*Extracts from*

# A New and Simple Definition of Myalgic Encephalomyelitis and a New Simple Definition of Chronic

As presented at the Invest in ME
London Conference of May 12, 2006

by Dr. Byron Hyde MD

This is an abridged version of Dr. Byron Hyde's booklet entitled

**A New and Simple Definition of Myalgic Encephalomyelitis and a New Simple Definition of Chronic Fatigue Syndrome**

**&**

**A Brief History of Myalgic Encephalomyelitis & An Irreverent History of Chronic Fatigue Syndrome**

Which is available from the **Nightingale Foundation** at
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# A new and simple definition of Myalgic Encephalomyelitis and a new simple definition of Chronic Fatigue Syndrome

As presented at the Invest in ME London Conference of May 12, 2006 by Byron Hyde MD:

This material will be available on the Nightingale's new website http://www.nightingale.ca from the summer of 2006 or earlier.

I would like to start by proposing that M.E. be defined simply as being consistent with the majority of the ten following diagnostic features:

**Myalgic Encephalomyelitis is**:

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

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

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

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

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

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

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

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

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

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

a. **POTS:** severe postural hypotension.

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

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

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

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

a. **Pituitary-thyroid axis:** This is common. Changes in serum TSH, FTI, FT4, Microsomal Ab., PTH, Calcium and phosphorus rarely occur until one or more years after illness onset and usually only after several years. This can be followed by ultrasound of the thyroid gland where a steady shrinking of the thyroid gland occurs with or without the development of non-serum positive Hashimoto's thyroiditis (a seeming contradiction of terms) and a significant increase in thyroid malignancy. Serum positive changes occur only after years.
b. **Pituitary-adrenal axis changes:** this finding is infrequent.
**c. Pituitary-ovarian axis changes:**
d. **Pituitary- (adrenal?)-Bladder dysfunction:** occurs frequently in the early disease in some people. It is unknown if the cause is due to this link.

I would like to propose that Chronic Fatigue Syndrome should be simply diagnosed as follows:

## NEW DIAGNOSTIC CRITERIA FOR CHRONIC FATIGUE SYNDROME

The patient has:

1. A gradual onset fatigue syndrome,

2. This is usually due to a missed major disease in which common things are common: i.e. the patient has:

    a. Missed cardiac disease,
    b. Missed malignancy,
    c. Missed vascular disease,
    d. Missed brain lesion either of a vascular or space occupying lesion,
    e. Missed test positive rheumatologic disease,
    f. Missed test negative rheumatologic disease,
    g. Missed endocrine disease,
    h. Missed physiological disease,
    i. Missed genetic disease,
    j. Missed chronic infectious disease,
    k. Missed pharmacological or immunization induced disease,
    l. Missed social disease,
    m. Missed drug use disease or habituation,
    n. Missed dietary dysfunction diseases,
    o. Missed psychiatric disease.

The reasons for these two proposals are implicit in the history of these two terms and are as follows:

## A HITCH HIKER'S GUIDE TO THE HISTORY OF MYALGIC ENCEPHALOMYELITIS & CHRONIC FATIGUE SYNDROME

(This section may be seen in full by visiting the Nightingale Foundation's site at **www.nightingale.ca**)

Our history starts with the first known recorded modem history of Myalgic Encephalomyelitis.

**Atypical Poliomyelitis 1932:** The first recorded major epidemic of a disease phenomenon identical to Myalgic Encephalomyelitis occurred in 1932 in California. Dr Alexander G. Gilliam who later became Professor of Epidemiology of Johns Hopkins Medical School documented this epidemic that destroyed the lives of a multitude of doctors, nurses and health care workers in the Las Angeles County General Hospital, at the time, the largest hospital in the world.

Gilliam called this disease atypical poliomyelitis. It was a logical conclusion since it was associated with a large epidemic of close to 100,000 people from San Francisco to the Mexican border at San Diego. The curious thing about this epidemic of "polio" is that very few people actually died.

Dr Alberto Marinacci, employing a Universal Electromyography Machine that unfortunately no longer exists, was able to demonstrate diffuse peripheral nerve changes in these patients, different from, but similar to a mild form of Guillam-Barre Syndrome.

**Disease Resembling Poliomyelitis: 1947-1948: Akureyri Disease:** This epidemic was described by J Sigurjonsson in Iceland as a Disease Resembling Poliomyelitis. Much to the disagreement of Sigurjonsson and other physicians in Iceland this disease came to be called Akureyri Disease. It was very similar if not identical to later epidemics of Myalgic Encephalomyelitis.

This epidemic started in a local residential school that was located a few hundred feet north of the hospital refuge dump. The epidemic started shortly after the return to school for the autumn session and probably followed significant immunization. Since the epidemic started among school children before it spread to the adults and then to neighbouring towns, there should be no controversy that this type of illness effects children. As in the LA epidemic, the disease manifested both diffuse central and peripheral nervous system symptoms. It was termed an "itis" or an inflammation of the nervous system. This was the first epidemic to demonstrate that we were dealing with a diffuse brain injury and specifically in the area that affected normal sleep and normal muscle physiology. Almost no patients died but all were left disabled and in many cases the disability persists until today, 55 years later.

Three children from this epidemic in the town of Friedrickshavn, became moribund and were unable to leave their beds, they eventually died of Parkinson's-like illness and were autopsied. Parkinson's Disease is almost unheard of in children. There can be no doubt that we were dealing with a diffuse inflammatory brain injury and at least some of these cases, involved the basal ganglia. However for the large number of those who fell ill in Akureyri and neighbouring towns on the north shore of Iceland, the symptoms and signs were those of Myalgic Encephalitis and it was a disease that trapped both child and adult in its icy grips. All true disease processes have variability from minor to major illness, from acute to chronic sequelae. I have seen at least two children in the UK who fell ill with M.E. but could have just as well be diagnosed as Von Economo's Encephalitis-like or Parkinson's-like illness.

## Encephalomyelitis of Unknown Origin 1955: Cumberland:

Dr Wallis did autopsies in the 1955 Cumberland epidemic. This was an epidemic that once again started with children in a boarding school in the early fall and spread to the near-by residents. This publication of one of the most extensive early investigations will appear on our Nightingale website later this year. It might be of interest in repeating the onset symptoms of children and adults who fell ill in 1955:

## Wallis' Onset Symptoms

1. The patients usually stated they thought they had caught a chill or a touch of the "flu", the illness starting immediately or on some occasions, a few days after this infectious illness,
2. Excessive tiredness, finding normal work a burden to them,
3. Difficulty in walking due to legs feeling a loss of power,
4. Sweating, unrelated to work done or the ambient temperature,
5. Difficulty in keeping warm,
6. Bouts of dizziness and unsteadiness,
7. Intermittent headaches and neuralgic pains, often around the eye or down the neck,
8. Insomnia,
9. Loss of clarity of thought and concentration,
10. Aching in the legs and back of an intermittent nature,
11. Pins and needles in hands and feet,
12. Blurred Vision.

## Wallis' Frequent, Later Neurological Signs and Symptoms

1. Paraesthesiae (burning, prickling or formication often in the absence of an external stimulus),
2. Hyperaesthesia (increased pain to touch) and hyperthesiae (increased pain),

3. Impairment of taste or smell,
4. Vertigo,
5. Blurred vision,
6. Loss of concentration or depression,
7. Poor recent memory, diminished powers of abstract thought,
8. Impairment of co-ordination and unsteadiness,
9. General weakness,
10. Inversion of sleep rhythm,
11. Abnormal perception of taste and smell,
12. Visual acuity considered impaired but no objective evidence,
13. Diplopia (double vision) in upward and lateral gaze,
14. Sluggish pupil reactions to light and accommodation,
15. Motor dysfunctions, generally muscular weakness,
16. Ataxia with positive Romberg,
17. Syncope without any epileptic evidence,
18. Tremor in half the cases that usually disappeared after several months,
19. Co-ordination impaired with breaking of crockery, difficulty walking through a doorway, pouring a cup of tea,
20. Reflexes: normal or decreased initially, normal or increased or brisk later.

Anyone with Myalgic Encephalomyelitis will recognize these epidemic features as a classical example of M.E.

**Brain Pathological Changes:** Deaths never occurred with the onset illness but three cases followed shortly after onset. Two patients with M.E. who died subsequent to the M.E. illness demonstrate diffuse micro-haemorrhagic injury of the brain and this occurred around the small blood vessels. In the second case, changes occurred around the basal ganglia as in Akureyri, but this second case was associated with Wilson's disease. Wilson's disease can be confused with M.E./CFS like illness since it has many of the same clinical characteristics. Nevertheless, these are not unlike some of the areas of abnormality noted in brain SPECT scans. But the samples are not sufficient or clear enough to be certain that these are from the ME disease itself.

## Discussion of the Akureyri and the Cumberland Epidemic Features

Both epidemics started with children and went on to involve both children and adults. Both epidemics involved an infectious disease onset and a following neurological illness that had features of both central and peripheral neurological anomalies. Both epidemics involved injuries consistent with the basal ganglia injury (as well as frontal and anterior left parietal lobe injury).

It might therefore be worthwhile to briefly mention some of the basal ganglia associated diseases. These illnesses are not the same as M.E. and in general are much worse but they do have some similar characteristics. The basal ganglia associated illnesses include (a) Parkinson's Disease, (b) Wilson's Disease (the copper metabolism illness), (c) Von Economo's Encephalitis, (See the book Awakening) (d) nerve gas injury, (e) carbon monoxide poisoning and (f) some Gulf War illnesses. Though different from Myalgic Encephalomyelitis the non-progressive features do share a very similar spectrum of symptoms and signs. All are associated with various degrees of sleep dysfunction, rapid exhaustion, muscle pains and weakness, loss of coordination, language difficulty, and neurological symptoms.

**Brain Injury Associations:** It is curious, that no neurologists or physicians have any problem associating these symptoms with brain pathology in the above disease processes but when they occur in M.E. patients they can make the obvious association.

**Children Fall Ill with Myalgic Encephalomyelitis:** Knowing about the Akureyri epidemic and the Cumberland epidemic, it is curious that there should be any debate in the UK or anywhere else that M.E. is also a disease of children. In March 2006 I was in court giving evidence for a M.E. patient and Dr Salit who frequently sees M.E. patients for the insurance

industry and who attended against the patient, made the statement that children do not fall ill with M.E. He uses the term CFS since as he stated, no physician of merit uses Myalgic Encephalomyelitis. He obviously does not appear to know the history of this illness.

The diagnostic problem of identification of M.E. illness is totally social. Why are these children and their parents too often conveniently diagnosed as psychiatric cases? Rather than separating the children from the mothers, rather than calling the parents examples of Munchausen's-by-proxy they could be and should be investigated and they are not.

How should these children and adults be investigated? If they truly have brain dysfunction as I say they do, then their brain function should be abnormal, this should be measurable and these patients should be investigated. Only if we subgroup these patients can we begin to scientifically treat them.

It should also be remembered that 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.

Today, any children or adults with the symptom picture of chronic ongoing Myalgic Encephalomyelitis do not have to die in order for us to examine their brains; we can examine their brains with technological instruments. Let us for this moment only consider the children. All of these children, and the medical and scientific community as well, could benefit if funds were dedicated for the complete and integrated physical and technological examination of these victims. I call them victims since I believe the medical community as a whole have abandoned them, both children and adults. Lack of progress in developing both scientific and medical understanding and treatment protocols are what I believe to be the result of medical and psychiatric arrogance.

It is my belief that the technological medical community should be funded to do a complete cardiovascular, neurovascular, thyroid and endocrine, and neurophysiological assessment of say:

- 250 of these children patients and
- 250 adults with acute onset M.E. and
- 250 gradual onset CFS type patients

The neurophysiological examination side should include (a) brain SPECT scans, (b) Xenon brain SPECT scans, (c) PET scans, (d) QEEG neuropsychological scans as well as (e) MRI scans since some of these children will be missed cases of multiple sclerosis or other space occupying processes or CNS injuries. This might seem excessive to some, but only if such an organized and structural approach is taken will we ever be able to come to grips with these disease processes and separate them out, one nom another.

I have heard so many physicians say that this is too expensive. Yet the cost to examine all 750 patients would definitely be a fraction of the cost of building one atomic bomb, and less than the cost of building one jet war plane. Where are our priorities?

- In the USA $5.5 trillion have been spent on building atomic weapons since 1940. That is more than the Americans have spent on Medicare, veterans' benefits and the total outlays on Social Security in the USA since 1940. (Source: Peter Passell," *New York Times,* July 9, 1998.)
- An F/A-18 in 1997 cost the military $28 million, according to the Blue Angels' official Web site. You can buy this used F/A 18 without armaments on EBay today for only 9 million dollars. Or you can purchase a Russian Sukhoi-30MKK as just sold to the Chinese for $35 million each. (Source: Google)
- Estimated outside cost of a comprehensive program to examine 750 ME patients: 500,000 pounds sterling yearly for 5 years.

## Where are our priorities?

Myalgic Encephalomyelitis 1955-1957: The term was jointly invented by Dr A Melvin Ramsay who coined this name in relation to the Royal Free Hospital epidemics that occurred in London from 1955 to 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 the physicians at the Royal Free Hospitals and in Newcastle-upon- Tyne that they were dealing with the consequences of an epidemic and endemic infectious disease. It was at this same epidemic period that Dr Wallis described the Cumberland Epidemic as Encephalomyelitis of Unknown Origin. It is difficult to imagine that these three physicians and their associates got the name wrong. They were dealing with an encephalomyelitis. What does this term signify?

**Myalgic Encephalomyelitis:** is a simple term that translates into English in the following manner:

- My = muscle
- Algic = pain

- Encephalo = brain
- Mye = spinal cord
- Itis = inflammation

**Myelitis:** In part the name myelitis was a logical association with the illness poliomyelitis that in 1955 was being tamed by the Jonah Salk Poliomyelitis Immunization.

**Criticism of the name, Myalgic Encephalomyelitis:** The critics of this term have had no problem with the Myalgic part referring to muscle pain.

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.

Inflammation is often associated with increased sedimentation rate, fever, inflammatory blood cells but these are not usually seen in paralytic poliomyelitis and yet no one doubts that this is in part an inflammation of the capillaries supplying the anterior horn cells.

Circa 1996, an autopsy was performed on a woman with Myalgic Encephalomyelitis in Newcastle-upon- Tyne by Dr John Richardson and the brain tissue examined by Dr. James Mobray at St Mary's Paddington. This woman had a history of typical Myalgic Encephalomyelitis, was well known by Dr Richardson and accidentally died when her car fell off the side of the pier into the North Atlantic, the cold water preserving the brain tissue. Dr Mowbray was able to demonstrate an autoimmune inflammatory injury at the capillary level of the brain and basement membrane, the area that separates the capillaries from the neurons and brain tissue. In effect the same juxtaposition as in poliomyelitis but in this case in the brain and not in the spinal cord. (Poliovirus also injures the sub cortical areas of the brain.)

Recently an M.E. patient's spine has been examined in the UK and the inflammatory nature was also discovered. Myalgic Encephalitis is a diffuse inflammatory injury of the capillaries at the level of the basement membrane of the brain. It makes no sense to rename the horse and call it Myalgic Encephalopathy. All brain pathologies involving brain tissue are encephalopathies. Let us stop fussing around and get back to the real problem and that is investigating the patients, segregating them into sub-type injuries and working on the treatment of these children and adults.

Were these epidemics that I have spoken about cases of Myalgic Encephalomyelitis? They were. I have personally visited all of these cases except for the Cumberland epidemic and

Wallis left us such a good description of that epidemic that there can be no doubt. I have personally gone to Los Angeles and examined patients from the Los Angeles epidemic. I have gone to Iceland and examined patients from the Akureyri epidemic. I have examined patients from the Royal Free Hospital epidemics, from the Newcastle sporadic illnesses. Many are the same or similar and many of them had been rejected or shunned because they were not true poliomyelitis. However they were all cases of Myalgic Encephalomyelitis.

### So what did we know about M.E. by 1958?

Myalgic Encephalomyelitis (M.E.) could be categorized as follows:

1. M.E. follows a contagious epidemic and endemic infectious disease,

2. M.E. represented a diffuse Central Nervous and in some cases a Peripheral Nervous System Injury in which different organs and different systems were also sometimes involved.

3. M.E. is an illness that follows an infection, probably viral in nature, but different epidemics appear to have been the result of different neurotropic diseases. Some were definitely ECHO and some were other enteroviruses but most were never categorized. (When we studied 100 cases, 40 acute onset and 60 gradual onset cases we found no suggestion of enteroviruses in the gradual onset and only 10 of the 40 acute onset cases were recoverable enteroviruses. Two of the 10 were post-transfusion and 8 of the acute onset were post infectious.) The cause in 90 % of the cases remained a mystery.

4. The incubation period from time of contact with the infection until the appearance of the illness is approximately 4-7 days,

5. In its epidemic form M.E. was most commonly seen in (a) Health Care Workers, (b) children and older students in residential schools, nurses residences and hospitals, (c) in military barracks where students or soldiers were housed in close proximity further supporting the belief in its infectious nature.

6. Although M.E. was not caused by poliovirus in the Akureyri epidemic, infection with M.E. somehow protected the patients from the polio epidemic that swept though Iceland in the 1950s. Polioviruses represent three of approximately 100 different enteroviruses. This was the reason why many in the UK believed that some of these epidemics were probably caused by a less lethal non-polio form of enteroviruses such as ECHO, Coxsackie, the numbered and new enteroviruses.

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## THE CENTRE FOR DISEASE CONTROL DEFINITION OF CHRONIC FATIGUE SYNDROME (CFS)

Do not for one minute believe that CFS is simply another name for Myalgic Encephalomyelitis (M.E.). It is not. Though CFS is based upon a typical M.E. epidemic, in my opinion it has always been a confused and distorted view of reality.

The invention of Chronic Fatigue Syndrome has to be one of the most curious cases of inventive American scientific imperialism that one could imagine.

### Children and Students

Many of the M.E. epidemics started out among children or students. This occurred in 1936 Fond du Lac epidemic, the 1946 to 1949 Akureyri epidemics, the 1950 St Joseph Infirmary epidemic, the 1952 Middlesex epidemic, the 1955 Cumbria epidemic, the 1955 Addington and Durban epidemics, the 1970-1971 Great Ormond Street Children's Hospital. It was not then surprising that the Incline Village epidemic should also start among students.

## The Lake Tahoe Epidemic

The Lake Tahoe epidemic that started in August 1984 also started amongst students. In this case the epidemic began in a high school girls' basketball team that was travelling in a bus to play various other teams. The epidemic spread rapidly with an incubation period of approximately a week. As in many of the other epidemics, it then spread to the general community. After the epidemic started it then involved three high schools, both students and teachers and ultimately spread to the community. For some reason it was considered to be an epidemic of infectious mononucleosis. This is an illness caused by a virus Epstein Barr Syndrome. Associating the Lake Tahoe epidemic with Epstein Barr Syndrome was frankly ridiculous and you will see why almost immediately.

Dr Paul Cheney and Dr Daniel Peterson were inundated by the number of rapidly developing cases of seriously ill patients and called the Centre for Disease Control (CDC) in Atlanta for back up.

## First International Symposium on Immunology and Pathogenesis of Persistent Virus Infections

Fast-forward to April 1987 and the First International Symposium on Immunology and Pathogenesis of Persistent Virus Infections held in Atlanta Georgia. This was a symposium hosted by the CDC and Dr Carlos Lopez. At this meeting Dr Gary Holmes gave out his new paper, "A cluster of patients with a chronic mononucleosis-like syndrome," that had just been published in JAMA. (See Holmes, Kaplan, Stewart et al: JAMA 1987:287:2297-2302)

The publication essentially stated that Epstein Barr Virus was not the apparent cause of this illness in the 130 patients from which they took blood samples. But they weren't sure and suggested that further study be done.

## Epstein Barr Virus (EBV)

Now anyone who realizes that infectious mononucleosis is caused by the herpes family virus, Epstein Barr Virus (EBV), and that the incubation period of this illness is approximately 40 days, should have realized that you simply cannot have a rapidly spreading viral epidemic with a virus with a latent period of 40 days.

Neither Dr Straus nor Dr Holmes, senior government physicians, should have fallen into such a trap. They only had to go to the excellent CDC library to realize that rather than spending half a million dollars or so on a publication that they should have known would not have incriminated EBV.

Yet this epidemic somehow spread the myth that this illness was caused by EBV. Today, as I write this short history of M.E. and CFS the vast majority of physicians and the public still associate Epstein Barr Virus with CFS. Such is the perseverance of error.

## Human Herpes Virus 6 (HHV6)

This virus was not associated with CFS until after the 1990 period. HHV6 is the virus that causes the benign childhood illness, Roseola. By 1986 HHV6 was already known to have an incubation period of 9 days due to human experimentation when the actual virus was injected into several children. See (Gorbac, Second Edition, Infectious Diseases, page 1335). When acquired by random infection, the incubation period of HHV6 Roseola was more like 12 days. So once again anyone with access to a library or a computer would have soon dispelled any view that HHV6 was a cause of M.E./CFS epidemics where the incubation was approximately 7 days or less.

Is it possible that Steven Strauss and the other intelligentsia of the National Institute of Health (NIH) in Bethesda and CDC in Atlanta and elsewhere didn't have access to libraries and the Internet? Maybe we should start a public request to ask for donations for them.

### What did we know about M.E. in 1984 after the Lake Tahoe epidemic?

- The CDC investigators and the physicians of Lake Tahoe were dealing with **a rapidly spreading infectious disease with a short one week or less incubation period**. Obviously this was consistent with the epidemics of Myalgic Encephalomyelitis already documented in this brief history.

- Like the several epidemics noted that **started with children or students**, so did this.

- Like the patients in all of the epidemics discussed, the **effects of the infection involved the Central Nervous System** but unlike a stroke caused by an embolism, or malignancy, or arterial obstruction, the CNS involvement that occurred in these patients were not focal but consistent with **a diffuse CNS injury**.

- In the Lake Tahoe epidemic as in the previous epidemics described, the type of Central Nervous System involvement was obviously of a more diffuse nature and the type of peripheral involvement that caused so many troubling symptoms in all these epidemics was consistent with **a very low grade vasculitis** (See Mercy San Juan Hospital Epidemic) or in many cases **a classical radiculopathy** (spinal nerve root involvement) or even **a very low grade Guillam Barre Syndrome** as was described by Alberto Marinacci when he examined the Las Angeles County Hospital patients. (See Dr Marinacci's book Applied Electromyography. Lea & Febiger, 1968: Chapter 9). However, I should note that the mere mention of Guillam Barre Syndrome drives many neurologists crazy. They say that GB Syndrome is a severe disease that if not treated effectively may kill or leave the patient permanently disabled. However, all real diseases have a wide variety of penetration from so mild that they may be missed to, in some diseases, having potentially mortal consequences.

If we consider the Lake Tahoe epidemic alone we have the primary definitional determinant of Myalgic Encephalomyelitis.

*The Lake Tahoe Epidemic represented an illness*

a. *With an acute onset,*

b. *With an incubation period of* 4-7 *days*

c. *Occurring in both students and adults,*

d. *Involving the central nervous system in a diffuse, non focal manner,*

e. *The onset of a Raynaud's disease with a peripheral coldness, blanching and pain syndrome of fingers, hands and feet or significant postural hypotension or instability. A non-traumatic, acute onset of these two syndromes is consistent with an injury or a significant diffuse change in the autonomic physiology of the sub cortical brain.*

f. *rapidly developing flaccid muscle weakness with minimal effort or activity, (The Lake Tahoe epidemic was initially called Raggedy Anne Syndrome due to this finding.)*

*g. There were two illnesses, an acute viral like illness and a secondary persisting illnesses that in the more severe cases left permanent persisting sequelae*

*h. With peripheral pain symptoms that have variable features resembling in some cases, a radiculopathy, is some cases a vasculitis, and even a very low grade Guillam Barre Syndrome,*

Although the final terminology of conclusion *"h"* is subject to debate, are features *"a to g"* a very difficult set of conclusions to come to? I don't thinks so. There is a consistent similarity of the Lake Tahoe epidemic patients to all of the previous epidemics mentioned in this short history and the many others that are documented in our textbook, The Clinical and Scientific Basis of Mvalgic Encephalomyelitis / Chronic Fatigue Syndrome.

Yet retain these above Lake Tahoe features in mind when we come to the first CDC definition that was largely based upon this very same Lake Tahoe epidemic illness.

## Major Problems of the 1988 CDC definition

It is my opinion that the CDC 1988 definition of CFS describes a non-existing chimera based upon inexperienced individuals who lack any historical knowledge of this disease process. The CDC definition is not a disease process. 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. Let us try to decipher this definition.

1. **The principal author:** Dr Gary Holmes is one of those men who it is difficult not to like. From my limited knowledge of Dr Holmes it is my opinion that he is well organized, brilliant, a kind man and the sort of person any university would want to have on staff. To my knowledge he never continued to show any interest in this disease process and Pub Med and Google search fail to reveal any subsequent scientific papers concerning M.E. or CFS.

2. **The other authors:** So curious was the 1988 CDC definition that if you review the authors, you will find that the majority had never published on M.E. or CFS either before or after this definitional publication and the majority had never ever to my knowledge ever before or since examined or investigated any serious number of CFS patients. In fact, I would estimate that the majority had never actually examined and investigated as single M.E. patient.

3. **The curious name:** The authors named the disease Chronic Fatigue Syndrome: Fatigue is a totally indefinable concept. Fatigue is impossible to measure or quantify. Fatigue is so non-specific that it can be a common element in any acute or chronic disease and many psychiatric diseases. Worse, it redirects the medical and public attention to the totally indefinable fatigue and away from the obvious Central Nervous System changes in these patients. Much worse, it makes fun of a serious illness since most people and most physicians tend to equate fatigue with laziness, work avoidance, something that a bit of effort will chase away. It has turned out to be a damning indictment to all M.E. patients.

4. **The first Major Criteria:** This 1988 CDC definition contains (a) two major criteria, (b) 11 Minor Criteria, (c) three physical criteria. Let us start with the first major criteria:

   > *"A new onset of persistent or relapsing, debilitating fatigue or easy fatigability in a person who has no previous history of similar symptoms, that does not resolve with bed rest, and is severe enough to reduce or impair average daily activity below 50% of the patients premorbid activity level for a period of at least 6 months."*

   This major criterion does not clearly distinguish between acute or gradual onset diseases. In all M.E. epidemic or endemic patients the patients represent acute onset illnesses. The fatigue criteria listed here can be found in hundreds of chronic illnesses and clearly defines nothing.

5. **The second Major Criteria:** This makes the illness CFS a disease of exclusion. The definitional statement is:

   > *"Exclude all other disease processes. "*

   Any disease process that has major criteria, of excluding all other disease processes, is simply not a disease at all; it doesn't exist. In effect, by either the first or second major criteria this is nor a measurable illness and a disease that is not measurable or testable simply does not exist. What did Dr Holmes and his colleagues miss? They missed the fact that M.E. is (a) an acute onset illness, (b) the fact that M.E. is a measurable diffuse brain injury, (c) in a complete form. M.E. has a dual inception, an infectious illness followed by the diffuse neurological aspects of this disease.

6. **The Minor Criteria** are consistent with M.E. but unfortunately for the greater part, are also consistent with Infectious Mononucleosis that I believe the authors of these diagnostic criteria thought they were describing.

7. **The Three Physical Criteria of the CDC 1988 Definition:** These findings are totally related to infectious mononucleosis and not to the normal or average Myalgic Encephalomyelitis. The criteria fail to distinguish the biphasic nature of M.E. as mentioned before, the initial infectious illness that often resembles the minor infection that heralds another biphasic disease, paralytical poliomyelitis. The infectious disease process varies but is usually minor and after three or four days is usually unverifiable so that any researcher who quotes the patient as having the three physical criteria when he or she examines the patient probably at the very least can be accused of being very imaginative. First it is not possible to examine any patient in the first days of illness unless it is an epidemic situation. In several chronic thousand patients I have examined the three physical criteria simply do not exist in more than 1 % of the patients examined. What are the CDC Physical Criteria?

   a. Low-grade fever with an oral temperature between 37.6 and 38.6 centigrade,
   b. Non-exudative pharyngitis (without any pus or discharge),
   c. Palpable or tender anterior or posterior cervical or axillary lymph nodes less than 2 cm in diameter.

In the chronic patients the temperature tends to be normal or subnormal. Most chronic patients have no pharyngitis, they may have a dry pharynx, they may have an injected pharyngeal area around the tonsilar pillars, (Anne Mildon effect) but generally they don't have a classical pharyngitis as seen in any acute infectious disease. As to the palpable lymph nodes, all healthy patients well or otherwise unless they are severely obese have palpable lymph nodes. Since many M.E. patients have hypersensitive skin or fibromyalgia of course they have tenderness. But painful lymph nodes scarcely are different from what is found in any acute upper respiratory track infection. If you are going to list physical findings then you have to first specify whether this is in the first few days of the illness or in the chronic phase and as mentioned almost no physician will ever see acute onset illness unless in an epidemic. In other words these physical criteria are at best of no diagnostic importance and in general, useless.

8. **The Insurance Company** - psychological bias: the direction given in the name Chronic Fatigue Syndrome has opened the door for insurance companies to invent and support a pseudo-psychological treatment of physical and cognitive therapy that in my view has been used to push the patients so far that they then quit the program and this allows the insurance company to define the disabled patient as non-compliant and allows the insurance company to stop insurance payments. Since many if not most insurance policies also cut the patient off after two years of disability, this psychological interpretation has been destructive to the many patients disabled by M.E.

9. **The pharmaceutical companies bias:** These companies have also jumped into the door opened by this name of chronic fatigue - depression association in recommending a non-stop series of "new and better" anti-depressive medications that not only have added little if anything to the patients recovery but in many cases have caused suicides and even greater fatigue. Since many of these medications have a side effect of causing obesity, the patient's self worth is often further deteriorated.

## THE UK DEFINITIONS AND THE LURE OF AMERICAN GOLD

Starting well prior to 1988 a deepening crisis loomed in both US and North American Research funding, in fact, there was no place in the world where there was sufficient funds to support the scientific community who did not work for commercial interests.

With the publication of the 1988 CFS definition, NIH made it public that there was going to be millions' of dollars distributed to worthy scientists and clinicians who wished to investigate CFS, not just in North America but also in the world. Generally speaking it was not true, of course, and, as mentioned earlier, most of the first 38 million dollars went to existing projects on alcoholism, herpes virus research and other projects that had nothing to do with M.E. or CFS. Nightingale was able to document this in 1992 but later it became a generally openly published scandal. Of course the financially starved UK physicians and researchers did not know the history of the NIH funding. From the early 1900s or even earlier, access to American Government Research medical funding was highly concentrated in the north east states and for all purposes didn't even approach the mid west and western states. If there were funding exceptions to this general rule, these were funds that went to researchers who had done their training at Harvard or the other blue blood eastern Universities and at CDC. Never the less, the British trout jumped at the bait and organized what was published as the Oxford Guidelines in February 1991.

There were some good very good clinicians and researchers on this definitional committee. Of the 21 researchers and clinicians who attended, the meeting was chaired by Professor Anthony Clare, a psychiatrist to constitute a total of 8 psychiatrists or individuals working in the field of psychology all who reputedly had studied patients with CFS.

The composition of this definitional meeting was a commercially rational decision. Psychiatrists don't require expensive labs with expensive technology and they don't have to examine patients or cause the Government any expense in doing expensive "useless tests". A diagnosis

of hysteria, psychosis, neurosis can be made as fast as it takes to open one's mouth and what is even better, there if there is no test to prove a psychiatric diagnosis correct, there is no test to prove them wrong either. Perhaps, it is for this reason that psychiatrists are used so frequently by the insurance industry to deprive the individual disabled M.E./ CFS patient from their disability pension.

Let us to a quick accounting of the distribution of specialists in this committee:

1. Psychiatrists or psychologists: 38 %
2. Infectious Disease: 4 persons or 19 %
3. Biochemist: 2 persons or 10 %
4. Internal Medicine: 5%
5. Pharmaceutical Corporation: 5%
6. Immunopathologic: 1 person or 5%
7. MRI Specialist: 1 person or 5%
8. GP: I person or 5%
9. Neurologist: I person of 5%

There was no nuclear medicine specialist in either SPECT or PET, nor were there any QEEG specialists, all who can map diffuse CNS injuries.

Since it would appear that we are definitely dealing with a disease that injures the immune system it is curious that only one person in this field was invited. We find a lot of secondary endocrine dysfunction or injury in M.E. patients and there was no endocrinologist. However, what is really criminal about the Oxford Definition is the composition of the committee and the presence of this overwhelming psychiatric lobby. In effect the Oxford definitions are not only bad since they are in effect a copy and a variation of the CDC definition and the authors in general do not appear to understand the definitions of Myalgic Encephalomyelitis but in addition they are onerous because they further lead the way with the psychiatric-sation of physical medicine in general and the psychiatrization of M.E. in particular or the technical confusion implicit in the CDC diagnostic criteria.
Psychiatrists are essential in modem medicine and in the evaluation and treatment of psychiatrically ill patients but psychiatrists have no primary role in the medical evaluation of physically injured patients: they only have place in the obscuring of the complex medical problems of M.E. patients.
I believe that no psychiatrist has ever cured an M.E. patient using psychiatric treatments and what we are looking for in a patient is diagnostic understanding and a cure where and when that is possible. Diagnostic understanding will only come with the scientific investigation of patients and the scientific treatment when and if these treatments are possible.

Essentially, like any subspecialty, M.E. is not a place for part time workers except in consultation but a discipline that requires physicians who are totally dedicated full time to the understanding of these patients, as are the specialists in any area of medicine.

You may believe that I am negative about the views of psychiatry. In part this is true.
When in my practice I see Canadian psychiatrists putting in writing that an M.E. patient has no psychiatric illness when the patient has a disability pension that pays the psychiatrically ill patients it makes me question where psychiatry is going. When the same psychiatrists then say that another M.E. patient has psychiatric disease when the insurance policy states that they don't pay pensions for psychiatric disease I know where psychiatry is gone.
They have become the handmaids of commercial interests not of medicine.

One grows sceptical of the bad faith of psychiatry. Psychiatric treatment is very useful and essential for psychiatric patients. Primary M.E. patients are simply not psychiatric patients. Unfortunately, it is not only psychiatrist physicians that have made themselves the tools of insurance companies.

Good medicine, all comes down to carefully examining the patient, by careful history, careful physical and careful scientific investigation to the best of our abilities. Only by considering the individual and finding out what exactly is making them ill and understanding the complexities of their illnesses is going to solve the M.E. problem. Medicine is not about twisting the facts to support some addled psychiatric theory. Essentially, if you cannot first prove a disease by careful examination and scientific reproducible testing and upon this search for adequate treatments, if we cannot do this English physicians essentially are simply sending all chronically ill Britons and British medicine back into the dark ages.
What we really require to investigate M.E. type disease is a battery of neuro-physiologists with another group of physicians to do a total body and illness mapping of the patient.

We certainly require a better association of treatment investigation funded by governments and not only by the pharmaceutical industry. We know a lot about some of the components of M.E. that should be treatable such as the vascular destabilization of many M.E. patients. However, I am not aware of anyone working on this aspect of the disease process.
None of these physicians were present, 'nor was the will, nor was the means to develop such a program. Once again, this definition or two definitions were really a dead end.

Essentially, for some of the attendants, I believe that the Oxford Definition was part of belief structure, that it would aid those physicians to obtain US funds for research when research funds in the UK was rapidly drying up.
If that was the case, it didn't succeed. Much of the US funds for CFS research was a myth.

The UK was left with a definition that few if any have ever used.

## CDC's 1992 DEFINITION OF CHRONIC FATIGUE SYNDROME

The CDC website defines this CFS definition as:

> *This case definition was authored by a group of international CFS experts, convened by the U.S. Centers for Disease Control & Prevention (CDC) in* 1994, *to update and refine an earlier (1*988) *case definition. Its purpose was to provide standard criteria for researchers who were investigating the illness.*

How truthful is this statement?

Were the 16 CFS experts actually CFS experts? It does not appear so. Let us start there.

Only three of the 16 authors of the 1992 CDC definition is well known in the M.E./CFS world and of those three, one was a NIH bureaucrat. In the 1992 CDC definition the two principal authors were Keiji Fukuda, M.D., M.P.H and Dr Stephen E. Straus. Dr Fukuda, the primary author was a very learned expert in Hansen's disease (Leprosy) not CFS and had never to my knowledge ever previously examined or investigated a single M.E./CFS patient.

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Like the ghosts on the 1988 definition, Fukuda disappeared in CFS obscurity. Nobody has ever used this mammoth definition of CFS. It was totally unusable. It removed patients as being ill with CFS with multiple disease conditions associated with M.E. It took the psychiatric approach of not permitting the intensive investigation of patients who essentially had an unknown complex disease process. The definition failed to address the findings that we already knew from the Lake Tahoe epidemic mentioned in this history or in any of the previous epidemics. This definition was a waste of good money.
The second author was Dr. Stephen E. Straus, M.D. He was essentially a NIH bureaucrat physician who I believe, was largely responsible in distributing funds for M.E/ CFS research.

The next two on the masthead were psychiatrists. If in the first CDC definition, it was unclear where the misinformed authors were taking this definition, i.e. to the world of medicine or to the world of psychiatry, then like in the Oxford definitions, we have no doubts as to the

direction of this 1992 CDC definition. In fact it was increasingly obvious where the definitional direction was going in a psychiatric direction. In fact, the only disease you don't have to test for is a clear-cut psychiatric disease that is if you are also totally sure that the patient doesn't have any other major illness.

Dr Ian Hickie, a learned and charming Australian psychiatrist was the third on the masthead of this definition. I had dinner with him in Australia and he was quite convinced that CFS was essentially a psychiatric disease. He was very well liked by his Australian colleagues but he did not seem to know or be interested in the investigation of physiological CNS dysfunction.

I have met him at a conference but don't really know Dr Michael C. Sharpe, M.R.C.P., M.R.C. Psych.; He is also an Oxford psychiatrist like Beard and McEvedy and fourth on the masthead. I assume like Dr Hickie, he believed that CFS was a psychiatric disease.

Dr James G. Dobbins, Ph.D. the fifth on the masthead as far as I am concerned, is a learned and brilliant medical bureaucrat associated with CDC Atlanta. Although he has his name on many CFS publications I do not know his views and he is not a physician.
Of all of the authors on the masthead of the 1992 definition, to my knowledge, only Anthony L. Komaroff, M.D., F.A.C.P of Harvard has spent any ongoing serious time examining and testing CFS patients. He is a learned and brilliant researcher and clinician and medical historian and to the best of my knowledge was the only person listed as principal author who was truly knowledgeable concerning M.E. and any understanding that gave rise to the 1988 CDC definition of CFS.

It is understandable that their laborious and endless definition, to my knowledge, has never actually been used except in lip service for researchers attempting to obtain grants from the NIH.

## THE 2003 CANADIAN M.E. AND CFS DEFINITION

I will not discuss this definition in any detail since one of the authors will be doing so. Let me say, that this is the first definition constructed by specialists who have spent collectively over 100 years studying M.E. and CFS. It is the first definition and introduction of M.E. and CFS that makes a bit of sense, but like I in the first years of study, I confused M.E. and CFS as being the same. As I have explained, they are not. However, until a better set of definitions is constructed, we should go with the so-called Canadian definition. In effect, the Canadian definition represents some of the best, the most experienced and the most learned of the North American physicians who have studied M.E. and CFS. It is also the first major definition to bring back the term Myalgic Encephalomyelitis.

## THE FUTURE OF M.E. AND CFS DEFINITIONS

I believe that M.E. and CFS should be separated as definitions. They are not the same.

I would like to propose that M.E. be defined simply as being consistent with the majority of the ten following diagnostic features:

**Myalgic Encephalomyelitis is:**

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

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

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

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

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

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

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

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

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

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

    a. POTS: severe postural hypotension.

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

    c. Raynaud's Disease: vasoconstriction, blanching, coldness and pain of extremities

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

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

a. Pituitary-thyroid axis: This is common. Changes in serum TSH, FTI, FT4, Microsomal Ab., PTH, Calcium and phosphorus rarely occur until one or more years after illness onset and usually only after several years. This can be followed by ultrasound of the thyroid gland where a steady shrinking of the thyroid gland occurs with or without the development of non-serum positive Hashimoto's thyroiditis (a seeming contradiction of terms) and a significant increase in thyroid malignancy. Serum positive changes occur only after years.
b. Pituitary-adrenal axis changes: this finding is infrequent.
c. Pituitary-ovarian axis changes:
d. Pituitary- (adrenal?)-Bladder dysfunction: occurs frequently in the early disease in some people. It is unknown if the cause is due to this link.

## SUGGESTED DIAGNOSIS OF CHRONIC FATIGUE SYNDROME

I would like to suggest the following radical new definition of Chronic Fatigue Syndrome but first I would like to give my reasons for this concept.

**Argument rationale:**

Increasingly over the past 60 years there has been a migration of rural inhabitants to the city. In 1945, approximately 60% of all people lived in the country or in small villages and towns. Town physicians were often well paid. In the post WWII period there has been a severe shift of populations in both the UK and in North America to large urban centres. In the United States there has' also been a shift of central urban white populations to the suburbs or peripheral areas of cities but these populations still are anchored to the urban centres by the automobile.

During this population shift various forms of state medicine and insurance medicine has become part of every day medical life. During this same period, particularly during the past 35 years most physicians real after tax salaries have plummeted. This loss of real income of physicians may have fallen as much as 50% to 70% of the original income buying power. Physicians moved to the population centres.

To compensate for the rapidly falling real income, physicians, at least in North America, have increased the number of patients that they see in any given hour. Instead of the examining room there are now a string of examining rooms where the physician runs between the cells, dispenses shoot from the hip diagnosis and rapid dispensing of pharmaceuticals that the industry suggest are the latest miracle. Careful history taking, examination and diagnosis have become too expensive for the average physician to maintain a superior income.

In this drive to maintain income levels it has become simply too costly for the physician to deal with complex medical issues where the patient cannot be gotten out of the physician's presence in less than 7-8 minutes, usually less. Due to this financial fact of life, several changes have occurred:

a. Physicians do not want to see sick people, only routine healthy people can be got in and out of the office efficiently in the allotted time.

b. Physicians above all do not want to see patients with complex problems and complex illnesses.

c. To assist this process of, time is money, physicians prescribe more pharmaceuticals many of which have adverse side effects that can make the ill patient even worse and over time, this has lead to the patient seeking alternative medicine answers, many of which are even more dangerous than the physician quick diagnosis and even quicker pharmaceutical treatment.

d. Diagnostic medical technology, particularly during the past 15 years has increased in excellence and has outstripped government willingness to pay for it. Accordingly,

technology in North America and the UK has become increasingly accessible only to the relatively wealthy citizens.

e. Also, physicians are increasingly avoiding many technologies simply due to the fact that it may take up to an hour to write out a detailed description of the patient's illness for the technical expert performing the test. Also it takes time for the physician to understand these tests and discuss them with the technical experts until the diagnostic physicians have built up their own diagnostic abilities. Due to financial considerations, this is simply not being done. Physicians are avoiding these powerful technologies.

f. Diagnostic technology for an increasing number of physicians means writing a few blood or urine tests. These are fast and cheap but rarely effective in uncovering complex medical illnesses.

g. Most university medical schools have also failed the public in that they teach the 1890 Oslerian view of medicine, an excellent aspect of medicine in its time and still the only way to go in acute onset single entity illness. This consisted of reducing all medical problems to acute illnesses or single organ or single system injuries or pathologies. Osler essentially stated find the unit cause of the illness, treat it and you have a chance of recovery. Simple as it may seem today, this was a radical new approach to diagnostic medicine. At a University Medical School level, the concept of Osler's still pervades. Chronic illness tends to be concerned with old people with multiple medical problems. In general the concept of young people with multiple factor illnesses simply does not appear on the busy learning schedule.

h. These and other factors have given rise to the concept of Chronic Fatigue Syndrome as much as the CDC itself. Chronic Fatigue Syndrome has become a convenient coat hanger for any patient who is fatigued. Most fatigued patients represent complex medical disease, missed major disease, chronic disease, or psychiatric disease, for which the physician simply has not time. Most physicians best deal with these patients by sending them to a psychiatrist.

i. A good psychiatrist in Canada will take a good history and also examine the patient and decide that the patient is either physically ill or psychiatrically ill. If this patient is considered physically ill, they often end up in limbo between physicians since few physicians wish to take the time to examine these patients properly.

j. What I am talking about is the general failure of much of modem medicine to act using the age-old medical principals of (a) careful history, (b) detailed physical examination and (c) appropriate investigation.

## NEW, CHRONIC FATIGUE SYNDROME DIAGNOSTIC CRITERIA

The patient has:

1. **A gradual onset fatigue syndrome**,

2. This is usually due to a missed major disease in which common things are common: i.e. the patient has:

    a. Missed cardiac disease,
    b. Missed malignancy,
    c. Missed vascular disease,
    d. Missed brain lesion either of a vascular or space occupying lesion, e. Missed test positive rheumatologic disease,
    e. Missed test negative rheumatologic disease,
    f. Missed endocrine disease,
    g. Missed physiological disease,
    h. Missed genetic disease,
    i. Missed chronic infectious disease,
    j. Missed pharmacological or immunization induced disease,
    k. Missed social disease,
    l. Missed drug use disease or habituation,
    m. Missed dietary dysfunction diseases,
    n. Missed psychiatric disease.

You will notice that

1. My diagnostic criteria for Myalgic Encephalomyelitis are little changed from the older experts diagnostic criteria for M.E. discussed briefly in this discussion paper and in more detail in our book The Clinical and Scientific Basis of Myalgic Encephalomyelitis and Chronic Fatigue Syndrome.

2. My diagnostic criteria restore Myalgic Encephalomyelitis to a CNS disease entity, stripping it away from the CDC Chronic Fatigue Syndrome Diagnosis collage of disease entities.

3. My diagnostic criteria for M.E. firmly note it as an acute onset disease and add technological diagnostic techniques to its understanding.

4. My diagnostic criteria for CFS are vastly different from all previous CDC diagnostic criteria including the new Canadian diagnostic criteria and place it where most physicians leave it, as a gradual onset missed major diagnosis.

5. Both diagnostic criteria take up less than a page, something that a modem physician will actually read and possibly act upon.

This material will be available on the Nightingale's new website http://www.nightingale.ca in September 2006 or earlier.