

## The HUMMINGBIRDS' FOUNDATION for M.E. (HFME)

Fighting for the recognition of Myalgic Encephalomyelitis based on the available scientific evidence, and for patients worldwide to be treated appropriately and accorded the same basic human rights as those with similar disabling and potentially fatal neurological diseases such as Multiple Sclerosis.

### The effects of CBT and GET on patients with M.E.

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No evidence exists which shows that cognitive behavioural therapy (CBT) or graded exercise therapy (GET) are appropriate, useful or safe treatments for Myalgic Encephalomyelitis patients. Studies involving miscellaneous psychiatric and non-psychiatric 'fatigue' patients which qualify for a diagnosis of 'CFS,' and their response to these treatments, have no more relevance to M.E. sufferers than they do to diabetes patients, cancer patients, patients with multiple sclerosis or any other illness. Thus, patients with M.E. are being prescribed these treatments on what amounts to a random basis medically and so the questions need to be asked:

1. What is the effect of graded exercise therapy (GET) on Myalgic Encephalomyelitis (M.E.) patients?
2. What is the effect of cognitive behavioural therapy (CBT) on Myalgic Encephalomyelitis (M.E.) patients?

#### 1. What is the effect of graded exercise therapy (GET) on Myalgic Encephalomyelitis (M.E.) patients?

As (bad) luck would have it, graded exercise programs are probably the single most inappropriate treatment that a M.E. sufferer could be recommended to undertake. This is because one of the unique features of authentic M.E. is exercise intolerance – that patients worsen with even trivial levels of activity beyond their individual post-illness limits. Exercise or exertion intolerance is one of the many things which separates Myalgic Encephalomyelitis so distinctly from various post-viral fatigue states or other illnesses involving 'chronic fatigue' as the defining or primary feature. People with M.E. do not improve with exercise. They cannot; exercise intolerance is a large and essential part of *what M.E. is*. Veteran M.E. expert Dr Ramsay explained that this unique characteristic: 'is virtually a sheet-anchor in the diagnosis of Myalgic Encephalomyelitis and without it a diagnosis should not be made' (1986, [Online]).

This essential feature of M.E. is characterised by a unique form of paralytic muscle weakness whereby muscles perform normally to begin with but after even a minor degree of physical effort; three, four or five days, or longer, elapse before full muscle power is restored. This is quite distinct from the 'chronic fatigue' seen in many other illnesses.

Fatigue' and feeling 'tired all the time' are not at all the same thing as the very specific type of *paralytic muscle weakness* or *muscle fatigue* which is characteristic of M.E. (and is caused by mitochondrial dysfunction) and which affects every organ and cell in the body; including the brain and the heart. This causes – or significantly contributes to – such problems in M.E. as; cardiac insufficiency (a type of heart failure), orthostatic intolerance (inability to maintain an upright posture), blackouts, reduced circulating blood volume (and pooling of the blood in the extremities), seizures (and other neurological phenomena), memory loss, problems chewing/swallowing, episodes of partial or total paralysis, muscle spasms/twitching, extreme pain, problems with digestion, Raynaud's phenomenon, vision disturbances, breathing difficulties, and so on. These problems are exacerbated by even trivial levels of physical and cognitive activity, sensory input and orthostatic stress beyond a patient's individual post-illness limits leaving M.E. patients extremely disabled (Bassett 2009, [Online]).

People with M.E. are experiencing a form of heart failure which can be exacerbated by even relatively low levels of activity. Many patients are housebound and bedbound and often are so ill that they feel they are about to die. Some M.E. patients do die due to overexertion. People with M.E. would give *anything* to instead only be severely 'fatigued' or tired all the time.

Fatigue or post-exertional fatigue (or malaise) may occur in many different illnesses such as various post-viral fatigue states or syndromes, Fibromyalgia, Lyme disease, and many others – but what is happening with M.E. patients is an entirely different (and unique) problem of a much greater magnitude. These terms are not accurate or specific enough to describe what is happening in M.E.

The paralytic muscle weakness seen in M.E. affects all muscles including the heart and causes what is commonly known as exercise intolerance; that patients relapse with excessive physical and cognitive exertion, as well as with orthostatic stress. These features are a core part of what M.E. is as they are responsible for causing much of the symptomatology and disability associated with the disease (Hyde 2006, [Online]) (Hooper 2006, [Online]) (Hooper & Marshall 2005a, [Online]) (Hyde 2003, [Online]) (Dowsett 2001, [Online]) (Hooper et al. 2001, [Online]) (Dowsett 2000, [Online]) (Dowsett 1999a, 1999b, [Online]) (Dowsett 1996, p. 167) (Dowsett et al. 1990, pp. 285-291) (Dowsett n.d., [Online]).

Doctors who have experience with M.E. (and can tell the difference between authentic M.E. and various unrelated fatigue states) and the leading M.E. experts all concur; physical, cognitive or orthostatic overexertion can have many harmful effects on patients both in the short- and long-term. The following comments which illustrate this point are provided by some of the world's leading M.E. experts, all of whom have been specialising in M.E. for many years and each of whom has seen literally *thousands* of M.E. patients;

**1. Dr Melvin Ramsay**, a UK doctor who specialised in M.E. for more than thirty years, from the Royal Free Hospital M.E. outbreak of 1955 until his death in 1990, and who is credited with having written some of the most accurate description of the illness to date, explains, 'The degree of physical incapacity varies greatly, but the [level of severity] is directly related to the length of time the patient persists in physical effort after its onset; put in another way, **those patients who are given a period of enforced rest from the onset have the best prognosis.** Those who are given complete rest from the onset do well. Those whose circumstances make adequate rest periods impossible are at a distinct disadvantage, but no effort should be spared to give them the all-essential basis for successful treatment.

Since the limitations which the disease imposes vary considerably from case to case, the responsibility for determining these rests upon the patient. Once these are ascertained the patient is advised to fashion a pattern of living that comes well within them' (Ramsay 1986, [Online]).

**2. Dr. Elizabeth Dowsett** explains, 'There is ample evidence that M.E. is primarily a neurological illness although non neurological complications affecting the liver, cardiac and skeletal muscle, endocrine and lymphoid tissues are also recognised. Apart from secondary infection, the commonest causes of relapse in this illness are physical or mental over exertion. The prescription of increasing exercise in such a situation (or in the early stage of the illness when the patient desperately needs rest) can only be counter-productive' and 'This illness is distinguished from a variety of other post-viral states by a unique clinical and epidemiological pattern characteristic of enteroviral infection. Prompt recognition and advice to avoid over-exertion is mandatory' and 'The prescription of increasing exercise can only be counter-productive.'

Also from Dr Elizabeth Dowsett:

The brain has often been likened to a computer. However, there are fundamental differences in its essential function of processing, comparing and storing information. Unlike a computer, which can be switched on and off and is programmed to give set answers to a single question, the chemical transmitter bridging the synapse introduces a variability into the on-going message and "Neuronal Plasticity" into the receiving/transmitting network. It has been shown that similar modifications in response may be induced by virus infection. The brain contains some 100 billion neurons connected to some 10,000 relay stations and this enormous electrical activity creates a massive need for energy, using up 20% of the entire body's demand for oxygen and glucose. Recent studies of the brain stem by SPECT scan, indicate hypoperfusion and low metabolic activity in subjects with M.E.

Modern research indicates disturbed metabolism in many areas essential to motor control in the brain stem of patients with M.E., the majority of whom have evidence of inco-ordinated muscle twitching after slight exertion.

A good memory demands normal functioning of almost all areas of the cerebral cortex, the basal nerve centres of the mid brain (eg the thalamus and hippocampus) and their interconnecting pathways through the brain stem.

Fluctuations of metabolic activity in these areas (often made worse by physical and mental [overexertion]) have been reported in SPECT scans of patients with M.E., the vast majority of whom complain of difficulty with short-term memory (n.d.c, [Online]).

Dr Dowsett states about M.E. patients that, '**20% have progressive and frequently undiagnosed degeneration of cardiac muscle which has led to sudden death following exercise.**'

According to Dr. Elizabeth Dowsett, any M.E. patient can also be stopped from deteriorating further and at least stabilised (if not in time experiencing some level of improvement) through receiving appropriate care and being allowed to get the needed level of rest (providing that the patient has not already been exposed to unrecoverable levels of overexertion) (Dowsett & Ramsay et al. 1990) (Dowsett 2000, [Online]) (Dowsett 2001a, [Online]) (Dowsett n.d.b., [Online]). Dr. Elizabeth Dowsett also explains that:

Scientific discoveries recently reported, indicate that embryonic stem cells left over from foetal development, remain in the brain tissue during adult life and are capable of "running repairs" (thus patients are able to recover after head injury, stroke and relapse in ME). However, overuse of these repairs, as in ME (when the patients are overstressed [overexerted] physically or mentally) will cause unnecessary deterioration which may then become irreparable. Intervention in the form of financial, rehabilitation and nutritional support can do much to prevent the physical, occupational and other deterioration in the quality of life for a large group of patients now between 40 and 60 years of age, to say nothing of educational loss in children.

HEALTH SERVICE INTERVENTIONS: It is sad to read that these are said to be of dubious priority in the present state of the NHS when it is known that the correct type of rehabilitation can stabilise the illness. This requires access to local facilities

without discrimination against patients with a diagnosis of ME, together with a domiciliary nursing service for the bed-bound who are unable to travel ( 2002b, [Online]).

**3. Dr Byron Hyde** explains in his M.E. textbook that it has been found that those patients with M.E. who returned to work soon after becoming ill or while they were still seriously or severely ill – instead of having an extended period of rest and recovery – are at risk of causing an abnormal increase in damage ‘to a heart muscle already vulnerable and under attack from an acute viral infection’ and that those who do not, or cannot, rest in the early stages of M.E. potentially create ‘a physical injury to the myocardium, cardiac pacemaker cells or their autonomic control.’ Dr Hyde explains that:

This is not just clinical supposition, there is a strong basis for this belief of work or exercise potentiated heart damage in the literature. It is well known that enteroviruses may cause chronic cardiac disease as well as major neurological injury. Kandolf states that "enteroviruses are capable of causing dilated cardiomyopathy of sudden onset or lead to a variety of common arrhythmias." Utilizing mouse models, Wilson and again Reyes demonstrated that Coxsackie infected [enterovirus infected] mice, forced to swim to the point of exhaustion during the acute phase of infection, developed chronic heart disease whereas Coxsackie infected mice who were allowed to rest during the acute phase, did not develop chronic heart disease.

M.E. represents a possibility of serious cardiac injury primarily in patients who exercise or maintain exhaustive work efforts during the onset of their illness. It is possible that some of these patients who die and other that develop major cardiac changes are never recognised as M.E.

With both CNS and CVS disease, chronicity may be provoked by maintaining strenuous exercise and work levels.. Early patient activation may represent serious cardiovascular danger to patients [with M.E.]. The strange concept of waiting 6 months to diagnose a classical case of M.E. [brought about by the confusion between M.E. and ‘CFS’] is unnecessary and fraught with potential danger to the patient. Such a diagnostic delay may create legal consequences for the physician. Physicians who take an early aggressive approach in physically activating these acute stage patients may do so at both their and their patient’s peril (Hyde & Jain 1992a, pp. 375-383).

M.E. is an infectious neurological disease and represents a major attack on the central nervous system (CNS) by the chronic effects of a viral infection. The world’s leading M.E. experts, namely Ramsay, Richardson, Dowsett and Hyde, (and others) have all indicated that M.E. is caused by an enterovirus. (This also includes doctors such as A. Gilliam, W.H. Lyle, Elizabeth Bell of Ruckhill Hospital, James Mowbray of St Mary’s, and Peter Behan). The evidence which exists to support the concept of M.E. as an enteroviral disease is compelling (Hyde 2007, [Online]) (Hyde 2006, [Online]).

Dr Hyde explains that enteroviral infections are able to cause:

- a. a chronic host infection
- b. major or no cardiac disease depending on the virulence of the subtype
- c. cardiac injury dependent upon the sex of the patient and of the level of physical activity of the patient during the acute or infectious stage
- d. cardiac disease depending upon the immunological variability of the host (Hyde & Jain 1992a, p. 40).

An enterovirus would also explain the; age variation, sex variation, obvious resistance of some family members to the infection and the effect of physical activity (particularly in the early stages of the illness) in creating more long-term/severe M.E. illness in the host (Hyde & Jain 1992a, p. 40). There is also the evidence that; M.E. epidemics very often followed polio epidemics, M.E. resembles polio at onset, serological studies have shown that communities affected by an outbreak of M.E. were effectively blocked (or immune) from the effects of a subsequent polio outbreak, evidence of enteroviral infection has been found in the brain tissue of M.E. patients at autopsy, and so on (Hyde 2007, [Online]) (Hyde 2006, [Online]) (Hyde 2003, [Online]) (Dowsett 2001a, [Online]) (Dowsett 2000, [Online]) (Dowsett 1999a, 1999b, [Online]) (Hyde 1992 p. xi) (Hyde & Jain 1992 pp. 38 - 43) (Hyde et al. 1992, pp. 25-37) (Dowsett et al. 1990, pp. 285-291) (Ramsay 1986, [Online]) (Dowsett & Ramsay n.d., pp. 81-84) (Richardson n.d., pp. 85-92) (Richardson 1999, [Online]).

Dr Byron Hyde, also explains that the vascular and cardiac dysfunctions seen in M.E. are often the most obvious set of dysfunctions when looked for, and are the cause of a significant number of M.E. symptoms:

The subject of vascular pathology is not new. The fact of the children dying of a Parkinsonian-like vascular injury to the basal ganglia in Iceland during the Akureyri M.E. Epidemic is an obvious indication of the CNS vascular effects in M.E. Vasculitis has been well documented by Dr. E. Ryll in his description of the epidemic in the San Juan Mercy, Sacramento California Hospital in 1975. He described this M.E. epidemic as an epidemic vasculitis. He was correct. Following my 21 years of examining M.E. patients and 16 years of subjecting M.E. patients to brain imaging techniques, it has become obvious to me that we are dealing with both a vasculitis and a change in vascular physiology. Numerous other physicians have supported this finding.

The recent interpretation of the cause of Multiple Sclerosis (MS), as an injury of the microvasculization causing the injury of the schwann cells that in turn causes the demyelination injuries of MS has been added to that of paralytic poliomyelitis as an essential vascular injury. Paralytic poliomyelitis was thought to be a primary injury to the anterior horn cells of the spinal cord but is now recognized as a vasculitis injuring the circulation to the anterior horn cells. Poliomyelitis is generally a non-progressive, specific site injury, although post-polio syndrome with demonstration of subcortical brain changes has challenged that belief. MS is a recurrent more fulminant physiological vascular injury. M.E. appears to be in this same family of diseases as paralytic polio and MS. M.E. is definitely less fulminant than MS but more generalized. M.E. is less fulminant but more generalized than poliomyelitis. This relationship of M.E.-like illness to poliomyelitis is not new and is of course the reason that Alexander Gilliam, in his analysis of the Los Angeles County General Hospital M.E. epidemic in 1934, called M.E. atypical poliomyelitis (2007, [Online]).

Dr Byron Hyde also writes, 'I have some M.E. patients with a circulating red blood cell volume less than 50% of expected and a very large number with the range of 60% to 70%. What this test means is that blood is pooling somewhere in the body and that this blood is probably not available for the brain. When blood flow to the heart decreases sufficiently, the organism has an increased risk of death. Accordingly, the human body operates in part with pressoreceptors that protect and maintain heart blood supply. When blood flow decreases, pressoreceptors decrease blood flow to noncardiac organs and shunt blood to the heart to maintain life. This, of course, robs those areas of the body that are not essential for maintaining life and means the brain, muscles, and peripheral circulation are placed in physiological difficulty.' This physiological difficulty is exacerbated by physical and mental activity and orthostatic stress.

Dr Byron Hyde goes on to say that, '**In MRI spectography of arm muscle of M.E. patients, it has been shown that because of an abnormal buildup of normal metabolites, the muscle cell actually shuts down to prevent cell death.**' Dr Hyde explains that this is what is happening to the true M.E. patient's cell physiology in the brain, and in muscle as a result of certain levels of physical and mental activity; there is 'cell field shutdown' to prevent the death of the cell (Hyde 2003, [Online]).

Dr Byron Hyde explains in The Nightingale Definition of M.E. that,

Possibly due to the fact that some Fibromyalgia patients can be improved by a gradual increase in exercise, or possibly due to the so called protestant ethic that all you have to do to get better is to take up your bed and walk, some physicians have extended the concept of passive or forceful increased exercise to Myalgic Encephalomyelitis patients. This is a common and potentially dangerous, even disastrous misconception. If the M.E. patient conforms to the guidelines set out in this definition, the insurance company can only make the patient worse by instituting progressive aggressive forced physical and intellectual activity. M.E. is a variable but always, serious diffuse brain injury and permanent damage can be done to the M.E. patient by non-judicious pseudo-treatment (2007, [Online]).

We also have ample evidence from other doctors who have a significant involvement with M.E. patients (although for various reasons they cannot be considered M.E. experts, as such), indicating that M.E. patients have an abnormal and negative response to exertion. This includes the following:

1. In April 2003, **Arnold Peckerman MD** from New Jersey reported findings to the annual meeting of the American Physiological Society that demonstrated via a sophisticated test that after exercise, the heart of those with M.E.\* pumped less blood than it did at rest. Peckerman is on record as saying that it is a 'progressive disease' and that, 'Basically we are talking about heart failure. A drop in [blood pumped by the heart] during exercise is not a typical response.'

This important research showed that, without exception, every disabled M.E. patient is in heart failure. The New Jersey team found evidence of the "Q" problem in M.E. "Q" stands for cardiac output in litres per minute. In M.E. patients, Q values correlated, with great precision, with the level of disability. Q was measured using impedance cardiography, a clinically validated and Government agency-recognised algorithm. (Impedance cardiography is not experimental.)

Normal people pump 7 litres of blood per minute through their heart, with very little variance, and when they stand up, that output drops to 5 litres per minute (a full 30% drop, but this is normal). Those two litres are rapidly pooled in the lower extremities and capacitance vessels. Normal people do not sense the 30% drop in cardiac output when they stand up because their blood pressure either stays normal or rises when they stand up, the body will defend blood pressure beyond anything else in order to keep the pulse going.

What the New Jersey team found in people with M.E. was astonishing – when these disabled patients stand up, they are on the edge of organ failure due to extremely low cardiac output as their Q drops to 3.7 litres per minute (a 50% drop from the normal of 7 litres per minute).

The disability level was exactly proportional to the severity of their Q defect, without exception and with scientific precision. In this Peckerman study, the data on the disabled M.E. patients reveals that even when they are lying down, their Q is only 5 litres per minute. The lower the Q, the more time the patient will spend lying down because lying down is the only time they come close to having sufficient cardiac output to survive (Peckerman et al. 2003, [Online]) (Hooper et al. 2007, [Online]) (Web M.D. 2009, [Online]).

**2. Dr Cheney** (following on from the Peckerman study) explained recently that because it takes more metabolic energy for the heart to relax and fill with blood than it does for it to squeeze and pump blood, the hearts of people with M.E. don't fill with the proper amount of blood before they pump which is what causes the reduced cardiac output and many of the symptoms of M.E. and much of the disability of M.E. (*The following summary of Cheney's work (most of which was made public only in the form of recorded lectures) is taken from the Corporate Collusion paper by Professor Malcolm Hooper et al.*)

Cheney comments that patients with M.E. suffer from cardiac problems since they cannot pump sufficient blood to the heart. He explains that the inability of very ill patients to stand up is the body protecting itself from cardiac stress and possible death. **Cheney explains that if patients draw down their lifestyle to live within the means of the reduced cardiac output, then progression into congestive cardiac failure (CCF) is slowed down**, but if things continue to progress, a point will be reached where there is no adequate cardiac output, and dyspnoea will develop, with ankle oedema and other signs of congestive cardiac failure. In order to stay relatively stable, it is essential for the patient not to create metabolic demand that the low cardiac output cannot match. Attempts to push beyond limits will cause injury or death.

Cheney also explains that M.E. patients have a high heart rate but a low cardiac output. In M.E. there is a cardiac dimension that is independent of (but not excluding) autonomic function or blood volume. A mismatch between metabolic demand and cardiac output, even very briefly, will kill. If the cardiac output goes down, in order not to die, there is a rise in noradrenergic tone (also involving the adrenal glands) to bring the output back up. This is a serious problem, because when the adrenals are exhausted, there will be low cardiac output. There is no such thing as an M.E. patient who is NOT hypothyroid: this has nothing to do with thyroid failure, but everything to do with matching metabolic demand and cardiac output.

Half of patients exhibit atrial cavitation, and when these patients stood up, the filling volume collapsed. M.E. patients "squeeze the hell" out of their left ventricle, resulting in a "whopping" 70% increase in left ventricular wall motion thickness. The reason why patients are squeezing so hard is because they do not have enough energy to fill the chambers of the heart properly so they are trying to compensate by squeezing a lot harder (ie. the way patients are compensating for this loss of cardiac output is by squeezing the left ventricle much harder). There are significant consequences of this. One consequence is that M.E. patients become asynchronised (ie. the heart can be filling and ejecting at the same time). If out of synchrony, the ventricle cannot cope, so cardiac output is severely degraded.

Cheney posits that when faced with a low Q, the body sacrifices tissue perfusion in order to maintain blood pressure: ie. microcirculation to the tissues of the body is sacrificed to maintain blood pressure so that the person does not die in the face of too low a cardiac output. This compensation is what is going on in the M.E. patient. Cheney states that it is important to note that the body does not sacrifice tissue perfusion equally across all organ systems: instead, it prioritises the order of sacrifice and one can observe the progression of M.E. in a patient by noting this prioritisation.

Two organ systems in particular have a protective mechanism (the Renin Angiotensin System, or RAS) against restricted tissue perfusion: the lung and the kidneys. These organs can sustain the greatest degree of Q problems because of this extra protection. Additionally, the heart and the brain also have this extra protection, even in the face of an extremely low Q. Therefore the lung, the brain, the kidneys and the heart are a bit more protected from a drop in Q than the liver, the gut, the muscles and the skin.

**a. The first to be affected is the skin:** if the microcirculation of the skin is compromised, several problems can arise. The body cannot thermoregulate anymore: the patient cannot stand heat or cold and if the core temperature rises, the patient will not be able to sleep and the immune system will be activated. In order to regulate that problem, the body will kick in thyroid regulation which will down-regulate in order to keep the body temperature from going too high. The patient then develops compensatory hypothyroidism, which means that now the patient will have trouble with feeling cold. Also, the body will not be able to eliminate VOCs (volatile organic compounds), which are shed in the skin's oil ducts, so VOCs build up in the body's fat stores and the patient becomes progressively chemically poisoned by whatever is present in the environment.

b. The second effect: the next microcirculation to be sacrificed is that to the muscles and the patient will have exercise intolerance. If things get still worse, the patient begins to experience pain in the muscles. If the microcirculation to the joints becomes compromised, the patient starts to have arthralgia linked to this circulatory defect.

c. The next system to be compromised is the liver and gut. One of the first things the patient may notice in this stage of disease progression is that there are fewer and fewer foods that can be tolerated, partly because microcirculation is necessary for proper digestion. Also the body will not secrete digestive juices so whatever food is tolerated will not be digested: if food cannot be digested, there will be peptides that are only partially digested and therefore are highly immune-reactive; they will leak out of the gut into the bloodstream, resulting in food allergies or sensitivities. The body will be unable to detoxify the gut ecology, so the gut will begin to poison the patient, who will feel as if poisoned, with diarrhoea, constipation, flatulence and other gut problems.

d. The fourth affected system is the brain: Cheney posits that there is a devastating effect in the brain as a result of liver / gut dysfunction, which can quickly toxify the brain, resulting in disturbances of memory and of processing speed. Also, the hypothalamus begins to destabilise the patient from the autonomic nervous system perspective. In all probability, the brain and heart suffer simultaneous compromise, but patients usually notice the brain being affected much earlier than the heart – this is because heart muscle cells have the greatest mitochondrial content of any tissue in the body, so when the mitochondria are impaired, the heart muscle has the greatest reserve. Even if the patient is sedentary with not too much demand on the heart, they can still think and make great demands on the brain, and energy is energy, whether it is being used physically or cognitively.

The fifth affected system is the heart: Cheney posits that the effect of compromised microcirculation upon the heart has an “a” part and a “b” part: part “a” is the manifestation of microcirculation impairment and part “b” is “the event horizon”.

Part “a”: manifestation of microcirculation impairment: the initial manifestation of microcirculatory impairment of the heart is arrhythmia with exercise intolerance: when the patient goes upstairs, more cardiac output is needed but the patient cannot sustain it. When there are even more severe microcirculatory problems, the patient starts to get chest pain as the myocardial cells die because they cannot get adequate oxygen.

Part “b”: the event horizon: (once this line is passed, there is no going back): Cheney’s view is that when the microcirculation defect within the heart itself begins to impact Q, a vicious circle begins – microcirculation impairment reduces the Q, which produces more microcirculation impairment, which produces even more Q problems, so down goes the patient into the next phase of cardiac failure, which involves the lungs.

The sixth affected system is the lung and kidney: this leads to congestive heart failure and pulmonary oedema, then the kidney is affected (the kidney is the last to go because it has the RAS back-up system). Combined with liver impairment, this stage is known as hepatorenal failure. A patient will know if s/he eventually loses the ability to compensate if, when they lie down, they are short of breath. Cheney’s view is that cardiac muscle has lost power because the mitochondria are dysfunctional (ie. there is an energy-production problem in the cells).

The red blood cells of patients with M.E. have been found to be deformed. When deformed, they cannot get through the capillary bed, causing pain. An indication of such deformity is a drop in the sedimentation rate (SED, or ESR) and Cheney (along with Dr Hyde and other M.E. experts) has observed that when measured in a laboratory, M.E. patients’ sedimentation rate is the lowest he has ever recorded, which confirms that M.E. patients have an induced haemoglobinopathy. Cheney has stated that the M.E. patients with the lowest sedimentation rate may have the greatest degree of pain. The more deformed the red blood cells, the more pain may be experienced. Some M.E. patients have a problem similar to that of sickle cell anaemia in this regard, and sickle cell patients have unbelievable pain. Cheney emphasises that it is bad enough when patients do not perfuse their muscles and joints (because of poor microcirculation) but it is even worse when red blood cells are so deformed that they can barely get through the capillaries or are blocked entirely. Cheney notes that in the Laboratory Textbook of Medicine, there are only three diseases that lower the sedimentation rate to that level: one is sickle cell anaemia (a genetic haemoglobinopathy); the second is M.E. (an acquired haemoglobinopathy) and the third is idiopathic cardiomyopathy. (The latter being one way in which the cardiac problems of M.E. are described.)

Cheney observes that in order to improve cardiac output, patients need to lie down, as this increases the cardiac output by 2 litres per minute. He notes that some patients need to lie down all the time to augment their blood volume in order to survive (Cheney 2006, [video recording]) (Peckerman et al. 2003, [Online]) (Hooper et al. 2007, [Online]).

Findings which showed mitochondrial dysfunction similar to mitochondrial encephalomyopathy also led Dr Cheney to comment, ‘The most important thing about exercise is not to have [patients with ME] do aerobic exercise. I believe that even progressive aerobic exercise is counter-productive. **If you have a defect in mitochondrial function and you push the mitochondria by exercise, you kill the DNA**’ (Williams 2004, [Online]).

- *Note that Dr Cheney cannot be said to be a M.E. expert, although he does deal primarily with M.E. patients and his comments on cardiac insufficiency can (and do) only relate to genuine M.E. patients as this finding is unique to M.E. patients. Unfortunately Cheney uses the terms ‘CFS’ and ‘CFIDS’ to refer to M.E. patients and, worse, unfortunately mixes in some medical and political facts about ‘CFS’ and ‘CFS’ patients (patients with diseases other than M.E.) into his 20 years of M.E. research. Thus not all of his work relates 100% to M.E. unfortunately. See: [Is Cheney talking about M.E. or ‘CFS’?](#) for more information.*
- *Dr Peckerman, like Cheney, has been involved in the study of the abnormalities unique to M.E. Unfortunately however he has used the terminology and definitions of ‘CFS’ and has included a vast amount of ‘CFS’ propaganda in his work. Thus while Dr Peckerman has some legitimate knowledge of the M.E. disease process, he cannot be considered a M.E. expert. Note also that both of these doctors do not use anything like the most severely affected M.E. patients in their research.*

As these comments clearly indicate, the adverse response to physical activity in M.E. patients is not ‘medically unexplained’ – research has found a number of sound medical reasons why M.E. sufferers are so physically disabled and limited, and unable to maintain an upright posture. These include; evidence of damage to the central nervous system (and autonomic and sympathetic nervous systems, causing a loss of normal internal homeostasis), damage to cardiac muscle (and many other cardiac and cardiovascular abnormalities including evidence of cardiac insufficiency), abnormalities and damage to muscle, immune system abnormalities, respiratory abnormalities and also a variety of abnormalities at a cellular level (eg. mitochondrial defects).

It is also worth noting that *none* of these abnormalities can be explained by so-called ‘deconditioning’ – the supposed reason for the recommendation of therapies such as GET.

- To read more articles, research and books by these authors (and others) which explain these abnormalities in more detail see: [Articles sorted by author](#) and [Myalgic Encephalomyelitis research and articles](#).
- For more information on why exercise programs are so dangerous for M.E. patients see also the medical overviews given in: [Profits Before Patients?.](#) [CRITICAL CONSIDERATIONS](#), [Science or Psychology?](#) and [Corporate Collusion](#) by Eileen Marshall and Margaret Williams and/or Professor Hooper.

Surveys of M.E. patients on the effects of GET illustrate the accuracy of these findings by experts only too well unfortunately:

- In 1998 a survey of over 3000 UK M.E. patients found that the single most harmful strategy was graded exercise therapy. 50% of respondents who had tried GET indicated that graded exercise had made their condition worse. This was the highest negative rating of any of the pharmacological, non-pharmacological and alternate approaches of management covered in the questionnaire. The most helpful strategies were: a) Pacing activity with rest: 90% b) Bed rest: 89% (Jones 1998, [Online]).
- In 2004 a survey of severely affected M.E. sufferers (conducted by the 25% M.E. Group) again found that graded exercise was by far the single most harmful treatment of any of the pharmacological, non-pharmacological and alternate approaches of management covered in the questionnaire. 95% of those that had tried GET said that graded exercise was ‘unhelpful’ while a shocking 82% reported that it had made their condition worse.’ A significant number of those surveyed indicated that they were not severely affected *before* GET (25% M.E. Group 2004, [Online]).

The way the bodies of people with M.E. react to exercise is abnormal in a number of different ways. These abnormalities are so pronounced that exercise tests are one of the series of tests which can be used to confirm a suspected M.E. diagnosis. Abnormalities found so far include the following:

Response to Exercise	Healthy People	M.E. Patients
Sense of well-being	Invigorating, anti-depressant effect	[Pain, exacerbation of many of all symptoms accompanied by overwhelming sensations of being intensely ill (see the description below)]
Resting heart rate	Normal	Elevated
Heart rate at maximum workload	Elevated	Reduced heart rate
Maximum oxygen uptake	Elevated	Only ½ that of sedentary controls

Age-predicted target heart rate	Can achieve it	Can NOT achieve it
Heart functioning	Increased	Sub-optimal level
Cerebral blood flow	Increased	Decreased
Cerebral Oxygen	Increased	Decreased
Body temperature	Increased	Decreased
Respiration	Increased	Breathing irregularities: shortness of breath, and irregular breathing
Cognitive processing	Normal, more alert	Impaired
Oxygen delivery to the muscles	Increased	Reduced
Gait Kinematics	Normal	Abnormalities
Recovery period	Short	[Days, weeks or months, or longer (or recovery may not occur and the relapse/damage may be semi-permanent or permanent. In a small percentage of cases, overexertion of the M.E. patient causes death)]

- This (modified) chart is taken from an article by M. van de Sande . See [Testing for M.E.](#) for more information about exercise testing in M.E.

If patients with M.E. exceed their individual physical, cognitive, orthostatic and other limits, they will experience some combination of the following:

- A mild-severe (acute or delayed) worsening of one or more symptoms for hours, days or longer afterward
- A mild-severe (acute or delayed) worsening of virtually every symptom for hours, days or longer afterward
- A severe (acute or delayed) worsening of the base level of illness/disability for hours/ weeks/ months or even years afterward, or
- A permanent worsening of the base level of illness/disability (i.e. permanent physical damage is caused and chances for significant recovery are adversely affected or lost entirely. Painstaking gains made slowly over many months or years may also be lost.)

It is also important to be aware that repeated or severe overexertion can also result in the death of the M.E. patient. (Death in M.E. is most often caused by heart failure or multiple organ failure.) (Bassett, 2009, [Online]).

The main characteristics of the pattern of symptom exacerbations, relapses and disease progression (and so on) in M.E. include:

- People with M.E. are unable to maintain their pre-illness activity levels. This is an acute (sudden) change. M.E. patients can only achieve 50%, or less, of their pre-illness activity levels post-M.E.
- People with M.E. are limited in how physically active they can be but they are also limited in similar way with; cognitive exertion, sensory input and orthostatic stress.
- When a person with M.E. is active beyond their individual (physical, cognitive, sensory or orthostatic) limits this causes a worsening of various neurological, cognitive, cardiac, cardiovascular, immunological, endocrinological, respiratory, hormonal, muscular, gastrointestinal and other symptoms.
- The level of physical activity, cognitive exertion, sensory input or orthostatic stress needed to cause a significant or severe worsening of symptoms varies from patient to patient, but is often trivial compared to a patient's pre-illness tolerances and abilities.
- The severity of M.E. waxes and wanes throughout the hour/day/week and month.
- The worsening of the illness caused by overexertion often does not peak until 24 - 72 hours (or more) later.
- The effects of overexertion can accumulate over longer periods of time and lead to disease progression, or death.
- The activity limits of M.E. are not short term: a gradual (or sudden) increase in activity levels beyond a patient's individual limits can only cause relapse, disease progression or death in patients with M.E.
- The symptoms of M.E. do not resolve with rest. The symptoms and disability of M.E. are not just caused by overexertion; there is also a base level of illness which can be quite severe even at rest.

- J. Repeated overexertion can harm the patient's chances for future improvement in M.E. M.E. patients who are able to avoid overexertion have repeatedly been shown to have the most positive long-term prognosis.
- K. Not every M.E. sufferer has 'safe' activity limits within which they will not exacerbate their illness; this is not the case for the very severely affected.
- For the full-length version of this text (which expands on each of these points) and for a full list of references for this text see: [The Ultra-comprehensive Myalgic Encephalomyelitis Symptom List](#). (An excerpt of this text is included in the Word/PDF file of this document.)

### **Can GET at least help some of those with mild M.E.?**

It is sometimes claimed that while exercise programs are not safe or appropriate for the severely affected, that mild or moderately affected M.E. sufferers can benefit from such interventions. But this assertion is NOT supported by the evidence. Some 'fatigue' sufferers have been shown to benefit from GET, but the results of these studies are no more relevant to mild M.E. sufferers than they are to severe M.E. sufferers; people with 'fatigue' do NOT have mild M.E. any more than they have mild multiple sclerosis, or mild cancer or any other illness. They are an entirely unrelated patient group. Thus graded exercise programs may help some fatigue sufferers but this is *irrelevant* to those who have M.E. Again, it has been shown that graded exercise programs are the actual reason many with M.E. are so severely affected ie. they were not severely affected before they were given advice to exercise or enrolled in formal GET programs. Thus GET should not be considered safe or useful for M.E. sufferers of any severity (25% M.E. Group 2004, [Online]).

Research has also proven that how much physical and cognitive overexertion a person can tolerate without serious damage depends on the severity of their illness. For example, we know that moderately affected patients can die from exercise sessions. For example, there is the case of the UK MP [Brynmor John](#) who had M.E. and was advised to 'exercise himself back to fitness' and who as a result of complying with this advice collapsed and died coming out of the House of Commons gym. Then there is the case of [Sophia Mirza](#), in the UK who died from M.E. after being forced into inappropriate and abusive psychiatric care. [Sophia](#) had severe M.E. and was of course not capable of any exercise. Nonetheless, she was inappropriately removed from her home and given inappropriate care. She was cruelly killed by being forced into what to most people would have been only very minor or trivial exertions.

For all of these reasons, it is vitally important that patients are allowed to judge *for themselves* how much activity it is safe and wise for them to attempt. Patients are the best judges of their own limits, and patients' judgements must not be over-ruled. Patients should never be advised, encouraged or forced to be more active than their severely damaged bodies can handle; these decisions cannot safely or ethically be made by any third party.

It is vital that M.E. patients avoid physical over-exertion and are never encouraged to exercise (or be mentally active, or cause orthostatic stress) beyond their individual limits particularly in the early and acute stages of the illness, but also at *any* stage of the illness as this can greatly damage a patients chances for future improvement or recovery. Graded exercise *cannot* improve authentic M.E.; disabled patients who improve with exercise do not qualify for a diagnosis of authentic M.E. There is nothing to be gained by people with M.E. pushing themselves beyond their individual physical limits; this can only result in unnecessary and sometimes very severe and prolonged relapses, disease progression, or even death (Ramsay 1986, [Online]) (Hyde 2003, [Online]) (Hyde 1992 p. xi) (Hyde & Jain 1992 pp. 38 - 43) (Dowsett 2001, [Online]).

- For more information on why exercise programs are so dangerous for M.E. patients see also the medical overviews given in: [Profits Before Patients?. CRITICAL CONSIDERATIONS, Science or Psychology?](#) and [Corporate Collusion](#) by Eileen Marshall and Margaret Williams and/or Professor Hooper.
- See [Testing for M.E.](#) for more information about the series of tests which can be used to confirm a suspected M.E. diagnosis. If you have M.E. see [Treating Myalgic Encephalomyelitis - The basics](#) and [Treating Myalgic Encephalomyelitis - avoiding overexertion](#) for more on the importance of avoiding over-exertion.

## **2. What is the effect of cognitive behavioural therapy (CBT) on Myalgic Encephalomyelitis (M.E.) patients?**

Compared to the physical devastation caused by GET, CBT would seem at first glance to be the softer option of the two; but this is not always the case. There are two different types of CBT that M.E. sufferers may be given and the effect on patients varies greatly depending on which type is used:

1. The first type of CBT respects that there is an organic illness present which is largely irreversible (and which cannot be improved by CBT), but aims to help a patient cope better with the limitations caused by their illness. This type of CBT is also given to patients with cancer and a wide array of other chronic illnesses (Carruthers et al. 2003, [Online]).

2. The second type of CBT is based on the premise that the patient's impairments are entirely due to 'wrong thinking' and that the pathophysiology of the illness is entirely reversible and perpetuated solely by a patient's 'false illness beliefs.' ie. 'Patients are sick only because they believe they are sick.' According to this theory of CBT, this therapy is potentially curative (Carruthers et al. 2003, [Online]).

Surveys of M.E. patients on the effects of cognitive behavioural therapy found:

- The (aforementioned) 1998 survey of over 3000 UK M.E. patients found that CBT was the least effective treatment of any of the pharmacological, non-pharmacological and alternate approaches of management covered in the questionnaire. Of those who had tried CBT, 55% indicated that the treatment had made no difference while 22% indicated that they had been made worse by CBT (Jones 1998, [Online]).
- The (aforementioned) 2004 survey of severely affected M.E. sufferers (conducted by the 25% M.E. Group) also found that cognitive behavioural therapy was one of the most unhelpful treatments for M.E. Fully 93% of those who had tried CBT said that it was unhelpful (the only treatment with a worse rating was GET) (25% M.E. Group 2004, [Online]).

**The hypothesis behind the first type of CBT** is reasonable. This type of CBT may do the vast majority of mild - moderately affected sufferers little harm (if also very little good), while a small percentage may find it useful in improving the way they cope with the illness emotionally. A significant percentage of patients will also be made worse by CBT. As with other chronic illnesses, the indications are that this type of CBT should be recommended or provided on a patient by patient basis only to those patients who have a specific need for such an intervention. CBT should not be considered essential for all – or even most – M.E. patients.

Even this type of CBT however (or any other), is not appropriate for any severely affected sufferer who is not physically able to cope with the physical and cognitive rigours of such a treatment ie. they cannot travel out of the house, speak or listen to speech for more than a few seconds or minutes etc. either without severe relapse or at all.

One of the main misconceptions is that while walking a few steps must of course require additional bodily resources and additional cardiac output, time spent thinking, looking, listening or experiencing other sensory stimuli does not. But this is not the case. Not only physical effort, but also cognitive effort, requires additional resources which an M.E. patient may not have. The brain contains some 100 billion neurons connected to some 10,000 relay stations and this enormous electrical activity creates a massive need for energy and other bodily resources. The brain uses up to 25% of the entire body's demand for glucose, 25% of the blood pumped from the heart goes to the brain and the brain also needs 25% of the body's oxygen supply. (Blood supplies nutrients like glucose, protein, trace elements, and oxygen to the brain.) So of course, every extra second of 'electrical activity' – every thought, every feeling, every noise heard or sight seen – requires additional cardiac output, makes additional oxygen and glucose demands, and so on, in just the same way as does a physical activity such as walking; if not more so. So in addition to physical activity, the list of things that can cause similar severe relapse in M.E. patients also includes cognitive exertion, sensory input and orthostatic stress. Anything that makes the body work harder or have to adjust in some way, in effect (Dowsett n.d. d, [Online]). (See: [Why patients with severe M.E. are housebound and bedbound](#) for more information.)

Any type of CBT will cause severe relapse in those who are too severely affected to safely participate. This relapse may last many weeks or months, or even be life-long or result in death. CBT can NOT be considered safe for all M.E. sufferers.

**The hypothesis behind the second type of CBT** however, is far from reasonable. Despite the large body of research which compellingly and conclusively disproves this hypothesis, the assumption of its truth by some has led to this treatment being forced on many M.E. sufferers particularly in the UK, The Netherlands and to a lesser extent, Australia. This unscientific and unethical form of CBT (which ignores the demonstrated biological pathology of the illness) seeks to disregard the patient's autonomy and experience of their illness. It tells them to ignore their symptoms. When, inevitably, this causes significant physical relapse, patients are told that this is entirely their own fault; that they must not be trying hard enough to get well and must still not be thinking 'correctly' about their illness. Patients are blamed entirely for their illness and accused of 'choosing' to remain unwell because they are supposedly 'enjoying the sick role' (Carruthers et al. 2003, [Online]).

CBT to convince a physically ill person that he/she does not have a physical disorder is disrespectful, inappropriate and cruel. It places an additional (and bogus) psychological burden on a person already suffering with severe physical illness, and can cause significant psychological harm.

M.E. expert [Dr. Elizabeth Dowsett](#) explains about CBT: 'Whereas any regime which can encourage patients with depression to discard or distract their damaging unrealistic morbid thoughts is helpful, patients with ME are usually capable of greater insight and understanding about their illness. Unfortunately, ME sufferers are too often

denied care in our society, so it is essential that they should remain as well informed as possible about treatment options and not ‘brainwashed’ into disbelieving their own symptoms’ (n.d.a. [Online]).

It is undoubtedly children with M.E. and their families who pay the highest price where CBT is involved however. Children with M.E. are not exempt from such ‘therapy’ and this is often far more detrimental to children as compared to adults. As M.E. authors [Verillo and Gellman](#) explain:

Misdiagnosing [M.E.] as school phobia, depression, or separation anxiety or chalking it up to family problems places the blame squarely on the shoulders of the child. When adults experience this kind of scepticism, they usually are able to defend themselves against the mistaken ideas of others. Children are unable to do so; they depend on adults for information, explanations, sympathy and advice. To throw disbelief in the face of a child who not only has all the symptoms of [M.E.] but is terribly frightened and in profound need of reassurance is not only cruel, it is detrimental to the child's future emotional growth (Verillo & Gellman 1997 p. 327).

The rate of clinical depression seen in M.E. is similar to, and not higher than, that seen in comparable illnesses such as rheumatoid arthritis. (Of course, depression is a common disease, and it does not make you immune from other diseases. So some patients with depression will also end up having other conditions as well, over time. This includes M.E., plus MS and Parkinson's and all other diseases.) Feelings of sadness and grief in M.E. are caused the loss of health, lifestyle, social role and financial means as well as the social stigma and severe abuse and neglect from friends and family and the medical profession that is so often an inescapable part of having M.E. (Stein 2005, [Online]).

Equally concerning is the fact that because it is harder to pin the blame for the illness on depression or anxiety with children, the parents are often blamed instead. The ‘family dynamic’ may be blamed for causing the child's illness and parents of these ill children have actually been charged with neglect or accused of actually making their children ill themselves (Munchausens by proxy). Some parents have lost custody and their children have been placed in foster care. Children have also been forcibly removed from the home and forced to undergo CBT and GET (and worse). All of this while the child continues to be seriously physically ill and not receive any sort of appropriate medical care (Hooper et al. 2001, [Online]).

Although a minority of M.E. patients will have a clinical depression, more often some patients are instead dealing with natural and expected levels of grief and sadness for what they have lost. If these feelings are present, they are not evidence of a psychiatric disease but simply is a normal and healthy reaction to an extremely distressing life experience and extreme levels of physical suffering. The only ‘treatment’ needed is an improvement in the severity of the condition, and in many cases probably also greater levels of appropriate medical, financial and/or social support. As one longtime M.E. sufferer explained, ‘The desperation one gets periodically from being so ill is not at all the same thing as ‘clinical depression’. Give me an even somewhat better day physically – and my mood improves quickly and dramatically!’

- For more information about forced exercise and other ‘treatments’ used on M.E. children and adults (which have in some cases resulted in death) see: [What is Myalgic Encephalomyelitis? Extra extended version](#)

This medically unsupportable and abusive form of CBT can undoubtedly cause significant psychological harm, but it is these additional associated burdens; physical relapse lasting months, years or longer, the risk of death through overexertion in some severely affected patients, the withholding of basic medical care, the removal of children from their parents and parents being falsely charged with making their children ill themselves (etc.) which combine to make this form of CBT so harmful. Thus the negative effects of CBT can sometimes be equally as devastating as those of GET, or in some cases, worse (for sufferers and their families).

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**Clearly, CBT and GET are at best useless and at worst extremely harmful for M.E. patients.** M.E. is not a short-term or ‘hit and run’ viral attack; it is not a self-limiting post-viral fatigue syndrome caused by mononucleosis/glandular fever, Q fever or hepatitis, or any other common infection. Nor is M.E. a psychological or behavioural condition, or a problem of mere ‘chronic fatigue’ or deconditioning. M.E. is also not medically unexplained, or the same thing as ‘CFS.’ M.E. cannot be improved through psychotherapy or graded exercise therapy. These theories have been comprehensively disproven many times over with regard to authentic M.E. patients.

Despite this, people with M.E. are routinely being recommended these treatments while also being assured that they are completely safe. These treatments are also not just being offered to M.E. patients solely on a voluntary basis; many have been treated as psychiatric patients against their will. (Or against the will of the parents of children with M.E., as described previously). It is also of great concern that many M.E. patients are ONLY offered ‘treatments’ such as CBT and GET – while access to even basic appropriate medical care is withheld. Enough

people with M.E. have had their long-term quality of life destroyed – or have been killed – by inappropriate use of these interventions.

If any drug caused even a very small percentage of the devastation GET causes in M.E. patients – let alone that it also had a zero percent chance of success – it would be immediately recalled. It would be an enormous worldwide scandal, and there would be some form of inquiry and serious criminal charges may well be laid. Yet the rate of people with M.E. recommended or even *forced* to exercise continues to rise, and with the full support of government, the mainstream medical community and the media.

This is despite the fact that legitimate research and evidence clearly shows that it has a ZERO percent chance of providing any benefit to people with authentic M.E. Patients with M.E. are regularly coerced or forced to undertake a huge level of risk, including significant risk of death or severe long-term disablement and permanent damage, **for zero chance of any gain**. All because of financial vested interests controlling science, and completely different mixed patients groups being used to determine the treatments appropriate for an entirely different and unrelated homogenous patient groups.

That this can be allowed to happen in such a supposedly enlightened day and age as ours defies belief: It amounts to legalised medical torture and horrific long-term abuse of some of our most vulnerable members of society.

People with M.E. must again be treated as is ethically and scientifically appropriate, and not merely in a way designed to suit certain political and financial considerations. What is happening today to people with Myalgic Encephalomyelitis is a gross violation of basic human rights. This has to stop, it has to BE stopped.

#### **For more information:**

- See the paper [Smoke and Mirrors](#) for information on why patients with M.E. are being treated based on theories motivated by financial and political considerations rather than the available medical evidence. This text forms the introduction to a 100 page + [CBT and GET database](#). The database contains excerpts and links to literally hundreds of articles and research studies which expose the lack of scientific legitimacy (and the hidden financial and political motivations) underlying the 'behavioural' paradigm of M.E. and the use of CBT and GET on M.E. patients – as well as a large number of patient accounts of CBT and GET.

To print or save a copy of this text (or the entire database) in a printer-friendly Word or PDF format, see the Downloads section. A shorter/condensed version of this text is also available: [The effects of CBT and GET – Condensed](#).

- See [What is Myalgic Encephalomyelitis? A historical, political and medical overview](#) for more information on all aspects of M.E.
- For whose benefit was 'Chronic Fatigue Syndrome' created, and for whose benefit is it so heavily promoted despite its utter lack of scientific credibility? Who benefits from Myalgic Encephalomyelitis and 'CFS' being mixed together through unscientific concepts such as 'CFS/ME' and 'ME/CFS' and Myalgic 'Encephalopathy'? Who benefits from the facts of M.E. remaining ignored, obscured and hidden in plain sight? See: [Who benefits from 'CFS' and 'ME/CFS'?](#)
- To learn more about the extreme limits imposed on M.E. patients see: [Why patients with severe M.E. are housebound and bedbound](#)
- The terminology is often used interchangeably, incorrectly and confusingly. However, the DEFINITIONS of M.E. and CFS are very different and distinct, and it is the definitions of each of these terms which is of primary importance. *The distinction must be made between terminology and definitions.* For more information see: [Who benefits from 'CFS' and 'ME/CFS'?](#), [The Terminology Explained](#) and [What is Myalgic Encephalomyelitis?](#) and [Problems with the so-called "Fair name" campaign: Why it is in the best interests of all patient groups involved to reject and strongly oppose this misleading and counter-productive proposal to rename 'CFS' as 'ME/CFS' and Problems with the use of 'ME/CFS' by M.E. advocates, plus The misdiagnosis of CFS, Why the disease category of 'CFS' must be abandoned.](#) In short:
  1. **Chronic Fatigue Syndrome** is an artificial construct created in the US in 1988 for the benefit of various political and financial vested interest groups. It is a mere diagnosis of exclusion (or wastebasket diagnosis) based on the presence of gradual or acute onset fatigue lasting 6 months. If tests show serious abnormalities, a person no longer qualifies for the diagnosis, as 'CFS' is 'medically unexplained.' A diagnosis of 'CFS' does not mean that a person has any distinct disease (including M.E.). The patient population diagnosed with 'CFS' is made up of people with a vast array of unrelated illnesses, or with no detectable illness. According to the latest CDC estimates, 2.54% of the population qualify for a 'CFS' (mis)diagnosis. Every diagnosis of 'CFS' can only ever be a misdiagnosis.

2. **Myalgic Encephalomyelitis** is a systemic neurological disease initiated by a viral infection. M.E. is characterised by (scientifically measurable) damage to the brain, and particularly to the brain stem which results in dysfunctions and damage to almost all vital bodily systems and a loss of normal internal homeostasis. Substantial evidence indicates that M.E. is caused by an enterovirus. The onset of M.E. is always acute and M.E. can be diagnosed within just a few weeks. M.E. is an easily recognisable distinct organic neurological disease which can be verified by objective testing. If all tests are normal, then a diagnosis of M.E. cannot be correct.

M.E. can occur in both epidemic and sporadic forms and can be extremely disabling, or sometimes fatal. M.E. is a chronic/lifelong disease that has existed for centuries. It shares similarities with MS, Lupus and Polio. There are more than 60 different neurological, cognitive, cardiac, metabolic, immunological, and other M.E. symptoms. Fatigue is not a defining nor even essential symptom of M.E. People with M.E. would give anything to be only severely 'fatigued' instead of having M.E. Far fewer than 0.5% of the population has the distinct neurological disease known since 1956 as Myalgic Encephalomyelitis.

- See also: [Problems with 'our' M.E. \(or 'CFS' 'CFIDS' or 'ME/CFS' etc.\) advocacy groups](#) (also available in an animated video format) and the new paper: [M.E. vs MS: Similarities and differences](#)
- For more information on scams aimed at M.E. patients (similar to CBT) such as the Lightning process, Reverse therapy, Mickel therapy, EFT and so on, see [Comments on the 'Lightning Process' \(etc.\) scam page](#).
- To read a list of all the articles on this site suitable for different groups such as M.E. patients, carers, friends and family, the 'CFS' misdiagnosed, doctors or severe M.E. patients and so on, see the [Information Guides page](#).

#### **Additional notes on this text:**

- **A note about antidepressant drugs and M.E.:** Along with CBT and GET, antidepressants are another treatment also commonly recommended to M.E. patients based on evidence involving non-M.E. patient groups and produced by vested interest groups. M.E. patients are commonly recommended or verbally forced to take these drugs on what amounts to a random basis medically. As with CBT and GET, patients are almost always incorrectly told that these drugs are a safe and effective treatment for M.E. So what effect do these drugs have on Myalgic Encephalomyelitis patients?

As with CBT and GET, they cannot improve the core problems of M.E. and can also very commonly cause serious adverse reactions. The number of M.E. patients that cannot tolerate these drugs, and for whom these drugs cause a worsening of the condition (including serious cardiac events) is very high. This is explained in more detail in the new paper: [The effects of antidepressants on Myalgic Encephalomyelitis patients](#). (This paper is due to be completed late 2009)

#### **What can you do to help?**

People with Myalgic Encephalomyelitis have only a tiny minority of the medical, scientific, legal and other potentially supporting professions – or the public – on their side. What is needed is people from all over the world to stand up for Myalgic Encephalomyelitis – whether they are affected yet by M.E. or not. That is the only way change will occur, through education and people simply refusing to accept what is happening any more. This appalling abuse and neglect of so many severely ill people on such an industrial scale is truly *inhuman* and has already gone on for far too long. People with M.E. desperately need your help. See [What is Myalgic Encephalomyelitis?](#) and the [Information Guides page](#)

#### **References**

All of the information concerning Myalgic Encephalomyelitis on this website is fully referenced and has been compiled using the highest quality resources available, produced by the world's leading M.E. experts.

More experienced and more knowledgeable M.E. experts than these – [Dr Byron Hyde](#) and [Dr. Elizabeth Dowsett](#) in particular – do not exist. Between Dr Byron Hyde and Dr. Elizabeth Dowsett, and their mentors the late Dr John Richardson and Dr Melvin Ramsay (respectively), these four doctors have been involved with M.E. research and M.E. patients for well over 100 years collectively, from the 1950s to the present day. Between them they have examined more than 15 000 individual (sporadic and epidemic) M.E. patients, as well as each authoring numerous studies and articles on M.E., and books (or chapters in books) on M.E. Again, more experienced, more knowledgeable and more credible M.E. experts than these simply do not exist.

This paper is merely intended to provide a brief summary of some of the most important facts of M.E. It has been created for the benefit of those people without the time, inclination or ability to read each of these far more

detailed and lengthy references created by the world's leading M.E. experts. The original documents used to create this paper are essential additional reading however for any physician (or anyone else) with a real interest in Myalgic Encephalomyelitis. For more information see the [References](#) page.

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This paper will be continue to be updated regularly (at least annually). Please check back at the website periodically to make sure that you have the most up-to-date version of this paper available.

'People in positions of power are misusing that power against sick people and are using it to further their own vested interests. No-one in authority is listening, at least not until they themselves or their own family join the ranks of the persecuted, when they too come up against a wall of utter indifference.'

Professor Malcolm Hooper 2003

'Do not for one minute believe that CFS is simply another name for Myalgic Encephalomyelitis (M.E.). It is not. The CDC definition is not a disease process. It is (a) a partial mix of infectious mononucleosis /glandular fever, (b) a mix of some of the least important aspects of M.E. and (c) what amounts to a possibly unintended psychiatric slant to an epidemic and endemic disease process of major importance' Dr Byron Hyde 2006

'The term myalgic encephalomyelitis (means muscle pain, my-algic, with inflammation of the brain and spinal cord, encephalo-myel-itis, brain spinal cord inflammation) was first coined by Ramsay and Richardson and has been included by the World Health Organisation (WHO) in their International Classification of Diseases (ICD), since 1969. It cannot be emphasised too strongly that this recognition emerged from meticulous clinical observation and examination.'

Professor Malcolm Hooper 2006

'M.E. is a systemic disease (initiated by a virus infection) with multi system involvement characterised by central nervous system dysfunction which causes a breakdown in bodily homeostasis. It has an UNIQUE Neuro-hormonal profile.'

Dr Elizabeth Dowsett

'Deaths are not anecdotal and are a matter of public record. Patients with Myalgic Encephalomyelitis are dying and [also] developing complications (known to be fatal) such as heart disease and cancer at considerably younger ages than the statistical norm. This is significant and needs to be looked into.'

Jill McLaughlin

'M.E. appears to be in this same family of diseases as paralytic polio and MS. M.E. is less fulminant than MS but more generalized. M.E. is less fulminant but more generalized than poliomyelitis. This relationship of M.E.-like illness to poliomyelitis is not new and is of course the reason that Alexander Gilliam, in his analysis of the Los Angeles County General Hospital M.E. epidemic in 1934, called M.E. atypical poliomyelitis.'

Dr Byron Hyde

'The degree of physical incapacity varies greatly, but the [level of severity] is directly related to the length of time the patient persists in physical effort after its onset; put in another way, those patients who are given a period of enforced rest from the onset have the best prognosis.'

Dr Melvin Ramsay on Myalgic Encephalomyelitis

'The vested interests of the Insurance companies and their advisers must be totally removed from all aspects of benefit assessments. There must be a proper recognition that these subverted processes have worked greatly to the disadvantage of people suffering from a major organic illness that requires essential support of which the easiest to provide is financial. The poverty and isolation to which many people have been reduced by ME is a scandal and obscenity.'

Professor Malcolm Hooper 2006

'What all this amounts to is that we have lost any semblance indeed any pretence of pursuing scientific inquiry (into) what is true. This is almost classic in its near-phobic avoidance of considering anything that could possibly be construed as speaking the truth.'

Margaret Williams on Myalgic Encephalomyelitis

‘Never in the field of human illness have so many been betrayed by so few’  
RiME Sept. 2007

‘Thirty years ago when a patient presented to a hospital clinic with unexplained fatigue, any medical school physician would search for an occult malignancy, cardiac or other organ disease, or chronic infection. The concept that there is an entity called chronic fatigue syndrome has totally altered that essential medical guideline. Patients are now being diagnosed with CFS as though it were a disease. It is not. It is a patchwork of symptoms that could mean anything.’  
Dr Byron Hyde 2003

Since Professor Cheney has shown that in M.E. patients, cardiac output struggles to meet metabolic demand, how can forced aerobic exercise help such patients remain as functional as possible? In the light of the Peckerman et al paper that was published in 2003, are the psychiatrists and their peer reviewers at the MRC who approved the PACE trial protocol still convinced that these trials (and the exercise regimes to be meted out by the new Centres) pose no harm for those with M.E.? Perhaps they are content to rely on the certainty that they themselves can never be held accountable for any harm to any patient because all participants must sign a compulsory waiver which means that no participant can ever pursue any claim for medical negligence or damages?  
Professor Hooper 2007

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*The HUMMINGBIRDS' FOUNDATION for M.E. (HFME)*

*Fighting for the recognition of Myalgic Encephalomyelitis based on the available scientific evidence, and for patients worldwide to be treated appropriately and accorded the same basic human rights as those with similar disabling and potentially fatal neurological diseases such as Multiple Sclerosis.*

myalgic

## A one-page summary of the facts of M.E.

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Taken from [www.hfme.org](http://www.hfme.org)



- Myalgic Encephalomyelitis is a disabling neurological disease that is very similar to multiple sclerosis (M.S.) and poliomyelitis (polio). Earlier names for M.E. were 'atypical multiple sclerosis' and 'atypical polio.'
- Myalgic Encephalomyelitis is a neurological disease characterised by scientifically measurable post-encephalitic damage to the brain stem. This is always damaged in M.E., hence the name M.E. The term M.E. was coined in 1956 and means: My = muscle, Algic = pain, Encephalo = brain, Mye = spinal cord, Itis = inflammation. This neurological damage has been confirmed in autopsies of M.E. patients.
- Myalgic Encephalomyelitis has been recognised by the World Health Organisation's International Classification of Diseases since 1969 as a distinct organic neurological disease with the ICD code G.93.3.
- Myalgic Encephalomyelitis is primarily neurological, but also involves cognitive, cardiac, cardiovascular, immunological, endocrinological, metabolic, respiratory, hormonal, gastrointestinal and musculo-skeletal dysfunctions and damage. M.E. affects all vital bodily systems and causes an inability to maintain bodily homeostasis. More than 64 individual symptoms of M.E. have been scientifically documented.
- Myalgic Encephalomyelitis is an acute (sudden) onset, infectious neurological disease caused by a virus (a virus with a 4-7 day incubation period). M.E. occurs in epidemics as well as sporadically and over 60 M.E. outbreaks have been recorded worldwide since 1934. There is ample evidence that M.E. is caused by the same type of virus that causes polio; an enterovirus.
- Myalgic Encephalomyelitis can be more disabling than MS or polio, and many other serious diseases. M.E. is one of the most disabling diseases there is. More than 30% of M.E. patients are housebound, wheelchair-reliant and/or bedbound and are severely limited with even basic movement and communication.
- *Why are Myalgic Encephalomyelitis patients so severely and uniquely disabled?* For a person to stay alive, the heart must pump a certain base-level amount of blood. Every time a person is active, this increases the amount of blood the heart needs to pump. Every movement made or second spent upright, every word spoken, every thought thought, every word read or noise heard requires that more blood must be pumped by the heart. However, the hearts of M.E. patients only pump barely enough blood for them to stay alive. Their circulating blood volume is reduced by up to 50%. Thus M.E. patients are severely limited in physical, cognitive and orthostatic (being upright) exertion and sensory input. This problem of reduced circulating blood volume, leading to cardiac insufficiency, is why every brief period spent walking or sitting, every conversation and every exposure to light or noise can affect M.E. patients so profoundly. Seemingly minor 'activities' can cause significantly increased symptom severity and/or disability (often with a 48-72 hour delay in onset), prolonged relapse lasting months, years or longer, permanent bodily damage (eg. heart damage or organ failure), disease progression or death. If activity levels exceed cardiac output by even 1%, death occurs. Thus the activity levels of M.E. patients must remain strictly within the limits of their reduced cardiac output just in order for them to stay alive. *M.E. patients who are able to rest appropriately and avoid severe or prolonged overexertion have repeatedly been shown to have the most positive long-term prognosis.*
- Myalgic Encephalomyelitis is a testable and scientifically measurable disease with several unique features that is not difficult to diagnose (within just a few weeks of onset) using a series of objective tests (eg. MRI and SPECT brain scans). Abnormalities are also visible on physical exam in M.E.
- Myalgic Encephalomyelitis is a long-term/lifelong neurological disease that affects more than a million adults and children worldwide. In some cases M.E. is fatal. (Causes of death in M.E. include heart failure.)

For more information, and to read a fully-referenced version of this text compiled using information from the world's leading M.E. experts, please see: What is M.E.? Extra extended version. Permission is given for this unedited document to be freely redistributed. Please redistribute this text widely.

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myalgic

## The ultra-comprehensive M.E. symptom list

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An excerpt:

### **Section 3: ON THE PATTERN/CAUSE OF SYMPTOM EXACERBATIONS, RELAPSES, AND DISEASE PROGRESSION IN MYALGIC ENCEPHALOMYELITIS**

What characterises M.E. every bit as much as the individual neurological, cognitive, cardiac, cardiovascular, immunological, endocrinological, respiratory, hormonal, muscular, gastrointestinal and other symptoms is the way in which people with M.E. respond to physical and cognitive activity, sensory input and orthostatic stress, and so on. In other words, the pattern of symptom exacerbations, relapses and of disease progression.

The way the bodies of people with M.E. react to these activities/stimuli post-illness is unique in a number of ways. Along with a specific type of damage to the brain (the central nervous system) this characteristic is one of the defining features of the illness which must be present for a correct diagnosis of M.E. to be made. The main characteristics of the pattern of symptom exacerbations, relapses and disease progression (and so on) in Myalgic Encephalomyelitis include:

- A. People with M.E. are unable to maintain their pre-illness activity levels. This is an acute (sudden) change. M.E. patients can only achieve 50%, or less, of their pre-illness activity levels post-M.E.
- B. People with M.E. are limited in how physically active they can be but they are also limited in similar way with; cognitive exertion, sensory input and orthostatic stress.
- C. When a person with M.E. is active beyond their individual (physical, cognitive, sensory or orthostatic) limits this causes a worsening of various neurological, cognitive, cardiac, cardiovascular, immunological, endocrinological, respiratory, hormonal, muscular, gastrointestinal and other symptoms.
- D. The level of physical activity, cognitive exertion, sensory input or orthostatic stress needed to cause a significant or severe worsening of symptoms varies from patient to patient, but is often trivial compared to a patient's pre-illness tolerances and abilities.
- E. The severity of M.E. waxes and wanes throughout the hour/day/week and month.
- F. The worsening of the illness caused by overexertion often does not peak until 24 - 72 hours (or more) later.
- G. The effects of overexertion can accumulate over longer periods of time and lead to disease progression, or death.
- H. The activity limits of M.E. are not short term: a gradual (or sudden) increase in activity levels beyond a patient's individual limits can only cause relapse, disease progression or death in patients with M.E.
- I. The symptoms of M.E. do not resolve with rest. The symptoms and disability of M.E. are not just caused by overexertion; there is also a base level of illness which can be quite severe even at rest.
- J. Repeated overexertion can harm the patient's chances for future improvement in M.E. M.E. patients who are able to avoid overexertion have repeatedly been shown to have the most positive long-term prognosis.
- K. Not every M.E. sufferer has 'safe' activity limits within which they will not exacerbate their illness; this is not the case for the very severely affected.

#### **A. People with M.E. are unable to maintain their pre-illness activity levels. This is an acute (sudden) change. M.E. patients can only achieve 50%, or less, of their pre-illness activity levels post-M.E.**

Only being able to achieve 50% or less of your pre-illness activity level immediately upon becoming ill is very common – if not universal – in Myalgic Encephalomyelitis. (Although a small percentage of sufferers may possibly be somewhat less severely affected at onset.) This is not a gradual change in ability levels which occurs over weeks, months or years; it is an acute change. The onset of M.E. is frequently very dramatic, M.E. patients can very often tell you not just the day that they became ill, but the exact *hour* they became ill.

- M.E. can commonly be diagnosed within just a few weeks if the doctor has experience with M.E. (If all tests are normal, M.E cannot be the correct diagnosis.) See: [Testing for M.E.](#) for more information. For more information on

the viral infection evident at onset in people with M.E., and the outbreaks of M.E. etc. see: [The outbreaks \(and infectious nature\) of M.E.](#)

- M.E. is an acute onset illness, however it should be noted that: (a) some sufferers will be unsure of their onset type (they may not recall it, or may not recall it accurately, for various reasons) and (b) in some cases, acute onset M.E. is preceded by a series of unrelated minor infectious episodes (in a previously well patient) which may be misinterpreted as being a gradual onset of the M.E. (These minor infectious episodes may be due to the immune system being under temporary or chronic stress from events such as; recent immunisation, repetitive contact with a large number of infectious persons, or the effect of travel; as in exposure to a new subset of virulent infections. This pre-existing temporary or chronic immune system weakness is not seen in all patients and is *not* what causes M.E., although a compromised immune system will of course make the body more vulnerable to all types of infections, including M.E.)

**B. People with M.E. are limited in how physically active they can be but they are also limited in similar way with; cognitive exertion, sensory input and orthostatic stress.**

The bodies of people with Myalgic Encephalomyelitis respond inappropriately to anything that forces the body to have to react in some way or work harder in some way, in order to maintain internal homeostasis, including (but not limited to): physical activity, cognitive exertion (including emotional stress), sensory input and orthostatic stress. It should also not be assumed that a person with M.E. will necessarily react more severely to (or have greater limits on) physical activity than with cognitive exertion, sensory input or orthostatic stress. Some patients find that their most severe relapses come from orthostatic stress, while others will have to be more careful with their levels of sensory input or cognitive exertion as compared to physical activity. Other patients may be equally limited with each of these activities or stimuli, and so on. It varies from patient to patient and can also change over the course of the illness.

One of the main misconceptions about M.E. is that while walking a few steps must of course require additional bodily resources and additional cardiac output, time spent thinking, looking, listening or experiencing other sensory stimuli does not. But this is not the case. Not only physical effort, but also cognitive effort, requires additional resources which an M.E. patient may not have. The brain contains some 100 billion neurons connected to some 10,000 relay stations and this enormous electrical activity creates a massive need for energy and other bodily resources. The brain uses up to 25% of the entire body's demand for glucose, 25% of the blood pumped from the heart goes to the brain and the brain also needs 25% of the body's oxygen supply. (Blood supplies nutrients like glucose, protein, trace elements, and oxygen to the brain.) So of course, every extra second of 'electrical activity' – every thought, every feeling, every noise heard or sight seen – requires additional cardiac output, makes additional oxygen and glucose demands, and so on, in just the same way as does a physical activity such as walking; if not more so.

- *What is Homeostasis?* Homeostasis is the ability of a living organism to regulate its internal environment to maintain a stable, constant condition, by means of multiple dynamic equilibrium adjustments, controlled by interrelated self-regulation mechanisms. Homeostasis is one of the fundamental characteristics of living things. It is the maintenance of the internal environment within tolerable limits. M.E. causes a loss of the ability of the CNS (the brain) to adequately receive, interpret, store and recover information which would enable it to control vital body functions. There is a loss of normal internal homeostasis; the individual can no longer function systemically within normal limits.

Metabolic problems at a cellular level also contribute to this inability to maintain homeostasis in M.E. M.E. expert [Dr Byron Hyde](#) explains, 'In MRI spectography of arm muscle of M.E. patients, it has been shown that because of an abnormal build-up of normal metabolites, the muscle cell actually shuts down to prevent cell death.' This is what is happening to the M.E. patient's cell physiology in every muscle (including the heart) and in the brain as a result of physical and cognitive activity and/or overexertion; there is 'cell field shutdown' to prevent the death of the cell. See: [Treating Myalgic Encephalomyelitis - Avoiding Overexertion](#) for more information and for references.

- Physical activity in this context does not just mean aerobic exercise; it includes any physical movement or activity, including stretching and even very small movements. Cognitive activity refers to any type of thinking, or mental processing. Sensory input includes exposure to light, noise and movement etc. Orthostatic stress or postural stress includes sitting or standing, but also things like having a few pillows under your head when lying down or sitting up in bed; orthostatic stress is caused by any posture other than lying down flat (perhaps with legs raised to reduce the load on the heart; unless the patient is wearing pressure stockings, which achieve the same goal.).

**C. When a person with M.E. is active beyond their individual (physical, cognitive, sensory or orthostatic) limits this causes a worsening of various neurological, cognitive, cardiac, cardiovascular, immunological, endocrinological, respiratory, hormonal, muscular, gastrointestinal and other symptoms.**

When a person with M.E. is active beyond their individual post-illness limits, the result is not tiredness, fatigue or even exhaustion – nor is 'malaise' an accurate word to describe what occurs. There simply is no one symptom

caused by overexertion in M.E. What does happen is that there is a worsening of all sorts of different symptoms and of the severity of the illness generally with overexertion. (Repeated or severe overexertion can also cause disease progression, permanent damage (eg. to the heart), or death in M.E.) It is an entirely different problem of a much greater magnitude.

Overexertion causes an exacerbation of all sorts of combinations of neurological, cognitive, cardiac, cardiovascular, immunological, endocrinological, respiratory, hormonal, muscular, gastrointestinal and other symptoms which can be mild, moderate, severe, or even life threatening (eg. seizures and cardiac events). Many of the symptoms involved are present at a lower level at rest, but overexertion causes them to worsen. (Although some patients may also have some symptoms that only appear after overexertion.)

The types of symptoms produced in response to certain levels of physical activity, cognitive activity, sensory stimuli or orthostatic stress may or may not vary depending on the type (and severity) of the activity or stimuli involved. But very often the types of symptoms worsened or produced by overexertion are fairly similar regardless of which exertion or input was involved. Overexertion can sometimes cause just one or two symptoms to worsen (eg. cardiac problems) but often a large cluster of symptoms are worsened. The cluster of symptoms made worse by excessive exertion or stimulus is often very similar from patient to patient, as generally it is a worsening of the most common symptoms of the illness. Patients commonly experience a combination of the following symptoms:

Profound cognitive dysfunctions (and various other neurological disturbances), muscle weakness (or paralysis), burning eye pain or burning skin, subnormal temperature or low-grade fever, sore throat or painful lymph nodes (and/or other signs of inappropriate immune system activation), faintness, weakness or vertigo, loss of co-ordination, dyspnea, an explosion of sensory phenomena (low level seizure activity), cardiac and/or blood pressure disturbances, facial pallor and/or a slack facial expression, widespread severe pain, nausea or feeling as if 'poisoned,' feeling cold and shivering one minute and hot and sweating the next, anxiety or even terror (as an organic part of the attack itself rather than as a reaction to it) and hypoglycaemia. Often the patient will feel an urgent need to retreat from all homeostatic pressures. The types of symptoms triggered vary widely from patient to patient, but some combination of these is common. There may also be an accompanying exacerbation of other symptoms. These symptoms often combine to create an indescribable and overwhelming experience of terrible illness that is unique to M.E, and can be profoundly incapacitating. At its most severe, the patient feels as if they are about to die.

Each of the symptoms caused or exacerbated by overexertion can be clearly articulated without difficulty whether they be; seizures, cardiac events, labile blood pressure, tachycardia, shortness of breath, muscle pain, muscle weakness or muscle paralysis, facial paralysis, black outs, flu-like symptoms, nausea, inability to speak or to understand speech, problems with memory, and so on. It makes no scientific or logical sense to subsume these very specific symptoms, and very specific and varied combinations of symptoms, under a vague and inaccurate label of mere 'fatigue.' To say that all of these very different and very specific – and in some cases very serious – symptoms can be accurately summarised as being a problem of mere 'fatigue,' 'malaise' or 'exhaustion' is absurd.

- A large number of illnesses cause significant fatigue or malaise after activity (for example post-mononucleosis or glandular fever fatigue syndromes, Lyme disease and Fibromyalgia and so on) but what is happening in M.E. is simply not the same; the symptomatology and pathology – and the effect of physical, cognitive and orthostatic overexertion on long-term prognosis – is very different in M.E.
- *Also note that:* repeated or severe overexertion can also cause disease progression, permanent damage (eg. to the heart), or death in M.E. patients. Again, to suggest that these very serious and long-term effects – and fatalities – could be accurately summarised as being a problem of mere 'fatigue' is clearly absurd
- *An additional note on 'fatigue':* The diagnosis of M.E. is determined upon the presence of certain neurological, cognitive, cardiac, cardiovascular, immunological, muscular, gastrointestinal and other symptoms and characteristics (and so on) – the presence or absence of mere 'fatigue' is irrelevant. In addition to these other (far more serious) symptoms, some M.E. sufferers may also suffer with mild, moderate or severe fatigue some of the time, while others will not. Thus the symptom of fatigue is not an essential symptom of M.E. and does not define M.E. (Although the symptom of fatigue *is* essential to qualify for a misdiagnosis of 'CFS'). For more information see: [M.E. is not defined by 'fatigue'](#) and [The misdiagnosis of CFS](#). The point to be most aware of is not that M.E. is 'more than fatigue' – but that M.E. *ISN'T FATIGUE AT ALL*.

**D. The level of physical activity, cognitive exertion, sensory input or orthostatic stress needed to cause a significant or severe worsening of symptoms varies from patient to patient, but is often trivial compared to a patient's pre-illness tolerances and abilities.**

When there is talk of 'overexertion' leading to an exacerbation of symptoms in M.E. what is being referred to is not hard exercise, it is not anything resembling what healthy people would recognise as 'overexertion.' This term just refers to any activity which goes beyond a person's individual post-M.E. limits.

There is a lot of variation from patient to patient but very often the levels of activity required to cause relapse are trivial compared to a patient's pre-illness tolerances and abilities. For example, what constitutes overexertion for someone with severe M.E. could be something as small as rolling over in bed, walking or talking for a few minutes, or eating a meal. The severity and duration of relapses varies depending on the severity of a person's illness, but relapses in M.E. are very often way out of all proportion to the actual activity. Relapses can be very severe and prolonged (or even permanent) even if a person with M.E. has only gone past their individual limits in a seemingly minor way.

- *A note on M.E. and other illnesses:* This extreme and out of all proportion reaction to even trivial levels of activity is just not seen in those illnesses causing fatigue (and other symptoms) after exertion which may commonly be misdiagnosed as 'CFS.' People with post-viral fatigue syndromes, Fibromyalgia and Lyme disease etc. are not affected by small activities for many weeks, months, or permanently, in this way. While people with M.E. and people with these other illnesses may all not improve with a graded exercise regime, the way people with M.E. respond to physical and cognitive activity, sensory input and orthostatic stress is profoundly different than in these other illnesses. The two problems are quite distinct.

### **E. The severity of M.E. waxes and wanes throughout the hour/day/week and month.**

One can probably observe people with some illnesses carefully for an hour or so and collect a lot of good information about what they can and can't do, how severe their illness is, and what their usual symptoms are from day to day, and so on. However M.E. is not one of those illnesses. M.E. is *not* a stable illness.

Observing the average M.E. sufferer for an hour – or even a week or more – will not give an accurate indication of their usual activity level because the severity of M.E. can wax and wane throughout the month, week, day and even hour. Also, people with M.E. can sometimes operate significantly above their actual illness level for short periods of time thanks to surges of adrenaline – albeit at the cost of severe and prolonged worsening of the illness afterward. Relapses and worsening of symptoms are also very often also significantly delayed (there may be both an acute AND a delayed reaction).

Just observing someone with M.E. do a certain task should not be taken to mean (a) that they can necessarily repeat the task anytime soon, (b) that they would have been able to do it at any other time of day, (c) that they can do the same task every hour, day or even every week, or month, or (d) that they won't be made very ill afterwards for a considerable period because they had to really push themselves (and make themselves ill) to do the task. Often a considerable rest period is needed before and after a task, which may be hours, days, weeks or months long. For example, someone may need 2 weeks rest before an outing, for example, and may then spend 3 weeks extremely ill afterwards recovering from it. Just observing them in the 2 hours they were 'out and about and mobile' is of course not at all representative of their usual ability levels.)

Most importantly, because the worsening of the illness caused by overexertion may not even begin until 48 or more hours afterwards (when most observers are long gone), it's impossible to tell by seeing an M.E. patient engaged in an activity, whether that activity is so far beyond the patient's limits that it will end up causing a severe or even permanent worsening of the illness (or 'relapse'). To be blunt, the activity may even end up killing the patient. This isn't common (the death rate is estimated at 3%), but deaths can and do occur. Thus, observers who see an M.E. patient engaged in an activity have no idea what the consequences of this activity may be.

- What is an adrenaline surge? Adrenaline is often referred to as the 'fight or flight' hormone as it kicks into action in situations of potential danger. However, adrenaline also kicks in when the body is in physiological difficulty, which is very often what is happening to severe M.E. sufferers. Adrenaline surges make the heart pump faster and raise the blood pressure, forcing blood around the body with greater force to supply the muscles with more oxygen, so that they can make a greater effort. Surges of adrenaline increase the metabolism. They also relax and dilate the airways so that more oxygen than usual can be taken in. Adrenaline surges can also decrease the amount of pain felt. As a result of all of these factors, adrenaline surges – while they last – have the ability to increase physical speed, strength and other physical abilities.

Unfortunately, when these bursts of adrenaline wear off – as they must – people with M.E. are left far more ill as a result for many days, weeks, months or even years. People with M.E. are harmed by adrenaline surges, both by the physiological stress to the body of the changes caused by adrenaline, and by the extra activity which adrenaline enables, which may be far beyond the body's normal limits so that such activity causes damage. For every short term 'gain' there is a far greater loss overall.

For more information on adrenaline surges in M.E., and the different order in which certain bodily systems may be affected by M.E. (and by overexertion), see the [Dr Cheney](#) section in [The effects of CBT and GET on patients with Myalgic Encephalomyelitis](#) or [Treating Myalgic Encephalomyelitis - Avoiding Overexertion](#).

- *A note on M.E. and other illnesses:* This is another one of the characteristics which clearly differentiates authentic M.E. from various self-limiting post-viral fatigue syndromes and so on – the striking variability of symptoms not only in the course of a day but often within the hour. As many M.E. experts have noted, this variability of the intensity of the symptoms is simply not found in post-viral fatigue states or syndromes (etc).

- There is also a waxing and waning of the *physical signs* of M.E. throughout the day, as Dr Hyde and Dr Jain explain, “A patient examined in the morning might have nystagmus, which would disappear at midday, recur later, disappear later and recur the next day.”

#### **F. The worsening of the illness caused by overexertion can be acute, but often does not reach its peak until 24 - 72 hours (or more) later.**

Another reason that short-term and superficial judgements of ability and disability levels in people with M.E. are ill-advised and often very misleading – and are in fact almost guaranteed to give a falsely more optimistic view of daily ability levels – is because the relapses caused by exertion very often do not appear until 48 or more hours afterward, when the average observer is long gone.

The onset of the worsening of symptoms caused by overexertion is sometimes be acute but often will not peak until 48 hours or more afterward (this is particularly true with regard to physical, cognitive and orthostatic exertions). Symptoms will then persist for hours, weeks or many months, or longer. For many M.E. sufferers, the effects from significant overexertion will very often peak on day three.

Sometimes there is a significant worsening of symptoms evident at the time of overexertion. At other times, there may only be a minor worsening of symptoms at the time of overexertion, but the delayed effects will be severe. Sometimes the acute effects and the delayed effects will both be severe. It varies depending on the type and severity of the overexertion involved etc.

- *A note on M.E. and other illnesses:* The ‘CFS’ definitions state that post-exertional symptoms ‘may take up to 24 hours to resolve.’ But to say that this is true of M.E. patients betrays an ignorance of the most basic facts of M.E. Post-exertional symptoms very often take far longer than 24 hours to even APPEAR in people with M.E., let alone be completely resolved in that time. These symptoms can take days, weeks, months or even several years to resolve. Overexertion can also cause a worsening of the base level of illness in M.E. and so the effects of overexertion can also be semi-permanent or permanent. Death can also occur due to overexertion in M.E.

This significant delay in the onset of post-exertional symptoms is not seen in those illnesses causing fatigue (etc.) after exertion. Nor do the effects of even minor overexertion very often last for weeks, months, years or permanently in people with these various fatigue syndromes as they do with M.E. sufferers. There is also not the same risk of overexertion leading to death in these other illnesses, as there is with M.E (The cardiac insufficiency seen in M.E., which causes much of the symptomatology and the limits with activity and orthostatic stress and so on in M.E., is simply *not seen* in these other illnesses.)

#### **G. The effects of overexertion can accumulate over longer periods of time and lead to disease progression, or death.**

In addition to the effects of overexertion commonly being delayed by 48 hours or so, the worsening of symptoms caused by overexertion can also sometimes be delayed (and accumulate) over weeks or even many months at a time until they are realised in a ‘crash.’ This is a period of intense worsening of the overall condition followed by a gradual return to the patient’s base level of illness over weeks, months or even years.

When the body is confronted with activity (or inputs) beyond the patient’s individual limits severely and/or repeatedly over time, these effects can also become cumulative in the long term; the patient becomes unable to return to their base level of illness at all. What this means is that long-term or permanent worsening of the overall severity of the condition is caused. Thus some patients are still dealing with the severe physical effects of inappropriate advice to exercise or to be more physically or mentally active etc. five, ten, fifteen or more YEARS afterward and for some patients the damage caused is permanent. Overexertion has also resulted in death in some cases of M.E.

Strong evidence exists to show that overexertion can have extremely harmful effects on M.E. patients. Patient accounts of leaving exercise programs much more severely ill than when they began them; wheelchair-bound or bed-bound or needing intensive care or cardiac care units, are common. (Recent research has shown that postural stress and physical and mental overexertion exacerbate cardiac insufficiency in this disease; see the notes below for more information.) In addition to the risk of relapse, permanent damage, and disease progression, there have also been reports of sudden deaths in M.E. patients following exercise. As M.E. expert Dr. Elizabeth Dowsett explains, ‘20% have progressive and frequently undiagnosed degeneration of cardiac muscle which has led to sudden death following exercise. Prompt recognition and advice to avoid over-exertion is mandatory.’

- For more information on the question of “Can M.E. patients really die just from being forced out of bed, or to leave the house etc.?” please see the paper: Why patients with severe M.E. are housebound and bedbound
- Cardiac and vascular abnormalities have been documented from the earliest outbreaks of M.E. to the present day. Dr. Paul Cheney explains that when M.E. patients stand up, they are on the edge of organ failure as their cardiac

output has dropped to the extremely low level of 3.7 litres per minute, a 50% drop from the normal output of 7 litres per minute. Without exception, says Cheney, every M.E. patient ‘is in heart failure.’

Recent research shows that mitochondrial and other dysfunction leads to diastolic dysfunction and reduced stroke volume/low cardiac output in M.E. – and that certain levels of orthostatic stress and physical and mental activity etc. exacerbate this cardiac insufficiency. Dr Cheney explained recently that because it takes more metabolic energy for the heart to relax and fill with blood than it does for it to squeeze and pump blood, the hearts of people with M.E. don’t fill with the proper amount of blood before they pump which is what causes the reduced cardiac output and many of the symptoms of M.E. (and much of the disability of M.E.). So the tachycardia – fast heart rate – often seen in M.E. in response to orthostatic stress and so on is actually compensating for low stroke volume to help increase cardiac output. The heart doesn’t fill with enough blood before each beat of the heart so it is forced to beat faster to try to make up some of the shortfall, but people with M.E. are still left with reduced cardiac output which leaves them very ill and disabled. If this problem is severe enough it can result in death.

As one M.E. advocate explains: ‘Cardiac output is sometimes too low to meet the demands of movement, and any attempt to exert oneself beyond one’s own capacity for cardiac output - that is when demand exceeds cardiac capacity - would indeed result in death. Studies on dogs have shown that when the demands of the body exceed cardiac output by even 1%, the organism dies. M.E. patients [must] reduce demand and reduce their exertion level to stay within the bounds of their low cardiac output to stay alive.’

- *A note on M.E. and other illnesses:* It is sometimes claimed that while exercise programs are not safe or appropriate for the severely affected, that mild or moderately affected M.E. sufferers can benefit from such interventions. But this assertion is NOT supported by the evidence. (Some miscellaneous ‘fatigue’ sufferers have been shown to benefit from graded exercise programs, but the results of these studies are no more relevant to mild M.E. sufferers than they are to severe M.E. sufferers; people with ‘fatigue’ do NOT have mild M.E. any more than they have mild multiple sclerosis, mild Lyme disease, mild cancer or any other illness.) Recent studies have shown that graded exercise programs are the actual reason many with M.E. are so severely affected in the first place, thus exercise programs should not be considered safe for M.E. sufferers of any severity. Graded exercise *cannot* improve authentic M.E.; disabled patients who improve with exercise do not qualify for a diagnosis of authentic M.E.

#### **H. The activity limits of M.E. are not short term, a gradual (or sudden) increase in activity levels beyond a patient’s individual limits can only cause relapse, disease progression or death in patients with M.E.**

Increasing the activity levels of someone with M.E. beyond their individual limits, can only ever be counterproductive. It really doesn’t matter if you do this gradually or all at once. Raising the limits gradually may well delay the onset of the relapse in some patients, but the end result will still be relapse and/or disease progression, or death. None of the various cardiac, cardiovascular, immunological, neurological, cognitive, muscular, and other abnormalities present in M.E. sufferers – which together cause the high level of disability associated with M.E. – can be explained by mere ‘deconditioning.’ *Patients who improve with graded activity programs do not qualify for a diagnosis of M.E.*

- M.E. is not a short-term or ‘hit and run’ viral attack; it is not a self-limiting post-viral fatigue syndrome caused by mononucleosis/glandular fever, Q fever or hepatitis, or any other common infection. Nor is M.E. a psychological or behavioural condition. Authentic M.E. cannot be improved through psychotherapy or graded exercise therapy. These theories have been comprehensively disproven many times over with regard to authentic M.E. patients (as have the many other similar theories). M.E. is a chronic illness which affects the vast majority of sufferers for many years or decades at a time, or for the rest of their lives. A person who has been correctly diagnosed with M.E. will naturally raise their activity levels when/if they have had an improvement in their illness – but it can never work the other way around. See: [Smoke and mirrors](#) for more information.
- *A note on M.E. and other illnesses:* M.E. can be progressive, degenerative, chronic, or relapsing and remitting. As many M.E. experts have noted, the chronicity of M.E. is another characteristic which clearly separates the illness from various self-limiting post-viral fatigue syndromes.

#### **I. The symptoms of M.E. do not resolve with rest. The symptoms and disability of M.E. are not just caused by overexertion, there is also a base level of illness which can be quite severe even at rest.**

There is a base level of illness that is always present in M.E., even at rest. (This is true of all sufferers except perhaps that small percentage who have improved enough over time to be only mildly affected, or who have had a total or almost total remission of their M.E.) This is because the metabolic problems of M.E. are only one part of M.E., they are not the only cause of symptoms or of the worsening of the illness.

But even those symptoms which are caused by the metabolic problems of M.E. (etc.) do not always resolve with rest. For severely affected patients, just keeping the body going at the lowest possible level can count as ‘overexertion’ – not only can the bodies of these people not cope with extra activity, but they also cannot even cope with keeping the bodily systems and organs going at the lowest possible level – at rest. Because even when we are resting as much as we can be; hearts have to keep pumping, lungs have to keep drawing air in and out constantly, kidneys have to keep working, and so on. It takes a lot of metabolic power to keep all the complex systems in the body working, even at the lowest possible level. Forcing the body to do more work when it is

already not coping with the most basic level of functioning causes these problems to become even more severe as the quality of function achieved across various bodily systems is lowered even further, but even at rest these same problems can be quite severe because of course so many different bodily systems never can ‘rest.’

Virtually all bodily systems are affected in some way by both the damage to the central nervous system and the metabolic problems of M.E. (including the cardiac insufficiency this causes) etc. so it is no wonder people with M.E. feel so ill, have such a reduced level of functioning in so many different bodily systems and have so many restrictions and limits on how active they can be. Even with complete rest – and some people with M.E. can do almost nothing else – many M.E. sufferers are still very ill and disabled.

**J. Repeated overexertion can harm the patient’s chances for future improvement in M.E. M.E. patients who are able to avoid overexertion have repeatedly been shown to have the most positive long-term prognosis.**

It is vital that M.E. patients are never encouraged to be active beyond their individual limits. As Dr Melvin Ramsay explains; ‘The degree of physical incapacity varies greatly, but the [level of severity] is directly related to the length of time the patient persists in physical effort after its onset; put in another way, *those patients who are given a period of enforced rest from the onset have the best prognosis*. Since the limitations which the disease imposes vary considerably from case to case, the responsibility for determining these rests upon the patient. Once these are ascertained the patient is advised to fashion a pattern of living that comes well within them.’

Patients with M.E. must be allowed to determine for themselves a level of daily activity which is not needlessly restrictive, but which is also sustainable in the long term without causing a worsening of symptoms or disease progression (and which also holds back a small amount of ability to cope with occasional unplanned or unavoidable overexertions, to prevent these from causing significant setbacks). People with M.E. must also be allowed to determine for themselves how much rest they need. Giving people with M.E. the support they need to limit their activities in this way is actually the best way to ensure that they each get to be as active as possible in the long term. The importance of getting appropriate rest and avoiding overexertion in M.E. cannot be overstated. Forcing or encouraging people with M.E. to engage in even low levels of physical and cognitive activity, sensory input and orthostatic stress beyond their individual limits can have catastrophic long-term consequences.

- For more information about the effects of overexertion on M.E. patients, including statements/research from some of the world’s leading M.E. experts about why overexertion is so physically harmful, see: Smoke and Mirrors. (This paper also includes links to many different patient accounts of the effects of overexertion on people with M.E.). If you have M.E. see Treating Myalgic Encephalomyelitis - The Basics and Treating Myalgic Encephalomyelitis - Avoiding Overexertion for more on the importance of avoiding overexertion.

**L. Not every M.E. sufferer has ‘safe’ activity limits within which they will not exacerbate their illness, this is not the case for the very severely affected.**

For very severely affected M.E. sufferers there is virtually no ‘safe’ level of physical or mental activity, orthostatic stress or sensory input; no level which does not produce a worsening of symptoms, and perhaps also contribute to disease progression. Even the most basic actions – speaking a few words, being exposed to moderate light or noise for a few minutes, turning over in bed, having hair or body washed in bed by a carer or chewing and swallowing food – cause severe and extended symptom exacerbations in such patients. It is not uncommon to hear of very severely affected sufferers who are unable to bathe themselves (or even be bathed by a carer) more often than once a week, or even once every few weeks, or even less. Some sufferers cannot chew or swallow food any longer and need to be tube fed. Many patients with severe M.E. are no longer able to toilet themselves, and so on. Either sufferers are just too ill to do these things at all, or they cannot tolerate the very long and severe relapses that come after such activities.

Even the smallest movement, thought, touch, light, noise or period upright etc. can the already very severe symptoms far, far worse. Thus few illnesses demand such isolation and loss of quality of life as severe M.E. Very often people with very severe M.E. can barely communicate, or even tolerate the presence of another person. This is what makes M.E. such a cruel disease and such an isolating disease. The illness can cause a level of disability and isolation that is just unimaginable to anyone not familiar with very severe M.E.

- For more information on severe M.E. see The severity of M.E. and M.E. Fatalities plus Why patients with severe M.E. are housebound and bedbound.