or chronic course and manifestations vary in length from hours to years. Recurrent brief neurasthenia (of a few days' duration) and extended neurasthenia of two weeks' or more duration were operationally described and have also been called prolonged fatigue Both represent shorter manifestations than the three months required by ICD-10 (WHO 1993).

In the WHO general health care study 8.7% of patients were found to be suffering from neurasthenia and 8.7% from depressive episodes, which represented the two most common psychological disorders. Most studies also reported that 70% or more of the cases of neurasthenia were associated with psychological disorders.

In the Zurich cohort study of a community sample, which assessed morbidity through five interviews (each covering one year) from the ages of 20 to 35, we found a longitudinal prevalence rate of 4.3% for ICD-10 neurasthenia and 6.3% for extended (prolonged) neurasthenia. Recurrent brief neurasthenia was observed in a further 11.4%, The prevalence in females was three to five times higher than in males.

Most neurasthenic syndromes were found to lead to subjective work and social impairment and to be associated with a positive family history of the syndrome. The validity of several neurasthenic subgroups can also be demonstrated by the degree of suffering/distress, treatment seeking, prescribed medication and diminished quality of life. Longitudinally ICD-10 neurasthenia was associated in 78% of the cases with major depressive episodes (especially with atypical major depression), with anxiety disorders in 70% and with substance abuse in 12% of the cases.

The data support a descriptive approach for the definition of the spectrum of neurasthenia from brief through extended to more chronic forms and suggest that they should be analysed in their complex association with all subgroups of psychological disorders.

S9-3

DIAGNOSIS, ASSESSMENT AND MANAGEMENT OF CHRONIC FATIGUE SYNDROME

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In this paper I shall review the recent epidemiological studies on the prevalence of chronic fatigue syndrome (CFS) in the general population and primary care, concluding that it is by no means uncommon, and is also a common cause of personal morbidity. However, patients who present to doctors with the label of CFS are less common, and also frequently present management problems. I shall consider the current diagnostic criteria, when and how to make the diagnosis of CFS, and what to do next. I will review the limited number of investigations necessary, and then conclude with a discussion of practical treatment strategies and the evidence to support them.

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S9-4

CHRONIC FATIGUE SYNDROME, SEROTONIN AND DEPRESSION: HOW STRONG THE LINK?

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There is a high degree of co-morbidity between chronic fatigue syndrome (CFS) and major depression (MD). Indeed, several symptoms in the diagnostic criteria for the two conditions overlap. However, there is now emerging data to suggest a neurobiological distinction between CFS and MD. First, the consensus of studies using neuropharmacological challenge tests reveal reduced central serotonergic function in MD, consistent with the serotonin hypothesis of MD. In contrast, studies in CFS show the opposite effect, with enhanced responses to serotonergic challenge, suggesting enhanced central serotonergic function. Second, MD has long been known to be associated with hypercortisolaemia and a range of abnormalities related to hypothalamo-pituitary-adrenal (HPA) axis overdrive. Emerging evidence in CFS points to a reduction in HPA axis output, with low circulating cortisol levels, and abnormal responses to dynamic testing of the HPA axis components. There is now much evidence of an inverse link between cortisol levels and serotonergic function; whether serotonergic abnormalities cause the HPA axis changes or vice versa is not yet known. However, since low cortisol levels lead to fatigue and other symptoms in Addisons disease, we tested the hypothesis that low cortisol levels in CFS were related to some symptomatology by giving low-dose cortisol replacement with 5 mg or 10 mg of hydrocortisone in a randomised double blind placebo controlled crossover. Both doses of cortisol led to significant improvements in fatigue and disability, suggesting that low cortisol levels may be a significant factor in maintaining symptoms in CFS.

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S9-5

A COGNITIVE BEHAVIOUR FORMULATION AND TREATMENT OF CHRONIC FATIGUE SYNDROME

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A variety of treatments have been tried for chronic fatigue syndrome. The only one to be shown to be efficacious in replicated randomised controlled trials is rehabilitative cognitive behaviour therapy (CBT).

The cognitive behavioural model emphasis the interaction of patient beliefs, emotional arousal and physiological disturbance within an interpersonal context. Particular importance is paid upon the belief that activity will be harmful and on the behavioural change of stabilising and increasing activity.

To date there have been three randomised controled trials of CBT published and one of a behavioural (exercise) programme.

The first of these by Lloyd et al. used a brief CBT and did not find this to be superior to good medical care. The next study by our own group used an intensive sixteen session cognitive behaviour therapy with a strong emphasis on rehabilitation and found a clinically and statistically significantly greater improvement in the functioning of patients who had received this treatment from that obtained by routine medical care. It is of considerable interest that the patient improvement was gradual and persisted after therapy had been completed. These results were substantially replicated in a further trial by Deale et al. which compared a similar form of cognitive behaviour therapy with time matched relaxation.

More recently Fulcher et al. have shown that graded aerobic exercise (accompanied by a considerable explanation and support) is also superior to simple flexibility exercises.