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Old wine in new bottles: neurasthenia and 'ME'

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SYNOPSIS The history of neurasthenia is discussed in the light of current interest in chronic fatigue, and in particular the illness called myalgic encephalomyelitis ('ME'). A comparison is made of the symptoms, presumed aetiologies and treatment of both illnesses, as well as their social setting. It is shown that neurasthenia remained popular as long as it was viewed as a non-psychiatric, neurological illness caused by environmental factors which affected successful people and for which the cure was rest. The decline in neurasthenia was related to the changes which occurred in each of these views. It is argued that similar factors are associated with the current interest in myalgic encephalomyelitis. It is further argued that neither neurasthenia nor 'ME' can be fully understood within a single medical or psychiatric model. Instead both have arisen in the context of contemporary explanations and attitudes involving mental illness. Future understanding, treatment and prevention of these and related illnesses will depend upon both psychosocial and neurobiological explanations of physical and mental fatigability.

INTRODUCTION

It seems impossible to open a newspaper without finding a reference to myalgic encephalomyelitis ('ME') or postviral fatigue, the 'Malaise of the Eighties' (Seligmann *et al.* 1986). The features of the disease are now well known: profound physical and mental fatigue, especially after physical or mental exercise, together with a variety of other symptoms especially neuromuscular, cardiovascular and gastrointestinal. It is usually stated that the condition results from a virus, and was first recognized at the Royal Free Hospital in 1955, although others have traced it as far back as 1934 (Behan & Behan, 1980; Ramsay, 1986). The illness is now recognized world-wide (Anonymous, 1986; Sinclair, 1988).

The thesis of this essay is that the origins of 'ME' lie not in 1955 or 1934, but in the last century, and in the condition known as neurasthenia (White, 1989*a*). The symptoms, social setting, treatment, and decline of neurasthenia will be considered in relation to 'ME'. This is not a review of 'ME' or 'postviral fatigue' syndrome (David *et al.* 1988; Behan & Behan, 1988; Wessely & Thomas, 1989), nor of fatigue (Berrios, 1989; White, 1989*b*).

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George Beard, an American neurologist, published his essay introducing neurasthenia in 1869 (Beard, 1869). He produced the definitive clinical text in 1880 (Beard, 1880), and the following year elaborated his theories on aetiology in *American Nervousness* (Beard, 1881). Despite Beard's claims, neurasthenia does have a history before 1869 (Sicherman, 1977; Berrios, 1989; Richmond, 1989). Nevertheless, it was Beard who gave the condition its peculiar flavour, and was responsible for its popularity. His books enjoyed extraordinary success, and his ideas rapidly spread to Europe (Rosenberg, 1962).

There are five essential themes to Beard's neurasthenia. First it was a disease in which profound fatigability of body and mind were the principal symptoms. Second, it was entirely organic: 'it is a physical, not a mental state' (Beard, 1881). Third, it was a commoner in the educated and professional classes. Fourth, it resulted largely from environmental factors. Finally, the treatment was rest. I shall discuss each of these themes in relation to the current revival of interest in fatigue states.

SYMPTOMS AND SIGNS

Neuromuscular

Perhaps because he was a neurologist Beard did not suggest that gross neurological abnormalities

or physical signs existed in neurasthenia: 'Nervous exhaustion is compatible with the appearance of perfect health' (Beard, 1880), and 'physical signs are conspicuous by their absence' (Savill, 1906). However, Beard explained such absence by developing the idea of a functional disorder, as opposed to abnormality of gross structure. It was never doubted by Beard or his followers that neurasthenia was an organic disease, and that the absence of macroscopic features simply reflected the limitations of contemporary investigative techniques (Rosenberg, 1962). 'It is caused by changes in the nervous system so minute that our present methods of investigation fail to detect them' (Oppenheim, 1908). Some refinements were necessary: De Fleury (1901) was forced to reply to the increasing numbers of sceptics by posing the rhetorical question 'does neurasthenia have a genuine pathology like aortic insufficiency or Parkinson's disease?'. Not surprisingly he answered in the affirmative, but the pathology was general, rather than specific.

It is similarly assumed that ME is an organic disorder of the peripheral or central nervous system. In the initial reports this was indicated by frank neurological signs. 'The clinical impression was of a disease producing a disorder of the nervous system with a combination of irritative and paralytic signs which were frequently transient' (Ramsay, 1986). Early descriptions emphasized the neurological features of the condition, and such signs as facial nerve palsies, foot drop and paresis were regularly reported. Whatever their merit, which has been disputed, they are no longer found. The concept of ME has shifted from an acute paralytic state to one of chronic fatigability, and current experts emphasize the absence of abnormal neurological signs (Behan *et al.* 1985; Archer, 1987).

Instead, as in neurasthenia, the emphasis is on muscle fatigability. 'This phenomenon of muscle fatigability is the dominant and most persistent feature of the disease and in my opinion a diagnosis should not be made without it' (Ramsay, 1986). It is 'cardinal' (Archer, 1987), 'principal' (Behan *et al.* 1985) or 'pathognomic' (Smith, 1989). All the current authors describe a unique pattern of fatigability, stating that with effort a sufferer may be able to exert himself, but will later suffer severe fatigue continuing several hours, days or even weeks. Kraepelin (1902) wrote that 'upon demand they are able to pull themselves together for a special occasion: but the following day witnesses an exacerbation of the symptoms' while Waterman (1909) described how the 'slightest muscular activity produces a feeling of exhaustion lasting for days'. Beard (1881) felt that neurasthenics could take 'months... to make up the deficiency' that occurred after exertion of any form.

Although the clinical and pathological concepts have changed over twenty years, the conviction of an exclusively physical origin is unchanging: it remains 'purely organically and virologically based' (Smith, 1989).

Mental fatigue

Mental fatigue was an integral feature of neurasthenia. Kraepelin (1902) wrote that 'The accustomed work is carried out with increasing difficulty, requiring greater exertion and more frequent rests. They are easily distracted by little things and are inattentive. Twice the usual time is spent in reading the paper... they are forgetful with names and figures... They assert that the memory is becoming profoundly affected, and that the judgement is failing.'

Mental fatigability is also integral to modern ME (Behan & Behan, 1988), since all agree that 'exhaustion also occurred after emotional and mental strain' (Fegan *et al.* 1983). Other symptoms, such as poor concentration, word-finding difficulties and frequent slips of tongue are included within this description. The recent guidelines issued by the ME Association explain these symptoms in terms of 'functional central nervous system disturbance' (Smith *et al.* 1988), a direct echo of Beard. In an effort to establish organic authenticity others erroneously label the same clinical features as 'encephalitic' (MacIntyre, 1989; Shepherd, 1989*a*, *b*; Yousef, 1989).

Somatic symptoms

'Sufferers from neurasthenia often time wonder and complain that they have so many symptoms; that their pain and distress attack so many parts and organs' (Beard, 1880). Beard listed over 70, with special attention being paid to specific areas: cardiac, gastrointestinal, temperature regulation, paraesthesiae and pain syndromes. Oppenheim (1908) wrote that 'the symptoms of neurasthenia are so numerous that it is hardly

possible to describe them in detail', but then devoted 17 pages to such a description. Kraepelin (1902) emphasized gastrointestinal symptoms: 'Nervous dyspepsia is the most prominent digestive disorder', but felt that any organ may be affected. Beard stressed the particular role of cardiac symptoms and abnormalities. Others agreed, and the term 'neurocirculatory asthenia' became synonymous with neurasthenia (Wheeler *et al.* 1950; Chatel & Peele, 1970). Indeed, the new label proved more durable than the old, and continues to receive serious attention from cardiologists (Mantysaari *et al.* 1988), especially under the heading 'effort syndrome'.

Identical symptom lists will be found in the current literature (Ramsay, 1986; Wilkinson, 1988; MacIntyre, 1989), the concordance with Beard's original account reaching as far as alterations in temperature regulation (Dawes & Downing, 1989) and difficulties in accommodation (Yousef *et al.* 1988), also known as 'hippus' (Oppenheim, 1908).

The ubiquity of these symptoms, and the absence of a satisfactory case definition attracted critical attention both in Beard's time (Bunker, 1930; Rosenberg, 1962) and nowadays (David *et al.* 1988). Others criticized the tendency to divide neurasthenia into 'gastric, or cardiac, or pulmonary or others according to the organ to which symptoms are mainly referable', which was 'not scientific, for it is undoubtedly a general disorder' (Savill, 1906). Similar criticisms have been levelled at the modern tendency to divide fatigue and myalgia syndromes according to medical speciality, leading to such names as fibromyalgia, effort syndrome, pseudomyasthenia and postinfectious fatigue syndromes (Wessely, 1989).

PATHOPHYSIOLOGY

Where is the fatigue?

Pathophysiology remains the appropriate starting point for an explanation of chronic fatigue of whatever cause. 'When we speak of a sense of fatigue we must necessarily be in doubt, not withstanding the fact that the symptoms are referred to the muscle, whether brain, nerve or muscle, one or all of them may be really at fault' (Poore, 1875). Vivian Poore, a London physician, first classified fatigue by pathophysiology. He discussed 'local fatigue', due to disease of muscle or the nerve supplying it, associated with loss of power, contrasted with 'general' fatigue, in which 'There is a disability for performing either mental or physical work... first in work requiring attention or sustained effort... The symptoms of general fatigue are referable to the brain and nervous system.'

This division sidestepped the classification of fatigue by diagnosis, which could include most medical illnesses. However, its empirical value was lost until the work of Edwards and colleagues on muscle physiology (Edwards, 1981, 1986). They separated peripheral fatigue due to disease at or beyond the neuromuscular junction, from central fatigue, manifested by deficits in the organization, integration and motivation of muscle action.

How should the fatigue of neurasthenia or ME be classified? Most of the early physicians avoided the issue. It was due to a disease of nerves, but precisely which was uncertain (as there was no valid way of measurement). However, once modern neurophysiological techniques became available, it became clear that organic disease of muscle and nerve was absent in neurasthenia.

Currently, it is often assumed that the fatigue of ME is due to a muscle disease (Ramsay, 1986; Goldman, 1987; Shepherd, 1989*a*). However, although a disorder of neuromuscular functioning occurs in some acute viral illness (Schiller *et al.* 1977), the evidence for such disorder in chronic fatigue is conflicting (Jamal & Hansen, 1985; Wessely & Thomas, 1989). Dynamic muscle function is normal in the majority, with muscles being neither weak nor fatigable (Lloyd *et al.* 1988; Stokes *et al.* 1988). Since evidence of 'inconsistent effort' occurs only in a small minority (Fullerton & Munsat, 1966), and twitch interpolation techniques have shown that voluntary muscle contraction is usually maximal, the implication is that muscle fatigue may be the result of a disorder of perception and integration of function rather than motivation (Wessely & Thomas, 1989).

Clinical studies also confirm that chronic fatigue is rarely a primary neuromuscular problem. The significance of mental fatigability was re-emphasized by David *et al.* (1988), and it was later confirmed that mental fatigability is characteristic of both postviral fatigue and affective disorder, but usually occurs in neuro-

muscular diseases only in the presence of co-existing psychiatric illness (Wessely & Powell, 1989). Wessely & Powell also showed the lack of specificity of the 'neuromuscular' symptoms, since the frequency of muscle pain both at rest and after exercise did not substantially differ between ME patients and psychiatric controls with major depression.

Measurement of fatigue

The impossibility of measuring objectively a personal experience such as fatigue was first pointed out by MacDougall (1899), who made the distinction between subjective and objective fatigue, and stated that measuring the latter gave little information on the former. Despite these warnings, an Industrial Fatigue Board was formed to produce a definitive fatigue test. It reported in strong terms (Muscio, 1921), demanding 'that the term fatigue be absolutely banished from precise scientific discussion, and consequently that attempts to obtain a fatigue test be abandoned'. In the USA a fatigue laboratory existed at Harvard until the Second World War. However, its director (Forbes, 1943) reported that 'our failure to solve the problem of measurement... has continuously twisted us aside from [our objectives]'.

The problem is the nature of fatigue itself (Berrios, 1989). On the one hand is the symptom described by Henry Miller (1987) as 'The vague sense of being under the weather is what most people, if asked, will admit to most of the time', which, translated into epidemiological terms, is the same as 'the fact that a large proportion of the population has the occasional symptom of dysphoria, fatigue or insomnia probably accounts for the high rates reported by earlier surveys' (Goldberg & Huxley, 1980) confirmed by large-scale modern community surveys in the USA (Chen, 1986) or the UK (Cox *et al.* 1987). On the other hand is a profound disabling fatigue state. Is there a clear cut-off between these two extremes?

A realization that fatigue was not a single entity present or absent led Wells (1908) to argue in favour of 'shifting the viewpoint from the measurement of discrete states of fatigue to continuous determinations of susceptibility'. Subjective vigour and fatigue show a normal distribution in both depressed patients and normal controls (Monk, 1989). Using data from the 1987 US General Social Survey it has been shown (Wesseley & Thomas, 1989) that chronic fatigue is present in the community with a skewed distribution, but no point of rarity. Some degree of fatigue is found in nearly all the population, but only a very small minority report severe fatigue. The number of fatigue symptoms endorsed is more important than any single item, supporting the concept of fatigue as a dimensional rather than a categorical variable. Fatigue tests remain an illusion, and future work must adopt a multifactorial rather a dichotomous approach.

NEURASTHENIA AND SOCIAL CLASS

Beard's neurasthenia was an affliction of the rich and successful. 'Neither in the medical school nor in the hospital has [the doctor] received any suggestions relating to any of these functional diseases of the nervous system, although, should he engage in private practice among the better classes he will meet these diseases every day and every hour.... The miseries of the rich, the comfortable and intelligent have been unstudied and unrelieved' (Beard, 1880), a view shared by Weir Mitchell (Drinka, 1984). There is no doubt that neurasthenia began as a disease of the upper social classes (Sicherman, 1977).

One of the reasons for this positive social class gradient was its alleged aetiology. In Britain, George Savage (1875), later to be knighted and achieve notoriety as the doctor so dismissive of Virginia Woolf (Porter, 1987) (for whom he prescribed the rest cure), reported that fatigue was commonest in professions requiring an unflagging devotion to work, or a high degree of emotional stress (he listed architects, surveyors, policeman, artists, schoolteachers, musicians, inventors and bootmakers among those afflicted). Since then over-representation of higher social classes has been a constant finding (Taylor, 1907; Dowden & Johnson, 1929; Macy & Allen, 1934). Kraepelin (1902) described neurasthenia as 'one of the products of civilisation, confined largely to the professional and clerical callings, and to women of the middle classes'.

All current work on the postviral fatigue syndrome acknowledges a positive socio-economic gradient, with an over-representation of upper social classes (Smith *et al.* 1988), and health service professions. Indeed, this has led to

the unpleasant term 'yuppie flu' (Seligmann *et al.* 1986). However, the only valid epidemiological study of chronic fatigue found the opposite: there is a negative socio-economic gradient to the symptom of chronic fatigue (Cox *et al.* 1987), emphasizing the role of selection bias in published studies.

DOCTORS, NEURASTHENIA AND CHRONIC FATIGUE

Acceptance of the diagnosis by doctors

Beard was constantly accusing doctors of refusing to acknowledge the existence of neurasthenia. 'Physicians, imitating the unscientific example of the laity, have denied the existence of such symptoms, just as they formerly denied the existence of diphtheria and hay fever' (Beard, 1880). Beard often referred to the disbelief that greeted his early work, and the difficulties he faced in publication (Beard, 1881; Rosenberg, 1962). Even when his reputation was secure, Beard continued to campaign about the neglect of neurasthenia in the medical school curriculum and the indifference shown to the subject and sufferers by the medical profession. Mitchell (1904) described the hurtful criticism and derision that greeted his original descriptions of therapy, and it was true that not all reviews were enthusiastic: some contemporaries compared Beard to that other great showman, Phineas T. Barnum (Rosenberg, 1962).

Such views are frequently expressed today. The principal theme of most of the self-help literature is the absence of recognition accorded to patients by relatives, colleagues and the medical profession (MacIntyre, 1989). Sufferers refer to a 'long uphill struggle against ignorance and inertia' (Francis, 1988), and claim that 'most... doctors... are still lamentably ignorant of even the most basic facts of the disease'. Research workers concur, 'I had some difficulty in persuading the DHSS that ME was a very real disease' (Ramsay, 1986).

There is ample anecdotal evidence that patients often feel their doctors do not take them seriously (Jeffreys, 1982; MacIntyre, 1989). Using questions from both the Midtown Manhattan and the Stirling County community surveys Dohrenwend & Crandell (1970) asked doctors and patients to rate the importance of various symptoms, and showed that the two groups viewed different symptoms with differing degrees of concern. 'Feeling weak all over for much of the time' was regarded as 'very serious' by only 6% of psychiatrists and 9% of physicians, and was among the least important of 46 quoted symptoms. This was in contrast to the views of the non-professionals. 'Feeling weak all over' was viewed as one of the most serious out of 46 symptoms in the community sample, and similarly in the out-patient sample. The contrasting importance afforded fatigue by patients and professionals may account for some of the fraught interactions between patients with chronic fatigue and their doctors.

Doctors with the illness

Doctors have always appeared susceptible to chronic fatigue forming 10% of Beard's patients. Doctors remain susceptible to 'ME' (Field, 1989*a*). 'The number of doctors who are victims of the disease is quite out of proportion to their numbers in the population as a whole' (Ramsay, 1986), while 'a curious susceptibility is shown by nursing and medical staff' (Bell *et al.* 1988). It is acknowledged that in most institutional outbreaks there is a remarkable difference in attack rates between professional staff and patients (Medical Staff Report, 1957; Ramsay, 1978). This is a striking feature, since it was a similar (albeit opposite) observation, of an illness that affected patients but never staff, that stimulated Goldberger's classic studies refuting the infectious origin of pellagra (Shepherd, 1978).

Doctors are also over-represented among those who write about the condition. Both Beard and Mitchell had suffered from neurasthenia, and it has been argued that many of the former's theories developed from an effort to explain his own condition (Gildea & Gildea, 1945; Sicherman, 1977; Drinka, 1984). Nowadays, many of the articles in the professional literature (and even more in the popular press) continue to be written by medical sufferers.

Doctors views on patients

Beard often stated that neurasthenics were usually treated without sympathy, especially as they usually looked healthy (Beard, 1880). For a while the medical literature became more sympathetic towards neurasthenics, especially in contrast with hysterics. Physicians then (and

now) displayed sympathy towards their patients based on their perceptions of whether the illness was acquired through praiseworthy or contemptible means. Neurasthenia, the disease of overwork, came into the former category (Gosling & Ray, 1986).

Furthermore, neurasthenics were seen to be more appropriate in their behaviour, perhaps as they initially came from the same class as the physician. Reflecting on his student days, Brill (1930) observed that neurasthenics were eager to cooperate, wanting to be helped, unlike hysterics. Playfair (1892), a Professor of Obstetrics, wrote in Tuke's Dictionary that patients 'give all they possess to be well, and heartily long for good health if they only knew how to obtain it. A condition such as this, in such women, is as far removed as possible from the state that is known as hysterical'. Summing up, Sicherman (1977) wrote 'neurasthenics seemed deeply concerned about their condition and eager to cooperate, hysterics were accused of evasiveness – la belle indifference – and even intentional deception'.

Identical descriptions have been applied to ME patients. 'Many of these girls were known to me. Illness was alien to their nature' (Judge, 1970), and in the context of another epidemic, the 'fact that they were all known to have good pre-morbid personalities made us consider an organic cause for their illness' (Fegan *et al.* 1983). Patients are the 'last types to stay away from work without good reason' (Shepherd, 1989*b*).

Hysteria remains controversial in the modern era. The use of the term by two psychiatrists (McEvedy & Beard, 1970) to describe the original Royal Free epidemic remains a source of anger. Virtually every article on ME includes an often bitter refutation of this theory. Few have acknowledged that whatever the merits of the hysteria theory, it has no relevance to the current situation, since the overwhelming majority of patients did not acquire their very different illnesses in an epidemic (David *et al.* 1988; Wessely & Thomas, 1989). Nevertheless, the absence of the 'classic' features of hysteria is emphasized in most of the current writing, for example, when discussing the reluctance of patients to talk about their illness, it is stated that such 'denial is the opposite of what one would anticipate from a patient with hysteria' (Hyde & Bergmann, 1988).

Similar motives had caused many authors to feel impelled to distinguish neurasthenia from hysteria (Beard, 1880; Playfair, 1892; De Fleury, 1901; Dutil, 1903). However, Beard also included hysteria among the features of neurasthenia. This logical inconsistency was pointed out in the article preceding that by Playfair in the 1892 *Dictionary of Psychological Medicine*. 'It is not surprising that neurasthenia has, according to Bochut and Beard, often been confounded with hysteria and hypochondriasis, and that, in spite of this, Bouchut and Beard do the same, describing distinctly hysterical, hypochondriacal and epileptic conditions as belonging to neurasthenia' (Arndt, 1892). Similar confusions remain today. 'The symptoms of 'hysteria' are present, although not the diagnosis' (Ramsay, 1986), or that 'were it not for the immunological studies... it would have been easy to concur that the illness is entirely a manifestation of mass hysteria' (Behan & Behan, 1980). On the one hand it is not hysteria, but alternatively, hysteria is a sign of ME. Such confusion and conflict are further proof of the failings of the term hysteria.

With the decline in neurasthenia, and its partial replacement by the concept of neurosis, patients were treated less sympathetically by the non-psychiatrist. By World War I both neurasthenia and hysteria were held in low medical esteem (Stone, 1985), although this was tempered by the fact that neurasthenia was the disease of officers, and was sometimes attributed to excessive zeal and responsibility, while hysteria was confined to other ranks (Graves, 1960; Showalter, 1987). This situation failed to improve after the war: 'The average doctor will see they are neurotic, and he will often be disgusted with them. Often he sends them away with as little ceremony as possible' (Alvarez, 1935). Perhaps the term neurosis (or any psychiatric label) was interpreted by the unsophisticated doctor as implying that the patient was responsible for their disease, and thus less liable to sympathy. Contemporary first person accounts continue to give disturbing accounts of condescension, levity and often condemnatory attitudes encountered from some doctors (Jeffreys, 1982; Francis, 1988).

PSYCHIATRY AND ANTIPSYCHIATRY

Depression and neurasthenia

The relationship of chronic fatigue and depression is fundamental to our understanding of the condition. Early descriptions overcame this problem by assuming depression within neurasthenia, but as the concepts of depression and affectivity became more refined (Berrios, 1988) the relative emphasis changed, and by 1893 depression was a 'first order' symptom of neurasthenia (Cowles, 1893), although others were convinced that any affective changes were secondary to the general organic pathology (De Fleury, 1901). The subsequent changes in psychiatric nosology that resulted in the separation of depression and neurasthenia are well known, but depression continued to be important in neurasthenia proper. Kraepelin (1902) recognized that low mood was characteristic, but noted 'It rarely happens that the feeling of despair becomes intense enough to lead to suicidal attempts', while Oppenheim (1908) wrote that 'mental depression is usually present, but is neither deep nor persistent'. Eventually, consideration was given to the possibility that all neurasthenics were depressed. Lane (1906) denied the then current notion that overwork was the cause of neurasthenia, instead blaming the 'prolonged influence of depressing emotions', while Bleuler (1924) wrote: 'What usually produces the so-called neurasthenia are affective disturbances. These can, however, be enhanced by exhaustion.'. Myerson (1922) wrote that 'the feeling of energy is low so that effort is painful, fatigue following rapidly upon exertion and having a peculiar painful component not present in ordinary fatigue', but called this anhedonia rather than neurasthenia. Myerson did compare the two, but was one of the last authors to do so. Finally, in a current leading neurology textbook chronic fatigue, neurasthenia and depression are seen as synonymous (Adams & Victor, 1985). The reasons why depressed patients have such profound physical and mental fatigue are beyond the scope of this paper.

Depression and ME

In the current debate some have denied that any psychological symptoms exist: 'Efforts to find evidence of depressive illness within this group have been unrewarding' (Read, 1988), or 'absent' (Bell & Bell, 1988). If such symptoms are found, they are seen as the natural consequence of physical impairment caused by the postviral syndrome (Ramsay, 1986). Others acknowledge that such symptoms occur in the majority of patients (Behan & Behan, 1988), and are even 'cardinal' (Fegan *et al.* 1983) or 'characteristic' (Bell *et al.* 1988), but neither collect this information systematically, nor use it in a meaningful way. Although it is only rarely suggested that patients actually deny symptoms (Fullerton & Munsat, 1966), more often doctors fail to enquire about them. A recent editorial on lassitude (Harvard, 1985) stated that 'failure to diagnose depression is usually due to failure to seek it rather than to any confusion in diagnostic symptoms'. Little has changed since Alvarez (1935) wrote that 'It is unfortunate that the average doctor who sees these patients doesn't ask some three or more questions.'

Modern systematic evidence mainly supports Bleuler. Of 135 self-referrals to a special fatigue clinic in a University hospital (Manu *et al.* 1988), 67% had psychiatric diagnoses (of which the majority had affective disorder), 3% had medical diagnoses, leaving 25% unexplained. These findings are confirmed by other studies of patients with neuromyasthenia (Taerk *et al.* 1987) or 'chronic Epstein Barr infection', the American version of ME (Katon *et al.* 1988; Kruesi *et al*, 1989). Finally, of 47 medical referrals to the National Hospital for Nervous Diseases with chronic unexplained fatigue, 72% had a psychiatric diagnosis, using research diagnostic criteria modified to exclude fatigue, of which again most had affective disorder (Wessely & Powell, 1989). Comparisons with neuromuscular controls matched for length of illness showed that 'secondary' depression as a result of physical disability was only a partial explanation of the observed psychiatric morbidity. Kraepelin and Oppenheim's view that low mood in neurasthenic patients was phenomenologically different from that found in melancholia was confirmed, since patients with ME who satisfied diagnostic criteria for major depression differed from psychiatric controls on measures of guilt, low self-esteem and attributional style (Powell *et al.* 1989).

Although many patients with either neurasthenia or ME fulfil criteria for affective disorder, one must beware of assuming either

condition is simply depressive illness. Schweder (1988) has forcibly argued against such a reductionist view, as it ignores the many differences between neurasthenia and affective disorder, which may in practice be as important as the psychobiological similarities. Furthermore, it must not be forgotten that all systematic studies confirm that a substantial minority do not fulfil criteria for depression or any psychiatric disorder.

Other psychiatric disorders and ME

Mood disorder is thus found in many cases of ME, but it is not the only psychiatric disorder. Anxiety, both simple and phobic, was included within Victorian neurasthenia. Beard (1880) listed 12 separate phobias, while Kraepelin (1902) observed 'They cannot trust themselves in public'. Nowadays some claim that hyperventilation is the sole pathogenesis of ME (Rosen *et al.* 1988). Few share this view, but some patients do satisfy criteria for anxiety and phobic disorders (Manu *et al.* 1988; Wessely & Powell, 1989), and more have such features in the context of depressive illness. Undifferentiated minor psychiatric morbidity is found in the majority of non-hospital patients (McDonald *et al.* 1989).

In the context of neurasthenia both physicians and psychiatrists included descriptions of patients in whom the principal feature was preoccupation with illness and symptoms. Kraepelin (1902) wrote 'There is a tendency to pay undue attention to trifling symptoms which may appear in any organ' and to 'become chronic invalids of a most distressing type.' Others suggested ways of decreasing such constant symptomatic vigilance that was restricting functioning: 'They should not be educated to worrisome self-observation' (Bleuler, 1924). Oppenheim (1908) wrote that great harm resulted from patients meeting in sanatoriums and 'spreading the mental infection by constant conversation and comparison of their complaints'. Such patients would now be considered as 'somatizers'. Recent studies report that between 10 and 15% of hospital patients with chronic fatigue syndrome fulfil diagnostic criteria for somatization (Manu *et al.* 1988; Wessely & Powell, 1989).

In 1904 Charles Dana proposed that neurasthenia was a heterogeneous condition, and many suffered from psychiatric illness, a view echoed in a recent editorial in the *New England Journal of Medicine* (Swartz, 1988). However, Dana concluded 'I shall be very much disappointed if those who read this paper should flippantly express their interpretation of it by saying "Well, he just wants to make out that all neurasthenics are crazy people and ought to be locked up".' Much of the research outlined in this section has already attracted similar criticism. The next section will consider some of the reasons why.

Patients dislike of psychiatry

Patients with chronic fatigue have long disliked psychiatry. 'Patients frequently resent the suggestion they see a psychiatrist' (Allan, 1945). 'They resent the term nervousness, and feel that such a diagnosis means they are silly, and hysterical, and without good morals or judgement... they may become deeply resentful of you' (Alvarez, 1935).

Antipathy towards psychiatry remains rife. A recent publication by the 'ME Action Campaign', a group formed to increase public awareness of ME, contained a satirical article entitled 'How to Survive a Psychiatric Consultation' (Bartlett, 1989), while the leading article in the previous newsletter stated 'It [psychiatry] is the dustbin of modern medicine' (Francis, 1988). Another sufferer has pointed out that 'if you are convinced that you are physically ill, naturally you resist and resent having your mind probed.... ME is an organic illness. Don't let anyone tell you there's something wrong with your mind' (Jeffreys, 1982). In the USA there is now a scientific consensus that the most appropriate term is the 'chronic fatigue syndrome' (Holmes *et al.* 1988), yet this apparently non-controversial label has attracted severe criticism from patients in the USA (Cuozzo, 1989), as it seen as 'reinforcing its psychiatric nature... trivialising and grossly misrepresented the disease' (Radford, 1988).

What are the reasons for such antagonism? In 1908 Ballet pointed out that patients 'spoke abundantly about their headaches and their muscular weakness, but deliberately concealed their emotionalism... symptoms it would offend their self-esteem to confess'. There is evidence of dislike of psychiatry: patients who fulfil criteria for psychiatric disorder and present with somatic

symptoms are less depressed, but more hostile to mental illness than controls presenting with more typical psychological symptoms (Goldberg & Bridges, 1988), while ME patients have been described as resentful and hostile towards all doctors (Jenkins, 1989). Studying chronic brucellosis Imboden *et al.* (1959) found patients who retained their symptoms after infection to be characterized by a conviction of organic illness, a preoccupation with symptoms and a refusal to discuss emotional issues. Stewart (1989) concludes that these patients view psychological difficulties as weakness, and therefore such diagnoses lead to anger and resentment.

Doctors writing about, and treating, ME and neurasthenia have often believed that a psychiatric diagnosis implies a denial of the reality of distress and illness. De Fleury (1901) vehemently opposed 'the idea, now strongly held, that the illness is basically psychiatric, almost imaginary'. This recurring tendency to juxtapose psychiatric and imaginary was noted by De Fleury's compatriot Dutil (1903), who believed although neurasthenia was a psychological condition, it was not the same as the 'malades imaginaires, or any subjective creation of the spirit' (author's translation). Nevertheless, modern writers on ME note that 'The majority of patients are given the dismissive diagnosis of psychoneurosis' (Fegan *et al.* 1983) or 'As a result of the widespread impression that they are neurotic some have received scant sympathy or understanding from their doctors' (Ramsay, 1986). 'It may not be a psychiatric problem – Royal Free disease really does exist' (Shepherd, 1986) sums up a large number of publications.

Ideas concerning organic *versus* functional lie at the heart of the both neurasthenia and ME. Beard's neurasthenia began as a physical disease, which may have accounted for its success. Sicherman (1977) wrote 'whether accurate or not, the diagnosis proved as satisfactory to the patient as it is easy to the physician.... It provided the most respectable label for distressing, but not life-threatening, complaints, one that conferred many of the benefits – and fewest of the liabilities – associated with illness'. It was preferable to the alternatives – hypochondria, malingering and insanity. There is little evidence of any change in the current era.

AETIOLOGY

Fatigue, overwork and civilization

'The prime cause of modern nervousness is modern civilisation with its accompaniments' (Beard, 1880), or 'the ever growing hurry and restlessness of social life' (Oppenheim, 1908). This broad explanation consisted of two related themes. The first was of external, environmental causes. Beard listed newspapers, steam, science and wireless telegraphy as the principal offenders. Even after the decline of neurasthenia, later variants, such as nervous exhaustion, were ascribed similar aetiologies, including 'bootleg liquor, carbon monoxide from cars, the stress of World War I and the residium of the influenza pandemic' (Dowden & Johnson, 1929).

The second theme linked neurasthenia to the pace of the modern life. This was the theme of *American Nervousness* (Beard, 1881), but had been suggested by several physicians writing in the *Lancet* during 1875, none of who appeared aware of Beard. George Johnson, the Professor of Medicine at King's College Hospital, lectured on 'Some nervous diseases that result from overwork and anxiety', as did Savage (1875) and Poore (1875). This explanation had more appeal to psychiatrists. Kraepelin (1902) ascribed neurasthenia to 'rapid, irregular and extravagant manner of living... in individuals actively engaged in business'. Neurasthenia resulted from 'overload' (Beard, 1881) when 'demand exceeded supply' (Sicherman, 1977): the exhaustion of any one bodily system could by reflex irritation spread to all other organs. In a sophisticated analysis Rabinbach (1982) has proposed a slightly different view. Fatigue and exhaustion were less the result of overwork, but more of the work-ethic itself, the drive to succeed. He shows that fatigue was seen in professions demanding an 'unflagging devotion to the task or a high degree of emotional pressure'. Showalter (1987) extends this to show how the links between illness and ambition, drive and financial acquisitiveness made it possible for men to develop neurasthenia. One of Oppenheim's suggested prophylactic measures against neurasthenia was 'a return to the simple life... a war against the desire of money'.

Many have ascribed similar aetiologies to ME. It has been succinctly described as a 'disease of the twentieth century' (MacIntyre,

1989), or 'an overload disease unique to this century' (Steincamp, 1989). Just as Beard was convinced that neurasthenia could not occur in less advanced societies, ME is restricted to 'developed nations' (MacIntyre, 1989), With the exception of the education of women, each of Beard's suggested aetiologies can be found in recent articles on ME. Obviously some modern causes were unknown to Beard, for example 'agent orange' (Hall & MacPhee, 1985) or pesticides (Dawes & Downing, 1989), but the idea of illness created by an external 'toxin' was entirely familiar. Even current theories implicating the immune system are consistent with Beard's views, since the alleged dysfunction is blamed on the excesses of modern living and the 'deteriorating quality of the world we live in' (Dawes & Downing, 1989), via notions of overstressing of the immune system. One medical sufferer has blamed the increased incidence on 'increased virulence of the virus, widespread use of antibiotics... or altered environmental factors such as pollution with tobacco, petrol or other allergens making victims more susceptible' (Wookey, 1987), echoed by many others (Field, 1989*b*: MacIntyre, 1989: Steincamp, 1989). Two contemporary newspapers published articles with the identical title ('The ME Generation') on the same day (Byran & Melville, 1989), one of which began 'What is modern life doing to us?' (Askwith, 1989), and was able to link ME with the recent deaths of North Sea seals.

Related to over-exertion is the theory that such illnesses can be caused by excessive exercise. Poore (1875) wrote 'athletes who overtrain run the risk of cardiac troubles... if such offences against the laws of nature be persisted in, general paralysis or other form of "breakdown" is likely to result'. Nowadays, the professional and lay literature regularly discuss the frequency with which athletes seem to be affected (Askwith, 1989; Nichols, 1989; Wessely *et al.* 1989).

One particular aetiological theory also undergoing a revival in ME is autointoxication: the idea that decayed and poisonous products in the colon leak into the system and cause disease, appropriately treated by some form of colonic washout. Although largely discredited by the beginning of the century, it was often revived. In 1919 the surgeon Sir Arbuthnot Lane quoted autointoxication as a cause of neurasthenia, but also schizophrenia, diabetes, angina and alcoholism (Lane, 1919). At the Mayo Clinic in the 1930s patients continued to ask for colonic therapy. The local gastroenterologist wrote 'Sometimes I have kept the colon perfectly clean with enemas for a week or two, only to show the patient he was not particularly improved' (Alvarez, 1935). Autointoxication has now been revived by the most active of the current campaign groups, and Arbuthnot Lane's paper has been reprinted and circulated, and even quoted in the *Guardian* (Dawson, 1989). Colonic lavage is still available privately in the UK (Wilkinson, 1988; MacIntyre, 1989).

The unifying theme underlying this section is that neurasthenia and its modern variants are the product of success. 'These exhaustion states were called to our attention because they occurred in great part in successful people... in contrast we presented the series of neuroses which we believe occur in constitutionally inferior people, to show we are dealing with people who have been apparently healthy but have been running their engines at high speed against brakes' (Dowden & Johnson, 1929). Barsky (1988) has included the modern revival of interest in chronic fatigue within an analysis of the relationship between improvements in absolute health but decline in the perception of well being. An editorial in the *British Medical Journal* in 1909 entitled 'Neurasthenia and Modern Life' suggested that rather than identifying a major health problem, neurasthenia was the problem, noting 'it is neurasthenia which breeds the almost universal anxiety about health which is one of the signs of the times. This leads to a corresponding prevalence of quackery of every kind' (Editorial, 1909).

Fatigue and infections

The original descriptions of neurasthenia did not suggest an infectious origin. However, with the revolution in bacteriology that occurred between 1870 and 1900 such ideas changed. Neurasthenics were thought to have increased susceptibility to infections (Arndt, 1892) and sporadic cases were linked to infectious outbreaks: 'At a time when dysentery, cholera or typhoid fever is prevalent, or when influenza prevails all over the globe... the slight affections mentioned are undoubtedly connected with the epidemics, but are the simulation only of the more severe forms' (Arndt, 1892). Kraepelin

(1902), Dutil (1903) and Oppenheim (1908) felt that neurasthenia could follow certain infections, in particular influenza. The latter was also singled out by Savill (1906), although he viewed dental sepsis as more significant. Four years later a meeting on neurasthenia concluded that neurasthenia not only occurred after infection as well as after 'mental stress', but that the two factors showed a significant interaction (Lane, 1906).

At the same time physiologists became preoccupied with the concept of a fatigue toxin. Mosso (1904) believed that a toxic product from fatigued muscle was the source of fatigue, and that blood from a fatigued individual could transmit fatigue to experimental animals. The efforts to link physiology with infection culminated in Weichardt's 1906 'discovery' of a fatigue antitoxin (Burnham, 1908). Rabinbach (1982) describes the excitement that this caused, and many scientists began work on a fatigue vaccine. The enthusiasm surrounding the vaccine did not abate until 1914, when scientists finally concluded that the previous tests had been faulty.

The links with ME are obvious, since it is now regularly stated that ME is 'caused by' (Wilkinson, 1988) or 'the result of' (Shepherd, 1989*a*) a virus. The description preferred in the scientific literature, 'postviral' fatigue, confirms current views on aetiology. Fatigue toxin has not disappeared either: in one outbreak an acidic toxic metabolite was found in the urine of patients (Hill *et al.* 1959). 'Fatigue vaccine' based on neutralizing antigens is available via a private allergy hospital (Byran & Melville, 1989). More subtle links are also evident in the relationship between epidemic Royal Free Disease and poliomyelitis. One view is that the illness protected against poliomyelitis (Ramsay, 1986), echoing Arndt, others believed that the fear of poliomyelitis in the prevaccination era facilitated the spread of a psychological illness (McEvedy & Beard, 1970).

Theories of infection provide a link between ME and 'disorders of fashion' (Stewart, 1989). The symptoms of ME overlap with those of chronic brucellosis, candidiasis and many allergy syndromes (Stewart, 1987; Straus, 1988). Such illnesses are not exclusive: ME is frequently alleged to be caused by candida and associated with severe allergy (Dawes & Downing, 1989; Jenkins, 1989; MacIntyre, 1989), although orthodox practitioners are more sceptical (Shepherd, 1989*a*, *b*; Smith, 1989). Stewart (1989) has shown that of 50 patients who previously felt they had 'environmental hypersensitivity', 32 went on to develop post-infectious neuromyasthenia. Most acquired their diagnoses after reading newspaper articles, and had multiple self-diagnoses concurrently. Suggestible patients with a tendency to somatize will continue be found among sufferers from diseases with ill defined symptomatology and external (usually infective) causation until doctors learn to deal with them more effectively.

One of the reasons for the rapid rise of ME (the ME Association is Britain's fastest growing charity, attracting 150 new members per week (Smith, 1989)) lies in the nature of the principal alleged aetiological agent. Viruses are among the commonest explanations given for non-specific transient illnesses that abound in the community (Pill & Scott, 1981). Such attribution has many features relevant to ME. The agent is external, and is beyond the subject's control. There is no 'maleficium' (Helman, 1978), nor guilt or self-blame. External attributions of illness to entirely organic causes distinguish ME patients from matched psychiatric controls (Wessely & Powell, 1989).

REST AND EXERCISE

The beginning

At its inception, the cure for neurasthenia was rest, formalized in the 'Rest Cure' of Beard's disciple and fellow neurologist Weir Mitchell. The rest was absolute, the patient was not even permitted to leave the bed for several weeks (Mitchell, 1889; Playfair, 1892). It was extremely popular and private sanatoria offering the cure rapidly appeared in the USA, although not in England. Mitchell's main book was an immediate success, selling out in 10 days and going through five editions between 1871 and 1889 (Sicherman, 1977).

However, this extreme position was soon modified. In the same volume as Playfair's 'hard line' approach Arndt (1892) wrote that 'for some patients exercise is needed, for others rest is beneficial'. By the following year views had further softened: 'Exercise may be pressed up to the limits of not causing effects' and 'they must

also be taught that some degree of fatigue thus manifested must be regularly incurred as wholesome tire' (Cowles, 1893). Absolute rest was indicated for a short period, but was soon followed by restoration of activity (De Fleury, 1901; Taylor, 1907).

Disillusionment with the rest cure spread. It was said that Mitchell achieved good results, but Waterman (1909) suggested this was less to do with his therapy and more with his charisma, which was considerable (Gildea & Gildea, 1945; Drinka, 1984). Instead rest was seen not only as irrelevant in anything but the short-term, but as counterproductive. 'Frequently these patients have indulged in rest for months, or even years, without beneficial results. This has been so much recognised by many of the sanatoria during recent years that facilities for exercise and occupation have to a large extent replaced those of rest and seclusion with gratifying results' (Waterman, 1909). Taylor (1907) was more abrupt 'Disuse is a hurtful as misuse... The most potent cure of ailments is correct, consistent use, exercise. From this truth there is no escape.' Others wrote that 'Daily, systematic, but not forced tramps in the mountains continued for weeks do more good [than other treatments]' (Hirt, 1899). For Dutil (1903) the inevitable consequence of Mitchell's regime was 'the patient, condemned to complete inactivity, lying on her chaise-longue for days and nights... constantly alert to the most minute sensations, surrounded by those excessively concerned for her health'. Eventually 'nothing interests them but the illness and its treatment'. An exception was the French physician Adrien Proust, who wrote a book on neurasthenia (Proust, 1902) in which he did not counsel activity, and followed more closely the standard approach. However, this brought no relief to one of his patients, his son, Marcel (Drinka, 1984).

Exercise, not rest

If rest was no longer advised, but exercise, how was this achieved? The answer was with difficulty. Waterman (1909) wrote 'It is all too easy for the memory of a previous breakdown and fear of its reoccurrence to render one loth to resume his former life again'. He described those who repeatedly attempted to 'take up the duties of life, but never seeming to get sufficient reserve to carry on the struggle'. They represent the 'psychological element of fatigue in its fullest development. The conviction that exhaustion will follow any amount of effort, physical or mental, is already a guarantee that it will result.' Fatigue presented a barrier to recovery which seemed unsurmountable. 'Any effort on the part of the patient to struggle against this symptom so increases the fatigue as to accentuate other symptoms, and cause great discomfort.' Clark (1886) also noted the involvement of the family: his cure was 'diet and discipline, work and will', but noted that 'it is difficult to follow, and provokes the antagonism of the patient's friends, who are usually constrained by some fatal necessity to destroy those whom they love'.

The first part of Waterman's programme was education: a 'careful and truthful statement of the existing condition', together with an explanation of how symptoms had arisen in the past. The intention was to ensure that 'The knowledge that experience has shown that certain sensations have resulted from certain activities must be replaced by a conviction that these efforts may be made without harm' (Waterman, 1909).

Finally, a behavioural approach ensured a gradual increase in activity. Fernand Lagrange's name was associated with a graded, careful but steady increase in activity (Lagrange, 1901): 'physical exercise, gentle and not excessive, but progressive in nature, provides the best method of treatment' (author's translation). Bryant prescribed a special form of slow exercise for the 'reeducation of the control mechanism', since he recognized that over exertion in the early stages would lead to further pain. Twenty-years earlier Hough (1902) showed that in the untrained muscle fibres are damaged by exercise, causing late soreness, but this 'tearing' does not occur in the trained muscle. This observation was lost, but recent neurophysiological studies, have shown that when untrained individuals over-exert themselves harm may result from eccentric muscle contractions (when the active muscle lengthens while doing work), which are potentially damaging and produced delay muscle pain (Editorial, 1987; Newham, 1988).

Physicians had no doubt that these methods were successful both physically and psychologically: 'A secondary result, of which patients almost always speak, is the evaporation of mild phobias' (Bryant, 1920). At least part of the

approach eventually adopted would now be called 'cognitive behavioural', and has indeed been suggested for treatment of modern chronic 'postviral fatigue' (Wessely *et al.* 1989).

Nowadays, the debate about the merits of rest *versus* activity is again raging. On the one hand those who write about postviral fatigue usually conclude with a statement along the lines of 'physical and mental exertion is to be avoided' (Anon, 1988). In particular, it is stated that exercise may induce relapse, since 'any exercise will make them worse' (Dawes & Downing, 1989). Many neurologists and physiologists take the contrary view (Edwards, 1986; Editorial, 1987). American experts on chronic fatigue syndrome have concluded that 'there is no evidence that forced rest or inactivity ameliorates the illness or that physical activity worsens the underlying process' (Schooley, 1988), as long as return to activity proceeds gradually.

THE END OF NEURASTHENIA

The decline of neurasthenia

From one of the most frequently diagnosed conditions in medical practice, neurasthenia disappeared almost as rapidly as it appeared. There are several reasons for this.

First, medical scepticism concerning neurasthenia continued to increase. In 1906 Savill reaffirmed the seriousness of the illness, but wrote that 'it will be noted that this statement is somewhat at variance with the generally held opinion'. Similar sentiments had been exposed by De Fleury (1901). Nevertheless, De Fleury's compatriot Dutil expressed the prevailing view when he wrote 'Beard's illness must be recognized as being of mental origin' (Dutil, 1903), although, as already stated, he continued to see it as a genuine illness. It was clear that neurasthenia was shifting from being the concern of neurology to psychiatry. This change was of critical importance, since once neurasthenia was viewed as psychiatric, a principal social function was lost.

Second, the increasing sophistication of psychiatric nosology rendered the diagnosis untenable. In the USA Dana (1904) began the protest against the broad definitions and lack of precision. He referred to the work of Janet, who had detached obsessional compulsive disorders from neurasthenia (Berrios, 1985), and Freud (1985), who detached both anxiety neurosis and hysteria.

Very little was left. Eventually Freud restricted neurasthenia to the following typical symptoms: headaches, spinal irritation, dyspepsia with flatulence and constipation. Freud himself once believed he suffered from neurasthenia (Freud, 1961), and during his stay in Paris had bought a dynamometer to study 'the variations in his muscular strength as a barometer of his nervous condition' (Carlson, 1970). Nevertheless, Freud wrote that only two specific aetiological factors existed: excessive masturbation and spontaneous emissions (Freud, 1895). Ernest Jones (1961) estimated that fewer than 1% of those labelled as neurasthenic were correctly diagnosed. 'The neurasthenic model, valuable because it gave credence to non-verifiable symptoms and to emotional distress that was not outright insanity, was dismantled when it proved too large and cumbersome to be subdivided into more specific categories' (Gosling & Roy, 1986).

By 1906 Blumer was able to talk about 'so called neurasthenics lulled into a comfortable sense of security by a name that was a misfit' (Blumer, 1906). Neurasthenia became the 'garbage can' of medicine (Brill, 1930). The process was summed up by the English neurologist Farquahar Buzzard (1930): 'Taken as a whole, we doctors carry on our trade in an honourable fashion and are imbued with a genuine desire to cheat neither our clients nor ourselves: but it can hardly be denied that the label of neurasthenia is often in order to evade a duty – the duty imposed on us to declare a correct diagnosis. Whether such action should be regarded as a crime or only a pardonable misdemeanour is a moral question I do not propose to discuss; the object of my discourse is to show that it is not a good business. In the first place it conveys little or no pathological meaning to your mind; in the second place it gives your patient the impression that he is suffering from a disease the cause, course and outcome of which is equally mysterious to both of you'.

The third reason for the decline in neurasthenia was a change in its social demography. It has been emphasized that neurasthenia, like ME, was a disease of the upper professional classes. However, this began to alter. Charcot (1889) began one of his Tuesday lectures by saying that 'whenever one speaks of neur-

asthenia, it seems one is almost exclusively talking about a man from the privileged classes', but continued 'it is not the exclusive right of the good and the great, but has extended its empire to the labouring classes' (author's translation). By 1906 two works were published dealing specifically with neurasthenia in lower social classes (Glorieaux, 1906; Savill, 1906). The latter was particularly interesting, since Savill made a virtue of being the first to study neurasthenia in a 'Poor Law Infirmary', and stated that the illness was actually more common in that setting: neurasthenia was now 'a disease of clerks'. The preferred aetiologies showed a similar change, Savill blaming, among others, 'the fashion of eating ice-creams, which first became prevalent among the children of the lower orders about the middle of the last century'. Going back to contemporary case-records Gosling (1987) has shown that by 1900 it was no longer the sole province of the prosperous, but was frequently diagnosed in lower classes and state hospitals.

In conclusion, several related factors contributed to the eclipse of neurasthenia. It had become an illness of lower social classes. It was no longer the concern of neurologists, but psychiatrists, although it is as yet unclear how much this was reflected in clinical practice and not just professional literature. It was coming into disrepute as a diagnosis in both specialties. Nowadays the current leading neurological textbook states that 'The great majority of patients who enter a hospital because of unexplained chronic fatigue and lassitude are found to have some type of psychiatric illness. Formerly this state was "neurasthenia", but since fatigue rarely exists as an isolated problem the current practise is to label such cases according to the total clinical picture' (Adams & Victor, 1985). By 1970 American psychiatrists had little knowledge or interest in the subject (Chatel & Peele, 1970).

Neurasthenia in England

Neurasthenia dominated the American medical scene for the last 20 years of the nineteenth century, and achieved substantial success in France and Germany (Drinka, 1984), where most of the experiments on the biochemistry and physiology of fatigue took place. Textbooks flourished, including titles by Krafft-Ebbing, Binswanger, Ballet, Moebius, De la Tourette and a German multi-author handbook. However, it was less successful in England, although the correspondence columns of the *British Medical Journal* and *Lancet* indicate that neurasthenia was certainly being discussed and diagnosed, while some physicians, in particular the influential society doctor William Playfair (Drinka, 1984), popularized the rest cure. Nevertheless, neurologists, then undergoing a rapid period of scientific progress and discovery, remained sceptical. Gowers (1888) devoted only a page of his major textbook to the topic: 'The use of the word [neurasthenia] has brought with it a tendency to regard the condition thus denoted as a definite disease. Books have even been written about it.' In the next edition he was even briefer 'Such symptoms occur especially in those of a neurotic disposition' (Gowers, 1899). This was in contrast to the 39 pages devoted to the topic in Oppenheim's equally monumental German neurology text. Before even Buzzard's farewell to neurasthenia, however, the savagest attack had occurred in the pages of the *Lancet* (Clark, 1886). Clark, a medical baronet, described it as 'vicious, inaccurate and therapeutically misleading'. It was a 'an assemblage of incoherent indications of disorder borrowed more or less freely from inchoate forms of insanity, and from almost every disease of the nervous system'. Neurasthenia did not 'merely hinder' scientific progress, it actually 'throws back the progress of true knowledge'. Neurasthenia was no less than 'mere and sheer nervousness'. It is not surprising that neurasthenia was less successful in England than the continent.

Modern neurasthenia

There is one exception to this process. Neurasthenia flourishes in some non-Western cultures, especially China. It is viewed as a mainly organic condition, but most of those affected fulfil research criteria for major depression (Kleinman, 1982), although the proportion appears lower in India (Jindal *et al.* 1978). This is the reason for the retention of neurasthenia in the current International Classification of Diseases (ICD-10), even if not used internationally (White, 1989*a*).

What will happen to ME? Again the American example is instructive. The current renaissance of chronic fatigue began in the USA, with a

series of reports from reputable investigators of a link between chronic Epstein–Barr infection and chronic fatigue, resulting in intense media and patient interest. However, after more careful research the same investigators who had raised the possibility were the first to suggest that the link accounts for only a small minority of those with chronic fatigue (Straus, 1988; Schooley, 1988; Swartz, 1988). Little evidence could be found of any relationship between clinical symptoms and laboratory findings, or between clinical recovery and the resolution of any serological or immunological abnormalities, even in patients specifically selected for serological abnormalities (Straus, 1988; Schooley, 1988; Kruesi *et al.* 1989). Instead, Lane's view (1906) of an interaction between susceptibility to psychiatric illness and infection is among the most promising of current lines of enquiry (Imboden *et al.* 1961; Straus, 1988; Katz & Andiman, 1988; Swartz, 1988; Wessely, 1989; White, 1989*c*).

It remains to be seen whether the same process happens in the UK. It is already clear that objections exist to theories that viruses convey a substantial attributable risk to fatigue states (Wessely & Thomas, 1989). Pasteur is reported to have said '*La germe n'est rien, c'est la terre qui est toute*': future research is likely to shift from the virus to the role of the host, including such risk factors as genetics, immune function and psychological vulnerability, and postmorbid variables as coping strategies, attributions and appropriate treatment.

CONCLUSIONS

This essay has traced the history of ME, and drawn parallels with the rise and fall of neurasthenia, by means of clinical, therapeutic and social comparisons. Evidence is presented of the striking resonances between neurasthenia and ME. A simple explanation is that clinicians in both the modern and Victorian periods are describing a similar neurobiological syndrome, of excessive fatigability: supported by the similarity of the clinical case histories. Current medical research into the relationship of viruses to fatigue states (Yousef *et al.* 1988), which is of undeniable importance, may therefore be seen as an renewed effort to solve a clinical problem common to both contemporary and nineteenth century medicine. Such work attempts to answer the question posed by Wechsler (1930): 'The suspicion is justified that "true" neurasthenia is an organic disease in the sense that as yet undemonstrable pathologic changes are the cause of the symptom and not the result of psychogenic processes. How much truth there is in such a view only further studies will determine.' However, further studies have failed to fully answer the question, and will continue to fail as neither neurasthenia nor ME fits into such a simple medical model.

The failures of a medical model are not corrected by studies that ascribe psychiatric diagnoses to many (but not all) of patients with ME syndromes (Taerk *et al.* 1987; Katon *et al.* 1988; Wessely & Powell, 1989; Kruesi *et al.* 1989), although such studies are preferable to those that ignore such information. Establishing psychiatric diagnoses when appropriate adds to the clinical information and permits better defined research and treatment, but also is an inadequate explanation of the social and symbolic phenomenon of neurasthenia, ME, and their successors.

Instead, both neurasthenia and ME can only be understood within their social context. A physiological explanation of fatigability will not explain the emergence of the diagnoses, nor, in the case of neurasthenia, its disappearance. Some of the themes common to both have been outlined, others require more detailed historical research. What happened between the end of neurasthenia and the arrival of ME? Physicians wrote occasional papers on 'Chronic nervous exhaustion' (Macy & Allen, 1934) or neurocirculatory asthenia (Wheeler *et al.* 1950), but more often patients were simply described by their symptoms, such as 'exhaustion' (Dowden & Johnson, 1929), 'tired, weak and toxic' (Alvarez, 1935) or simply 'weak and fatigued' (Allan, 1944). No attempt was made to describe specific syndromes, and physicians generally resorted to psychological or psychosomatic explanations. It seems unlikely that the needs of those patients previously viewed as neurasthenic and later as ME would have been met by such descriptions. The fate of these patients is thus unclear, and it is plausible that the illnesses represented by neurasthenia and ME were actually less prevalent during this period. The social processes that govern the creation of such

illnesses remain obscure (Eisenberg, 1988), but one may argue that they represent culturally sanctioned expressions of distress, in effect culture bound syndromes. It is thus possible that in the changing climate of this period such illnesses were actually less common, as opposed to simply grouped under another label. It is also difficult to ignore the parallels between attitudes to work and material success between the late Victorian age and now, the periods of neurasthenia and ME, and to suggest that the intervening years were characterized by different attitudes to both work and the expression of emotional distress.

This paper has a further purpose. Despite the advent of social psychiatry and community care, psychiatric illness remains as stigmatizing as ever. Perhaps the most consistent theme of this essay is the dislike and distrust shown by patients and doctors towards psychiatry. This may be direct, but is more often indirect, indicated by the many statements affirming the 'genuine' or 'real' nature of ME, contrasted with the unreal, fake and malingered illnesses with which ME might be confused, and which are the province of psychiatry.

It has been shown that some patients have always preferred to receive, and well-meaning doctors to give, a physical rather than a psychological explanation for ill-defined illnesses associated with fatigue. This is not surprising, and in the absence of definitive explanations for fatigability, is not necessarily mistaken. However, an insistence on solely 'organic' explanations may have unforseen and regrettable consequences (Archer, 1987; Wessely & Thomas, 1989). Patients may be denied the most appropriate treatment available, and may instead receive 'new' diagnoses which are later found to be spurious, as exemplified by both neurasthenia and chronic brucellosis (Eisenberg, 1988). Such uncritical diagnoses may reinforce maladaptive behaviour, and may create more severe and persistent morbidity than the initial illness which lead to the consultation (Eisenberg, 1988; Wessely, 1989).

I shall conclude with a description of the Section on Practice of Medicine held at the 94th Annual Session of the American Medical Association in Chicago on 14 June 1944 (Allan, 1945). Allan began by presenting a series of 300 fatigued patients (Allan, 1944) of whom the majority had minor psychiatric conditions, and emphasized the poor yield of further detailed investigations. Alvarez from the Mayo Clinic spoke next, agreeing with the paucity of clinical signs. 'Many have a fever, but this is 99·6 in the afternoons... many have a basal metabolic rate of minus 15%, but that again is normal... many are supposed to have some endocrine disturbance, but they show no sign of it that I can recognise.' Dr Sieve from Boston then presented the same data, but reached the opposite conclusion. 'Detailed study reveals minute changes which constitute a definite physiological deficiency. The basal metabolic rate may range from minus 8% to plus 15%... there may be just a slight change in the red cell count... a white blood cell count 4 to 5000, etc... I believe that many of Dr Allan's so-called neurotic group, if studied in detail from the point of view of physiologic deficiency, could be helped by substitution therapy.'

Dr Freeman spoke next. Whereas Alvarez had spoken in psychological, and Sieve in organic terms, he linked the two. The answer was hyperventilation: 'If you want to demonstrate to the patient how these symptoms develop just have him stand by your desk, open his mouth and breath for... three minutes and he will be ready for collapse'.

As usual in these meetings, the chairman tried to end with a consensus: 'The cooperation of physicians in all fields of medicine is essential to secure the best solution to this problem.'

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