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Annotation

Fatigue and Fatigability

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> "One spring, towards the end of March, I happened to be in Rome, and, hearing that the migration of the quails had begun, I went down to Palo on the sea coast in order to ascertain whether these birds, after their journey from Africa, showed any of the phenomena of fatigue."

So begins Mosso's (1904) monograph on fatigue. Influenced by the German school of sensory physiologists, Mosso went beyond the measurement of muscle fatigue to take account of individual factors and to link these with intellectual fatigue. He describes neurasthenic subjects as "people [who] quickly exhaust the energy of their nervous centres, and who are slow in repairing the loss of that energy" (p. 124). He quotes cases, some of the afflicted being his colleagues and students, in which the major complaint is of poor sleep, and headache following any intellectual effort. Many of these cases are attributed to infections of the upper respiratory tract.

Kraepelin (1902) described acquired neurasthenia. Emotional irritability is often the first symptom to appear. The capacity for mental work diminishes rapidly, so that work is slower, requiring greater exertion, and more frequent rests. Patients are easily distracted, take several attempts to understand a newspaper article or add a column of figures, cannot recall the names of well-known acquaintances, and lose the trend of thought in conversation. The ability to originate and create disappears, and the patient is confined to the purely routine in thought and action. Physical symptoms include headache, insomnia, and rhythmic twitchings and tremor. There is a tendency to exaggerate the real symptoms, so that hypochondriasis is a characteristic symptom which Kraepelin describes as "a tendency to pay undue attention to trifling symptoms which may appear in any organ".

It is perhaps this last feature which has led to the eclipse of neurasthenia by hypochondriasis. The reviews by Wood (1941) and by Jones & Lewis (1941), of functional disorders in solders, concentrated on the somatic complaints of hypochondriasis, while the psychological symptoms, described above by Kraepelin, were largely forgotten by psychiatrists. The constellation of symptoms of fatigue described at the turn of the century has not gone away but has undergone sea changes in the hands of neurologists, immunologists, and epidemiologists (Behan & Behan, 1980; Behan, 1985; Ramsey, 1986), among whom post-viral fatigue, or myalgic encephalomyelitis, enjoys considerable interest.

Definition

Fatigue is the decline in performance that occurs in any prolonged or repeated task. Fatigue of muscle contraction is familiar to all and well described in the physiological literature. Fatigue as a decline in attention or concentration is also familiar, and can be measured by the selectivity and capacity of attention, the rate of change of the contents of attention, and susceptibility to distraction.

Epidemiology

A Danish population study (Norrelund & Holnagel, 1979) showed that in a stratified sample of 1050 40-year-olds, 41% of the women and 25% of the men felt 'tired at present'. In the USA, the National Ambulatory Medical Care Survey (1978) found that, in primary care, fatigue was the seventh most common presenting symptom, and that 25% of those symptoms were new concerns. It is probable that 40–50% of these have psychological causes (Morrison, 1980). Few of these would have what psychiatrists recognise as 'major depression' and fewer still 'endogenous' depression with biological features.

Physiology

Muscle performance and fatigue has been described in mechanical terms by physiologists. For physical work, it can be shown that fatigue to breaking point occurs long before the nerve–muscle unit is incapable of further performance. This may be seen as establishing the primacy of the central nervous system in mediating fatigue. It can also be shown that central integration of physical effort, which favours efficiency of effort, occurs (Marsden *et al*, 1983). This may overlap with learnt strategies, as in athletic training.

Psychology

Fatigue can be measured in normal people as paradigms for physical exertion, mental work, and vigilance tasks, all of which can have detrimental effects on the performance of perception tasks (Jain, 1983); this has relevance to safety studies (Brown, 1982; Dodge, 1982).

Personality factors may partially explain why a patient complains of mild symptoms. It is in general true that those who present to doctors with complaints that in population samples are common but not distressing are those who score highest on the N-scale of the Eysenck Personality Questionnaire (Costa & McCrea, 1985). This is especially true in depression (Mathew *et al*, 1981). This measure, better thought of as 'emotionality' rather than neuroticism, is not necessarily a fixed trait and may be a state-dependent attribute that rises in depression (Coppen & Metcalf, 1965; Katz & McGuffin, 1987). It can also be shown that those with the type 'A' personality report less subjective awareness of effort than others, even when exercised to their limit (Carver *et al*, 1976).

Early experimental work on fatigue was prompted by concerns about 'eye strain' following the use of microfiche. Bartley & Chute (1947) devised a theory of visual fatigue which distinguished between physical changes at the tissue level and a mental state which was in no way related to the physical state. They regarded fatigue purely as a mental state and concluded that it was impossible to measure because of the complex interaction of subjective feelings, tissue impairment, and performance decrement.

The introduction of information theory to psychology led to the equating of fatigue with declining information-processing capacity (Brown, 1982). It was assumed that man behaved as a single information channel with finite capacity. Experiments usually gave the subject a primary task that remained constant, and a secondary task that made variable demands so as to measure reserve capacity. This approach failed owing to the subject's ability to vary working strategy, thereby freeing reserve capacity, and because the secondary task provided a source of stimulation which itself seemed to offset the effects of fatigue and lassitude (Ogden *et al*, 1979).

The next phase of research was directed towards 'vigilance', in the theoretical framework of signal detection theory (reviewed by Eysenck, 1982). This holds that increasing 'arousal' leads to improved performance up to an optimum level, beyond which further rises in arousal are counterproductive, the 'inverted U'. There is also evidence that the more difficult the task, the lower the optimum level of arousal. This paradigm might have yielded valuable information on fatigue; could all performance decrements be related to physiological measures of arousal? In fatigue and lassitude, does the arousal mechanism fail, or does the optimum level of arousal for a given task vary with states such as sleep deprivation or major depression? Unfortunately, the lack of agreement between different measures of arousal has weakened the concept. It was found, however, that repetitive tasks led to lowered arousal and diffusion of attention with increased errors in performance. Conversely, increases in emotional arousal led to greater selectivity of attention (Easterbrook, 1959; Hockey, 1970). It might follow that the emotional reaction to prolonged working, which often includes irritability, complaints of somatic symptoms, and a tendency to take risks, represents a homeostatic mechanism intended to maintain arousal, but at the cost of increasingly restricted cue utilisation.

Pathophysiology

More recent research has been directed at pharmacological agents and their ability to produce subjective tiredness and objective impairment of driving skills. The British National Formulary attributes 'fatigue' as a side-effect to virtually every category of centrally active drug, including antihypertensives, sedative hypnotics, antihistamines, anticonvulsants, opiates, neuroleptics, and tricyclic and MAOI antidepressants. Although these do not share any single receptor mechanism, they have in common the effect of reducing motor and autonomic activity and lowering the sleep threshold. Amphetamine and its congeners, as well as nicotine, caffeine, and other theophyllines, improve both physical and mental performance, postpone the onset of fatigue and elevate mood (reviewed by Weiss & Laties, 1962). As for the first group, no constant receptor mechanism can be inferred.

This might be seen as supportive of a unitary concept of physiological arousal, in which fatigue of performance in one modality would impair performance in others. This has received limited recent support (Weber *et al*, 1975; Jain, 1983). It has also been established that physical training sufficient to produce a moderate increase in maximum oxygen uptake produced significant reduction in depression scores in depressed in-patients when compared with controls (Martinsen *et al*, 1985). In normal subjects, the subjective rating of physical exertion is closely related to heart rate and oxygen uptake. Physical training lowered the subjective rating of physical exertion at a given rate, but was the same when related to the relative (percentage of maximum) oxygen uptake (Ekblom & Goldbarg, 1971).

Endocrine changes might be expected in fatigue and lassitude, which are prominent symptoms in Addison's disease, and can be reproduced by reducing or abruptly stopping corticosteroids in patients on long-term treatment (Hennemann *et al*, 1955). Exercise and anxiety are often shown to produce similar endocrine changes, and exercise can be shown to cause elevations of circulating catecholamines, growth hormone, corticotrophin, cortisol, and glucagon, with a decline in insulin and testosterone (Dessypris *et al*, 1976).

This physiological response has wide-ranging effects, including the mobilisation of metabolites and altered distribution of cardiac output so as to facilitate work. If the response failed, one might expect a rapid decline in performance accompanied by subjective discomfort. This discomfort on effort would lead to learnt inactivity, apathy, and slowness, mimicking depression. Although the cortisol response to chronic stressful situations rapidly diminishes, this represents habituation rather than impairment, as a new stimulus can immediately evoke a maximal response. The secretion of adrenaline corresponds closely with vigilance tasks, and appears not to adapt even when other endocrine responses have habituated (reviewed by Rose, 1984). There is little evidence for failure of these endocrine responses in acute or chronic fatigue, or in psychiatric illnesses. There is one report of low morning cortisol levels in a series of 25 patients with 'chronic fatigue' when compared with matched controls (Poteliakhoff, 1981). It has in addition been reported that the normal elevation in plasma cortisol induced by amphetamine is blunted in depressed patients (Checkley & Crammer, 1977).

Psychopathology

Performance shows a diurnal rhythm, as measured by tests of psychomotor ability, symbol cancellation, reaction time, and digit summation (Klein *et al*, 1972). The state that produces impaired initial performance, for example, that due to sleep deprivation, is lassitude. Whether this is associated with increased fatigue (rapid decline of performance) is not known. Nor is it clear whether cognitive fatigue is associated with a refractory period analogous to physical fatigue.

Kraepelin (1902, p. 42) believed that retardation of thought could occur even in healthy individuals as the result of physical and mental fatigue, or unpleasant emotional states. Other causes include intoxications produced by alcohol or tobacco. He also believed that "the capacity for mental work is independent of the rapidity of thought". It could be increased by practice, and "in morbid states the effects of practice are usually lessened and rapidly disappear". This "capacity for mental work stands in inverse ratio to susceptibility to fatigue, [and] increased susceptibility to fatigue is very general in most forms of insanity". Although learning effects are readily demonstrated, for example, on continuous performance tasks, the ability of practice to enhance the capacity for mental work by analogy with weight training or long-distance running is intriguing, but unconfirmed.

Organic disorders and fatigue

The differential diagnosis of patients presenting to physicians with fatigue has been described (Havard, 1985). Tiredness, subjective anergia, and retardation are common in bulimia nervosa (Fairburn & Cooper, 1984) and also occur in chronically hyperventilating patients and alcoholics. These disorders produce complex changes in potassium, magnesium, calcium, and phosphate ions, which directly impair muscle and nerve, by effects on both electrical activity and metabolism (Brashear, 1983; Fonesca & Havard, 1985; Pearson *et al*, 1986). Can this be attributed to electrolyte disturbances? Patients with hypokalaemia can be shown to have objective muscle weakness, confusion, and depression (Lishman 1978, p. 659), but these are mostly elderly patients. Pure magnesium deficiency causes depression, irritability, vertigo, ataxia, and muscle weakness (Hanna *et al*, 1960). Whether in addition they would have 'cognitive fatigue', as measured experimentally, would be of interest.

Daytime somnolence, fatigue, morning headache, irritability, and impotence are prominent features of the sleep apnoea syndrome, in which profound hypoxia occurs repeatedly throughout the night (Orr, 1983; Guilleminault, 1985). Patients are typically obese, have a high alcohol intake and may show signs of frontal-lobe dysfunction. These features can be reversed by treating the nocturnal hypoxia (Sullivan *et al*, 1983). Symptoms do not always accompany nocturnal hypoxia, and it may be that an additional disorder, such as an impaired arousal response, is necessary to develop the full syndrome (Broughton, 1968; Orr *et al*, 1979; Issa & Sullivan, 1982).

Recently a post-viral syndrome of fatigue has been described. Some patients have evidence of recent or chronic viral infection (Behan, 1985; Roberts, 1985) but many do not (Thomas, 1987). Treatment with interferon, a substance released by lymphocytes as part of the normal response to viral infections, produces a syndrome of fatigue, slowness, drowsiness, and confusion, while at the same time

accentuating any existing neurotic symptoms (McDonald *et al*, 1987). Others have described unusual immunological defects in patients with prolonged illnesses after Ebstein–Barr virus infection. These patients complain of lassitude, but have in addition persistent fever and atypical lymphocytes in the blood film (Borysiewicz *et al*, 1986).

Fatigue is clearly a feature of active viral infection, but the status of the post-viral syndrome is less well established. It has recently been argued that at least some epidemic forms should be classified with psychological disorders (Wessely, 1987). Physical illness can cause fatigue and depression, but depression can cause a wide variety of immunological defects (Locke *et al*, 1984; Baker, 1987), which could in turn cause physical illnesses. This could encourage some physicians to define a syndrome of 'primary' fatigue, by exclusion of known physical causes, while disregarding coexisting depression as merely something to be expected. A Toronto group has recently reported a series of 24 patients with 'neuromyasthenia' (said to be synonymous with benign myalgic encephalomyelitis and the post-viral syndrome). Of these, 16 concurrently had major depression, and 12 had major depression before onset of neuromyasthenia, compared with 12% of matched controls (Taerk *et al*, 1987).

Psychiatric disorders and fatigue

Fatigue is considered a sufficiently valid concept to be included in the operational criteria for major depression, dysthymia, and cyclothymic personality disorder, in DSM–III (American Psychiatric Association, 1980). Fatigue is also included in the Beck (Beck *et al*, 1979) and Hamilton (1967) depression rating scales and the General Health Questionnaire (Goldberg, 1972). All of these also include items concerning weight loss and sleep loss, which might reasonably be expected to cause fatigue. Is fatigue in depression independent of other features of depression?

There is relatively little work on the constancy of the relationship of fatigue to specific mood states. The Profile of Mood States (POMS), a mood adjective checklist validated on 1000 university-hospital out-patients, found by factor analysis that independent factors for fatigue and vigour could be distinguished from other factors for anxiety, depression, and anger (McNair *et al*, 1971), implying that in normal subjects, depression and fatigue are independent. In clinical depression, the use of factor analysis to relate symptoms to syndromes usually assigns fatigue to a depression syndrome, but sometimes to neurotic rather than endogenous categories (Paykel, 1971; Overall, 1980). The complaint of fatigue in the absence of signs of physical disease cannot be dismissed as due to depression or neuroticism. Taking a psychiatric history and assessing the mental state may give positive evidence of depression, which is essential for such a diagnosis.

Conclusion

The status of fatigue as a physiological response, psychological perception, or symptom of physical and psychiatric diseases, remains unclear. In particular, the distinction between psychological and physiological aspects of fatigue has led to the conclusion that clear experiments could not be done in this field. This may arise from the artificial nature of the distinction. The decline in performance on tasks such as the rotor pursuit seems likely to represent the decline of a neurophysiological function. If interpersonal differences in such paradigms exist and can be related to neuroticism or depression scores, they cannot be dismissed as merely functional. There is a need to revive interest in the phenomenology of fatigue. Of particular interest to psychiatrists must be the relationship between fatigue of psychological functions and abnormal mental states. Future phenomenological research might try to differentiate between the fatigue symptoms of depression, anxiety, postencephalitic and postconcussional states and other causes of chronic 'fatigue', if any.

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