

## 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.

# Testing for M.E.

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Many articles which claim to be about Myalgic Encephalomyelitis (M.E.) in the mainstream media (and even some medical texts) write that not only are there no tests which can be utilised to help confirm an M.E. diagnosis. Despite their popularity, these claims are simply absurd.

### Part 1: Overview

M.E. is a distinct, recognisable disease entity that is not difficult to diagnose and can in fact be diagnosed relatively early in the course of the disease, within just a few weeks, providing that the physician has some experience with the disease. There is just no other disease that has all the major features of M.E.

Objective evidence of quantifiable organic abnormalities in M.E. patients has existed since the 1950s. As with a wide variety of illnesses – lupus, multiple sclerosis, and ovarian cancer for example – there is as yet no *single* test which can diagnose M.E. in all patients. Therefore, like these other illnesses, M.E. must be diagnosed by taking a detailed medical history, noting the type and severity of symptomatology and other characteristics of the illness and the type of onset of the symptoms. (An acute or sudden onset of symptoms is always seen in M.E. and this onset type rules out a wide variety of other illnesses associated with gradual onset). A *series* of tests may also then be necessary to rule out or confirm a suspected M.E. diagnosis.

One cannot test for ‘CFS’ but M.E. is not the same thing as ‘CFS’ (or ‘ME/CFS.’) The presence or absence of ‘fatigue’ is largely irrelevant in determining an M.E. diagnosis except in that its presence may of course make the diagnosis of a large number of well-known fatigue causing illnesses considerably more likely (depression, vitamin deficiency or malignancy for example) (Hyde & Jain, 1992). M.E. is not a diagnosis of exclusion or an untestable disease. Tests will only *all* be normal in M.E. patients – as with all illnesses – if the wrong tests are conducted, or if those tested do not in fact have M.E. (Hyde 2007, [Online]) (Hyde 2006, [Online]) (Chabursky et al. 1992, p.22).

Contrary to common disinformation erroneously linking M.E. with ‘CFS,’ it is not mere ‘fatigue’ that defines M.E. but central nervous system (CNS) dysfunction. M.E. represents a major attack on the CNS by the chronic effects of a viral infection which targets the brain: an enterovirus. As M.E. expert Dr Byron Hyde explains:

The one essential characteristic of M.E. is acquired CNS dysfunction. A patient with M.E. is a patient whose primary disease is CNS change, and this is measurable. We have excellent tools for measuring these physiological and neuropsychological changes: SPECT, xenon SPECT, PET, and neuropsychological testing (2003, [Online]).

Tests which together can be used to confirm an M.E. diagnosis include:

- SPECT and xenon SPECT scans of the brain.
- MRI scans of the brain.
- PET scans of the brain.
- EEG/QEEG brain maps.
- Neurological examination. Neuropsychological testing.
- The Romberg test.
- Immune system tests.
- Insulin levels and glucose tolerance tests.
- Erythrocyte Sedimentation Rate (ESR) tests.
- Circulating blood volume tests.
- 24 hour Holter monitor testing.
- Tilt table examination and standing/sitting/reclining blood pressure tests.
- Exercise testing and chemical stress tests.

- Physical exam.

These tests are the most critical in the diagnosis of M.E., although various other types of tests are also useful.

M.E. expert Dr Byron Hyde's [Nightingale Definition of M.E.](#) also now makes diagnosis easier than ever before even for those with no prior experience in diagnosing M.E. This is a pure M.E. definition and, most importantly, it defines M.E. as *testable* (Hyde 2007, [Online]) (Hyde 2006, [Online]) (Hyde 2003, [Online]) (Dowsett 2001a, [Online]) (Dowsett 2000, [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 n.d., [Online]) (Dowsett & Ramsay n.d., pp. 81-84) (Richardson n.d., pp. 85-92).

## **Part 2: More detailed testing information**

This text will now focus in more detail on the series of tests that can be used to confirm an M.E. diagnosis as well as detailing some of the physical signs common in M.E. patients which may also be useful for diagnosis. *(Some of the tests listed may however also be useful in proving illness for the benefit of social security or insurance company entitlements, or may be used to determine appropriate treatments in patients who have already been diagnosed with M.E. References useful in providing further information about all other aspects of diagnosis are provided at the end of this text.)*

### **SPECT and xenon SPECT scans of the brain**

The remarkable similarity in the brain images of patients with M.E. and multiple sclerosis patients has been noted for many years. M.E. was once known as 'atypical MS' due to the similarity between the two diseases.

SPECT scans have demonstrated decreased cortical/cerebellar regional cerebral blood flow most frequently in the frontal, parietal, temporal, occipital and brain stem areas of the brain, and throughout the cerebral cortex in M.E. This decrease in cerebral blood flow has also been found to have worsened further still 24 - 72 hours post-exertion. These abnormalities have also been shown to correlate with clinical status.

Dr Byron Hyde adds that, 'I do not describe a patient as having M.E. unless there is an abnormal SPECT. If the SPECT is normal, I often repeat it along with xenon SPECT. If the brain scans remain normal, I conclude that it is unlikely to be M.E.' (Hyde, 2003).

In 2007, Dr Byron Hyde's [Nightingale Definition of M.E.](#) further explained that brain scans can be used in diagnosing M.E. and to some degree can also be used to predict the future severity of the disease. He writes that:

The second and chronic phase that clearly defines M.E. is characterized by various measurable and clinical dysfunctions of the cortical and/or sub-cortical brain structures. If the patient's illness is not measurable using a dedicated brain SPECT scan such as a Picker 3000 or equivalent, then the patient does not have M.E. For legal purposes these changes may be confirmed by PET brain scans with appropriate software and/or QEEG. These changes can be roughly characterized as to severity and probable chronicity using the following two scales: **A:** Extent of injury and **B:** degree of injury of CNS vascular function.

#### **Extent of Injury**

**Type 1:** One side of the cortex is involved. Those patients labeled as 1A have the best chance of recovery.

**Type 2:** Both sides of the cortex are involved. These patients have the least chance of spontaneous recovery.

**Type 3:** Both sides of the cortex, and either one or all of the following: posterior chamber organs, (the pons and cerebellum), limbic system, the subcortical and brainstem structures are involved.

Type 3B are the most severely affected patients and the most likely to be progressive or demonstrate little or no improvement with time.

#### **Degree of injury**

**Type A:** Anatomical integrity is largely maintained in the Brain SPECT scan.

**Type B:** Anatomical integrity is not visible in the CNS SPECT scan. Type 3B are some of the most severely and chronically injured patients (Hyde 2007, [Online]).

Dr Byron Hyde's [Nightingale Definition of M.E.](#) also explains that the subject of vascular pathology in M.E. is not new and that:

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. In the late 1980s Drs. Jay Goldstein and Ismael Mena confirmed and proved this initial description by examining the changed brain microcirculation using brain SPECT imaging in M.E. patients. Following my 21 years of examining M.E. patients and 16 years of subjecting M.E. patients to brain imaging techniques suggested by Goldstein and Mena, 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]).

### ***MRI scans of the brain***

MRI scans have shown the presence of small white matter lesions predominantly in the frontal lobes and subcortical areas. Punctate, subcortical areas of high signal intensity consistent with edema or demyelination were identified by MRI in at least 78% of M.E. patients (similar to those seen in MS). The abnormalities in M.E. patients most closely resemble those seen in AIDS encephalopathy. Research has shown that at least 80% of M.E. patients will have abnormal MRI scans (Hyde, 2003). M.E. patients with abnormalities on MRI have been reported as being more severely impaired than those without such abnormalities. In a comparison of intracranial abnormalities in M.E. patients by MRI and SPECT, the SPECT scan abnormalities appeared to correlate with clinical status while the MRI changes were irreversible (Carruthers et al. 2003).

Researchers have found that the bright spots on MRI scans of M.E. patients are evidence of an ‘arteriolar vasculopathy’ and that M.E. is a ‘systemic micro-vascular inflammatory process.’ That is a process that affects not only the brain or the heart or the muscles, but potentially every organ system in the body. There is also evidence in M.E. of capillary inflammation, perivascular cuffing (the accumulation of immune cells that surround injured blood vessels) and remarkably reduced renal blood flow (NAME-US, 2008, [Online]).

MRI scans are not recommended for those patients who relapse when exposed to loud noises (a common symptom of M.E.). An MRI scan can cause severe and extended relapse in such patients, although newer and quieter machines may not cause a problem. Dr Byron Hyde also explains that a normal MRI does not conclusively prove that a person has no CNS dysfunction as the MRI demonstrates only abnormal *anatomy* and so a normal MRI should never be used to prove that a person does not have CNS dysfunction or is not ill (2003, [Online].)

### ***PET scans of the brain***

PET scans have shown decreased metabolism of glucose in the right mediofrontal cortex. PET scans have also shown generalised hypoperfusion of the brain with a particular pattern of decreased neuronal metabolism and hypoperfusion in the brain stem.

CT scans are not appropriate or useful for diagnosing M.E. or for investigating M.E. pathology. Most M.E. patients will have a normal CT scan, although CT scans may show reduced adrenal gland size in M.E. patients (Carruthers et al. 2003).

### ***Neuropsychological testing***

Of the CNS dysfunctions that make up M.E., severe cognitive dysfunction is easily one of the most disabling characteristics of the illness. This is nothing like the ‘brain fog’ seen with the flu, other transient or minor viral illnesses or overwork. M.E. patients may *at times* be unable to read or write, to understand speech or to speak, to recognise close friends and family members, may forget how to use a telephone or have to pull their car over to the side of the road as they cannot recall where they live. The cognitive and neurological changes seen in M.E. are

severe, long-term and profoundly life-changing. These changes also worsen further following overexertion and so they are not stable from one day to the next.

The cognitive dysfunction in M.E. can be extremely severe due to decreased cognitive performance overall, decreased processing speed, problems with working memory and information retention and learning etc. Neuropsychological testing can be used to identify cognitive dysfunction and/or to confirm an M.E. diagnosis. It should focus on the abnormalities known to differentiate M.E. from other causes of organic brain dysfunctions. (Carruthers et al. 2003). Dr Sheila Bastien Ph.D. is an expert in this field with over 20 years experience in neuropsychological testing and more than 6 years experience in applying these tests to M.E. patients. She explains that:

Deterioration of IQ levels, as well as cognitive and motor dysfunction in these patients, suggest a pathological process in the brain. The pattern of focal and lateral impairments is consistent with patients who have this particular neurologic dysfunction. The impairment pattern is consistent across the study group [of M.E. patients] although impairment levels vary. This pattern is not seen in other diseases or injuries, such as Alzheimers, stroke, head injuries, multiple sclerosis, systemic lupus erythematosus, personality disorders, depression, psychosis, malingering, anxiety or panic disorders, somatisation or situational stress disorders. The pattern of impairment is one of focal and lateral deficits, consistent with a multi-focal organic brain syndrome. Tests suggest that the most impaired focal areas are the left temporal, right parietal, and left temporal lobes; although there are lesser bilateral impairments in the opposite lobes as well. (1992, pp. 453 - 454)

In 2007, Dr Byron Hyde's Nightingale Definition of M.E. explained that:

There are neuropsychological changes that are measurable and demonstrate short-term memory loss, cognitive dysfunctions, increased irritability, confusion, and perceptual difficulties. There is usually rapid decrease in these functions after any physical or mental activity. Neuropsychological changes must be measured in relation to estimates of prior achievement. This feature may improve over a period of years in patients with adequate financial and social support and can be made worse by chronic stressors. The neurophysiological changes are those observed by a qualified Neuropsychologist with experience in examining this type of disease spectrum. Some of the deficits that a Neuropsychologist should consider examining include: (a) word finding problems, (b) Subtle problems with receptive and expressive aphasia, (c) Decreased concentration, (d) Distractibility and the decreased ability to process multiple factors simultaneously, (e) Dyscalculia, (f) Decreased fine and gross motor problems, (g) Dysfunction of spatial perception, (h) Abstract reasoning, (i) Compromised visual discrimination, (j) Sequencing problems. In Cochran's Q Neuropsychological tests there is an increased observation of significant problems in both immediate and delayed verbal recall. In Dr Sheila Bastien's investigations, over 50% of M.E. patients have delayed visual recall, TAP dominance, TPT N-Dominance and 40% or more have abnormalities of Immediate visual recall, Tap N-Dom, Grip N Dominance, & grip dominance problems (Hyde 2007, [Online]).

Dr Byron Hyde also adds that:

This is a complex type of testing, and a physician should attempt to locate an experienced neuropsychologist without ties to the insurance industry. Most neuropsychologists today are employed by the insurance industry, and if they find too much pathology, I suspect that they are no longer engaged. Do not be fooled by a negative insurance-paid neuropsychological report; psychologists whose primary training is not neuropsychology prepare many of these reports' (2003, [Online]).

- One of the most useful texts on neuropsychological testing in M.E. is chapter 51 of the book 'The Clinical and Scientific Basis of ME' written by Dr Sheila Bastien Ph.D. Several other chapters of this book also describe the various cognitive effects of M.E. in detail. (Details of this book are given below.) See also [The comprehensive M.E. symptom list](#) for more information about the specific cognitive deficits commonly seen in M.E.

### ***EEG brain maps and QEEG brain maps***

95% of M.E. patients have been found to have abnormal cognitive-evoked EEG brain maps (Hooper, 2001 [Online]). But Dr Byron Hyde argues that QEEG brain maps are even more accurate:

An EEG only records activity on the outer millimeter of the brain. A QEEG is simply an EEG attached to a computer that contains appropriate software. A QEEG will immediately demonstrate tumors and brain activity or lack of it related to specific stimuli that are simply not possible to detect on a non-computer-driven EEG. Using QEEG technology operated by an expert physician, we have been able to demonstrate not only lack of normal activity in ME patients but migration of the normal activity centers from injured areas to different parts of the brain. (Hyde, 2003, [Online])

QEEG brain topography has found evidence in M.E. of elevated EEG activity in theta and beta frequencies and increased intracerebral electrical sources in left frontal region delta and beta frequencies in eyes closed condition may be identified. Reduced sources in right hemisphere (beta) may also be noted during verbal cognitive processing (Carruthers et al. 2003 [Online]).

### ***Romberg or tandem Romberg test***

The test involves standing with eyes open and then with eyes closed with feet together or one behind the other. A positive Romberg is when the position is maintained with eyes open but not when they are closed. It is a useful test of brain stem function. Professor Malcolm Hooper explains that, ‘Dr Paul Cheney found that more than 90% of patients have an abnormal Romberg versus 0% of controls.’ (Hooper et al. 2001 [Online]).

### ***Neurological examination***

Most M.E. patients have abnormal neurological examination (Hooper et al. 2001 [Online]).

### ***Tests of the immune system***

The immune system abnormalities in M.E. patients indicate a viral infection. Specific findings include (but are not limited to):

- Increased numbers of activated cytotoxic T cells (most patients have evidence of T-cell activation)
- Reduced T-Cell count
- Low natural killer cell numbers/percentage and function (cytotoxicity)
- Elevated immune complexes
- Atypical lymphocyte count
- Significantly reduced CD8 suppressor cell population and increased activation marker (CD38, HLA-DR) on CD8 cells
- Abnormal CD4/CD8 ratio
- ANA
- Elevations of circulating cytokines (including IL-6) particularly after exertion (there is an inappropriate and negative immune response to exertion)
- Immunoglobulin deficiencies (most often IgG 1 and IgG 3)
- Th1/Th2 Imbalance (some patients appear to have an over activation of the anti-inflammatory (Th2) branch and an under activation of the pro-inflammatory (Th1) branch of the immune system. This would explain increased rates of allergies and sensitivities, and conversely, difficulty fighting off pathogens).
- Th1 –Th2 response to mitogen stimulation (high levels of Th2 indicate autoimmunity)
- Antilamin antibodies (indicate autoimmunity and brain cell damage. Lamin B antibodies are evidence of autoimmunity)
- Apoptosis is often raised (this is programmed cell death: known to be raised in infection)
- Monocytosis (raised monocytes are suggestive of infection) (McLaughlin, 2004 [Online]) (Carruthers et al. 2003) (Hooper et al. 2001 [Online]) (Cheney 2006, [video recording]).

Natural killer cells are clearly impacted in M.E. One of the most commonly observed abnormalities in M.E. patients, and also one of the most striking abnormalities, is that of low numbers and function of natural killer cells, perhaps as low as it is ever seen in other diseases. Actual NK cell function was found to be very, very low – 9% cytotoxicity. The mean for the controls was 25%. This is significant because in early HIV and even well into ARC (AIDS related complex, which often precedes the fully developed condition), NK cytotoxicity might be around 13 or 14 percent. It was found that M.E. patients ‘represent the lowest cytotoxicity of all populations we’ve studied’ (Klimas, 1990). Poor T cell functioning is also seen in M.E. Researchers believe that chronic immune activation due to an underlying chronic infection has caused these T and NK cells to ‘burn out’ due to overuse (Cheney 2006, [video recording]).

Professor Malcolm Hooper adds that: ‘There is mounting international evidence that M.E. is an autoimmune disorder, with similarities to systemic lupus erythematosus. Evidence of antilamin antibodies has been found in the blood of M.E. patients: antibodies against this protein are proof of autoimmunity and of damage to brain cells’ (2001 [Online]). In addition, these immune tests (especially NK cell tests) correlate with how ill a person is, and so can also be used to some extent to indicate the severity of the disease at a particular time.

### **Erythrocyte Sedimentation Rate (ESR)**

An unusually low sedimentation rate of <5mm/hr is common in M.E. and can occur in 40% or more of patients (although there may also be brief periods where there is an elevated rate >20mm/hr). ESR rates as low as 0 have been documented in M.E. patients, and levels of 1 and 2 are very common (Hooper et al. 2001 [Online]) (Johnston, 1996, p. 215).

Such low sedimentation rates indicate that M.E. patients may have difficulty forming red cell membranes, as is the case with sickle cell disease (where such low sedimentation rates are also seen), because of a distorted red cell pathology. Dr Byron Hyde reported in 1989 that, "To my knowledge, there are only five diseases that have a pathological low sedimentation level: Myalgic Encephalomyelitis, sickle-cell anemia, hereditary spherocytosis, hyper-gammaglobulinemia [and] hyper-fibrogenemia" (Johnston, 1996, p. 215).

- *A note to patients on the misinterpretation of this test by doctors:* Unfortunately, as problems with *high* sedimentation rates are very common and more well known, doctors may mistakenly be of the opinion that with this test – as with blood pressure tests – 'lower is better.' This is a real problem when the low sedimentation rate and the low blood pressure seen in M.E. are signs of serious abnormal pathology, debilitating symptoms and a potentially fatal disease. Although a very low sedimentation rate by itself should not be interpreted as diagnostic of M.E., this is a simple and inexpensive test that can be a very strong indicator of M.E., if a patient's symptoms and additional tests also point to the diagnosis. However, patients must be aware that the results of this test are prone to misinterpretation, unfortunately.

### **Insulin Levels and Glucose Tolerance Tests**

Derangement of insulin response and insulin levels is a frequent finding in M.E. patients. Glucose tolerance curves are often abnormal (Hooper et al. 2001 [Online]).

Hypoglycaemia in M.E. can also be caused by low cellular energy levels. This is why people with M.E. have very severe hypoglycaemia during and after overexertion. The GTT (and finger-prick tests of blood glucose levels) may also be normal in M.E. (and other diseases involving problem with ATP production such as post-polio syndrome) despite very clear problems with the patient tolerating foods high in carbohydrate and sugars. Patients with M.E. may also have a delayed effect with the GGT, registering a normal test result during the three hour duration of the test but then feeling extremely unwell and collapsing for an entire day an hour or more later. As Dr Wilson explains in his excellent article on [Hypoglycemia](#),

Confusion occurs regarding the definition of hypoglycemia. The standard medical definition is a serum glucose level of less than about 65 mg/ml. However, many patients undergoing a glucose tolerance test or GTT experience symptoms of hypoglycemia in spite of normal serum glucose levels.

I heard of one case in which a patient undergoing a 5-hour GTT ripped off her clothes and ran naked through the streets, although her serum glucose level was normal. In a less dramatic example, another patient fainted right in their chair during the test when the serum glucose was normal. Clearly the GTT is missing something.

What is missing is a better definition of hypoglycemia. It is not just low glucose in the blood. It is really related to *low energy production at the cellular level*. What happens during a GTT is that just giving a dose of sugar by mouth, as is done for this test, upsets glucose metabolism sufficiently that the entire glucose regulatory mechanism is occasionally thrown out of kilter and this produces the bizarre symptoms. It also produces false positives, false negatives and other aberrations on the GTT. If the laboratory measured the insulin levels during the test, as Dr. Robert Atkins, MD and others have suggested, it would give a much clearer picture. But even with this, it is only measuring sugar in the blood.

**What is required for energy production?** Adequate cellular energy production requires that enough glucose reaches the cells, not only an adequate supply of glucose, but also that it finds its way into the cells through the cell membranes. Once in the cells, it also requires that the mitochondria of the cells are able to burn or metabolize the glucose to form ATP. It also requires that the ATP is able to be utilized, meaning consumed or metabolized to ADP, and then recycled or reprocessed again into ATP. In short, any problem in these chemical pathways will cause hypoglycemic symptoms.

As the GTT is such an incomplete test and many patients are very aware before the test that fasting and drinking the sugar solution will make them feel terribly ill, M.E. patients are also most likely best off avoiding the relapse-inducing GTT and instead having a simple finger-prick blood glucose level test. If the GTT test is done, the glucose drink should be warmed first as this seems to reduce the relapse to some extent in M.E. patients.

- For more information on hypoglycaemia, and why glucose tolerance tests only tell part of the story and may be classed as normal even where hypoglycaemia is a very severe problem, see the article [HYPOGLYCEMIA](#) by Lawrence Wilson MD.

### **24 Hour Holter Monitor**

A 24 hour Holter monitor (a type of heart monitor) may show repetitively oscillating T-wave inversions and/or a flat T-wave (a standard 12 lead ECG is usually normal however) (Hooper et al. 2001 [Online]). Holter monitors may also show heart rates as high as (or higher than) 150 beats per minute as an immediate or delayed response to the patient maintaining an upright posture, or at rest (Carruthers et al. 2003). Heart rates as low as 40 beats per minute may also be observed (during sleep) (Hyde, 2003, [Online]). Dr Byron Hyde explains that:

I routinely use a Holter monitor on all patients. The cardiologist often reports these as normal. *Do not trust this report.* What the cardiologist or computer is basing the report on is the number of ischemic events [which is not relevant to the heart problems caused by M.E.]. However, read the lowest heart rate at night, and note that it sometimes falls to the low 40s. Though this may be normal in an athlete, it is not in a sedentary M.E. patient. For a patient who is not active all day long and has an average heart rate that flirts with 100 beats per minute or more, you know that this is not normal. These abnormal tests, however, are often reported as normal. (Hyde, 2003, [Online])

Dr Byron Hyde recommends that patients or doctors request to be informed of these specific patterns as they may not be reported (or may be subsumed under non-specific T-wave changes) by the interpreter (Hyde, 2003, [Online]).

Dr Hyde also adds that cardiac irregularity on minor positional changes or after minor physical activity, including inability of the heart to increase or decrease in speed and pump volume in response to increase or decrease in physical activity, is often seen in M.E. and that, 'In many M.E. patients there is an unusual daytime tachycardia, particularly since these patients are often very sedentary. In doing a 24-hour Holter monitor this may be missed since the 24 hour average is usually given. One should always ask for wake time and sleep time heart rates' (2007, [Online]).

### **Tilt Table Examination**

Dr Byron Hyde explains that testable vascular and cardiac dysfunction is the most obvious set of dysfunctions when looked for and is probably the cause behind a significant number of M.E. complaints, and that all moderate to severe M.E. patients have one or more of the following vascular dysfunctions:

- Severe postural orthostatic tachycardia syndrome (POTS)
- Cardiac irregularity on minor positional changes or after minor physical activity
- Raynaud's phenomenon (vasoconstriction of small arteries or arterioles of extremities, with change in colour of the skin, pallor and cyanosis)
- Circulating blood volume decrease
- Bowel dysfunction caused by vascular dysfunction (vascular dysfunction may be the most significant causal basis of the multiple bowel dysfunctions occurring in M.E.)
- Persantine effect in M.E. patients
- M.E. associated clotting defects (M.E. represents both a vasculitis and a central and peripheral change in vascular physiology)
- Anti-smooth muscle antibodies (This is an antibody to the muscle tissue in the arterial bed. It is elevated in about 5% of M.E. patients)
- Cardiac dysfunction (There are a large number of cardiac dysfunctions that can regularly appear in an M.E. patient).

Note however that the primary vascular change in M.E. is seen in abnormal SPECT brain scans. It should also be noted that a significant number of M.E. patients may show evidence of all, or almost all, of these vascular abnormalities (2007, [Online]).

Orthostatic intolerance is very common in M.E. patients and may manifest as one of, or a combination of the following:

- **Neurally mediated hypotension (NMH)**: Involves disturbances in the autonomic regulation of blood pressure and pulse. There is a precipitous drop that would be greater than 20-25 mm of mercury of systolic blood pressure upon standing, or standing motionless, with significant accompanying symptoms. The patient feels an urgency to lie down.

- **Postural orthostatic tachycardia syndrome (POTS):** Excessive rapidity in the action of the heart (either an increase of over 30 beats per minute or greater than 120 beats per minute during 10 minutes of standing); and a fall in blood pressure occurring upon standing. Syncope can but usually does not occur.
- **Chronic Orthostatic Intolerance (COI):** The inability to sustain upright activity (standing, sitting or walking), is very common and important component of M.E. Upon limited standing, the patient experiences an urgency to lie down, confusion, malaise, various cardiac/vascular symptoms and worsening of other symptoms. Sitting and light walking are tolerated better than standing still, but no upright activity is tolerated well. Lying down helps alleviate symptoms. Tilt-table testing may be helpful in diagnosis but some patients may have a normal tilt-table test and still have severe COI. Quiet standing in the office allows for observation and monitoring the blood pressure and pulse.
- **Delayed postural hypotension:** As above except the drop in blood pressure occurs many minutes (usually ten or more) after the patient stands rather than upon standing (Carruthers et al. 2003) (Bassett 2009, [Online]).

Dr Byron Hyde warns about tilt table testing in M.E. patients however that:

I frequently find gross abnormalities in M.E. patients with this test. [But] a circulating blood volume and a complete cardiac investigation should be done first. This is not a test to undertake lightly since the patient's heart sometimes stops and may have to be restarted. This test should only be done in major hospital centers in the presence of an appropriate physician where such emergency capabilities can be instituted.' (Hyde, 2003, [Online])

If a tilt table is not available, doctors can monitor pulse and blood pressure while the patient is standing and again while lying down. This may need to be repeated several times, and is known as the 'poor man's tilt table test.' More than 97% of patients demonstrate vasovagal syncope (NMH) on tilt table testing.

**Description of poor man's tilt table testing procedure:** Ask the patient to lie down and rest quietly for 15 minutes, then take the first blood pressure and pulse readings. Ask the patient to sit up for 10 minutes (or as long as they can manage without severe problems) and then take another set of readings. Direct the patient to stand up for 10 minutes (leaning against a wall, but without fidgeting or moving or talking which can affect the result) and then take another set of readings. After another 10 minutes of standing, take the readings again. Many patients will not be able to tolerate this much time upright and will need to stop the test partway through. If this happens, take another reading (if possible) and then ask the patient to lie down again. Failure to stop the tests when the patient becomes severely ill can lead to a loss of consciousness, or severe relapse lasting days, weeks or even months in the very severely affected. Someone should always be standing near the patient to catch them if they fall and serious requests to stop the test must be acted on in a timely manner (NAME-US, 2008, [Online]).

An additional important comment by Dr Byron Hyde about M.E. patients with POTS:

This group can be confused with diabetes insipidus due to the fact that they may have polydipsia from their attempt to increase their circulating blood volume by consuming large amounts of fluids. This group can be verified by the absence of pituitary adenoma or pathology and the fact that they can sleep through the night without waking to drink fluids (Streeten, David.) Despite the great steps forward in the understanding of this relatively common pathophysiology seen routinely in M.E. patients, a pathology which is really related to either an autonomic injury to the CNS, injury to the vascular receptors or both, very little of the present treatment protocol is of much use. The situation is so bad that few major centres have any well-funded expertise in either autonomic or vascular receptor injury. Many of the M.E. patients that are dismissed by physicians as suffering from lack of activity have significant proprioceptive injuries in these areas. Nor can we always rely on the few autonomic laboratories and their tilt table testing abilities. Many of the tilt table examination reports return as normal, many as grossly abnormal. Yet all the physician has to do is have each M.E. patient stand for 8-12 minutes to realize that a large number of these normal tilt table patients simply cannot maintain a normal blood pressure and normal heart rate. Compare this to non-M.E. patients and one immediately can tell the difference. A large number of M.E. patients have significant autonomic difficulties (2007, [Online]).

### ***Q scores (as measured by impedance cardiography)***

This is a test of heart function, as discussed by Dr Cheney and Dr Peckerman.

Dr. Paul Cheney explains that Dr Peckerman's study showed that when disabled M.E. 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). Without exception, according to Dr. Paul Cheney, every disabled M.E. patient 'is in heart failure' and the disability level is exactly proportional to the severity of their Q defect, without exception and with scientific precision (Cheney 2006, [video recording]).

Cardiac and vascular abnormalities have been documented in M.E. consistently from the earliest outbreaks to the current day. Recent research shows that there is diastolic dysfunction and reduced stroke volume/low cardiac output in M.E., and that certain levels of orthostatic stress and physical and cognitive 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 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 that leaves them very ill and disabled. If this problem is severe enough it can even result in death (Cheney 2006, [video recording]).

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' (MESA, 2007, [Online]).

Along with diastolic dysfunction, Dr Cheney also found evidence of another cardiac abnormality in M.E.: patent foramen ovale, or PFO. This results in hypoxia (low oxygen levels relative to metabolic needs). Dr Cheney stated that the cardiac index of M.E. patients is so severe that it falls between the value of patients with myocardial infarction (heart attacks) and those in shock. In 2006, Dr Cheney presented a seminar entitled "The Heart of the Matter". This important seminar will eventually be published. For more information on Q scores see the [Dr Cheney](#) page. (Note that this test is as yet not widely available.)

### ***Exercise testing and chemical stress tests***

Cardiopulmonary exercise testing (CPX) is widely used for the diagnosis (and functional assessment) of various cardiac and metabolic disorders and can also be used in the diagnostic evaluation of M.E. patients. Heart rate and blood pressure responses during the exercise test may reveal abnormalities specific to M.E. including: lower cardiovascular and ventilatory values at peak exercise (patients only being able to attain half the expected maximal workload and oxygen uptake compared to sedentary controls), elevated resting heart rates, and an inability to reach maximum age-predicted heart rates (suggesting cardiac or peripheral insufficiency and/or reduced blood volume) (Carruthers et al. 2003).

Many more severely affected M.E. patients however will be either too ill to complete such tests altogether, or may be able to complete these tests only at the cost of a potentially severe and unnecessary relapse (which usually peaks 24 – 48 hours post-exercise and will then persist for days, weeks or even months afterward depending on the patient's illness level). In addition to the risk of relapse, there have also been reports of sudden deaths in M.E. patients following exercise as M.E. expert [Dr Elizabeth Dowsett](#) explains: 'Some 20% [of M.E. patients] have progressive and frequently undiagnosed degeneration of cardiac muscle which has led, in several cases, to sudden death following exercise' (2000 [Online]). As exercise tests are not appropriate for many M.E. sufferers, [Dr Byron Hyde](#) writes:

Patients with ME frequently cannot do exercise tests, and so I then do chemical testing as a second best. Several of our patients have reacted severely to the chemical test with excruciating pain. This is not true angina, and although the pain sometimes ceases as soon as the chemical is stopped and the antidote given, sometimes it persists for weeks after the procedure with no sign of coronary artery disease. I do not understand this phenomenon, but it is obviously vascular. The cardiologists state that this pain does not occur with the same frequency in non-ME patients (2003, [Online]).

[Dr Byron Hyde](#) also adds that:

Persantine is a chemical manufactured by Boehringer Ingelheim. It is employed to perform chemical cardiac stress testing when a patient cannot exercise sufficiently to stress the heart. It is a particularly safe medication but when employed with many M.E. patients it can cause severe muscle pain over the extremities and entire musculature. Normally this can be reversed by injection of an antidote but this does not always work rapidly in M.E. patients. Severe pain can be intolerable and persist for minutes to days in some M.E. patients following Persantine use. Persantine works by dilating both peripheral and cardiac blood vessels and causing the heart rate to increase as in a POTS patient. Obviously one major pain factor in M.E. patients is caused by abnormal dilatation of peripheral blood vessels. The resulting pain may be related to reflex vasospasm as in severe Raynaud's phenomenon that I

note elsewhere is one of the causes of M.E. pain. To my knowledge, no testing of M.E. patients with Persantine has ever been published by Boehringer Ingelheim or others. It is one of the reasons I believe that pain syndromes in M.E. patients are due to a pathological vascular physiology.

Note that tests of exercise capacity must be repeated on day two or three if they are to more fully illustrate the level of disability in M.E. caused by exertion, *the onset of which is typically delayed by 24 to 72 hours*. See also: [Legal and Scientific Considerations of the Exercise Stress Test](#)

### ***Circulating Blood Volume (decrease):***

Dr Byron Hyde explains that:

This is a nuclear medicine test in which the circulating red blood cell levels in some M.E. patients can fall to below 50%, preventing adequate oxygenation to the brain, gut and muscles. These patients do not generally have anemia and are not blood deficient. This is undoubtedly a subcortical dysregulation. It is associated with serum and total blood volume measurements. This is a concept that many physicians have difficulty understanding. I have heard physicians repeatedly tell the patient they are not anemic and therefore dismiss this important finding.

**Note:** So where does the blood go? Body servomechanisms are genetically designed so that blood flow and oxygen to the heart are always protected. Thus, when the body of the M.E. patient is stressed, the blood flow to organs not necessary for short-term survival, such as the brain, the gut and skeletal muscles, can be temporarily decreased. This of course gives rise to many of the M.E. symptoms.

M.E. represents both a vasculitis and a central and peripheral change in vascular physiology. All such vascular illnesses should be potentially treatable. We do not yet know how to adequately treat the (i) genetic forms of vasculitis & vascular patho-physiology mentioned here, nor (ii) the probable viral triggered genetic vascular pathologies also mentioned. Nor do we know how to treat those (iii) centrally caused injuries causing the circulating blood volume defects that are demonstrated when we do the “nuclear medicine circulating blood volume tests. It is important to do this test on all [M.E.] patients (2007, [Online]).

Dr Byron Hyde also explains that:

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’ (2003, [Online]).

### ***PCR testing for enteroviral infection***

Positive PCR tests for enteroviral infection have been documented in a large percentage of M.E. patients that have been given this test. Enteroviruses become harder to pick up over time, although there are reports of some patients still testing positive for enterovirus infections 10 years or longer after the onset of M.E.

A patient that once tested positive for enteroviral infection may return a negative test some years later. But this negative test result should not be assumed to indicate that the patient’s disease status has necessarily changed as Dr Hyde reports that whether subsequent tests were positive or negative patients remained similarly disabled (Hyde 2011, [Online]).

### ***Miscellaneous other abnormalities commonly found in M.E. patients***

Cholesterol (high), 24-hour urine free cortisol (low), potassium (low), prolactin (high), elevated amino-hydroxy-N-methyl-pyrolidine, IAG-tryptopan metabolite is usually positive (and indicates a leaky gut, which in turn is indicative of a defective blood brain barrier), Hypercoagulability, flow cytometry (fibrinogen, thrombin/anti-thrombin complexes, etc.). There is also:

Thyroid status tests: Free T4, Free T3, TSH. Every M.E. patient should have their thyroid status tested and be treated with thyroxine (or similar) or amour thyroid as appropriate. Even if thyroid problems are not detected on the first test, M.E. undermines the thyroid gland and thyroid problems can develop later, so it's a good idea to test

thyroid function repeatedly. (Note that ‘armour thyroid’ may be far more beneficial/better tolerated than synthetic thyroxine in M.E. patients.)

Anti-smooth muscle antibodies: This is an antibody to the muscle tissue in the arterial bed. It is elevated in about 5% of M.E. patients but whether this is different in non-M.E. patients is unknown but unlikely. It rarely is over 1:40 (Hyde, 2007, [Online]).

Testable Endocrine Dysfunction: These features are common and tend to be of late appearance. Dr Byron Hyde explains that:

They are most obvious in: a. Pituitary-Thyroid Axis: Changes in serum TSH, FT3, FT4, Microsomal Ab., PTH, calcium and phosphorous rarely occur until several years after illness onset. This anomaly can best be followed by serial ultrasounds of the thyroid gland, where a steady shrinking of the thyroid gland may occur in some M.E. patients with or without the development of non-serum positive Hashimoto’s thyroiditis (a seeming contradiction in terms) and a significant increase in thyroid malignancy. In cases of thyroid wasting, serum positive changes tend to occur only after years and often not until the thyroid gland shrinks from the normal 13 to 21 cc. volume in an average adult female and 15-23 cc. volume in male patients to below a volume of 6 cc. (Mayo Clinic averages) (Rumack, Carol). The normal serum analysis of patients for thyroid dysfunction, TSH, FT4, microsomal antibodies etc., the golden rule of most physicians and endocrinologists, is simply not an adequate means of ascertaining thyroid dysfunction in most M.E. patients. Repeat thyroid ultrasound must be performed for all M.E. patients to observe the presence of dystrophic changes. It is also inadequate simply to accept the radiologist’s report of a normal thyroid. The volume of each lobe and its homogeneity must be requested and documented. Radiologists simply report normal thyroids when in effect they are hypo and hyper-trophic. Although the Mayo Clinic averages cited above may be criticised they are as good as any in ascertaining normal thyroid size.

Skin conductivity and skin temperature tests: The combination of a lower ability of the skin to conduct electrical current in response to visual and auditory stimuli, and a higher skin temperature of fingers indicate a down regulation of autonomic sympathetic tone.

RNase L (37kDa 25A RNase L immunoassay: protein, activity, PKR cleavage and elastase activity assays): RNaseL test results change as M.E. progresses and so this test could possibly be used to monitor progression of the disease. Initially it might be high, but as it struggles with pathogens, it gets exhausted and will decrease. Eventually it shifts to the Low Molecular Weight (LMW) form, and then disappears. Up to 80% of patients have evidence of an up-regulated 2-5A antiviral pathway (Carruthers et al. 2003).

Note that RNaseL will be affected in any disease that affects the immune system in this way, so this test is not specific to M.E. and is certainly *not* diagnostic of M.E. This test is expensive, non-specific and not recommended.

Some M.E. patients also display increased activity of another enzyme called protein-kinase R (PKR) that is involved in killing cells infected with pathogens (NAME-US, 2008, [Online]). Cell-free DNA in blood plasma is also a basic test of severity of illness that can be used in diagnosing M.E. When cells are dying (abnormally) they release DNA, so high levels of cell degeneration indicate severe illness (eg. M.E., cancer, stroke, etc.) Again, note that this test is not specific to M.E.

- What does ‘RNase L’ mean in layman’s terms?: The immune system has 2 types of cells, T helpers 1 and 2, and in M.E. and those other illnesses which share this abnormality, there is a switch away from Th1 to Th2. Th1 is suppressed and Th2 is activated. (The weapons of the Th1 system are NK cells, which is why NK count is decreased in M.E.) The role of RNaseL is to prevent pathogens from reproducing, ‘holding the line’ while waiting for Th1 cells to come and kill the pathogens. The problem is, when Th1 is suppressed, Th1 never comes to the rescue. RNaseL struggles, gets worn out, and shifts to the exhausted ‘afterburner’ mode – the Low Molecular Weight form discovered by Suhadolnik. Eventually RNaseL disappears altogether. For more information see: Th1 and Th2, cancer and M.E. and Balance the Immune System (Th1/Th2).

Testable Major Sleep Dysfunction: This can include all forms of sleep dysfunctions. All or any of the following may be present: (a) impaired sleep efficiency, (b) significant fragmented sleep architecture, (c) movement arousals, particularly if there is an associated pain syndrome, (d) absence or significant decrease of type 3 and 4 sleep, (e) abnormal REM sleep pattern (f) changes in daytime alertness and (g) sleep reversals (Hyde 2007, [Online]).

Ocular tests: Slowed and marked jerkiness of saccades; difficulty with and slowed changing of visual fixation, constricted peripheral fields; low and/or incomplete blinking; small pupils; light hypersensitivity, tear film abnormalities such as low tear break-up time, inadequate production of the oil or mucous layer in tears, rose Bengal corneal staining, and visual midline shift (Hyde 2007, [Online]).

## Physical Exam

There are also abnormalities visible on physical exam in M.E. patients. These abnormalities are not usual in healthy patients but they are also found in people with other illnesses (so they are not specific to M.E.). In cases of severe or acute M.E. there are always definite physical signs indicative of physical illness, but virtually all patients will have some abnormality on physical exam. Not all patients will have all signs, and as fluctuation of symptoms from hour to hour and day to day is one of the key characteristics of M.E., *signs* of the illness may *also* change or fluctuate during the course of a day.

As Professor Malcolm Hooper explains: ‘a patient examined in the morning might have nystagmus, which would disappear at midday, recur later, disappear and recur the next day; thus a once-only cursory examination could be misleading’ (Hooper et al. 2001 [Online]).

Physical signs of illness commonly observed in M.E. patients include:

- Nystagmus; nystagmus is jelly-like and variable (15% of M.E. patients will have nystagmus)
- Sluggish visual accommodation
- Unequal pupils and contrary pupil reaction to light
- A labile blood pressure (sometimes as low as 84/48 in an adult at rest)
- Shortness of breath (particularly on exertion)
- Sometimes marked falling pulse pressure in arterial pressures taken first when prone, then sitting, then standing
- Rapid heart rate on minor activity such as standing
- Subnormal temperature
- Patients show significant reduction in all lung function parameters tested
- Liver involvement (an enlarged liver or spleen)
- Abnormal tandem or augmented tandem stance
- Abnormal gait
- Hand tremor
- Incoordination
- Cogwheel movement of the leg on testing
- Muscular twitching or fasciculation
- Hyper-reflexia without clonus
- Facial vasculoid rash
- Vascular demarcation which can cross dermatomes with evidence of Raynaud's syndrome and / or vasculitis and spontaneous periarticular bleeds in the digits
- Mouth ulcers
- Hair loss
- Destruction of fingerprints is sometimes seen (atrophy of fingerprints is due to perilymphocytic vasculitis and vacuolisation of fibroblasts)
- Ghastly pallor of face with frequent lupus-like submaxillary mask
- Parkinsonian rigidity of facial expression
- Scanning, disjointed speech, or speech reversals
- Nasal passage obstruction and inflamed areas around tonsillar pillars
- Sicca syndrome of conjunctiva and mucous membranes
- Frequent equivocal Babinski/plantar reflex on one side
- Unusual sensitivity of cervical vertebrae area
- Nodular thyroid (Hooper et al. 2001 [Online]) (Hyde, 2003).

In addition, the diagnosis of M.E. should never be made without the post-exertional paralytic muscle weakness which is *unique to M.E.* being present. M.E. authority [Dr Melvin Ramsay](#) explains that this unique symptom: ‘is virtually a sheet-anchor in the diagnosis of Myalgic Encephalomyelitis and without it a diagnosis should not be made.’

Muscles may function normally to begin with but there is a continued loss of post-exertional muscle power after even a minor degree of physical effort; three, four or five days, or longer, elapse before full muscle power is restored (n.d. [Online]). (In sedentary controls full muscle power is restored after just 200 minutes.) All muscles are affected, including the brain, the heart and the eyes and so on (Hooper et al. 2001 [Online]).

Thus a patient may be easily able to lift/push/squeeze something with close to normal strength several times, or for a short period; but be unable to complete the same task (or even a trivial task) *repeatedly* (such as lifting a soup spoon or a glass of water many times for example).

Dr Byron Hyde comments on testable muscle dysfunction in M.E. that:

This feature may be due to vascular dysfunction or peripheral nervous or spinal dysfunction and includes both pain and rapid loss of strength of muscle function after moderate physical or mental activity. This feature tends to improve over a period of years but many patients frequently remain permanently vulnerable to new disease episodes. Few centres are equipped or funded to make these examinations. Unfortunately only a few major medical centres are equipped to study this type of dysfunction (2007, [Online]).

Professor Malcolm Hooper recommends testing for this by observing the patient repeatedly lifting something weighing around 2 pounds or one kilogram for a period of time. He explains that, 'M.E. patients can often manage a small number of repeats but performance rapidly falls off and recovery is very slow compared to healthy controls. More sophisticated treadmill tests will also show the same effect.' This sudden onset muscle weakness (or paralysis) and very slow recovery is uniquely characteristic of M.E. patients (2003, [Online]).

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

- **An important note to patients:** Objective scientific tests *are* available which can aid in the diagnosis of M.E. and easily prove the severe abnormalities across many different bodily systems seen in M.E. Unfortunately many (in fact most) patients are not given access to these tests. Problems also exist with doctors not being familiar with the abnormalities on testing seen in M.E. and so misinterpreting the results of some tests.  
The problem is not that these tests don't exist, but that doctors – and many patients – are unaware of this information on testing, that it is not generally accepted due to the nefarious influence of political and financial vested interest groups, and that there are overwhelming financial and political incentives for researchers to IGNORE this evidence in favour of the bogus 'CFS' (or 'subgroups of 'ME/CFS') construct.  
See [Testing for M.E.: Plan D](#) for discussion of the ways in which patients seek a diagnosis in practice, and a 'Plan D' for patients who are forced to diagnose themselves.
- **A note on 'CFS' testing and redefinitions:** Whilst various 'fatiguing conditions' with a variety of different aetiologies may be made up of vague and mild 'everyday' symptoms, with no observable signs and no tests which have shown abnormalities or that can aid diagnosis, M.E. shares none of these characteristics. M.E. is not described by any of the definitions of 'CFS' or 'ME/CFS' (including the Canadian 'ME/CFS' definition or the ICC). *Many* patients can and do fit these (wastebasket) definitions that have diseases other than M.E.  
For more information see [What is M.E.?](#) and [Who benefits from 'CFS' and 'ME/CFS'?](#) See [The misdiagnosis of CFS](#) for information on why 'CFS' is always a misdiagnosis or non-diagnosis.
- The standard battery of tests is inadequate in revealing the abnormalities seen in M.E. These standard tests may be normal in up to 90% of M.E. patients.
- This text provides merely a brief overview of some of the tests available which together aid M.E. diagnosis, see. See the section below for more information on all aspects of diagnosis (including more in-depth information about each of these tests).
- Other tests which may also be useful include: gut permeability tests, antioxidant status, buspirone-prolactin tests, ACTH-cortisol response tests, cardiac PET scans, circulating red blood cell and serum volume, tests of mitochondrial function (ATP levels, ATP->ADP conversion efficiency, ADP->ATP recycling efficiency etc. at [Biolabs](#) in the UK for example.) among others. ADP to ATP conversion can be tested. However, positive test results on some of these tests are not conclusive proof of a correct diagnosis of M.E.: a patient must also have the essential symptoms and other characteristics of M.E. as some of these tests may also be positive/relevant in other illnesses. Such tests should only be used following a careful clinical history and thorough examination.
- It is not necessary that every test listed here be performed for the diagnosis to be made, nor that a patient have an abnormal result on all of these tests.
- Note that all tests listed here are not suitable for all patients; the risk/benefit ratio must always be taken into account with each patient. Some patients will also be too ill to undergo some tests altogether (25% of M.E. patients are severely affected and are wheelchair-bound, housebound and/or completely bedbound and can relapse severely or permanently with even a very minor or trivial level of physical activity).
- See [The misdiagnosis of CFS](#) for information on some of the illnesses which are commonly misdiagnosed as 'CFS' and what a diagnosis of 'CFS' based on any of the CFS definitions actually means.

- Just as the list of diagnostic tests and physical signs of M.E. given here are not exhaustive, this is also *not* an exhaustive list of the hundreds of studies which have found a wide variety of different abnormalities in M.E. patients, in many bodily systems. For more information about some of these other studies see: [ME: The Medical Facts](#) and [The Clinical and Scientific Basis of Myalgic Encephalomyelitis](#) (book). See [Research and Articles](#) also for many different articles and medical studies on M.E.

***A note from [Dr Byron Hyde](#) on the frequent misinterpretation of test results:***

When I was a medical student at the University of Toronto, our radiology professor insisted that as physicians, it was important to go over the actual X-Rays of the patient with the radiologist in order to develop an understanding of how to read an X-Ray and how to keep the radiologist aware of the pathology that you are investigating. Over the years I have had multiple reasons to visit a radiologist to assist me with reading routine X-Rays, complex intestinal X-rays, Ultrasounds, MRI and CT scans as well as brain SPECT and brain PET scans. I cannot recall a single time that the radiologist did not take the time to go over the actual scans and X-rays with me and answer my sometimes very rudimentary and facile questions. However these trips to the hospital have also made me realize that the radiologist can miss major problems since they are not always aware of the individual patient's pathology.

Recently, many SPECT scan and other technological facilities in Canada have simplified their technology, limited their findings to reports and failed to reproduce print-outs of their findings. This is true in Carotid and Transcranial Doppler examination where the velocity of blood flow through the arteries is not given, yet this is a valuable aid to understanding diseases related to arterial spasm. Yet the work sheets of the technicians contain this data.

The same is true of the reading of EEGs. Neurologists too often simply say a test is normal since there is no evidence of a seizure disorder or a large space occupying lesion. Often the neurologists go no deeper than this and miss major observable pathology. It is most unfortunate that so few centers have adopted QEEG or Beam technology, i.e. quantitative computer driven EEG technology. It gives significant better understanding of brain function abnormalities.

The same is true of brain SPECT scans. These are very easy to learn how to read. I have already mentioned the problem with dropping Xenon scans. But recently, some Canadian centres have lost their experts in brain nuclear medicine and replaced them with individuals who are not expert in reading brain SPECTs. They have also in some cases simplified the systems to maximize profit so that the detail is not always there. The hospital is paid the same for a badly done rushed SPECT as for an expert SPECT. This is increasingly a problem. For the physicians who only read the typed report stating, "the findings are normal" and who does not take the time to look at the brain images themselves, SPECT can be a useless exercise. I have mentioned the problem with thyroid ultrasound imaging. It is essential to insist that the radiologist actually give the measurements of each thyroid lobe rather than simply saying, "the findings are normal." This attention to detail is time consuming but also rewarding for the physician who is truly interested in understanding pathology (2007, [Online]).

***More information on all aspects of M.E. diagnosis***

For more information on diagnosing M.E.; including discussions of the hallmark symptoms and characteristics of M.E., to more information about many of the tests listed above; the following resources are highly recommended and are some of the best sources of information on M.E. available. Each of these texts has been produced primarily for doctors – but may also be accessible by less severely affected sufferers (and their families or carers).

The paper by Dr Byron Hyde entitled [The Nightingale Definition of Myalgic Encephalomyelitis](#) is *the most essential paper* on this topic and is essential reading for any doctor with an interest in the correct diagnosis of M.E. This paper cannot be recommended highly enough.

It is also recommend that doctors read this paper together with [The Complexities of Diagnosis and A New and Simple Definition of Myalgic Encephalomyelitis and a New Simple Definition of Chronic Fatigue Syndrome & A Brief History of Myalgic Encephalomyelitis & An Irreverent History of Chronic Fatigue Syndrome](#) by Dr Byron Hyde.

**The Nightingale Definition of Myalgic Encephalomyelitis (M.E.) by Dr Byron Hyde 2006**

This is a modern definition of Myalgic Encephalomyelitis, created by the world's preeminent and most experienced M.E. expert, Dr Byron Hyde. This is *not* a redefinition of CFS but is instead a pure M.E. definition, something M.E. patients, advocates and researchers have long been waiting for.

It draws on the long history of M.E., collates the evidence from each of the world's leading M.E. experts (past and present) and combines this with details of the most modern medical tests. This definition also rightly gives no importance at all to the bogus notion of mere 'fatigue' having any importance in the diagnosis/definition of M.E. – unlike each of the 'CFS' definitions, including unfortunately the Canadian 'ME/CFS' definition which unfortunately mixes in a few M.E. facts with what is still primarily a 'CFS' redefinition. Dr Hyde observes that:

I believe it essential to define clearly Myalgic Encephalomyelitis, returning the definition to its clinical and historic roots and complementing this information with the certitude of modern scientific testing. That is what the Nightingale definition of M.E. sets out to do. But let me first ask you a very important question.

What is the purpose of any medical definition? What is the purpose of any disease definition if it is not to allow the physician to rapidly and accurately diagnose a specific illness in order to attempt to effectively treat the patient before the illness becomes chronic or to call in the appropriate specialists? Our definition solves this problem.

What then is the purpose of any disease definition, once the disease has become chronic, if it is not (a) to elicit clues for the immediate effective treatment of at least some of the patients, (b) to separate out illnesses with a similar symptom pictures in order to effectively treat them and finally (c) to direct research into reversing pathophysiological injuries that can be defined in terms of modern testing but for which, there is no effective treatment. Our definition solves this problem.

There is a third purpose for any disease definition. That is to clearly define the disease so that various physicians and researchers can clearly understand that they are talking about the same illness spectrum and so launch research into what will become an effective treatment. Our definition gives a clear baseline for investigation.

The Nightingale definition is based upon the following two criteria: (a) The excellent scientific and clinical work of respected physicians and scientists who investigated the various M.E. epidemics. (b) The results of modern scientific testing techniques and the knowledge accruing from examining thousands of M.E. patients using these and more historical techniques. The proposed M.E. definition is designed to improve early diagnosis and treatment for the tens of thousands of patients stricken with M.E. It is not a new definition of CFS nor should it be conceived as a rewording of any previous CFS definition.

The definition is set out in such a fashion as to enable the physician to make a bedside or office clinical diagnosis and then to scientifically test the hypothesis. This will allow the physician an early diagnostic understanding of this complex illness and a scientific and technological method to investigate and confirm the diagnosis. It is well known by all serious physicians that in order to assist any patient in a partial or full recovery the illness must be (a) prevented from occurring by either immunization or understanding and avoiding the causes, (b) or diagnosed and treated immediately following onset. The Nightingale Definition assists the physician both in diagnosis and early treatment.

What follows is the primary M.E. definition for adults. I believe it essential to define clearly Myalgic Encephalomyelitis. That is what the Nightingale definition of M.E. sets out to do ... To various degrees many if not all of the above historic findings have been observed and discussed by Doctors Alexander Gilliam, Bjorn Sigurdsson, Alberto Marinacci, Andrew Lachlan Wallis, A Melvin Ramsay (Elizabeth Dowsett), John Richardson, Elizabeth Bell, Alexis Shelokov, David C Poskanzer, W.H. Lyle, Sir E. Donald Acheson, Louis Leon-Sotomayor, J. Gordon Parish and many others.

**Again, this comprehensive text is also essential reading for any doctor with an interest in diagnosing M.E. correctly, and cannot be recommended highly enough.**

- Read more articles by [Dr Byron Hyde](#)
- This Nightingale Research Foundation's Definition will be available with any updates or corrections, on the Nightingale Research Foundation's Website at [www.nightingale.ca](http://www.nightingale.ca)

This definition may be copied, translated, distributed by electronic or hard copy and may be included, in whole or in part in any publication without permission from the Nightingale Research Foundation or the authors, provided that this last paragraph and referral back to our website are noted. A copy of any translation should be sent to Nightingale for possible inclusion in our website.

### **The Complexities of Diagnosis** by Dr Byron Hyde

A comprehensive text by leading M.E. expert Dr Byron Hyde which concentrates solely on all aspects of diagnosis. This text is a chapter taken from the book: Handbook of Chronic Fatigue Syndrome by Leonard A. Jason et al. but is now also available online as a free PDF. This text should be essential reading for any doctor with an interest in diagnosing M.E. correctly.

**A New and Simple Definition of Myalgic Encephalomyelitis and a New Simple Definition of Chronic Fatigue Syndrome & A Brief History of Myalgic Encephalomyelitis & An Irreverent History of Chronic Fatigue Syndrome** by Dr Byron Hyde 2003

Dr Hyde writes:

Do not for one minute believe that CFS is simply another name for Myalgic Encephalomyelitis (M.E.). It is not. Though CFS is based upon a typical M.E. epidemic, in my opinion it has always been a confused and distorted view of reality. The invention of Chronic Fatigue Syndrome has to be one of the most curious cases of inventive American scientific imperialism that one could imagine. It is my opinion that the CDC 1988 definition of CFS describes a non-existing chimera based upon inexperienced individuals who lack any historical knowledge of this disease process. The CDC definition is not a disease process.

This text is also essential reading for any doctor with an interest in diagnosing M.E. correctly.

**A simplified definition of Myalgic Encephalomyelitis**  
**Myalgic Encephalomyelitis is:**

**1 A variable and biphasic acute onset disease**

**2 Primary Infection Phase:** The first phase is an epidemic or endemic infectious disease generally with an incubation period of 4 to 7 days, where in most (but not all) cases an infection is evident.

**3 Chronic Phase:** The second and chronic phase follows closely on the first phase, usually within two to seven days, and is characterized by a measurable diffuse change in the function of the CNS. This is the persisting disease that most characterizes M.E. and is demonstrated by the following:

**4 Testable Brain Changes:** This second phase becomes chronic and is characterized by various measurable and clinical dysfunctions of the cortical or cortical and sub cortical brain. If the patient's illness is not persistently measurable using SPECT, PET or QEEG and/or Neuropsychological changes then it is not M.E. These changes can be roughly characterized as to severity:

1. **Type 1:** where one side of the cortex is involved. These patients have the best chance of spontaneous recovery.
2. **Type 2:** where both sides of the cortex are involved: These patients have the least chance of spontaneous recovery.
3. **Type 3:** where both sides of the cortex, and either one or all of the posterior chamber organs, the Pons and Cerebellum, the sub cortical and brain stem structures are involved. Type 3 are the most severely affected patients and the most likely to be progressive or see little or no improvement with time.

**5 Pain Syndromes:** The pain syndromes associated with the acute and chronic phases of M.E. may include (a) severe headaches of a type never previously experienced, (b) often associated with neck rigidity and occipital pain, (c) retro-orbital eye pain, (d) migratory muscle and arthralgia pain, (e) cutaneous hypersensitivity and (f) fibromyalgia type pain. These pain syndromes tend to decrease over time.

**6 Neuropsychological Changes:** There are neuropsychological changes that are measurable and demonstrate short-term memory loss, cognitive dysfunctions, increased irritability, confusion, and perceptual difficulties. There is usually rapid decrease in these functions after any physical or mental activity. This feature may improve over a period of years in patients with adequate financial and social support.

**7 Major Sleep Dysfunction:** including all forms of sleep dysfunction and day time alertness and sleep reversals.

**8 Muscle Dysfunction:** This feature may be due to vascular dysfunction or peripheral nervous or spinal dysfunction and includes both pain and rapid loss of strength of muscle function after moderate physical or mental activity.

**9 Vascular Dysfunction:** This is the most obvious dysfunction when looked for and probably is the cause behind a significant number of the above complaints. Vascular change is most evident in patients with:

- a. **POTS:** severe postural hypotension.
- b. **Cardiac irregularity:** on minor positional changes or after minor physical activity, including inability for the heart to increase or decrease in speed and pump volume in response to increase or decrease in physical activity.

c. **Raynaud's Disease:** vasoconstriction, blanching, coldness and pain of extremities. This is in part the cause for temperature dysfunctions seen in M.E.

d. **Bowel Dysfunction:** vascular dysfunction may be the single most causal basis behind bowel dysfunction when it occurs

**10 Endocrine Dysfunction:** This feature is common and tends to be a late appearance and is most obvious in the:

a. **Pituitary-thyroid axis:** This is common. Changes in serum TSH, FTI, FT4, Microsomal Ab., PTH, Calcium and phosphorus rarely occur until one or more years after illness onset and usually only after several years. This can be followed by ultrasound of the thyroid gland where a steady shrinking of the thyroid gland occurs with or without the development of non-serum positive Hashimoto's thyroiditis (a seeming contradiction of terms) and a significant increase in thyroid malignancy. Serum positive changes occur only after years.

b. **Pituitary-adrenal axis changes:** this finding is infrequent.

c. **Pituitary-ovarian axis changes:**

d. **Pituitary-Bladder dysfunction:** occurs frequently in the early disease in some people.

### **The Canadian Clinical Case Definition for ME/CFS** by Dr Bruce Carruthers et al

These guidelines are problematic. In a nutshell, this is yet another redefinition of 'CFS' that has had a small number of facts about neurological M.E. tacked onto it. Yes, it selects a more severely ill and less-likely-to-be-mentally-ill patient group than other 'CFS' definitions, but just like these other 'CFS' definitions, it cannot be said to select a 100% homogenous patient group consisting solely of M.E. patients (or even any other distinct patient group). As with all other 'CFS' definitions, it selects a mixed or heterogeneous patient group – and of course studies involving heterogeneous patient groups define nothing and help nobody; especially not any of the various patient groups involved.

The problem with this definition is that it focuses on mere fatigue instead of the genuine symptomatology and pathology that distinguishes M.E. from many other illnesses with which may simply share a few symptoms. It is entirely possible that patients with non-M.E. illnesses such as Lyme disease, Fibromyalgia, MCS and many others may again be subsumed under this vague and mostly fatigue or 'post exertional malaise' based definition. Many illnesses present with fatigue, and what might be described as 'post exertional malaise' and do worse with exercise. But in no way do these patients suffer the same symptomatology, pathology and disability as genuine M.E. patients, and they are *not* genuine M.E. patients. (For more information see: [What is Myalgic Encephalomyelitis?](#))

Not only does this definition fail to select a 100% M.E. patient population, it is also entirely possible that research could be conducted using this definition which contains *no M.E. patients at all*, which makes this definition a dangerous tool just waiting to be used against the best interests of patients and science by vested interest groups. *It should not and cannot be considered a definition of M.E.* It is at best an unscientific blend of M.E. and 'CFS'. Furthermore, the definition unfortunately makes use of the confusing and misleading term 'ME/CFS' which gives the false and harmful impression that M.E. and 'CFS' are essentially the same. This is hardly helpful when the number one goal of advocacy is to make people aware of the fact that M.E. is NOT 'CFS.'

The treatment advice associated with this definition is very weak, so much so as to be useless and even dangerous, as is the section describing the symptoms which supposedly define 'ME/CFS' (which are very different from those that define genuine M.E.) The case definition also misrepresents the connection between "ME/CFS" and Fibromyalgia: which is nowhere near as marked as stated. However, the research section is useful to some degree as is the three page section on how inappropriate CBT and GET are for M.E. patients, and the section which describes some of the tests which can be used to confirm the presence of the illness.

This paper confuses the issue of correct diagnosis for many patients, in the same way as each of the other 'CFS' definitions. This is unacceptable.

M.E. is not described by any of the definitions of 'CFS' or 'ME/CFS' (including the Canadian 'ME/CFS' definition or the updated version the ICC). *Many* patients can and do fit these (wastebasket) definitions that have diseases other than M.E.

- A summary of the clinical criteria is [available online](#).
- A PDF document containing the diagnosis and research summary sections of the definition is also [available for free online](#)
- Read [Comments from the Canadian Guidelines on Cognitive Behavior Therapy \(CBT\) and Graded Exercise Therapy \(GET\)](#)

- A summary of the entire document is also available in PDF format [online](#).
- Printed copies of the full text are available from [www.haworthpressinc.com](http://www.haworthpressinc.com)
- See also the new pure (and by far superior) M.E. definition by Dr Hyde [The Nightingale Definition of M.E.](#) This is NOT a redefinition of 'CFS' as are the Canadian Guidelines, but is a genuine testable M.E. definition.
- Read more about the limitations of the Canadian Guidelines at: [Canadian Guidelines Review](#) and [The Definitions of M.E.](#)

### **The Clinical and Scientific Basis of M.E.** Edited by Dr Byron Hyde

This textbook is essential reading for anyone with an interest in M.E. as it contains the accumulated knowledge of 80 of the worlds leading M.E. experts in one 725-page encyclopaedia. This book is a comprehensive account of current knowledge concerning the history, epidemiology, issues of diagnosis, children with M.E., investigation, virology, immunology, muscle pathology, host response, food intolerance, brain mapping, neurophysiology, neuropsychology, sleep dysfunction, treatment and management.

This is a simply essential reference book for doctors and M.E. patients and easily surpasses all others of its type (as there really *are* no others of its type). See The Nightingale Foundation Website and the Review of this book for more information and purchasing details. All funds from the purchase of this book also benefit further research into the illness and assist in the promotion of greater understanding about the illness.

### **Engaging with M.E.** by Professor Malcolm Hooper

This text contains an overview of much of the medical knowledge of M.E., including a detailed discussion of diagnosis and treatments for the illness. Hard copies may be obtained in the UK (price £4.00 plus £1.10 postage) from Malcolm Hooper, Emeritus Professor of Medicinal Chemistry, School of Sciences, University of Sunderland, Sunderland, SR2 3SD, UK. A DVD which contains some of this information (although not all) is also available for free online.

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#### ***\*A further note on terminology and definitions:***

The various definitions of 'CFS' *do not* recognise Myalgic Encephalomyelitis as an organic neurological disorder as defined at G.93.3 in the World Health Organization's International Classification of Diseases (ICD). The definitions of 'CFS' do not reflect this. The 'CFS' definitions are not 'watered down' M.E. definitions, as some claim. They are not definitions of M.E. at all.

Ever since an outbreak of M.E. in the US was mislabelled as 'CFS,' the name/definition 'CFS' has prevailed for political reasons. 'CFS' is widely though inappropriately applied to M.E. as well as to other diseases.

The overwhelming majority of 'CFS' research does not involve M.E. patients and is not relevant *in any way* to M.E. patients. However, a very small amount of research published under the name 'CFS' clearly does involve a significant number of M.E. patients as it details those abnormalities which are unique to M.E. (see the end of the references section for more on this topic.)

*It is important to be aware that M.E. and CFS are not synonymous terms.* For more information see [What is M.E.?](#) See also [Smoke and Mirrors](#) for a discussion of why the bogus disease category 'CFS' must be abandoned.

Of course this 'CFS' and M.E. confusion must be stopped. It is unreasonable in this day and age that studies on a vague, mixed patient group be used to determine the treatments and aetiology of an entirely different and unrelated and distinct patient group. The only way forward, for the benefit of society and *every* patient group involved, is that:

**1. The bogus disease category of 'CFS' must be abandoned completely.** Patients with fatigue (and other symptoms) caused by a variety of different illnesses need to be diagnosed correctly with these illnesses if they are to have any chance of recovery; not given a meaningless Oxford or Fukuda 'CFS' misdiagnosis. Patients with M.E. need this same opportunity. Each of the patient groups involved must again be correctly diagnosed and then treated as appropriate based on legitimate and unbiased science involving the *same* patient group.

2. The name Myalgic Encephalomyelitis must be fully restored (to the exclusion of all others) and the World Health Organization classification of M.E. (as a distinct neurological disease) must be accepted and adhered to in all official documentations and government

People with M.E. must also be given access to basic medical care, financial support and other appropriate services on an equal level to what is available for those with comparable illnesses (eg. multiple sclerosis or lupus). Funding should be made available for legitimate M.E. research involving 100% M.E. patient populations. The facts about M.E. must be taught to medical students, and must continue to be published in mainstream medical journals.

### 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 M.E. patients (sporadic and epidemic), as well as each publishing numerous studies, articles and books relating to M.E. Again, more experienced, more knowledgeable and more credible M.E. experts than these simply do not exist.

This paper is 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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**Before reading this research/advocacy information, please be aware of the following facts:**

1. Myalgic Encephalomyelitis and 'Chronic Fatigue Syndrome' are not synonymous terms. The overwhelming majority of research on 'CFS' or 'CFIDS' or 'ME/CFS' or 'CFS/ME' or 'ICD-CFS' does not involve M.E. patients and is not relevant *in any way* to M.E. patients. However, if the M.E. community were to reject all 'CFS' labelled research as '*only* relating to 'CFS' patients' (including research which describes those abnormalities/characteristics unique to M.E. patients), this would seem to support the myth that 'CFS' is just a 'watered down' definition of M.E. and that M.E. and 'CFS' are virtually the same thing and share many characteristics.

A very small number of 'CFS' studies/articles and books refer in part to people with M.E. but it may not always be clear which parts refer to M.E. The [A warning on 'CFS' and 'ME/CFS' research and advocacy paper](#) is recommended reading and includes a checklist to help readers assess the relevance of individual 'CFS' studies (etc.) to M.E. (if any) and explains some of the problems with this heterogeneous and skewed research.

2. The research referred to on this website varies considerably in quality. Some is of a high scientific standard and relates wholly to M.E. and uses the correct terminology. Other studies are included which may only have partial or minor possible relevance to M.E., use unscientific terms/concepts such as 'CFS,' 'ME/CFS,' 'CFS/ME,' 'CFIDS' or Myalgic 'Encephalopathy' and also include a significant amount of misinformation. Before reading this research it is also essential that the reader be aware of the most commonly used 'CFS' propaganda, as explained in [A warning on 'CFS' and 'ME/CFS' research and advocacy](#) and in more detail in [Putting Research and Articles on Myalgic Encephalomyelitis into Context](#).

**Acknowledgments:** Thank you to Emma Searle for editing this paper.

**Disclaimer:** The HFME does not dispense medical advice or recommend treatment, and assumes no responsibility for treatments undertaken by visitors to the site. It is a research-information and advocacy resource only. Please consult your own health-care provider regarding any medical issues relating to the diagnosis or treatment of any medical condition.

### **Relevant quotes**

“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 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

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

Dr Melvin Ramsay on Myalgic Encephalomyelitis: "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."

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

‘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

**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.

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

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- Myalgic Encephalomyelitis (M.E.) is a disabling neurological disease that is very similar to Multiple Sclerosis (M.S.) and Poliomyelitis. Earlier names for M.E. were 'atypical Multiple Sclerosis' and 'atypical Polio.'
- M.E. is a neurological disease characterised by scientifically measurable post-encephalitic damage to the brain stem. This damage is an essential part of 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, tis = 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. M.E. is classified in the current WHO International Classification of Diseases with the neurological code G.93.3.
- M.E. 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.
- M.E. 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.
- M.E. can be more disabling than M.S. or Polio, and many other serious diseases. M.E. is one of the most disabling diseases that exists. 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 M.E. 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 and 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 (e.g. 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.*
- M.E. 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 (e.g. MRI and SPECT brain scans). Abnormalities are also visible on physical exam in M.E. M.E. is a long-term/lifelong neurological disease that affects hundreds of thousands of adults and children worldwide. In some cases M.E. is fatal.



This paper is included in the new **Caring for the M.E. Patient** book by Jodi Bassett.

The book also includes a Foreword by the world's most experienced M.E. expert Dr Byron Hyde and is essential reading for anyone with an interest in M.E.

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