

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.

M.E.: The medical facts

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Myalgic Encephalomyelitis (M.E.) is a debilitating neurological disease which has been recognised by the World Health Organisation (WHO) since 1969 as a distinct organic neurological disorder. M.E. can occur in both epidemic and sporadic forms, over 60 outbreaks of M.E. have been recorded worldwide since 1934. M.E. is similar in a number of significant ways to illnesses such as multiple sclerosis, Lupus and poliomyelitis (polio). M.E. can be extremely severe and disabling and in some cases the disease is fatal.

Is Myalgic Encephalomyelitis a new illness? What does the name M.E. mean?

The illness we now know as Myalgic Encephalomyelitis is not a new illness. M.E. is thought to have existed for centuries. (Hyde 1998, [Online]) (Dowsett 1999a, [Online])

In 1956 the name Myalgic Encephalomyelitis was created. The term was invented jointly by Dr A Melvin Ramsay who coined this name in relation to the Royal Free Hospital epidemics that occurred in London in 1955 - 1957 and by Dr John Richardson who observed the same type of illness in his rural practice in Newcastle-upon-Tyne area during the same period. It was obvious to these physicians that they were dealing with the consequences of an epidemic and endemic infectious neurological disease (Hyde 1998, [Online]) (Hyde 2006, [Online]). The term Myalgic Encephalomyelitis means: My = muscle, Algic = pain, Encephalo = brain, Mye = spinal cord, Itis = inflammation (Hyde 2006, [Online]). As M.E. expert Dr Byron Hyde writes:

The reason why these physicians were so sure that they were dealing with an inflammatory illness of the brain is that they examined patients in both epidemic and endemic situations with this curious diffuse brain injury. In the epidemic situation with patients falling acutely ill and in some cases dying, autopsies were performed and the diffuse inflammatory brain changes are on record (2006, [Online]).

Professor Malcolm Hooper explains that:

The term myalgic encephalomyelitis has been included by the World Health Organisation (WHO) in their International Classification of Diseases (ICD), since 1969. The current version ICD-10 lists M.E. under G.93.3 - neurological conditions. It cannot be emphasised too strongly that this recognition emerged from meticulous clinical observation and examination (2006, [Online]).

The recorded medical history of M.E. as a debilitating organic neurological illness affecting children and adults is substantial; it spans over 70 years and has been published in prestigious peer-reviewed journals all over the world (Hooper 2003, [Online]). The term M.E. has stood the test of time for more than 50 years. As microbiologist and M.E. expert Dr Elizabeth Dowsett explains: 'There is ample evidence that M.E. is primarily a neurological illness, although non-neurological complications affecting the liver, cardiac and skeletal muscle, endocrine and lymphoid tissues are also recognised' (n.d.b, [Online]).

What is Myalgic Encephalomyelitis? What defines M.E.?

Myalgic encephalomyelitis is a systemic acutely acquired illness initiated by a virus infection which is characterised by post encephalitic damage to the brain stem; a nerve centre through which many spinal nerve tracts connect with higher centres in the brain in order to control all vital bodily functions – this is always damaged in M.E. (Hence the name Myalgic Encephalomyelitis.) The CNS is diffusely injured at several levels, these include the cortex, the limbic system, the basal ganglia, the hypothalamus and areas of the spinal cord and its appendages. This persisting multilevel central nervous system (CNS) dysfunction, and in particular, inconsistent CNS dysfunction is undoubtedly both the chief cause of disability in M.E. and the most critical in the definition of the entire disease process.

Myalgic Encephalomyelitis represents an acute change in the balance of neuropeptide messengers, and due to this, a resulting loss of the ability of the CNS (the brain) to adequately receive, interpret, store and recover information which enables it to control vital body functions (cognitive, hormonal, cardiovascular, autonomic and sensory nerve communication, digestive, visual auditory balance etc). It is a loss of normal internal homeostasis. The problem is one of maintaining systemic functioning within normal limits in the face of a chronic infectious stress. The resulting loss of normal internal homeostasis arises from the fact that a chronic viral infection provokes reactive changes in these peptides with the consequence of pathophysiological changes and autonomic

dysregulation. These powerful excitatory and inhibitory mechanisms for rapid physiological adjustment work well with short-term stressors. Frequently, these mechanisms shut down the biological system allowing for compensatory adjustments of the homeostatic mechanisms. When the stressor is an infectious agent, the messenger mechanisms stimulate compensating immune reactions to rid the body of this stressor and ultimately return the individual to a normal internal homeostasis. By definition, chronic infections have managed to escape these initial compensatory immune mechanisms. However, the neurochemical homeostatic events continue to be employed uselessly and to the detriment of the organism. This modulatory biochemical complex, biologically derived over the millennium to assist the organism, destabilises the autonomic neuronal outflow and the individual can no longer function systemically within normal limits. This dysfunction also results in the inability of the CNS to consistently programme and achieve normal smooth end organ response.

M.E. is primarily neurological, but because the brain controls all vital bodily functions virtually every bodily system can be significantly affected by M.E. Again, although M.E. is primarily neurological it is also known that the vascular and cardiac dysfunctions seen in M.E. are also the cause of many of the symptoms and much of the disability associated with M.E. – and that the well-documented mitochondrial abnormalities present in M.E. significantly contribute to both of these pathologies. There is also multi-system involvement of cardiac and skeletal muscle, liver, lymphoid and endocrine organs in M.E. Some individuals also have damage to skeletal and heart muscle. Thus Myalgic Encephalomyelitis symptoms are manifested by virtually all bodily systems including: cognitive, cardiac, cardiovascular, immunological, endocrinological, respiratory, hormonal, gastrointestinal and musculo-skeletal dysfunctions and damage.

M.E. is an infectious neurological disease and represents a major attack on the central nervous system (CNS) – and an associated injury of the immune system – by the chronic effects of a viral infection. There is also transient and/or permanent damage to many other organs and bodily systems (and so on) in M.E. M.E. affects the body systemically. Even minor levels of physical and cognitive activity, sensory input and orthostatic stress beyond a M.E. patient's individual post-illness limits causes a worsening of the severity of the illness (and of symptoms) which can persist for days, weeks or months or longer. In addition to the risk of relapse, repeated or severe (individually determined) overexertion can also cause permanent damage (eg. to the heart), disease progression and/or death in M.E. (See the sections below for more information.)

It is the combination of the chronicity, the dysfunctions, and the instability, the lack of dependability of these functions, that creates the high level of disability in M.E. It is also worth noting that of the CNS dysfunctions, cognitive dysfunction is one of the most disabling characteristics of M.E. (Chabursky et al. 1992 p. 20) (Hyde 2007, [Online]) (Hyde 2006, [Online]) (Hyde 2003, [Online]) (Dowsett 2001a, [Online]) (Dowsett 2000, [Online]) (Dowsett 1999a, 1999b, [Online]) (Hyde 1992 pp. x-xxi) (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.a, [Online]) (Dowsett & Ramsay n.d., pp. 81-84) (Richardson n.d., pp. 85-92).

All of this is not simply theory, but is based upon an enormous body of mutually supportive clinical information which has been published in prestigious peer-reviewed journals all over the world and spans over 70 years. Modern technology has now served to confirm and to detail the meticulous clinical and scientific observations made about M.E. before 1988. Confirmation of this hypothesis is now supported by electrical tests of muscle and of brain function (CT, MRI, SPECT and PET scans) and by biochemical and hormonal assays. Many aspects of the pathophysiology of the disease have been medically explained in volumes of research articles (Hyde 2007, [Online]) (Hyde 2006, [Online]) (Hooper 2006, [Online]) (Hyde 2003, [Online]) (Dowsett 2001a, 2001b, [Online]) (Hooper et al. 2001, [Online]) (Dowsett 2000, [Online]) (Dowsett 1999a, 1999b, [Online]) (Hyde 1992 p. xi) (Hyde & Jain 1992 pp. 38 - 43).

- *What is Homeostasis?* Homeostasis is the property of a living organism, to regulate its internal environment to maintain a stable, constant condition, by means of multiple dynamic equilibrium adjustments, controlled by interrelated regulation mechanisms. Homeostasis is one of the fundamental characteristics of living things. It is the maintenance of the internal environment within tolerable limits.

What are some of the symptoms of Myalgic Encephalomyelitis?

More than 64 distinct symptoms have been authentically documented in M.E. At first glance it may seem that every symptom possible is mentioned, but although people with M.E. have a lot of different minor symptoms because of the way the central nervous system (which controls virtually every bodily system) is affected, the major symptoms of M.E. really are quite distinct and almost identical from one patient to the next. Different people have a lot of different symptoms but the general pattern and evolution of major symptoms are remarkably coherent from patient to patient in M.E. (Hooper & Montague 2001, [Online]) (Hyde 2006, [Online]) Individual symptoms of Myalgic Encephalomyelitis include:

Sore throat, chills, sweats, low body temperature, low grade fever, lymphadenopathy, muscle weakness (or paralysis), muscle pain, muscle twitches or spasms, gelling of the joints, hypoglycaemia, hair loss, nausea, vomiting, vertigo, chest pain, cardiac arrhythmia, resting tachycardia, orthostatic tachycardia, orthostatic fainting or faintness, circulatory problems, ophthalmoplegia, eye pain, photophobia, blurred vision, wavy visual field, and other visual and neurological disturbances, hyperacusis, tinnitus, alcohol intolerance, gastrointestinal and digestive

disturbances, allergies and sensitivities to many previously well-tolerated foods, drug sensitivities, stroke-like episodes, nystagmus, difficulty swallowing, weight changes, paresthesias, polyneuropathy, proprioception difficulties, myoclonus, temporal lobe and other types of seizures, an inability to maintain consciousness for more than short periods at a time, confusion, disorientation, spatial disorientation, disequilibrium, breathing difficulties, emotional lability, sleep disorders; sleep paralysis, fragmented sleep, difficulty initiating sleep, lack of deep-stage sleep and/or a disrupted circadian rhythm.

Neurocognitive dysfunction may include cognitive, motor and perceptual disturbances. Cognitive dysfunction may be pronounced and may include; difficulty or an inability to speak (or understand speech), difficulty or an inability to read or write or to do basic mathematics, difficulty with simultaneous processing, poor concentration, difficulty with sequencing and problems with memory including; difficulty making new memories, difficulty recalling formed memories and difficulties with visual and verbal recall (eg. facial agnosia). There is often a marked loss in verbal and performance intelligence quotient (IQ) in M.E. (Bassett 2009, [Online]).

- For a more complete symptom list see: [The Ultra-comprehensive Myalgic Encephalomyelitis Symptom List](#)
- See also: [What it feels like to have M.E.: A personal M.E. symptom list and description of M.E.](#)

What other features define or characterise Myalgic Encephalomyelitis?

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

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

A summarised explanation of why Myalgic Encephalomyelitis patients are so severely and uniquely disabled, taken from the 'A one-page summary of the facts of Myalgic Encephalomyelitis' text, is as follows:

For a person to stay alive, the heart must pump a certain base-level amount of blood. Every time a person is active, this increases the amount of blood the heart needs to pump. Every movement made or second spent upright, every word spoken, every thought thought, every word read or noise heard requires that more blood must be pumped by the heart.

However, the hearts of M.E. patients only pump barely enough blood for them to stay alive. Their circulating blood volume is reduced by up to 50%*. Thus M.E. patients are severely limited in physical, cognitive and orthostatic (being upright) exertion and sensory input.

This problem of reduced circulating blood volume, leading to cardiac insufficiency, is why every brief period spent walking or sitting, every conversation and every exposure to light or noise can affect M.E. patients so profoundly. Seemingly minor 'activities' can cause significantly increased symptom severity and/or disability (often with a 48-72 hour delay in onset), prolonged relapse lasting months, years or longer, permanent bodily damage (eg. heart damage or organ failure), disease progression or death.

If activity levels exceed cardiac output by even 1%, death occurs. Thus the activity levels of M.E. patients must remain strictly within the limits of their reduced cardiac output just in order for them to stay alive. *M.E. patients who are able to rest appropriately and avoid severe or prolonged overexertion have repeatedly been shown to have the most positive long-term prognosis* (Hyde 2007, [Online]) (Hyde 2003, [Online]) (Cheney 2006, [video recording]) (Hyde & Jain 1992a, pp. 375-383) (Ramsay 1986, [Online]) (Peckerman et al. 2003, [Online]) (Dowsett 2002b, [Online]) (Hyde 1992 p. xi) (Hyde & Jain 1992 pp. 38 - 43) (Dowsett 2001, [Online]) (Dowsett 2001a, [Online]) (Dowsett 2000, [Online]) (Dowsett 1999a, [Online]) (Dowsett 1999b, [Online]) (Dowsett & Ramsay et al. 1990, pp. 285-291) (Dowsett & Ramsay n.d., pp. 81-84) (Dowsett n.d., [Online]) (Dowsett n.d.b, [Online]). (Dowsett n.d.d, [Online]) (Ryll 1994, [Online]) (Jones 1998, [Online]) (25% M.E. Group 2005, [Online]) (25% M.E. Group 2004, [Online]) (MESA 2007, [Online]) (Marshall & Williams 2005d, [Online]) (Williams 2004, [Online]).

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- *A note about low circulating blood volume findings in M.E.* Dr Byron Hyde explains that circulating blood volume levels of 60 - 70% of expected are very common in M.E. and that readings as low as 50% are common in more severely affected patients. However, it should be noted that as the vast majority of those with very severe M.E. are of course far too ill to have this test and so they are not represented in these statistics. Thus it should not be assumed that this 50% percentage represents the 'worst case scenario' as regards reduced circulating blood volume test results in M.E. The findings from patients with very severe M.E. (if these patients could safely be tested) would likely be even more startling. For more information on this test see: [Testing for M.E.](#)

What causes Myalgic Encephalomyelitis?

M.E. expert [Dr Byron Hyde](#) explains that:

[The] onset illness usually takes the form of either, or any combination, of the following, (a) an upper respiratory illness, (b) a gastrointestinal upset, (c) vertigo and (d) a moderate to severe meningitic type headache. These are only the most common onset illnesses or symptoms of which there are several. The onset illness is associated with either a low grade or subnormal temperature, headaches, sometimes persisting and accentuated by movement with intermittent attacks of vertigo or dizziness. The second and third phases of the illness are usually always different in nature from the onset illness and usually become apparent within 1-4 weeks after the onset of the infectious triggering illness (1998 [Online]).

What causes Myalgic Encephalomyelitis? Are there outbreaks of M.E.?

One of the most fundamental facts about M.E. throughout its history is that it occurs in epidemics. This fact conveys, among other things, the infectious and contagious nature of the disease. (Hyde 1998, [Online]) The usual incubation period of the virus infection involved is 4-7 days. There is a history of over sixty recorded outbreaks of the illness going back to 1934 when an epidemic of what seemed at first to be poliomyelitis was reported in Los Angeles. As with many of the other M.E. outbreaks the Los Angeles outbreak occurred during a local polio epidemic.

The presenting illness resembled polio and so for some years the illness was considered to be a variant of polio and classified as 'Atypical poliomyelitis' or 'Non-paralytic polio' (TCJRME 2007, [Online]) (Hyde 1998, [Online]) (Hyde 2006, [Online]). Many early outbreaks of M.E. were also individually named for their locations and so we also have outbreaks known as Tapanui flu in New Zealand, Akureyri or Icelandic disease in Iceland, Royal Free Disease in the UK, and so on (TCJRME 2007, [Online]) (Hyde 1998, [Online]). A review of early M.E. outbreaks found that clinical symptoms were consistent in over sixty recorded epidemics spread all over the world (Hyde 1998, [Online]). Despite the different names being used, these were repeated outbreaks of the same illness. It was also confirmed that the epidemic cases of M.E., and the sporadic cases of M.E. each represented the same illness (Hyde 2006, [Online]) (Dowsett 1999a, [Online]).

Myalgic Encephalomyelitis is an acutely acquired neurological illness (with systemic effects) initiated by a virus infection. This point of view is supported by **history** (M.E. epidemics have followed polio epidemics and serological studies have shown that communities affected by an outbreak of M.E. were effectively blocked (or immune) from the effects of a subsequent polio outbreak) **incidence** (correlation with a flu-like prodromic illness), **symptoms** (painful or tender lymph nodes, low-grade fever, sore throat), and **similarities** with other viral ailments (including post-polio syndrome). A large body and a large body of medical **research** also supports a viral causation for the illness (Gellman & Verillo 1997, p 19) (Dowsett 1999a, [Online]).

M.E. is an infectious neurological disease and represents a major attack on the central nervous system (CNS) by the chronic effects of a viral infection. A significant number of the world's leading M.E. experts believe that M.E., like poliomyelitis, is caused by an enterovirus. (This includes doctors such as A. Gilliam, A. Melvin Ramsay and Elizabeth Dowsett, John Richardson of Newcastle-upon-Tyne, W.H. Lyle, Elizabeth Bell of Ruckhill Hospital, James Mowbray of St Mary's, Peter Behan and also the brilliant Byron Hyde of Canada.) The evidence which exists to support this theory is compelling (Hyde 2007, [Online]) (Hyde 2006, [Online]). M.E. epidemics very often followed polio epidemics, M.E. resembles polio at onset, serological studies have shown that communities affected by an outbreak of M.E. were effectively blocked (or immune) from the effects of a subsequent polio outbreak, evidence of enteroviral infection has been found in the brain tissue of M.E. patients at autopsy, and so on (Hyde 2007, [Online]) (Hyde 2006, [Online]) (Hyde 2003, [Online]) (Dowsett 2001a, [Online]) (Dowsett 2000, [Online]) (Dowsett 1999a, 1999b, [Online]) (Hyde 1992 p. xi) (Hyde & Jain 1992 pp. 38 - 43) (Hyde et al. 1992, pp. 25-37) (Dowsett et al. 1990, pp. 285-291) (Ramsay 1986, [Online]) (Dowsett & Ramsay n.d., pp. 81-84) (Richardson n.d., pp. 85-92) (Richardson 1999, [Online]). An enterovirus would also explain the; age variation, sex variation, obvious resistance of some family members to the infection and the effect of physical activity (particularly in the early stages of the illness) in creating more long-term/severe M.E. illness in the host (Hyde & Jain 1992a, p. 40).

M.E. expert [Dr Elizabeth Dowsett](#) writes about Myalgic Encephalomyelitis that: 'This illness is distinguished from a variety of other post-viral states by a unique clinical and epidemiological pattern characteristic of enteroviral infection. Prompt recognition and advice to avoid over-exertion is mandatory' (Dowsett et al. 1990, pp. 285-291).

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

How common is Myalgic Encephalomyelitis, who gets it and how?

Although the illness we now know as M.E. has existed for centuries, for much of that time it was a relatively uncommon disease. Following the mass polio vaccination programs of the 1960s cases of polio were greatly reduced and outbreaks of M.E. seemed to be similarly affected. It wasn't until the late 1970s that M.E. began (for reasons as yet not fully understood) its dramatic increase in incidence worldwide. Over 20 years later, M.E. is a worldwide epidemic of devastating proportions. Many people have died from M.E. and there are now many hundreds of thousands of people severely disabled by this epidemic. (TCJRME 2007, [Online]) (Hyde 1992, p. xi)

The main period of infectivity of M.E. peaks at the time just before symptoms appear through to the initial acute phase of the illness (which lasts for several months or in some cases years). M.E. appears to be highly infective but also highly selective. The major mode of infectivity is by airborne or respiratory route. Modes of transmission are thought to include: casual contact (respiratory), salivary transmission (eg. kissing), sexual transmission and transmission through blood products. (Hyde et al. 1992, pp. 25 - 37) (A recent study of 752 patients found that 4.5% of them – almost one in twenty – had had a blood transfusion days or a week before experiencing acute onset of M.E., for example.) (Carruthers et al. 2003, [Online]) There is also evidence that asymptomatic carrier of the illness may be able to pass the illness on to others for a brief period following their exposure to the illness. During the recovery and/or chronic stages of the illness however M.E. does not present a significant infective risk. There is no evidence in the literature that the infection can be waterborne or that potables (drinkable liquids) were the cause of the illness – although bodies of water, or sewage, may possibly sustain the causative infectious agent. (Hyde et al. 1992, pp. 25 - 37)

M.E. has a similar strike rate to multiple sclerosis (or possibly somewhat higher), and is estimated to affect roughly 0.2% of the population. Children and teenagers are also susceptible to the illness and children as young as five have been diagnosed with M.E. (M.E. can occur in children younger than five, but this is thought to be rare.) One hundred thousand kids are estimated to have M.E. in the US alone and a recent study in the UK found that M.E. was by far the most common reason for a child's long term absence from school (Munson 2000, p. 198) (Dowsett 1997, [Online]) (Hyde 1992, pp. x - xxi) (Hooper et al. 2001, [Online]).

There appears to be somewhat of an occupational bias towards teachers (students) and health care workers in the incidence of Myalgic Encephalomyelitis cases (and outbreaks). These are jobs which require higher rates of immunisation than others. This relationship with inoculation is often seen in infectious illnesses (Hyde et al. 1992, pp. 25 - 37). All ages are affected but most commonly sufferers are under 45 at onset. Women are affected around three times as often as men, a ratio common in autoimmune disorders (although in children the sexes seem to be afflicted equally). M.E. affects all ethnic and socio-economic groups and has been diagnosed all over the world. There are more than a million M.E. sufferers worldwide (Hooper et al. 2001 [Online]) (Hyde 1992, pp. x - xxi).

- See: [The outbreaks \(and infectious nature\) of M.E.](#) section for more information as well as many of the excellent articles by [Dr Byron Hyde](#) and [Dr Elizabeth Dowsett](#).

Is the onset of Myalgic Encephalomyelitis gradual or acute?

Again, only being able to achieve 50% or less of your pre-illness activity level immediately upon becoming ill is virtually universal in Myalgic Encephalomyelitis. (Although a small percentage of sufferers may possibly be somewhat less severely affected at onset.) It must be emphasised that this is not a gradual change in ability levels (etc.) which occurs slowly over weeks, months or years; it is an acute (sudden) change.

The onset of M.E. is always acute and is frequently very dramatic; M.E. patients can very often tell you not just the day that they became ill, but the exact *hour* they became ill (Chabursky et al. 1992, p.22) (Hyde 2007, [Online]) (Hyde 2006, [Online]) (Hyde 2003, [Online]) (Hyde et al. 1992, pp. 25-37) (Hyde & Jain 1992, pp. 38-65).

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

Is Myalgic Encephalomyelitis difficult to diagnose? What tests are used to diagnose M.E.?

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 – providing that the physician has some experience with the illness. There is just no other illness that is even remotely like M.E.

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, along with these other illnesses, M.E. must instead be diagnosed by a combination of: taking a detailed medical history (to rule out other possible causes of symptoms), noting the type and severity of symptomatology and other characteristics of the illness, the type of onset of the symptoms (a acute or sudden onset of symptoms is always seen in M.E. If present this characteristic rules out a wide variety of other illnesses associated with gradual onset) and looking for some of the physical signs of illness. A *series* of tests may also then be necessary both to rule out other illnesses, and/or to help confirm a suspected M.E. diagnosis.

Although there is (as yet) no single test which can be used to diagnose M.E. there are a series of tests which can confirm a suspected M.E. diagnosis. Virtually every M.E. patient will also have various abnormalities visible on physical exam. If all tests are normal, if specific abnormalities are not seen on certain of these tests (eg. brain scans), then a diagnosis of M.E. cannot be correct (Hyde 2007, [Online]) (Hyde 2006, [Online]) (Hooper et al. 2001, [Online]) (Chabursky et al. 1992, p.22). 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 CNS changes: SPECT, xenon SPECT, PET, and neuropsychological testing (Hyde 2003, [Online]).

Thus it is these tests which are therefore most critical in the diagnosis of M.E., although various other types of tests are also useful. Abnormalities are also visible on physical exam in Myalgic Encephalomyelitis.

How quickly can Myalgic Encephalomyelitis be diagnosed?

M.E. can commonly be diagnosed within just a few weeks, if the doctor has some experience with M.E. (Chabursky et al. 1992, p.22).

- See: [Testing for M.E.](#) for more information on the various tests which can aid M.E. diagnosis.

What is known about Myalgic Encephalomyelitis so far?

There is an abundance of research which shows that M.E. is an organic illness which can have profound effects on many bodily systems. These are well-documented, scientifically sound explanations for why patients are bedridden, profoundly intellectually impaired, unable to maintain an upright posture and so on. Nearly **a thousand** good articles now support the basic premises of M.E. Autopsies have also confirmed such reports of bodily damage and infection (Hooper & Williams 2005a, [Online]).

Many different organic abnormalities have been found in M.E. patients (in peer reviewed research). Patient advocates Margaret Williams and Eileen Marshall explain that:

- there is evidence of disrupted biology at cell membrane level
- there is evidence of abnormal brain metabolism
- there is evidence of widespread cerebral hypoperfusion
- there is evidence of central nervous system immune dysfunction
- there is evidence of central nervous system inflammation and demyelination
- there is evidence of hypomyelination
- there is evidence that Myalgic Encephalomyelitis is a complex, serious multi-system autoimmune disorder (in Belgium, the disorder has now been placed between multiple sclerosis and Lupus)
- there is evidence of significant neutrophil apoptosis
- there is evidence that the immune system is chronically activated (eg. the CD4:CD8 ratio may be grossly elevated)
- there is evidence that NK cell activity is impaired (ie. diminished)
- there is evidence that the vascular biology is abnormal, with disrupted endothelial function
- there is novel evidence of significantly elevated levels of isoprostanes
- there is evidence of cardiac insufficiency and that patients are in a form of cardiac failure (which is exacerbated by even trivial levels of physical activity, cognitive activity and orthostatic stress)
- there is evidence of autonomic dysfunction (especially thermoregulation; frequency of micturition with nocturia; labile blood pressure; pooling of blood in the lower limbs; reduced blood volume (with orthostatic tachycardia and orthostatic hypotension. Findings of a circulating blood volume of only 75% of expected are common, and in some patients the level is only 50% of expected.)
- there is evidence of respiratory dysfunction, with reduced lung function in all parameters tested
- there is evidence of neuroendocrine dysfunction (notably HPA axis dysfunction)
- there is evidence of recovery rates for oxygen saturation that are 60% lower than those in normal controls
- there is evidence of delayed recovery of muscles after exercise. (Affecting all muscles including the heart.)
- there is evidence of a sensitive marker of muscle inflammation
- there is evidence that the size of the adrenal glands is reduced by 50%, with reduced cortisol levels
- there is evidence of at least 35 abnormal genes, (these are acquired genetic changes, not hereditary), specifically those that are important in metabolism; there are more abnormal genes in Myalgic Encephalomyelitis than there are in cancer
- there is evidence of serious cognitive impairment. (Worse than occurs in AIDS dementia)
- there is evidence of adverse reactions to medicinal drugs, especially those acting on the CNS
- there is evidence that symptoms fluctuate markedly from day to day and even from hour to hour (2006, [Online])

(Note that this is only a sample of some of the research available, not an exhaustive list.) From Professor Malcolm Hooper:

In M.E. there is evidence of inflammation of the central nervous system (CNS). In some cases of ME, as in multiple sclerosis, there is evidence of oligoclonal bands in the cerebrospinal fluid. It is accepted by the most experienced M.E. clinicians that some degree of encephalitis has occurred both in patients with M.E. and in those with post-polio syndrome: the areas chiefly affected include the upper spinal motor and sensory nerve roots and the spinal nerve networks traversing the adjacent brain stem (which is always damaged).

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. The occurrence of autoantibodies to an intra-cellular protein like lamin B1 provides laboratory evidence for an autoimmune component in M.E.

A particularly important piece of research in these patients has demonstrated sensitivity of the vascular endothelium to acetylcholine (a major neurotransmitter and vascular dilator) and this finding may have implications for many other cholinergic pathways (which are extensive throughout the body). In M.E. there is evidence of disruption in ion channels in the cell membranes; changes in ion channel function from time to time offer a rational basis to explain the fluctuating symptoms, and such ion channel changes are known to be induced by physical activities, stress and fasting. If sodium channels are blocked in the open mode, this causes entry of sodium into neural tissues and muscles. This ingress of sodium is followed by water, which in turn leads to swelling of the neural tissues, a phenomenon observed both electron microscopically and by laser scanning microscopy. There is a continued loss of post-exertional muscle power (giving an additional loss of power), with delayed recovery for at least 24 hours, whereas sedentary controls recovered full muscle power after 200 minutes.

Autonomic nervous system testing has revealed abnormalities of the sympathetic and parasympathetic systems. There is considerable evidence from different investigators, using different technologies and studying different groups of patients, of a state of chronic immune activation (Hooper et al. 2001 [Online])

Dr Byron Hyde, explains that:

I have some ME 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. (Hyde 2003, [Online])

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

Dr Byron Hyde, also explains that:

The subject of vascular pathology is not new. The fact of the children dying of a Parkinsonian-like vascular injury to the basal ganglia in Iceland during the Akureyri M.E. Epidemic is an obvious indication of the CNS vascular effects in M.E. Vasculitis has been well documented by Dr. E. Ryll in his description of the epidemic in the San Juan Mercy, Sacramento California Hospital in 1975. He described this M.E. epidemic as an epidemic vasculitis. He was correct. Following my 21 years of examining M.E. patients and 16 years of subjecting M.E. patients to brain imaging techniques 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. Dr. David Bell, who rediscovered the work of Dr. David Streeten and his book, *Orthostatic Disorders of the Circulation*, advanced this understanding of M.E. The work of Dr. Vance Spence and his colleagues in Scotland have started to nail this CNS-vascular relationship down even further with a series of major research papers.

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

Dr. Paul Cheney explains 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 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 (Marshall & Williams 2005a, [Online]) (Cheney 2006, [video recording]). Findings which showed mitochondrial metabolic dysfunction similar to mitochondrial encephalomyopathy also led Dr Cheney to comment, 'The most important thing about exercise is not to have [patients with M.E.] do aerobic exercise. I believe that even progressive aerobic exercise is counter-productive. If you have a defect in mitochondrial function and you push the mitochondria by exercise, you kill the DNA' (Williams 2004a, [Online]).

It is known that Myalgic Encephalomyelitis is:

1. An acute onset (biphasic) epidemic or endemic infectious disease process
2. An autoimmune disease (with similarities to Lupus)
3. An infectious neurological disease, affecting adults and children
4. A disease which involves significant (and at times profound) cognitive impairment/dysfunction
5. A persistent viral infection (most likely due to an enterovirus; the same type of virus which causes poliomyelitis and post-polio syndrome)
6. A diffuse and measurable injury to the vascular system of the central nervous system (the brain)
7. A central nervous system (CNS) disease (with similarities to MS)
8. A variable (but always, serious) diffuse (acquired) brain injury
9. A systemic illness (associated with organ pathology; particularly cardiac)
10. A vascular disease
11. A cardiovascular disease

12. A type of cardiac insufficiency
13. A mitochondrial disease
14. A metabolic disorder
15. A musculo-skeletal disorder
16. A neuroendocrine disease
17. A seizure disorder
18. A sleep disorder
19. A gastrointestinal disorder
20. A respiratory disorder
21. An allergic disorder
22. A pain disorder
23. A life-altering disease
24. A chronic or lifelong disease associated with a high level of disability
25. An unstable disease; from one hour/day/week or month to the next
26. A potentially progressive or fatal disease (Hyde 2007, [Online]) (Hooper et al. 2001, [Online]) (Cheney 2007, [video recording]) (Ramsay 1986, [Online])

- For more information see the [General articles and research overviews](#) section. See also articles by: [Dr. Elizabeth Dowsett](#), [Byron Hyde](#), [Professor Malcolm Hooper](#), and [Dr. Paul Cheney](#).

Are there different stages of Myalgic Encephalomyelitis?

Different stages of M.E. have been identified. [Dr Byron Hyde](#) explains that, 'M.E. is an acute onset biphasic epidemic or endemic (sporadic) infectious disease process, where there is always a measurable and persistent diffuse vascular injury of the CNS in both the acute and chronic phases' (2007, [Online]).

- For more information on the stages of M.E. see: [The Nightingale Definition of M.E.](#) by Dr Byron Hyde and [The Late Effects Of M.E.](#) by Dr Elizabeth Dowsett

Are there any treatments for Myalgic Encephalomyelitis?

Whilst there is no cure as yet, or treatments which can dramatically influence the course of the illness due to the lack of funding into research; intelligent nutritional, pharmaceutical and other interventions can make a significant difference to a patient's life. Appropriate biomedical diagnostic testing should be done as a matter of course (and repeated regularly) to ensure that the aspects of the illness which are able to be treated *can* be diagnosed, monitored and then treated as appropriate. Testing is also important so that dangerous deficiencies and dysfunctions (which may place the patient at significant risk) are not overlooked. (Hooper et al. 2001 [Online]). For information on treatment see: [Treating M.E. - The Basics](#).

Similar Medical Conditions?

There are a number of post-viral fatigue states or fatigue syndromes which may follow common infections such as mononucleosis/glandular fever, hepatitis, Q fever, Ross river virus and so on. M.E. is an entirely different condition to these self-limiting fatigue syndromes however (and is *not* caused by the Epstein Barr virus or any of the herpes or hepatitis viruses), the science is very clear on this point. People suffering with any of these post-viral fatigue states or fatigue syndromes do not have M.E.

Myalgic Encephalomyelitis does have some limited similarities – to varying degrees – to illnesses such as multiple sclerosis, Lupus, post-polio syndrome and chronic Lyme disease, and others. But this does not mean that they represent the same etiological or pathobiological process. They do not. M.E. is a distinct neurological illness with a distinct; onset, symptoms, aetiology, pathology, response to treatment, long and short term prognosis – and World Health Organization classification (G.93.3) (Hyde 2006, [Online]) (Hyde 2007, [Online]) (Hooper 2006, [Online]) (Hooper & Marshall 2005a, [Online]) (Hyde 2003, [Online]) (Dowsett 2001a, [Online]) (Hooper et al. 2001, [Online]) (Dowsett 2000, [Online]) (Dowsett 1999a, 1999b, [Online]) (Dowsett 1996, p. 167) (Dowsett et al. 1990, pp. 285-291)

- See [M.E. and other illnesses](#) for more information. See also the new paper: [M.E. vs MS: Similarities and differences](#)

Recovery from Myalgic Encephalomyelitis

Myalgic Encephalomyelitis patients who are given advice to rest in the early stages of the illness (and who avoid overexertion thereafter) have repeatedly been shown to have the most positive long-term prognosis. As [Dr Melvin Ramsay](#) explains; 'The degree of physical incapacity varies greatly, but the [level of severity] is directly related to the length of time the patient persists in physical effort after its onset; *those patients who are given a period of enforced rest from the onset have the best prognosis*. Since the limitations which the disease imposes vary considerably from case to case, the responsibility for determining these rests upon the patient. Once these are ascertained the patient is advised to fashion a pattern of living that comes well within them' (1986, [Online]).

M.E. can be progressive, degenerative (change of tissue to a lower or less functioning form, as in heart failure), chronic, or relapsing and remitting. Some patients experience spontaneous remissions albeit most often at a greatly reduced level of functioning compared to pre-illness and such patients remain susceptible to relapses for the remainder of their lives – M.E. is a life-long disability where relapse is always possible. Cycles of severe relapse are common, as are further symptoms developing over time. Around 30% of cases are progressive and degenerative and sometimes M.E. is fatal. As Dr Elizabeth Dowsett explains:

After a variable interval, a multi-system syndrome may develop, involving permanent damage to skeletal or cardiac muscle and to other "end organs" such as the liver, pancreas, endocrine glands and lymphoid tissues, signifying the further development of a lengthy chronic, mainly neurological condition with evidence of metabolic dysfunction in the brain stem. Yet, stabilisation, albeit at a low level, can still be achieved by appropriate management and support. The death rate of 10% occurs almost entirely from end-organ damage within this group (mainly from cardiac or pancreatic failure) (2001a, [Online]).

‘When asked on CNN how many of his M.E. patients had fully recovered in fifteen years, Dr Peterson equivocally and chillingly stated, "None.”’ (Munson 2000, p. 5). M.E. expert Dr Byron Hyde, explains that the prognosis of M.E. also differs from patient to patient depending on the degree of damage to the brain:

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 (2007, [Online]).

- See: The 3 Part ME Ability and Severity Scale to measure your own illness severity over time.
- See Treating M.E. for more information on the importance of avoiding overexertion in M.E. and how to make sure your prognosis is as positive as possible. See also: Why patients with severe M.E. are housebound and bedbound and Hospital or carer notes for M.E.
- For information on adrenaline surges in M.E., and the different order in which certain bodily systems may be affected by M.E. (and by overexertion), see the Dr Cheney section in The effects of CBT and GET on patients with Myalgic Encephalomyelitis or Treating Myalgic Encephalomyelitis - Avoiding Overexertion.

Severity of Myalgic Encephalomyelitis

Although some people do have moderate versions of the illness, symptoms are extremely severe for at least 30% of the people who have M.E.; significant numbers of whom are wheelchair-bound, housebound and/or bedbound. M.E. specialist Dr. Paul Cheney stated before a US FDA Scientific Advisory Committee:

I have evaluated over 2,500 cases. At worst, it is a nightmare of increasing disability with both physical and neurocognitive components. The worst cases have both an MS-like and an AIDS-like clinical appearance. We have lost five cases in the last six months. 80% of cases are unable to work or attend school. We admit regularly to hospital with an inability to care for self. (Hooper et al. 2001 [Online])

Dr Dan Peterson found that: ‘M.E. patients experienced greater "functional severity" than the studied patients with heart disease, virtually all types of cancer, and all other chronic illnesses.’ An unrelated study compared the quality of life of people with various illnesses, including patients undergoing chemotherapy or haemodialysis, as well as those with HIV, liver transplants, coronary artery disease, and other ailments, and again found that M.E. patients scored the lowest. "In other words", said one M.E. expert in a radio interview, “**this disease is actually more debilitating than just about any other medical problem in the world**” (Munson 2000, p. 4).

Very often people with very severe M.E. can barely communicate, or even tolerate the presence of another person. This is what makes M.E. such a cruel disease and such an isolating disease. The illness can cause a level of disability and isolation that is just unimaginable to anyone not familiar with very severe M.E.

In the 1980s Mark Loveless, an infectious disease specialist and head of the AIDS and M.E. Clinic at Oregon Health Sciences University, found that M.E. patients whom he saw had far lower scores on the Karnofsky performance scale than his HIV patients even in the last week of their life. He testified that a M.E. patient, ‘feels effectively the same every day as an AIDS patient feels two weeks before death’ (Hooper & Marshall 2005a, [Online]). But in M.E., this extremely high level of illness is not short-term – it does not always lead to death – it can instead

continue uninterrupted for **decades**. **Myalgic Encephalomyelitis can be one of the most debilitating and devastating illness there is.**

People with M.E. must – as soon as possible – be given a correct diagnosis and the appropriate advice and support to ensure that they are given a chance at achieving their best possible prognosis.

For more information:

- For more information on all aspects of Myalgic Encephalomyelitis, including the political issues surrounding M.E., see: [What is Myalgic Encephalomyelitis? A Historical, Medical and Political Overview](#).
- See also the new paper: [M.E. vs MS: Similarities and differences](#).
- To read a list of all the articles on this site suitable for different groups such as M.E. patients, carers, friends and family, doctors or severe M.E. patients and so on, see the [Information Guides](#) page.
- For more information about some of the topics mentioned here, see also the longer/extended version of this text: [Myalgic Encephalomyelitis: The Medical Facts EXTENDED](#)

References

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

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

This paper is merely intended to provide a brief summary of some of the most important facts of M.E. It has been created for the benefit of those people without the time, inclination or ability to read each of these far more detailed and lengthy references created by the world's leading M.E. experts. The original documents used to create this paper are essential additional reading however for any physician (or anyone else) with a real interest in Myalgic Encephalomyelitis: see [What is Myalgic Encephalomyelitis? Extra extended version](#). A partial reference list follows:

- Dowsett, Elizabeth MBChB. 2001a, *THE LATE EFFECTS OF ME* [Online], Available: <http://www.hfme.org/wdowsett.htm>
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- Hyde, Byron M.D. & Anil Jain M.D. 1992, *Clinical Observations of Central Nervous System Dysfunction in Post Infectious, Acute Onset M.E.* in Hyde, Byron M.D. (ed) 1992, *The Clinical and Scientific Basis of Myalgic Encephalomyelitis*, Nightingale Research Foundation, Ottawa, pp. 38-65.
- Hyde, Byron M.D. 2007, *The Nightingale Definition of Myalgic Encephalomyelitis* [Online], Available: <http://www.hfme.org/whydepapers.htm#390403648>

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 (The brain can no longer receive, store or act upon information which enables it to control vital body functions, cognitive, hormonal, cardiovascular, autonomic and sensory nerve communication, digestive, visual auditory balance, appreciation of space, shape etc). 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 2006

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."*

There is ample evidence that M.E. is primarily a neurological illness. It is classified as such under the WHO international classification of diseases (ICD 10, 1992) although non neurological complications affecting the liver, cardiac and skeletal muscle, endocrine and lymphoid tissues are also recognised. Apart from secondary infection, the commonest causes of relapse in this illness are physical or mental over exertion. Dr Elizabeth Dowsett

The body, its systems (such as the gastrointestinal system, the muscular system, the endocrine system, the cardiovascular and vascular systems) and its organs are dependent and their actions largely controlled by the brain. If the brain is physiologically injured, then so is the body. Depending upon which parts of the brain are physiologically injured different parts of the body will also be caused to malfunction. Dr Byron Hyde 2006

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

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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Taken from www.hfme.org



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

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