251-267. 1986.

HANNS, Luiz. *Dicionário comentado do alemão de Freud*. Rio de Janeiro: Imago. 1996.

HODGKISS, Andrew.
From lesion to metaphor: chronic pain in British, French and German medical writings, 1800-1914. Amsterdam: Rodopi. 2000.

JANET, Pierre.
Les obsessions et la psychasténie. Paris: Baillière. 1903.

KLEINMAN, Arthur.
Social origins of distress and disease: depression, neurasthenia, and pain in modern China. New Haven: Yale University Press. 1986.

LAURENT, Émile.
La neurasthénie et son traitement. Paris: A. Maloine. 1897.

LEVILLAIN, Fernand.
La neurasthénie, maladie de Beard. Paris: A Maloine. 1891.

LÓPEZ-PIÑERO, José Maria.
Historical origins of the concept of neurosis. London: Cambridge University Press. 1983.

LUTZ, Tom.
American nervousness, 1903. An anecdoctal history. Ithaca: Cornell University Press. 1991.

LUTZ, Tom.
Neurasthenia and fatigue syndromes: social section. In: Berrios, German; Porter, Roy (Ed.). *A history of clinical psychiatry*: the origins and history of psychiatric disorders. London: Athlone. p.533-544. 1989.

MITCHELL, Sillas Weir.
Fat and blood: hints for the overworked. New York: Altamira Press. 1.ed., 1878. 2004.

MITCHELL, Sillas Weir.
Lectures on diseases of the nervous system especially in women. London: J. & A. Churchill. 1881.

MOSSO, Angelo.
La fatica. Milano: Treves. 1891.

PORTER, Roy.
Eighteenth and nineteenth century style: from luxury to labour. In: Gijswijt-Hofstra, Marijke; Porter, Roy (Ed.). *Cultures of neurasthenia*: from Beard to the First World War. Amsterdam: Rodopi. p.31-50. 2001.

PROUST, Adrien; BALLET, Gilbert.
L'higiène du neurasténique. Paris: Masson et cie. 1897.

RABINBACH, Anson.
The human motor: energy, fatigue, and the origins of modernity. California: University of California Press. 1990.

ROELKE, Volker.
Electrified nerves, degenerated bodies. In: Gijswijt-Hofstra, Marijke; Porter, Roy (Ed.). *Cultures of neurasthenia*: from Beard to the First World War. Amsterdam: Rodopi. p.177-188. 2001.

ROELKE, Volker.
Biologizing social facts: an early 20th century debate on Kraepelin's concept of culture, neurasthenia, and degeneration. *Culture, Medicine and Psychiatry*, Cleveland, v.21, p.383-403. 1997.

ROSENBERG, Charles.
Contested boundaries: psychiatry, disease and diagnosis. *Perspectives in Biology and Medicine*, Baltimore, v.49, n.3, p.407-24. 2006.

ROSENBERG, Charles.
Body and mind in nineteenth-century medicine: some clinical origins of the neurosis construct. *Bulletin of the History of Medicine*, Baltimore, v.63, n.2, p.185-197. 1989.

ROSENBERG, Charles.
The place of George M. Beard in nineteenth-century psychiatry. *Bulletin of the History of Medicine*, Baltimore, v.36, p.245-59. 1962.

SAVILL, Thomas Dixon.
Clinical lectures on neurasthenia. London: William Wood & Company. 1906.

SCHMIEDEBACH, Hein-Peter.
The public view of neurasthenia in Germany. In: Gijswijt-Hofstra, Marijke; Porter, Roy (Ed.). *Cultures of neurasthenia*: from Beard to the First World War. Amsterdam: Rodopi. p.219-238. 2001.

SHORTER, Edward.
From paralysis to fatigue: a history of psychosomatic illness in the Modern Era. New York: The Free Press. 1992.

SICHERMAN, Barbara.
The uses of a diagnostic: doctors, patients and neurasthenia. *Journal of the History of Medicine*, Oxford, v.32, p.33-54. 1977.

SLIJKHUIS, Jessica.
Neurasthenia as Pandora's box? In: Gijswijt-Hofstra, Marijke; Porter, Roy (Ed.). *Cultures of neurasthenia*: from Beard to the First World War. Amsterdam: Rodopi. p.257-278. 2001.

TAYLOR, Ruth.
Death of neurasthenia and its psychological reincarnation: a study of neurasthenia at the National Hospital for the Relief and Cure of the Paralysed and Epileptic, Queen Square. *British Journal of Psychiatry*, London, v.179, p.550-557. 2001.

VAN DEUSEN, Edwin.
Observations on a form of nervous prostration (neurasthenia) culminating in insanity. *American Journal of Insanity*, Arlington, supp. to the Annual Report of 1867 and 1868, v.25, p.445-461. 1869.

WESSELY, Simon.
The history of chronic fatigue syndrome. In: Straus, Stephen (Ed.). *Chronic fatigue syndrome*. New York: Marcel Dekker. p.3-44. 1994.

WESSELY, Simon.
Old wine in new bottles: neurasthenia and 'ME'. *Psychological Medicine*, Cambridge, v.20, p.35-53. 1990.

WARE, Norma; KLEINMAN, Arthur.
Culture and Somatic Experience: the social course of illness in neurasthenia and chronic fatigue syndrome. *Psychosomatic Medicine*, Inverness, v.54, p.546-560. 1992.

WILL, Barbara.
The nervous origins of the American Western. *American Literature*, Durham, v.70, n.2, p.293-316. 1998.