

Myalgic Encephalomyelitis – The Medical Facts

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Myalgic Encephalomyelitis is a debilitating acquired illness which has been recognised by the World Health Organisation (WHO) since 1969 as an organic neurological disorder with the code G93.3. M.E. can occur in both epidemic and sporadic forms (over 60 outbreaks have been recorded worldwide since 1934) and appears to be remarkably similar to post-polio syndrome (an enteroviral triggered disorder) (Hooper et al. 2001 [Online]).

Myalgic Encephalomyelitis is a systemic acutely acquired illness initiated by a virus infection which is characterised by post encephalitic damage to the brain stem. Symptoms are primarily caused by central nervous system dysfunction and a subsequent breakdown in bodily homeostasis. Therefore although M.E. is primarily neurological, symptoms may be manifested by cardiac, cardiovascular, immunological, endocrinological, respiratory, hormonal, gastrointestinal and musculo-skeletal dysfunctions and damage. M.E. can be extremely severe and in some cases the illness is fatal.

Is it Myalgic Encephalomyelitis a new illness?

Myalgic Encephalomyelitis is not a new illness, it has been documented as a neurological illness for centuries. The disorder used to be known as ‘atypical poliomyelitis’ but was renamed Myalgic Encephalomyelitis (M.E.) in 1956. In 1962 the distinguished neurologist Lord Brain included M.E. in the standard textbook of neurology, M.E. was then formally classified as a disease of the nervous system in the World Health Organisation International Classification of Diseases in ICD 8 in 1969. The current ICD, ICD-10, continues to classify M.E. as a distinct organic neurological illness with ICD code G93.3. (Hooper & Montague 2001 [Online]). 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’ (a [Online]).

What is Myalgic Encephalomyelitis? What is its symptomatology?

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. 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 is a 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 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. There is also multi-system involvement of cardiac and skeletal muscle, liver, lymphoid and endocrine organs. Some individuals also have damage to skeletal and heart muscle.

This is not simply theory, but is based upon an enormous body of clinical information. Confirmation of this hypothesis is supported by electrical tests of muscle and of brain function (including the subsequent development of PET and SPECT scans) and by biochemical and hormonal assays. Newer scientific evidence is increasingly strengthening this hypothesis.

It is the combination of the chronicity, the dysfunctions, and the instability, the lack of dependability of these dysfunctions, 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. (Hyde 1992 p. xi) (Hyde & Jain 1992 pp. 38 - 43) (Dowsett 2001, 2000, 1999.b, b [Online])

More than 64 distinct symptoms have been authentically documented in M.E. (Hooper & Montague 2001 [Online]) At first glance it may seem that every symptom possible is mentioned, but the seemingly random list of symptoms in

fact form unique and distinct patterns – they are anything but ‘random’ for those with knowledge of the illness and/or of how the illness effects the body’s various systems. As Montague and Hooper explain, ‘In M.E., different people have different symptoms but the general pattern and evolution of major symptoms are remarkably coherent.’ (2004, [Online]) Symptoms 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, hyperacuity, 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). (Bassett, 2005)

Having Myalgic Encephalomyelitis is like having parts of Multiple Sclerosis, AIDS, Alzheimer's, the flu, Arthritis and Epilepsy all mixed together at once, with some extra horrific symptoms thrown in that are entirely its own. It is a neurological illness of extraordinarily incapacitating dimensions that also affects virtually every bodily system.

- For a more complete symptom list see: [The Clinical and Scientific Basis of Myalgic Encephalomyelitis](#) (book) or [Verillo and Gellman's Treatment Guide](#) (book) or [The Ultra-comprehensive Myalgic Encephalomyelitis Symptom List](#) and [The M.E. Society of America Framework](#) and [Cheney on cardiac issues and ME](#) (among many other references). See [Wikipedia](#) for a short explanation of homeostasis. See [Research and Articles](#) for many different articles and medical studies into M.E.

What is the main feature of Myalgic Encephalomyelitis?

From [The Ultra-comprehensive Myalgic Encephalomyelitis Symptom List](#) (on this site):

M.E. is characterised primarily by damage to the central nervous system (the brain) which results in dysfunctions and damage to many of the body’s vital systems and a loss of normal internal homeostasis.

Therefore, although M.E. is primarily neurological, symptoms may be manifested by: cognitive, cardiac, cardiovascular, immunological, endocrinological, respiratory, hormonal, gastrointestinal and musculo-skeletal dysfunctions and damage. Symptoms are also caused by a loss of normal internal homeostasis; the body becomes unable to make all the appropriate physiological adjustments that allow it to maintain homeostatic equilibrium in response to the many changes to the internal and external environment that are part of everyday life. The body/brain no longer responds appropriately to homeostatic pressures, including (to varying extents): physical activity, cognitive exertion, sensory input, orthostatic stress, emotional stress and infectious stress.*

When certain levels of each of these homeostatic pressures occur (or are applied), homeostatic disequilibrium results. The result of this homeostatic disequilibrium is a period of time in which the patient experiences:

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

The level or intensity of each of these **internal and external homeostatic pressures** needed to cause the **M.E. homeostatic disequilibrium symptom complex** (or symptom ‘storm’) outlined above varies from patient to patient, but is often trivial compared to a patient’s pre-illness tolerances and abilities. The severity level of the symptoms produced varies widely between patients and ranges from mild to very severe. The symptoms produced may also be life-threatening (seizures and cardiac events). The severity of the attack and its symptomatology will

also vary depending on which particular homeostatic pressure is involved. (Most commonly, an intolerance to particular levels of physical and cognitive activity are the primary features of the illness; the diagnosis of Myalgic Encephalomyelitis should **never** be made without these features being present.)

The onset of these symptoms may be acute but often symptoms will not peak until 24 – 48 hours or more afterward (this is particularly true with regard to physical, cognitive and orthostatic exertions). Symptoms will then persist for hours, weeks or many months.

The symptomatic expression of these effects can also be delayed and accumulate over time (usually days or weeks) until they are realised in a ‘crash,’ a period of intense worsening of the overall condition followed by a gradual return to the patient’s base level of illness. When the body is confronted with homeostatic pressures beyond the patient’s individual limits severely and/or repeatedly over time, these effects can also become cumulative in the long term; the patient becomes unable to return to their base level of illness at all (long-term or permanent worsening of the overall severity of the condition is caused). This is particularly true with regard to physical activity. It is vital that patients avoid physical over-exertion and are never encouraged to exercise (or be active) beyond their individual limits at *any* stage of the illness. In addition to the risk of relapse, permanent damage (eg. to the heart), and disease progression, there have also been reports of sudden deaths in M.E. patients following exercise.

For the most severely affected sufferers there is virtually no ‘safe’ level of any of these homeostatic pressures, no level which does not produce a worsening of symptoms (and perhaps also contribute to disease progression). Even the most basic actions – speaking a few words, being exposed to bright light or moderate noise for a few minutes, turning over in bed, having hair or body washed in bed by a carer or chewing and swallowing food – cause severe and extended symptom exacerbations (or ‘storms’) in such patients.

***Note:** This is not ‘stress’ as the concept is commonly understood, but is a reference to anything which causes the homeostatic systems to have to react in some way. Even the category of ‘emotional stress’ is not solely concerned with ‘anxiety’ as symptoms may in fact be induced by ALL strong emotions, negative *and* positive. ‘Sensory input’ includes (to varying extents): light, noise, vibration, motion, touch, smell and temperature sensitivities. (Bassett, 2005)

Another of the defining characteristics of the illness is its waxing and waning pattern: the striking variability of symptoms (and of symptom severity) over 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’. (2001 [Online]). The chronicity of the illness, is another.

- For more information see: The Clinical and Scientific Basis of Myalgic Encephalomyelitis (book), The Canadian criteria or The Ultra-comprehensive Myalgic Encephalomyelitis Symptom List and The M.E. Society of America Framework. See Research and Articles for many different articles and medical studies into M.E. involving exercise intolerance.

What can trigger or cause the onset of Myalgic Encephalomyelitis?

Veteran M.E. specialist Dr Byron Hyde explains that:

[The] prodromal phase is associated with a usually short onset or triggering illness. This 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. Evidence of a previous immune insult [such as a recent immunisation] is found regularly in both epidemic and sporadic cases. The usual incubation period of the triggering illness is 4-6 days. 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 presumed infectious triggering illness. (1998 [Online])

What causes Myalgic Encephalomyelitis? Are there outbreaks?

Yes, there is a history of recorded outbreaks going back to 1934, when an epidemic of what seemed at first to be poliomyelitis was reported in Los Angeles. A review of early outbreaks found that clinical symptoms were consistent in over sixty recorded epidemics of M.E. spread all over the world (Dunn 2005, [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) (Dowsett 1999.a, [Online]) **incidence** (correlation with a flu-like prodromic illness), **symptoms** (swollen lymph nodes, low-grade fever, sore throat), and **similarities** with

other viral ailments, notably mononucleosis and post-polio syndrome (Gellman & Verillo 1997, p 19). **Research** also supports a viral causation for the illness.

In 1959 Dr. Donald Henderson (a CDC epidemiologist) and Dr. Alexis Shelakov (a NIH epidemiologist), published a comprehensive review paper in the New England Journal of Medicine describing several outbreaks. Dr. Henderson noted: ‘The pattern of the epidemic, the absence of any common exposure factors and the high incidence among medical and hospital personnel were consistent only with an infectious disease transmitted from person to person.’ (McLaughlin 2004, [Online])

- For more information see: [Recent Epidemics: Why are the Epidemics so important](#) and [Osler’s Web](#) (book) and [The Clinical and Scientific Basis of Myalgic Encephalomyelitis](#) (book) and many of the excellent articles by [Dr Byron Hyde](#) and [Dr Elizabeth Dowsett](#) and [The Committee for Justice and Recognition of Myalgic Encephalomyelitis](#) for more on M.E. epidemics and transmissibility.

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

Although the illness we now know as Myalgic Encephalomyelitis has existed for centuries, for much of that time it was a relatively uncommon disease. Following the mass polio vaccination programs of the 1960’s cases of polio were greatly reduced and outbreaks of M.E. seemed to be similarly affected. It wasn’t until the late 1970’s 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 millions of people severely disabled by this epidemic. (TCJRME, [Online]).

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. Modes of transmission are thought to include: casual contact (respiratory), salivary transmission (eg. kissing), sexual transmission and transmission through blood products. (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.) 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 appear to present a significant infective risk). (Hyde et al. 2002)

M.E. is three times more common than Multiple Sclerosis. It is also more common than lung cancer, breast cancer, or HIV (in women) with an estimated 2 million sufferers in the US (Bell 1995, p. 231), 250 000 sufferers in the UK and around 36 000 in Australia and many more worldwide. Children and teenagers are also susceptible to the illness and children as young as five have been diagnosed with M.E. One hundred thousand kids are estimated to have M.E. in the US alone (Munson 2000, p. 198) 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. (Dowsett 1997 [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 higher risk groups do not work in environments which are more stressful than the average job, but these are jobs which require higher rates of immunisation than others. This relationship with inoculation is often seen in infectious illnesses. (Hyde et al. 2002) 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 races and socio-economic groups and has been diagnosed all over the world (Hooper et al. 2001 [Online]).

- See: [Recent Epidemics: Why are the Epidemics so important](#) and [Osler’s Web](#) (book) and [The Clinical and Scientific Basis of Myalgic Encephalomyelitis](#) (book) and many of the excellent articles by [Dr Byron Hyde](#) and [Dr Elizabeth Dowsett](#) for more on M.E. epidemics and transmissibility.

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. Many aspects of the pathophysiology of the disease have, indeed, been medically explained in volumes of research articles (M.E. Society of America 2005 [Online]). Nearly **1000** good articles now support the basic premises of M.E. (Bell [Online]) Autopsies have also confirmed such reports of bodily damage and infection. (Short 2005, [Online])

As [Peggy Munson](#) explains in her book ‘Stricken’:

Many startling abnormalities have been found in M.E. patients in almost every bodily system- such as extremely low blood volume, enzyme pathway disruptions, cardiac disturbances, and malfunction of the hypothalamus-pituitary-adrenal axis. One remarkable study, utilising specific brain scan techniques, found the effects of M.E. on the brain to be strikingly similar to AIDS dementia. Earlier research discovered punctate

lesions in M.E. brains resembling those of Multiple Sclerosis patients. Dr Paul Cheney found that in dual chromatography analyses, many M.E. patients actually had more derangement of the brain, on a biochemical level, than Parkinson's or Alzheimer's patients. Dr Sheila Bastien, who studied a group of educated patients, was stunned to realise that patients who initially appeared very lucid had suffered tremendous drops in IQ points, so severe in some cases that "a few performance IQ's were startlingly close to the legal definition of idiocy." (2000, p. 1)

Jill McLaughlin further describes some of the other major laboratory findings in M.E. patients thus:

SPECT scans have demonstrated decreased cerebral blood flow, PET scans have shown decreased brain metabolism; MRI scans have shown the presence of small white matter lesions. These abnormalities have been shown to correlate with clinical status. The abnormalities in M.E patients most closely resemble those seen in AIDS encephalopathy.

Autonomic nervous system dysfunction includes orthostatic intolerance, neurally-mediated hypotension and postural tachycardia syndrome. Many patients have low blood volume.

The immune system abnormalities mimic the immune pattern seen in viral infections. Specific findings include increased numbers of activated cytotoxic T cells, low natural killer cell function, and elevated immune complexes. A large University of Miami study found that the array of immunological defects suggest that M.E. is a form of acquired immunodeficiency. A more specific immune system abnormality has been discovered in M.E. of increased activity and dysfunction of the 2-5A RNase-L antiviral pathway in lymphocytes. The dysregulation of the RNase-L pathway supports the hypothesis that viral infection [plays] a role in the pathogenesis of the illness.

Sub-optimal cardiac function and abnormal cardiovascular responses have also been demonstrated. One study found left-ventricular dysfunction following exertion and orthostatic stress in patients with M.E. and that the heart failed to pump enough blood following exertion and upright posture. Dr. A. Martin Lerner discovered persistent viral infection in the heart, causing left-ventricular dysfunction, resulting in exercise intolerance. (Exercise, in turn, worsens the cardiac dysfunction.) The disease in the early stages is consistent with a dilated cardiomyopathy that in later stages might result in progressive, end-stage dilated cardiomyopathy, a type of heart failure.

Abnormal laboratory values [in M.E.] patients may include: Immune complexes, Immunoglobulin G, low level Antinuclear antibody titer, Alkaline phosphatase, Cholesterol, Lactate dehydrogenase, Atypical lymphocyte count. These tests may support a diagnosis of M.E.

More recently, the University of Hawaii discovered the occurrence of an endogenous lipid, similar in structure to ciguatoxin, in M.E. The chronic phase lipids (CLP) may be comparable to "acute phase proteins" such as C-reactive protein, which increase in diseases such as inflammation and trauma. The test for CPL may be used to confirm the diagnosis. The testing procedure is on the NCF website at <http://www.ncf-net.org/> (2004, [Online])

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 ME 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).

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. 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. Acquired ion channel abnormalities in the myocardium... may form the basis of cardiac dysfunction in [M.E].

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.

Single photon emission computed tomography (SPECT) signal abnormalities also are found in [M.E.] patients, abnormalities like those seen in patients with encephalopathy due to the acquired immunodeficiency syndrome (AIDS). 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.

There may be significant and permanent damage to skeletal or cardiac muscle as well as to other end-organs including the liver, pancreas, endocrine glands and lymphoid tissues, with evidence of dysfunction in the brain stem. (Hooper et al. 2001 [Online])

Findings which suggest mitochondrial metabolic dysfunction similar to mitochondrial encephalomyopathy in Myalgic Encephalomyelitis patients led M.E. expert Professor Paul 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 2004, [Online]).

This is just a sample of some of the laboratory findings specific to Myalgic Encephalomyelitis patients, it is by no means exhaustive. Numerous documented heart, lung, brain, gene and other abnormalities also show strong evidence that exercise can have extremely harmful effects on M.E. patients in many different bodily systems including that permanent damage may be caused, as well as disease progression. (Williams 2004, [Online]).

Patients also show observable physical signs of the illness:

Observable signs include a typically swinging low-grade temperature, nystagmus; sluggish visual accommodation; abnormality of vestibular function with a positive Romberg test; 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; mouth ulcers; hair loss; a labile blood pressure (sometimes as low as 84/48 in an adult at rest); flattened or even inverted T-waves on 24 hour Holter monitoring (a standard 12 lead ECG is usually normal); orthostatic tachycardia; shortness of breath (patients show significant reduction in all lung function parameters tested); abnormal glucose tolerance curves; liver involvement (an enlarged liver or spleen may not be looked for in ME, so missed) and destruction of fingerprints: (atrophy of fingerprints is due to perilymphocytic vasculitis and vacuolisation of fibroblasts). (Hooper et al. 2001 [Online]).

Whilst there is as yet no *single*, definitive laboratory test for M.E., there are a specific *series* of tests which enable a M.E. diagnosis to be easily confirmed (MRI and SPECT scans of the brain for example). (Montague & Hooper 2001, [Online]) As M.E. authors Verillo and Gellman explain: 'Myalgic Encephalomyelitis is a distinct, recognisable entity that can be diagnosed relatively early in the course of the disease, providing the physician has some experience with the illness.' (p. 21) New clinical guidelines such as the Canadian Criteria now also make diagnosis easier than ever before; even for those with no experience with Myalgic Encephalomyelitis patients.

- For more information see: General Research and Articles, Cardiac and Cardiovascular Research, Exercise Research, Mitochondrial Muscle Research and General Muscle Research, Neurological and Cognitive Research, Genetic Research, Neuroendocrine and Endocrine Research, Immune System Research and RNase L Research General Viral Research, Enteroviral and Post-Polio Research, HHV6 Research and Stealth Virus Research Miscellaneous Research, The Severity of M.E., and Outstanding Research. See also: Putting Research and Articles into Context
- See also articles by: Dr. Elizabeth Dowsett, Byron Hyde MD, Professor Malcolm Hooper, Dr. Paul Cheney, Dr David Bell M.D., The Committee for Justice and Recognition of Myalgic Encephalomyelitis, and Margaret Williams and Eileen Marshall

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]).

(Some examples of the treatments which may help M.E. patients include: (1) various hormone levels can become dangerously low in M.E. and so need to be monitored regularly and hormone replacements therapies administered when needed (cortisol (an adrenal hormone), thyroid hormones, reproductive hormones etc.). (2) Medication may

be required to stop the muscles around the lungs going into spasm (which can cause a great difficulty with maintaining breathing.) (3) A diet low in sugar and salt may help to reduce symptoms of noise sensitivity.)

Similar Medical Conditions

Myalgic Encephalomyelitis is similar to varying degrees to other illnesses such as Multiple Sclerosis, Lupus, Lyme disease and Gulf War Syndrome; and M.E. may overlap or co-exist with Fibromyalgia, Multiple Chemical Sensitivities, and Irritable Bowel Syndrome. But as patient advocate [Jill McLaughlin](#) explains: ‘these [illnesses] may coexist with other conditions as well and are viewed as separate entities which stand on their own, regardless of whether a person has other medical problems. Even though there are similarities or overlap does not mean that they represent the same etiological or pathobiological process.’ (2004, [Online])

- See [M.E. and other illnesses](#) for more information.

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 veteran M.E. clinician Dr Melvin Ramsay MD explains: ‘The degree of physical incapacity varies greatly, but the [level of severity] is directly related to the length of time the patient persists in physical effort after its onset; put in another way, **those patients who are given a period of enforced rest from the onset have the best prognosis.**’ (1986 [Online])

- See [Treating M.E.](#) for more information on the importance of avoiding overexertion in M.E.

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. (M.E. Society of America [Online]). 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). (2001 [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)

- *If you are newly diagnosed with M.E. however, never lose hope that you will be one of the lucky ones that never has the severe version of the illness and that you will experience some level of recovery. It does happen, just not to everyone unfortunately, but as long as there's a chance, and there is, there should be hope.*
- See: [The 3 Part ME Ability and Severity Scale](#) to measure your own illness severity over time.

Severity of Myalgic Encephalomyelitis

Although some people do have moderate versions of the illness, symptoms are extremely severe for around 30% of the people who have M.E. According to patient surveys, it would appear that around half of those most severely affected by this illness are bed-bound and housebound (Dunn 2005, [Online]).

M.E. specialist [Dr. Paul Cheney](#) stated before an FDA Scientific Advisory Committee:

I have evaluated over 2,500 cases. At best, it is a prolonged post-viral syndrome with slow recovery. 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. The most difficult thing to treat is the severe pain. 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 Dr Leonard Jason in a radio interview, **"this disease is actually more debilitating than just about any other medical problem in the world."** (Munson 2000, p. 4)

An infectious disease specialist and head of the AIDS and M.E. Clinic at Oregon Health Sciences University, testified that a M.E. patient, 'feels effectively the same every day as an AIDS patient feels two weeks before death.' 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**.

Truly Myalgic Encephalomyelitis can be one of the most debilitating and horrific illness there is.

See www.ahummingbirdsguide.com for more information about any of the topics discussed. See www.ahummingbirdsguide.com/whatisme.htm for references, a recommended reading list and a summary of the some of the political issues surrounding Myalgic Encephalomyelitis.

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