Chronic Fatigue Syndrome

Introduction

Chronic Fatigue Syndrome (CFS) is a debilitating condition of persisting or relapsing fatigue with a prevalence of 0.5% in the U.K (White and Clare, 2002, p1234). In the past, research in this area has been fraught with difficulty due to vague and inconsistent diagnostic criteria. However, more recently the Center for Disease Control and Prevention (CDCP) in the United States has produced a standardised framework for a diagnosis of chronic fatigue.

The patient must have severe and disabling fatigue for a minimum of six months duration associated with a decrease of at least 50% in functioning and with at least four of the following eight symptoms being concurrently present over the same period

- Enlarged and tender axillary/cervical lymph nodes
- Recurrent sore throats
- New headaches
- Unrefreshing sleep
- Arthralgia
- Myalgia
- Post-exertional malaise
- Neuropsychological disturbance

Fatigue that can be explained by any medical condition precludes a diagnosis of CFS and this criteria does help to make distinctions from Epstein-Barr (Scott and Dinan, 1999, p3). However, this criterion could be considered a somewhat narrow view of a multifactorial syndrome in which a sufferer can be present with any of a wide range of symptoms alongside the persisting fatigue. Various other symptoms, including palpitations, arrhythmias and nausea, are not included and there is much overlap with fibromyalgia. Whether this criterion has been recently used to assess the prevalence in the UK is unclear.

The Terrain of the Patient – Predisposing factors

The fundamental terrain of the patient – i.e. the genetic endowment of the individual together with social and environmental factors will have a primary role in the predisposition to CFS. It has also been established that panic disorder has the highest rate of familial co-morbidity in CFS and personality traits such as those that tend to overwork and take little time for relaxation are predominant (Gupta, 2002, p727; Scott and Dinan, 1999, p8).

Excitatory factors

It appears that many chronic fatigue patients can recall a period of acute psychological stress which accompanied the onset of the CFS. This can also be in combination with other types of physical stressors, predominantly chemical or viral (Gupta, 2002, p727). Stimulation of the sympathetic nervous system in the alarm phase of the stress response causes the adrenal medullae to secrete adrenaline. This provokes many different responses in the body including the redirection of oxygen to the heart and muscles via peripheral vasoconstriction, bronchial dilatation, decreased urine production at the kidneys and elevated blood sugar and cholesterol levels and a myriad of other effects to enable the body to 'fight or flight.'

These processes can continue for some time and the terrain enters what is known as the resistance phase of the stress response with the increased breakdown of fats, proteins and carbohydrates to produce glucose. But because the majority of modern day stress is taken sitting down, these responses are not being seen through to their rightful end. Apart from the more obvious and documented problems that arise from these unfulfilled pathways, such as arteriosclerosis from raised cholesterol, arrhythmias from the extra stimulation of the heart and raised blood pressure from persistent vasoconstriction (Mills, 1993, p90) one is left to speculate what other effects on the body may take place if this carries on beyond what should essentially be an acute response culminating in physical exertion.

The parasympathetic rebound is known to occur after a period of sympathetic activation. This over activation of the parasympathetic nervous system occurs in order to compensate for a period where the terrain has been subjected to the stress response (Schedlowski and Tewes, 1999, p321). It is the autonomic nervous systems attempt to rebalance itself, and as such, sounds the bell for the commencement of sustaining factors.

Sustaining factors

A chronic sympathetic outpouring will suppress digestive functions by inhibition of the secretory glands and a decrease in blood supply to the digestive viscera. Essential IgA production could become compromised and leave the gut vulnerable to antigens. The pancreas is also affected, with decreased exocrine secretion and inhibition of insulin. This leads to poor assimilation, raised blood sugar levels, immunosurpression and subsequently - a vulnerable terrain. These disruptions will promote the parasympathetic rebound. Although various cranial nerves innervate this division, the digestive organs are specifically innervated by the vagus nerve. Over activation of this nerve, known as vagotonia, can in itself can produce some of the symptoms seen in CFS such as enlarged salivary glands and upper respiratory tract infections along with lymphadenopathy due to a lack of migratory IgA from the gut (Nicholls, 2003). This prolonged imbalance of the autonomic nervous system will help sustain some of the symptoms experienced by CFS suffers. There is a general consensus that dysregulation of the Hypothalamic-Pituitary-Adrenal (HPA) axis resulting in decreased plasma cortisol levels is a consistent finding in patients with

CFS (Demitrack et al, 1999; Polietakhoff, 1981; cited in Scott and Dinan, 1999, p5; Cleare et al, 1995, p283-289). There can be no doubt that this hypofunctioning is a sustaining factor and this type of irregularity can also be compared to the exhaustive phase of the stress response.

It is interesting to note that some of the symptoms of CFS are the same as primary hypoadrenalism or Addisons disease. This too has symptoms of weakness, malaise, myalgia and arthralgia (Drury and Howlett, 2002, p 1050) But the complexity of the feedback systems within the HPA system appears to be eluding researchers in this area.

A paper published by Ashok Gupta (2002, p727-735) provides further clues when considering sustaining factors. Gupta's hypothesis takes us deeper - into the unconscious emotional responses of CFS patients. The amygdala within the limbic system operates at an unconscious level and has two roles; the first is to determine whether immediate stimuli is a threat to well being. The second is to mount appropriate behavioural, autonomic (fight or flight) and endocrine (HPA axis) if the stimuli does pose a threat. Furthermore, the autonomic and endocrine responses to the stimuli imprint an emotional memory within the amygdala so that it can respond accordingly if presented with the same stimuli. Gupta suggests that the amygdala may become over sensitised to both exogenous and endogenous stressors. Thus the amygdala ends up hitting the panic button even when there is really nothing to panic about. This causes a chronic neuroendocrine outpouring from the amygdala via various brain pathways including the hypothalamus.

He proposes that the following excitatory factors may cause this unconscious and sustaining response.

- Overactive sympathetic nervous system in response to psychological stress
- Effects of a viral infection acting on a weakened immune system
- Effects of an active immune system (notably IL-1 & 6) which itself produces symptoms of general weakness. (i.e. autoimmunity causes the amygdala to become sensitised)
- Prolonged period of post viral fatigue.

Concern over symptoms may also add to sustaining this inappropriate response from the limbic system. When contemplating this hypothesis, it is once again clear why the diagnostic criteria for CFS remained confused for so long. From this, it can be seen that the excitatory factors of prolonged fatigue can be caused by endogenous or exogenous stressors - or both.

Although fatigue from autoimmunity is undoubtedly explainable from a medical condition it should be noted that according to Gupta the limbic system can sustain it further. There is evidence to suggest blood-brain-barrier permeability (BBBP) may have a role in sustaining chronic fatigue. Some factors which are known to compromise BBBP are stress, viruses, glutathione and essential acid deficiency, all of

which can be related to CFS. It is thought that the breakdown of BBBP may cause disruptions to neuronal transmission, exacerbating CFS further (Bested et al, 2001).

Treatment plan

Having considered all of the above, it would be unwise to suggest there is such a thing as a typical case of CFS . The course of CFS is multidimensional and cases can only be contemplated on an individual basis. An attempt has been made here to generalise and the following aims and actions along with a wide choice of herbal indications have been considered. Careful choice of herbs will enable a single herb to have a two or three fold mechanism in the treatment of CFS. It should be possible to treat a broad spectrum of presentations with the following suggestions.

Terrain Medicine

• Regulation of the HPA axis in order to help the terrain regain the ability to deal with presenting stressors efficiently and in relative terms. Use adaptogenic herbs; Panax ginseng, Eleutherococcus senticosus, Withania somniferons.

• Nourish the depleted adrenal cortex. Use adrenocorticotrophic herbs; Glycyrrhiza glabra, Borago officinalis, Ribes nigrum, Bupleurum falcatum. Atrophy of the adrenal glands maybe addressed using Panax ginseng.

• Correction of autonomic imbalance in order to reduce conflict within the terrain predisposing to GIT disturbances, heart disturbances, blood sugar disruption and immunosurpression - Decrease sympathetic nervous activity and reduce parasympathetic rebound. Use sympatholytic herbs – Crateagus oxyacantha , Thymus vulgaris parasympatholytic herbs; Lavandula angustifolia, Gentiana lutea.

• Support the nervous system and aid sleep; use trophorestoratives to the nervous system and antidepressants; Hypericum perforatum, Avena sativa, Withania somniferons, Lavandula angustifolia, Schisandra chinensis

• Stimulate gastric secretions and improve choleresis, stimulate exocrine pancreas. Enhance elimination. Use bitters - encompassing stomachics, choleretics, pancreatics and hepatoprotectives; Artemisia absinthium, Gentiana lutea, Cynara scolymus, Chelidonium majus, Taraxacum Radix, Carduus marianus, Bupleurum falcatum, Schisandra chinensis, Juglans regia

• Regulate endocrine pancreas. Use blood sugar regulators; Gymnema sylvestre, Juglans regia, Vaccinium myrtillus leaf, Galega officinalis.

• Provide antiviral activity. Use antivirals Eleutherococcus senticosus, Astragalus membranaceus, Glycyrrhiza glabra

• Improve circulation along with vascular permeability. Use circulatory herbs and vascular tonifiers Gingko biloba Vaccinium myrtillus fructus Symptomatic Treatment

• Reduce respiratory infections: – Use immunostimulants, antimicrobials and astringents: Echinacea purpurea, Baptisia tinctoria, Thymus vulgaris, Hydrastis canadensis, Commiphora molmol.

• Decrease lymphadenopathy. Use lymphatic drainers: Galium aparine, Phytolacca americana.

• Address palpitations and arrythmias. Use cardiotonics ; Crateagus oxyacantha, Leonorus cardiaca. • Ease Myalgia and Arthralgia. Use antispasmodics Virburnum opulus. Use analgesics Salix alba Use anti-inflammatory Harpagophytum procumbens.

• Reduce nausea – Cynara scolymus, Zingiber officinale

Sample Rx
Eleutherococcus senticosus 20ml
Baptisia tinctoria 15ml
Cynara scolymus 20ml
Artemisia absinthium 5ml
Lavandula angustifolia 20ml
Ribes nigrum 20ml

Anticipated Outcome-

Whatever is prescribed, indications that treatment is going well should take the form of a reduction in respiratory symptoms, lymphadenopathy, palpitations and nausea etc. If analgesics and antispasmodics are being used a reduction in myalgia and arthralgia will be swift and this will give hope to the patient and encourage compliance. However, in many cases of CFS these symptoms can only truly be addressed if the imbalance of the terrain is dealt with effectively and this may take some time. A gradual increase in energy is anticipated.

Further management

It may be of use to the patient to consider finding an experienced cranio-sacral therapist who can communicate with the amygdala to help correct the sensitisation that has occurred on an unconscious emotional level. A programme of gradually increasing activity is proposed as a form of management for CFS but is nevertheless noted as not curing the patient (White and Clare, 2002, p1234). Diet is naturally an issue to be considered in all cases. Encouraging the patient to gain an understanding of the glycaemic index will be helpful for blood sugar levels. A diet rich in fruit and vegetables will help ease the burden on GIT as well as providing many nutrients for the terrain as a whole.

Conclusion

The complexity of CFS has led to many problems in its management as the numerous symptoms and signs that appear can seem to have no rhyme or reason. Conversely, attempts to put CFS into any one box (e.g. the CDCP's recent diagnostic criteria) could also hinder understanding and may lead to the GP's diagnosis of 'nothing wrong with you'.

When considering a treatment plan it is imperative that the excitatory cause of the fatigue, e.g. prolonged psychogenic stress, viral stress, environmental or autoimmune stress or the combination presenting, is ascertained in order to ensure a successful therapeutic strategy. A thorough history of the presenting problem with a focus on circumstances may go far in elucidating this. Following that, the systemic enquiry should help reveal sustaining factors i.e. autonomic and endocrine imbalances demonstrated through symptoms such as lymphadenopathy, salivary gland enlargement, respiratory infections, heart symptoms and GIT disturbances. It should not be automatically assumed that a viral stressor is involved simply because there are respiratory symptoms presenting.

CFS is diverse, but if the fundamental principles of the terrain are applied, a greater clarity and understanding may be achieved of this syndrome's multifactorial nature.