# *Testing for M.E. - Summary*



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

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

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

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

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

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

- **SPECT and xenon SPECT scans of the brain.** SPECT scans have demonstrated decreased cortical/cerebellar regional cerebral blood flow most frequently in the frontal, parietal, temporal, occipital and brain stem areas of the brain, and throughout the cerebral cortex in M.E. This decrease in cerebral blood flow has been found to have worsened further still 24 - 72 hours post-exertion. These abnormalities have also been shown to correlate with clinical status.
- **MRI scans of the brain.** Punctate, subcortical areas of high signal intensity consistent with edema or demyelination have been identified by MRI in 80% or more of M.E. patients (similar to those seen in MS). M.E. patients with these MRI abnormalities have been reported as being more severely impaired than those without such abnormalities.
- **PET scans of the brain.** PET scans of the brain have shown decreased metabolism of glucose in the right mediofrontal cortex and generalised hypoperfusion of the brain with a particular pattern of decreased neuronal metabolism in the brain stem.
- **EEG/QEEG brain maps**. Almost all M.E. patients have abnormal cognitive-evoked EEG brain maps. QEEG brain maps are even more accurate as they have been able to demonstrate not only lack of normal activity in M.E. patients but migration of the normal activity centers from injured areas to different parts of the brain.
- **Neurological examination.** Most M.E. patients will have an abnormal neurological exam.
- **Neuropsychological testing.** This test can uncover the specific cognitive dysfunctions of M.E.
- **The Romberg test.** The Romberg test is a useful test of brain stem function. Almost all M.E. patients will have an abnormal Romberg test.
- **Immune system tests**. Natural killer cell number and function is particularly low in M.E., for example.
- **Insulin levels and glucose tolerance tests.**
- **Erythrocyte Sedimentation Rate (ESR) tests.** An unusually low sedimentation rate of less than 5mm/hr is common in M.E. ESR rates as low as 0 have been documented in M.E. patients. There are only five diseases that have a pathological low sedimentation level: Myalgic Encephalomyelitis, sickle-cell anemia, hereditary sperocytosis, hyper-gammaglobulinemia and hyper-fibrogenemia.
- **Circulating blood volume tests.** This test may show a reduced circulating blood volume of up to 50%.
- **24 hour Holter monitor testing** (a type of heart monitor). May show repetitively oscillating T-wave inversions and/or a flat T-wave and heart rates as high as (or higher than) 150 beats per minute as an immediate or delayed response to the patient maintaining an upright posture, or at rest. Heart rates as low as 40 beats per minute may also be observed (during sleep).

- **Tilt table examination and standing/sitting/reclining blood pressure tests**. Dr Byron Hyde explains that testable vascular and cardiac dysfunction is the most obvious set of dysfunctions when looked for and is the cause behind a significant number of M.E. complaints, and that all moderate to severe M.E. patients have one or more of the following vascular dysfunctions:
    - Severe postural orthostatic tachycardia syndrome (POTS), cardiac irregularity on minor positional changes or after minor physical activity, Raynaud's phenomenon, circulating blood volume decrease, bowel dysfunction caused by vascular dysfunction, Persantine effect in M.E. patients, M.E. associated clotting defects, anti-smooth muscle antibodies and other cardiac dysfunctions.
- **Exercise testing and chemical stress tests.** These tests are not appropriate for severely ill patients.
- **Physical exam.** Physical signs of illness commonly observed in M.E. patients include:
    - Nystagmus
    - Sluggish visual accommodation or unequal pupils and contrary pupil reaction to light
    - A labile blood pressure (sometimes as low as 84/48 in an adult at rest)
    - Shortness of breath (particularly on exertion)
    - Subnormal temperature
    - Cogwheel movement of the leg on testing
    - Muscular twitching or fasciculation
    - Hyper-reflexia without clonus
    - Destruction of fingerprints is sometimes seen (atrophy of fingerprints is due to perilymphocytic vasculitis and vacuolisation of fibroblasts)
    - Ghastly pallor of face with frequent lupus-like submaxillary mask or facial vasculoid rash
    - Parkinsonian rigidity of facial expression
    - Scanning, disjointed speech, or speech reversals
    - Sicca syndrome of conjunctiva and mucous membranes
    - Frequent equivocal Babinski/plantar reflex on one side
    - Nodular thyroid

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

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

years later. But this negative test result should not be assumed to indicate that the patient's disease status has necessarily changed as Dr Hyde reports that whether subsequent tests were positive or negative patients remained similarly disabled (Hyde 2011, [Online]).

M.E. expert Dr Byron Hyde's **Nightingale Definition of M.E.** also now makes diagnosis easier than ever before even for those with no prior experience in diagnosing M.E. This is a pure M.E. definition and, most importantly, it defines M.E. as *testable* (see the full-length text for details) (Hyde 2007, [Online]) (Hyde 2006, [Online]) (Hyde 2003, [Online]) (Dowsett 2001a, [Online]) (Dowsett 2000, [Online]) (Hyde 1992 p. xi) (Hyde & Jain 1992 pp. 38 - 43) (Hyde et al. 1992, pp. 25-37) (Dowsett et al. 1990, pp. 285-291) (Ramsay 1986, [Online]) (Dowsett n.d., [Online]) (Dowsett & Ramsay n.d., pp. 81-84) (Richardson n.d., pp. 85-92).

- See the full-length Testing for M.E. text for more information on this topic.
- **A note on 'CFS' testing and redefinitions:** Whilst various 'fatiguing conditions' with a variety of different aetiologies may be made up of vague and mild 'everyday' symptoms, with no observable signs and no tests which have shown abnormalities or that can aid diagnosis, M.E. shares none of these characteristics. M.E. is not described by any of the definitions of 'CFS' or 'ME/CFS' (including the Canadian 'ME/CFS' definition or the ICC). *Many* patients can and do fit these (wastebasket) definitions that have diseases other than M.E.

  For more information see What is M.E.? and Who benefits from 'CFS' and 'ME/CFS'? See The misdiagnosis of CFS for information on why 'CFS' is always a misdiagnosis or non-diagnosis.
- **A note on testing *availability* in M.E.:** Objective scientific tests *are* available which can aid in the diagnosis of M.E. and easily prove the severe abnormalities across many different bodily systems seen in M.E. Unfortunately many (in fact most) patients are not given access to these tests. Problems also exist with doctors not being familiar with the abnormalities on testing seen in M.E. and so misinterpreting the results of some tests.

  The problem is not that these tests don't exist, but that doctors – and many patients – are unaware of this information on testing, that it is not generally accepted due to the nefarious influence of political and financial vested interest groups, and that there are overwhelming financial and political incentives for researchers to IGNORE this evidence in favour of the bogus 'CFS' (or 'subgroups of 'ME/CFS') construct.

  For more information on the lack of access to appropriate testing for M.E. patients see Testing for M.E.

### 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. From the 1950s to the present day, Dr Byron Hyde and Dr.Elizabeth Dowsett along with their mentors, the late Dr John Richardson and Dr Melvin Ramsay (respectively). Collectively, these four doctors have been involved with M.E. research and M.E. patients for well over 100 years. Among them they have examined more than 15 000 individual (sporadic and epidemic) M.E. patients as well as each authoring numerous studies and articles and books (or chapters in books) on M.E.

This paper is merely intended to provide a brief summary of some of the most important facts of M.E. It has been created purely 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 M.E.? or the References page.

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

'The term myalgic encephalomyelitis (means muscle pain, my-algic, with inflammation of the brain and spinal cord, encephalo-myel-itis, brain spinal cord inflammation) was first coined by Ramsay and Richardson and has been included by the World Health Organisation (WHO) in their International Classification of Diseases (ICD), since 1969. It cannot be emphasised too strongly that this recognition emerged from meticulous clinical observation and examination.' Professor Malcolm Hooper 2006

'M.E. is a systemic disease (initiated by a virus infection) with multi system involvement characterised by central nervous system dysfunction which causes a breakdown in bodily homoeostasis. 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

*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.***



- Myalgic Encephalomyelitis (M.E.) is a disabling neurological disease that is very similar to Multiple Sclerosis (M.S.) and Poliomyelitis. Earlier names for M.E. were 'atypical Multiple Sclerosis' and 'atypical Polio.'
- M.E. is a neurological disease characterised by scientifically measurable post-encephalitic damage to the brain stem. This damage is an essential part of M.E., hence the name M.E. The term M.E. was coined in 1956 and means: my = muscle, algic = pain, encephalo = brain, mye = spinal cord, tis = inflammation. This neurological damage has been confirmed in autopsies of M.E. patients.
- Myalgic Encephalomyelitis has been recognised by the World Health Organisation's International Classification of Diseases since 1969 as a distinct organic neurological disease. M.E. is classified in the current WHO International Classification of Diseases with the neurological code G.93.3.
- M.E. is primarily neurological, but also involves cognitive, cardiac, cardiovascular, immunological, endocrinological, metabolic, respiratory, hormonal, gastrointestinal and musculo-skeletal dysfunctions and damage. M.E. affects all vital bodily systems and causes an inability to maintain bodily homeostasis. More than 64 individual symptoms of M.E. have been scientifically documented.
- M.E. is an acute (sudden) onset, infectious neurological disease caused by a virus (a virus with a 4-7 day incubation period). M.E. occurs in epidemics as well as sporadically and over 60 M.E. outbreaks have been recorded worldwide since 1934. There is ample evidence that M.E. is caused by the same type of virus that causes Polio; an enterovirus.
- M.E. can be more disabling than M.S. or Polio, and many other serious diseases. M.E. is one of the most disabling diseases that exists. More than 30% of M.E. patients are housebound, wheelchair-reliant and/or bedbound and are severely limited with even basic movement and communication.
- *Why are M.E. patients so severely and uniquely disabled?* For a person to stay alive, the heart must pump a certain base-level amount of blood. Every time a person is active, this increases the amount of blood the heart needs to pump. Every

movement made or second spent upright, every word spoken, every thought thought, every word read or noise heard requires that more blood must be pumped by the heart.

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

If activity levels exceed cardiac output by even 1%, death occurs. Thus the activity levels of M.E. patients must remain strictly within the limits of their reduced cardiac output just in order for them to stay alive. *M.E. patients who are able to rest appropriately and avoid severe or prolonged overexertion have repeatedly been shown to have the most positive long-term prognosis.*

- M.E. is a testable and scientifically measurable disease with several unique features that is not difficult to diagnose (within just a few weeks of onset) using a series of objective tests (e.g. MRI and SPECT brain scans). Abnormalities are also visible on physical exam in M.E. M.E. is a long-term/lifelong neurological disease that affects hundreds of thousands of adults and children worldwide. In some cases M.E. is fatal. (Causes of death in M.E. include heart failure.)