From: LK Woodruff
Sent: Monday, May 25, 2009 12:52 AM
To: OS OPHS CFSAC (HHS/OPHS)
Subject: Fw: the USA CDC, re: the CFS debacle, 4/17/2009
Importance: High

5/25/2009

TO: CFSAC

I know the mtg is over, but I think you need to also read my recent submission to the CDC (below).

'CFS' (Fukuda, et al) has helped no one; it is a tiny little 'syndrome' based on 'fatigue' and sore throats. A 'diagnosis of exclusion' only to be made after 6 months, when everything else has been crossed off the list. It is nothing more (tho God knows many are trying to make it so!) This current trend to 'blend' illnesses--or bits and pieces of them--together is absurd and not base don any science.

-->Scientific protocols must be adhered to at all times.

-->And each and every patient needs to be correctly diagnosed per the well-established WHO classification system. It is not the right nor the job of confused patients to self-diagnose, or to keep making up illnesses to suit their own situations, etc. And far too many diagnosed with 'CFS' actually have a myriad of OTHER things wrong with them, many of which are tretable.

Huge changes need to be made. *This nonsense, these past 20+ years, cannot continue. Especially the assumption that 'CFS' is the same thing as ME, G93.3. It most assuredly is not, and this thinking/approach is extremely detrimental to true ME patients, who are severely debiltitated with a CNS/multi-systemic illness. They are NOT 'fatigued' for a variety of reasons, as are the 'CFS' crowd.*

ME, G93.3, needs to be acknowledged and studied separately. On it's own. The WHO has formally acknolwdeged it since 1969!!!!
And 'CFS' should be dropped. It is too vague; too tiny; too broad. Correctly diagnose those patients, instead.

Sincerely,
LKWoodruff

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----- Original Message -----
From: LK Woodruff
To: CFSResearchPlan@cdc.gov
Sent: Friday, April 17, 2009 4:11 PM
Subject: TO: the USA CDC, re: the CFS debacle, 4/17/2009

4/17/2009

TO: The USA CDC
RE: The CFS Research Plan (public comments period)

****permission to share with interested parties****

I am unable to travel, or attend conferences. Most of us with ME, G93.3 are in that position, which is why you never see us in person.
We only have our pc's to help deliver our urgent message.

Therefore, please read this very lengthy communication very, very carefully, as much is at stake here and it contains valuable and important information that should impact your decisions greatly.

Remember that ME patient's health and lives are at stake here (although they shouldn't be!)

And that correct and proper illness diagnosis is every patient's right.

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Massive illness and patient confusion has ruled--these past 20+ years--since 'CFS' (Fukuda, et al) was first written b/n 1988-1994.
'CFS' was first made-up, written back then in response to what the USA CDC staff thought was a ***chronic Epstein Barr (EBV) situation****.*
-->In other words: it was never written to describe ME, G93.3.

The consensus NOW is that the Lake Tahoe outbreak was actually a ME, G93.3, outbreak.
Back then, it was reported to be something 'new' and 'emerging', by the USA CDC staff.
Tho others present told them it was another ME, G93.3, outbreak.
Also remember that ME, G93.3, has been formally acknowledge and classified by the WHO since 1969.

What has taken place since then, in differing ways, in different countries, and to varying degrees is:
an illogical and unscientific 'blending' of CFS (R53.82) with ME (G93.3) primarily in the forms of:

1 - 'CFS/ME' (a 'psychosocial illness MODEL' made-up by the UK Wessely School ~psychs~ to help insurance companies deny patient claims, and for which they have been well-compensated) and

2 - 'ME/CFS' (the 2003 Canadian Criteria, based on an illogical and unscientific 'blend' of CFS, R53.82, and ME, G93.3.)

NOTE that neither of these two made-up terms--CFS/ME and ME/CFS--has formal WHO recognition, acknowledgement, or classification.

NOR can any syndrome, illness or disease be classified by the WHO in two different places!
(Yet somehow the British CMO--sometime b/n the early 1990's and the early 2000's; the details remain elusive--somehow approved adding 'post viral' CFS (only) to G93.3?! That was a mistake that needs to be UNdone ASAP. If for no other reason than: there is no such thing as 'post viral' CFS! Too many folks get that 'post viral' part confused....)

Mistakes and misguided ideas and illness misperceptions like these have flourished, driven no doubt by the ease of information transfer on the internet, where droves of patients can believe whatever they read, rather than focus on actual scientific research abstracts and demand that SCIENTIFIC PROTOCOLS be adhered to, etc. Where patients--many of them clearly SELF-diagnosed and/or MIS-diagnosed--think they can drive this train. Well, to-date they are only managing to de-rail everything right off the rials and into the ditch:(

Desperate CFS patients--who are often MISdiagnosed and actually have a myriad of other things wrong with them, many of which are treatable, like Lyme (A69.2), thyroid (E00-E07), CMV & EBV (G05), or 'fatigue' and 'malaise' (R53)--see more below--have tried to make their illnesses sound more serious in an attempt to override Reeves and the WS's attempts. While this is understandable--to a point--they are also taking bits and pieces from here, there, everywhere...and making things up as they go along!! -->This cannot continue.

One of the ways they--and even some self-proclaimed 'experts'--have done this is to latch onto ME. G93.3, and now start saying that ME and CFS belong together and are actually the same thing.

- They most certainly are not!

- ME, G93.3, is classified under Brain, CNS, Neurology.... It starts with a 'sudden onset viral event'....

- CFS is a syndrome based on fatigue with a slow onset; a 'diagnosis of exclusion' that can only be made after 6 months, and when everything else has

been crossed off the list.... It has always been classified under vector-borne, zoontic, to....

Where is any similarity?!

- Furthermore, G's in the WHO clasifications (ME, G93.3), do not get mixed with R's (CFS, R53.82)!! They are different bodily systems.

Let's all get the pieces into proper order, and keep them there, shall we????

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It is beyond time for the world to take ME, G93.3, seriously and stop trying to subsume it with, or under, 'CFS'. That is both absurd and completely UNscientific. Those presently diagnosed with 'CFS' need to be better evaluated to determine precisely what it is that they have.

- CFS patients like this 'blending' and 'lumping together' of two disparate things because it makes their tiny 'syndrome' (i.e., a group of symptoms) based on 'fatigue' sound more serious.

- ME-defined patients are infuriated with this 'blending' and 'lumping together' of two disparate things because it waters down and weakens our severely debilitating CNS (Central Nervous System) and multi-systemic illness!!!

-->Let us all focus on scientific protocols, illness-specific databases, and keeping the facts and details straight, shall we???? It'd be a good first step toward improving and unraveling this tangled mess....

*And to add even more to this horrific mess now: since 2005, the USA CDC's Reeves and the UK WS's psychs are collaboratively pushing their **'empirical definition'** for CFS hard, in an attempt to both expand the numbers and funding AND to get 'CFS' classified under **'mental'**.*

And with all of this ludicrous blending and subsuming going on - that could well also land ME, G93.3 there!
That would be an even greater tragedy than what has already taken place so far. It would be absolutely unconscionable.

Here is critically important ME, G93.3 (only) information.

- Note that it is in no way comparable to CFS, R53.82.
- The incubation periods for each are very different.
- The prodomal phase of each is quite different.
- Each starts, presents and ends differently.
- The CFS criteria(s) do not begin to compare with the ME Definition.

- And Dr Byron Hyde, in Ottaowa, Canada, is the world's pre-eminent authority on ME, G93.3.
-->Start working with him!!!!!!!!!!!!!!!!!!

What follows:

1) Hyde's statement on the various incubation periods for proposed 'causes', followed by his
2) 'prodomal phase for ME', followed by his
3) newest (2006) ME Definition, which is excellent. And finally,
4) a paper on the many, many things 'CFS' patients are actually being diagnosed with, when evaluated thoroughly and completely.

Please read it all very carefully and pay full attention to the details, as they do matter incredibly, and are much more important than the blending 'CFS', 'CFS/ME' &/or 'ME/CFS' proponents seem to realize:(

Thank you very much.
And now please do the RIGHT THINGS to help all of us.

LaVonne K Woodruff, USA

P.S. I am a ME-defined patient who's done serious illness advocacy for 6+ years now. I got hit by ME 11 years ago (Feb 17th, 1998) and my health and my life as I knew them changed literally overnight, within a 12 hour period. This is not fatigue - it is severe CNS damage.

Hyde on incubation periods

*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. 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. - Dr Hyde M.D.*

*Associating the Lake Tahoe epidemic with Epstein Barr Syndrome was frankly ridiculous and you will see why almost immediately. 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.
Such is the perseverance of error. - Dr Hyde M.D.

*"[The] **prodromal phase of ME, G93.3** is associated with a 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.

The usual incubation period of the triggering illness is 4-7 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 infectious triggering illness

Hyde (1998 [Online])."

NOTE: highlights and underlines are mine. LKW

http://www.nightingale.ca/documents/NightingalesDefinitionofME.pdf

The Nightingale Definition of Myalgic Encephalomyelitis (M.E.)

Preface

Since the Nightingale Research Foundation's publication in 1992 of its textbook, The Clinical and Scientific Basis of Myalgic Encephalomyelitis / Chronic Fatigue Syndrome, there has been a tendency by some individuals and organizations to assume that M.E. and CFS are the same illness. Over the course of two International Association of Chronic Fatigue Syndrome (IACFS, formerly the American Association of CFS) conferences, there have been suggestions that the name CFS be changed to M.E., while retaining the CFS definitions as a basis for such change. This does not seem to me to be a useful initiative: it would simply add credence to the mistaken assumption that M.E. and CFS represent the same disease processes. They do not.
M.E. is a clearly defined disease process.
CFS by definition has always been a syndrome.

At one of the meetings held to determine the 1994 U.S. Centers for Disease Control and Prevention (CDC) definition of CFS, in response to my question from the floor, Dr. Keiji Fukuda stated that numerous M.E. epidemics—he

cited the Los Angeles County Hospital epidemic of 1934, the Akureyri outbreak of 1947-48 and the 1955-58 Royal Free Hospitals epidemics-- were definitely not CFS epidemics. Dr. Fukuda was correct.
The Psychiatric Label

Unfortunately many physicians and some senior persons in governments, including Great Britain, Norway and to a lesser degree the USA and Canada treat CFS as a psychiatric illness. This view has been arrived by some physician's readings of the CFS definitions from CDC. Indeed, despite clear signals in the 1994 CDC definition that CFS is not a psychiatric disease, each of the CDC definitions and their addenda referring to CFS remain open to interpretation as a psychiatric rather than a physical illness. This is not a view to which I subscribe. It is the CFS definitions themselves that give rise to this inaccuracy. Consider the following:

*(a) What other physical disease definitions essentially state that **if you discover the patient has any physical injury or disease, then the patient does not have the illness CFS?** In other words if you have CFS then it does not result in or cause any major illness. What else could CFS then be but any number of various psychiatric, social, hysterical or mendacious phenomena?*

*(b) The various CDC administrations dealing with the subject have clearly stated that CFS is a physical, not a psychiatric disease. However, is there any other definition of any physical disease that is not provable by scientific and clinical tests? Only **psychiatric diseases** are not clearly verifiable by physical and technological tests.*
*(c) What other physical disease definition requires a **six month waiting period** before the illness can be diagnosed? Any physician knows that to treat a disease adequately you have to be able to define the disease at its onset and treat it immediately in order to prevent chronic complications from arising. There are simply no other disease definitions that have ever been assembled similar to the CFS definitions.*

(d) If you are still not convinced, check the Internet for the Diagnostic and Statistical Manual of Mental Disorder definition of: DSMIII Somatization Disorder. You will find that there is little substantial difference to distinguish the DSMIII definition from the 1988 and 1994 CDC definitions of CFS. It is difficult to believe that the CDC medical bureaucracy is not aware of this similarity. It is thus understandable why the insurance industry, as well as some psychiatrists and physicians, have simply concluded that CFS is somatization disorder.

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I believe it essential to define clearly Myalgic Encephalomyelitis, G93.3. That is what the Nightingale definition of M.E. sets out to do.

The definition is based upon two criteria:

(a) The excellent scientific work of respected physicians and scientists who investigated the various M.E. epidemics

(b) Our Foundation's modern scientific testing techniques and the knowledge resulting from examining thousands of M.E. patients using these techniques.

The proposed M.E. definition is designed to improve early diagnosis and treatment for the patients stricken with M.E. It is not a new definition of CFS nor should it be conceived as a rewording of any previous CFS definition.

What follows is the primary M.E. definition for adults.

The Nightingale Definition of Myalgic Encephalomyelitis (M.E.)

Primary M.E. is an acute onset biphasic infectious disease process, where there is always a measurable and persistent ***diffuse vascular injury of the Central Nervous System in both the acute and chronic phases.*** *Primary M.E. is associated with immune and other pathologies.*

Primary M.E. is a chronic disabling, acute onset biphasic infectious disease process affecting both children and adults.

There are both central and peripheral aspects to this illness.
A: The ***Central Nervous System (CNS)*** *symptoms, as well as the clinical and technological abnormalities, are caused by a diffuse and measurable injury to the vascular system of the Central Nervous System. These changes in the organization of the CNS are caused by a combined infectious and immunological injury and their resulting effect on CNS metabolism and control mechanisms. Much of the variability observed in an M.E. patient's illness is due to the degree and extent of the CNS injury and the ability of the patient to recover from these injuries.*

B: A significant number of the initial and long-term peripheral or body symptoms, as well as clinical and technological body abnormalities in the M.E. patient, ***are caused by variable changes in the peripheral and CNS vascular system.*** *The vascular system is perhaps the largest of the body's organs and both its normal and pathological functions are in direct relationship to CNS and peripheral vascular health or injury, to CNS control mechanisms and to the difficulty of the peripheral vascular system and organs to respond to CNS neuro-endocrine and other chemical and neurological stimuli in a predictable homeostatic fashion.*

C: When pain syndromes associated with M.E. occur, they are due to a combined injury of:

(i) the posterior spinal cord and / or posterior root ganglia and appendages,
(ii) patho-physiological peripheral vascular changes, and
(iii) CNS pain reception homeostasis mechanisms.

Depending upon the degree and extent of the ongoing CNS and peripheral vascular injuries, these patho-physiological changes in turn may give rise to both transient and in many cases permanent systemic organ changes in the patient.

As with any illness, the diagnostic criteria of M.E. are divided into two sections:

(a) The clinical features and history of the ill patient that alert the physician to the initial diagnosis

(b) The technological examinations that confirm to the physician proof of the diagnosis.

Clinical Features

The clinical features of Myalgic Encephalomyelitis are consistent with the following characteristics that can easily be documented by the physician.

1. M.E. is an acute onset biphasic epidemic or endemic infectious disease: Both Epidemic and Non-Epidemic cases are often preceded by a series of repeated minor infections in a previously well patient that would suggest either a vulnerable immune system, or an immune system subject to overwhelming stressors such as:

(a) repetitive contact with a large number of infectious persons,

(b) unusually long hours of exhausting physical and / or intellectual work,

(c) physical traumas, (d) immediate past immunizations,

(e) epidemic disease cases whose onset and periodicity appear to occur cyclically in a susceptible population,

(f) the effect of travel, as in exposure to a new subset of virulent infections, or

(g) the effects of starvation diets.

(It should be noted that subsets c, d, e, f and g are all stressors associated with decreased immune adaptability plus an associated infection with an appropriate neurovascular infectious virus or other infectious agents. This may be due either to an immediate preexisting infectious disease or to a closely following infection, either of which may or may not be recognized.)

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

(See B. Hyde and A Jain, Clinical and Scientific Basis of M.E./CFS, Chapter 13, Nightingale Research Foundation, Ottawa, Canada, pp. 124-126)

3. Secondary Chronic Phase: The second and chronic phase follows closely on the first phase, usually within two to seven days; ***it is characterized by a measurable diffuse change in the function of the Central Nervous System****. This second phase is the persisting disease that most characterizes M.E.*
4. The Presence or Absence of Various Pain Syndromes is highly variable: The pain syndromes associated with the acute and chronic phases of M.E. may be described as Early and Late findings.

Early Findings:

*(a) severe headaches **of a type never previously experienced;***

*(b) these are often associated with **neck rigidity and occipital pain;***

(c) retro-orbital eye pain;

(d) migratory muscle and arthralgia pain;

(e) cutaneous hypersensitivity.

Late Findings:

(f) fibromyalgia-like pain syndromes.

When occurring, these various pain syndromes may include fibromyalgia-like pain syndromes. Many of the pain features tend to decrease over time but can be activated or increased by a wide range of external and chemical stressors. (See Clinical and Scientific Basis of M.E./CFS, Chapter 5, pp. 58-62)

Testable and Non-testable Criteria:

The technological tests listed below can be used to

(a) confirm the clinical diagnosis of Myalgic Encephalomyelitis and

(b) to some degree gauge its severity and probability of persistence.

The second and chronic phase that clearly defines M.E. is characterized by various measurable and clinical dysfunctions of the cortical and/or sub-cortical brain structures.

*5. Diffuse Brain Injury Observed on Brain SPECT: **If the patient's illness is not measurable using a dedicated brain SPECT scan such as a Picker 3000 or equivalent, then the patient does not have M.E.** For legal purposes, these changes may be confirmed by PET brain scans with appropriate software and / or QEEG. These changes can be roughly characterized as to severity and probable chronicity using the following two scales:*

A: extent of injury and
B: degree of injury of CNS vascular function.

Extent of Injury

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

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

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

severely affected patients and the most likely to be progressive or demonstrate little or no improvement with time

Degree of injury

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

Type B: Anatomical integrity is not visible in the CNS SPECT scan. Type 3B are some of the most severely and chronically injured patients.

6. Testable 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. Neuropsychological changes must be measured in relation to estimates of prior achievement. This feature may improve over a period of years in patients with adequate financial and social support and can be made worse by chronic stressors. The neurophysiological changes are those observed by a qualified neuropsychologist with experience in examining this type of disease spectrum. (See S. Bastien in Clinical and Scientific Basis of M.E. / CFS, Chapter 51, pp. 453-460.)

7. Testable Major Sleep Dysfunction: This can include all forms of sleep dysfunction. All or any of the following may be present:

(a) impaired sleep efficiency,

(b) significant fragmented sleep architecture,

(c) movement arousals if there is an associated pain syndrome,

(d) absence of type 3 and 4 sleep,

(e) abnormal REM sleep pattern

(f) changes in daytime alertness and

(g) sleep reversals.

8. Testable 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 a period of years but many patients frequently remain permanently vulnerable to new disease episodes. Few centres are equipped or funded to make these examinations.

9. Testable ***Vascular Dysfunction****: This is the most obvious set of dysfunctions when looked for and is probably the cause behind a significant number of the above complaints. As noted, the primary vascular change is seen in abnormal SPECT scans and clinically most evident in patients with:*

a. POTS: severe postural orthostatic tachycardia syndrome. Note: This group can be confused with diabetes insipidus due to the fact that they may

have polydipsia from their attempt to increase their circulating blood volume by consuming large amounts of fluids. This group can be verified by the absence of pituitary adenoma or pathology and the fact that they can sleep through the night without waking to drink fluids.

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

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. Circulating Blood Volume Decrease: this is a nuclear medicine test in which the circulating red blood cell levels can fall to below 50%, preventing adequate oxygenation to the brain, gut and muscles. This is undoubtedly a subcortical dysregulation. It is associated with serum and total blood volume measurements. Note: Body servomechanisms are genetically designed so that blood flow and oxygen to the heart are always protected. Thus blood flow to organs not necessary for short-term survival, such as the brain, the gut and muscles, can be temporarily decreased. This of course gives rise to many of the M.E. symptoms.

e. Bowel Dysfunction: vascular dysfunction may be the most significant causal basis of the multiple bowel dysfunctions occurring in M.E.

f. Ehlers-Danlos Syndromes Group: this is a group of illnesses with a genetic predisposition to M.E. or M.E.-like illness. In fact it probably represents a spectrum of illnesses that start with

(i) Hyper-Reflexia Syndrome, moving through any of the

(ii) various Ehlers-Danlos Syndromes and climaxing in

(iii) Marfan Syndrome where there tends to be early death if the aortic and cardiac changes are not repaired.

Ehlers- Danlos Syndromes can go undetected until what appears to be an infectious switch is turned on, usually in late teens to early thirties. Briefly, patients over the age of 16 who can

(i) touch their nose with their tongue,

(ii) touch their forearm with the thumb of the same extremity,

(iii) touch the floor readily with the full palm should be considered suspect for further examination.

See S.I. Magalini, S.C. Magalini, Dictionary of Medical Syndromes, pp. 251-252, Lippincott-Raven Publishers, Philadelphia, 4th Edition, 1997.

g. Persantine Effect in M.E. Patients: Persantine is a chemical manufactured by Boehringer Ingelheim. It is employed to perform chemical cardiac stress testing when a patient cannot exercise sufficiently to stress the heart. It is a particularly safe medication but when employed with many M.E. patients it can cause severe muscle pain over the extremities and

entire musculature. Normally this can be reversed by injection of an antidote but this does not always work in M.E. patients. Severe pain and fatigue can be intolerable and persist for minutes to days in some M.E. patients following persantine use. Persantine works by dilating both peripheral and cardiac blood vessels and causing the heart rate to increase as in a POTS patient. Obviously one major pain and fatigue factor in M.E. patients is caused by ***abnormal dilatation of peripheral blood vessels****. To my knowledge, no testing of M.E. patients with persantine has ever been published by Boehringer Ingelheim or others. It is one of the reasons I believe that pain syndromes in M.E. patients are due to a pathological vascular physiology.*

h. M.E. Associated Clotting Defects: ***M.E. represents both a vasculitis and a central and peripheral change in vascular physiology.*** *All such vascular illnesses should be potentially treatable. We do not yet know how to treat the*

(i) genetic forms of vasculitis and vascular patho-physiology mentioned here, nor

(ii) the probable viral triggered genetic vascular pathologies also mentioned. Nor do we know how to treat those

(iii) centrally caused injuries causing the circulating blood volume defects that are demonstrated when we do the "nuclear medicine circulating blood volume tests. It is important to do this test on all patients. POTS is poorly treatable and more often success in treatment presently escapes physicians' ability. Eventually, I have no doubt that these will be treatable causes of M.E. type disease.

However there is a significant group of M.E. patients who are ill due to a treatable form of vasculitis and can be treated if the physician takes the time to diagnose the subgroup. These patients are the clotting defect patients. Some of these clotting defects are genetic and some appear to be genetic with an age or viral switching mechanism; although they may develop in childhood, they are more frequently noted well after puberty and before the age of 40.

Many of these patients can be diagnosed by the following tests:

(1) Serum viscosity test,
(2) Antiphospholipid Ab.,
(3) Protein C defects,
(4) Protein S defects,
(5) Factor V Leiden defect,

to name the most common that we have uncovered. However, there are others for which we also test. These conditions are all potentially treatable and when treated will allow the patient to return to normal range of activities (e.g. school, career, etc).

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

a. Pituitary-Thyroid Axis: Changes in serum TSH, FT3, FT4, Microsomal Ab., PTH, calcium and phosphorous rarely occur until several years after illness onset. This can be followed by ultrasound of the thyroid gland, where

a steady shrinking of the thyroid gland may occur in some patients with or without the development of non-serum positive Hashimoto's thyroiditis (a seeming contradiction in terms) and a significant increase in thyroid malignancy. In cases of thyroid wasting, serum positive changes tend to occur only after years and often not until the thyroid gland shrinks from the normal 13 to 21 cc. volume in an average adult female and 15 to 23 cc. volume in male patients to below a volume of 6 cc. (Mayo Clinic averages). Serum analysis of M.E. patients for thyroid pathology is simply not adequate. Repeat thyroid ultrasound must be performed for all M.E. patients to ascertain presence of dystrophic changes. It is also inadequate simply to accept the radiologist's report of a normal thyroid. The volume of each lobe and its homogenicity must be requested and documented.

The following changes, while uncommon, ***may*** *also be related to an M.E. disease process:*

b. Pituitary-Adrenal Axis Changes: where changes and findings are infrequent.

c. Pituitary-Ovarian Axis Changes

d. Bladder Dysfunction Changes: This dysfunction occurs frequently in the early and in chronic disease in some people. In some instances this may be due to a form of diabetes insipidus, in other cases it is related to POTS type illness where the patient is compensating for an inability to maintain vascular pressure by attempting to increase fluid volume. In other cases this may be due to interstitial cystitis or a form of polio-type-bladder articularly if the cause of the individual disease is an enterovirus. Dr. John Richardson also associated this finding with adrenal dysfunction that he measured.

Discussion

To various degrees all of the above historic findings have been observed and discussed by Doctors Alexander Gilliam, Bjorn Sigurdsson, Alberto Marinacci, Andrew Lachlan Wallis, A Melvin Ramsay (Elizabeth Dowsett who assisted in much of his later work), John Richardson, Elizabeth Bell, Alexis Shelokov, David C Poskanzer, W.H. Lyle, Sir E. Donald Acheson, Louis Leon-Sotomayor, J. Gordon Parish and many others.

In varying degrees all of the following physicians have also noted the above historical and the more recent investigational findings. They include alphabetically, Doctors Peter Behan, David Bell, Dedra Buchwald, Paul Cheney, Jay Goldstein, Seymour Grufferman, Anthony L Komaroff, Russell Lane, Ismael Mena, Harvey Moldofsky, James Mowbray, Daniel Peterson, Vance Spence and scores of others.

I have examined patients with M.E. since the late 1970s but only at the urging of Dr. Charles Poser of Beth Israel Hospital at Harvard and John Richardson in Newcastleupon- Tyne did I take up the study of these unfortunate patients on a full time basis. The material in this definition is the cumulative result of my listening and interpreting the work of all of the above clinicians and my evaluation of over 3,000 M.E. and CFS patients since 1984. The essential concept of the indepth medical evaluation that is the basis of my work on M.E. and CFS since 1995 was crystallized in my

discussions in Seattle Washington State in 2002 with Dr. Leonard A. Jason, Patricia A. Fennell and Renee R. Taylor. This discussion was set down as Chapter 3, "The Complexities of Diagnosis", in their book, The Handbook of Chronic Fatigue Syndrome, John Wiley and Sons, Hoboken, New Jersey, 2003. I would also like to thank Elizabeth Dowsett and Jane Colby whose work with children in the UK as well as their advice have been instrumental in this definition. I must also thank each and every one of the members of John Richardson's Newcastle Research Group who have provided me with so much valuable information over the years and who have all supported my continued investigations of M.E. patients.

What is new and different about the Nightingale M.E. definition?

A: A Testable Definition

The definition is set out in both a clinical diagnostic and scientifically testable fashion. This will allow the physician both an early diagnostic bedside or office understanding of the illness and a scientific and technological method to investigate and confirm the diagnosis.

It is well known by all serious physicians that in order to assist the patient in a partial or full recovery the illness must be

(a) prevented from occurring by either immunization or understanding and avoiding the causes,

(b) diagnosed and treated immediately following onset.

The Nightingale Definition assists the physician in diagnosis and early treatment.

B: A Vascular Pathophysiology

The subject of vascular pathology is not new. The fact of the children dying of a Parkinsonian-like vascular injury to the basal ganglia in Iceland during the Akureyri Epidemic is an obvious indication of the CNS vascular effects. Vasculitis has been well documented by Dr. E. Ryll in his description of the epidemic in the San Juan Mercy, Sacramento California Hospital in 1975. He described this M.E. epidemic as an epidemic vasculitis. In the late 1980s Drs. Jay Goldstein and Ismael Mena confirmed and proved this initial description by examining the changed brain microcirculation using brain SPECT imaging in M.E. patients. Following my over twenty years of examining M.E. and CFS patients and sixteen years of subjecting the M.E. and CFS patients to brain imaging techniques suggested by Goldstein and Mena, ***it has become obvious to me that we are dealing with both a vasculitis and a change in vascular physiology.***

Numerous other physicians have supported this finding. Dr. David Bell, who rediscovered the work of Dr. David Streeten and his book, Orthostatic Disorders of the Circulation: Mechanisms, Manifestations and Treatment, New York: Plenum Medical, 1986, advanced this understanding of M.E. The work of Dr. Vance Spence and his colleagues in Scotland have started to nail this CNS-vascular relationship down even further with a series of major research papers. The recent interpretation of the cause of Multiple Sclerosis (MS) as an injury of the microvasculization causing the injury of the schwann

cells that in turn causes the demyelination injuries of MS has been added to that of paralytic poliomyelitis as an essential vascular injury.

- Paralytic poliomyelitis was thought to be a primary injury to the anterior horn cells of the spinal cord but is now recognized as a vasculitis injuring the circulation to the anterior horn cells. Poliomyelitis is generally a non-progressive, specific site injury, although post-polio syndrome has challenged that belief.

MS is a recurrent more fulminant physiological vascular injury.

M.E. appears to be in this same family of diseases as paralytic polio and MS.

M.E. is definitely less fulminant than MS but more generalized.

M.E. is less fulminant but more generalized than poliomyelitis.

This relationship of M.E.-like illness to poliomyelitis is not new and is of course the reason that Alexander Gilliam, in his analysis of the Los Angeles County General Hospital M.E. epidemic in 1934, called M.E. atypical poliomyelitis.

C: ***The Lack of Mention of Fatigue***

Myalgic Encephalomyelitis is not CFS. *Fatigue was never a major diagnostic criterion of Myalgic Encephalomyelitis. Fatigue, loss of stamina, failure to recover rapidly following exposure to normal physical or intellectual stressors occur in most if not all progressive terminal diseases and in a very large number of chronic non-progressive or slowly progressive diseases.*

Fatigue and loss of stamina are simply indications that there is something wrong. They cannot be seriously measured, are generally subjective and do not assist us with the diagnosis of Myalgic Encephalomyelitis or CFS or for that matter any disease process.

D: Cause of Myalgic Encephalomyelitis

It is obvious that all cases of epidemic M.E. and all primary M.E. are secondary to infectious / autoimmune phenomena. *Many M.E. and M.E.-like patients' illness is complicated by multiple other causes. This is why a complete technological investigation has to be made on each chronically ill M.E. or M.E.-like patient.*

Under epidemic and primary M.E. there is no consensus as to the viral or infectious cause. Much of this lack of consensus may be due to the non-separation of acute onset from gradual onset patients in the M.E. and CFS groups of patients.

Primary M.E. is always an acute onset illness.

Doctors A. Gilliam, A. Melvin Ramsay and Elizabeth Dowsett, John Richardson of Newcastle-upon-Tyne, W.H. Lyle, Elizabeth Bell of Ruckhill Hospital, James Mowbray of St Mary's and Peter Behan all believed that the majority of primary M.E. patients fell ill following exposure to an ***enterovirus*** *(Poliovirus, ECHO, Coxsackie and the numbered viruses are the significant viruses in this group).*

I share this belief. Unfortunately, it is very difficult to recover polio and enteroviruses from live patients. Dr. James Mowbray developed a test that

demonstrated enterovirus infection in many M.E. patients but I do not believe he qualified his patients by acute or gradual onset type of illness.

In my tests in Ruckhill Hospital in Glasgow, I found confirmation of enteroviral infection only in acute onset patients and not in any gradual onset patients.

Few physicians realize that almost all cases of poliovirus recovered from poliomyelitis victims came from cadavers. At the very least, these enteroviruses must be recovered from patients during their onset illness and this has rarely been done. An exception is in the case of the Newton-le-Willows Lancashire epidemic where Dr. W. H. Lyle's investigation recovered ECHO enterovirus. Recent publications by Dr. J. R. Kerr have also identified the fact that enteroviruses are one of the most likely causes of M.E. If this belief is correct, many if not most of the M.E. illnesses could be vanquished by simply adding essential enteroviral genetic material from these enteroviruses to complement polio immunization. I have not discussed non-infectious M.E.-type disease.

Similar M.E.phenomena can occur due to diffuse Central Nervous System injuries from toxic chemical injury. I have seen this in police officers who have fallen into toxic chemical ponds in pursuit of those suspected of criminal activity. I have seen it in farmers, in hospital and industrial workers and in military personnel in contact with toxic chemicals, specifically toxic gases. I plan to explore these in a future publication as Secondary M.E. They do have one thing in common, and that is the diffuse CNS injury as noted on brain SPECT scans. Often these Secondary M.E. diseases are more severe than the Primary M.E. cases.

E: Caution One

One should be careful in applying the diagnostic criteria discussed under the Nightingale M.E. Definition without also completing a thorough investigation. M.E., whether we are discussing primary or secondary forms, involves a significant diffuse injury of the Central Nervous System and an associated injury of the Immune System. This always implies the potential for secondary injuries or secondary disease or pathology caused by a dysfunctional brain and dysfunctional immune system. When the immune system is injured there is an impairment of the patient's ability to resist the development of malignancy or other important organ and systemic injuries.

Due to funding limitations, we have only been able to demonstrate in our work only two characteristics of this corollary injury. The first is the high incidence of thyroid cancer in M.E. patients. In the general public, cancer of the thyroid occurs in 1 case per 100,000. In our studies, in the case of the M.E. patient, thyroid cancer has an incidence of 6,000 cases per 100,000. We have already mentioned the pervasive vascular injuries. We believe that other pathological associations also occur. Failure to evaluate fully the M.E. patient may result in the physician missing important secondary pathology and possibly giving rise to patient death.

All M.E. patients as well as all chronic illness patients deserve a systematic and total body investigation. ***No individual should go through life, ill, disabled without knowing why he is ill.*** *Simply offering a label, whether M.E., or CFS, without looking at the pathophysiology, is both unacceptable and potentially dangerous both for the patient and the patient's physician.*

(See "The Complexities of Diagnosis" by Byron Hyde, in the Handbook of Chronic Fatigue Syndrome, Chapter 3)

F: Caution Two

Insurance companies regularly employ reputedly independent psychologists who demonstrate normal neuropsychological findings. This presents a grave problem in that neuropsychological testing by a truly independent neuropsychologist may be delayed for up to a year before the patient can be properly tested. The conflicting results may tend to confuse any trial judge in a legal case.

G: Depression, Anti-depressive Medications and Myalgic Encephalomyelitis

Myalgic Encephalomyelitis is not depression.
Myalgic Encephalomyelitis is not hysteria.
Myalgic Encephalomyelitis is not a conversion disorder nor is it a somatization disorder.

Myalgic Encephalomyelitis is an acute onset diffuse injury of the brain.

Psychiatrists should not ever be placed in charge of diagnosis and treatment of M.E. patients. It is simply not their area of expertise and their meddling has at times caused great harm to M.E. patients. Also, during the 20 years that I have investigated M.E. patients I have yet to see a single case of real M.E. that has responded to psychiatric pharmacological treatment such that the patient has recovered and been able to return to work or school.

This topic is a very large subject and demands a separate publication and this is not the place for it. However I would like to note again the vascular and cardiac pathologies that one encounters in M.E. patients and how M.E. patients are often made worse by one antidepressive medication that is considered benign. One of the most common antidepressive medications employed by psychiatrists and physicians in general for M.E. patients is an old pharmaceutical, Amitriptyline. Yet this medication may result in a condition referred to as Torsade de Pointes, a cardiac irregularity giving rise to resting tachycardia, QT interval prolongation and significant orthostatic hypotension. Since there is already a high frequency of these anomalies in M.E. patients, the use of Amitriptyline may assist sleep to some degree but may also simply worsen existing M.E. symptomology. I plan to return to this subject in another publication.

Definition Changes and Improvements

As with all definitions, the Nightingale Research Foundation's Definition of Myalgic Encephalomyelitis will have to be looked at by many clinicians and researchers and over the years, disagreed about, changed and improved upon. But what this definition does today is

(a) separate clearly M.E. from CFS and

(b) demonstrate that M.E. is an early diagnosable and provable disease - as are all true diseases, and

(c) assist in the early treatment and cure of M.E. patients.

This Nightingale Research Foundation's Definition will be available with any updates or corrections, on the Nightingale Research Foundation's Website, ***http://www.nightingale.ca*** *This definition may be copied, translated, distributed by electronic or hard copy and may be included, in whole or in part in any publication without permission from the Nightingale Research Foundation or the authors, provided that this last paragraph and referral back to our website are noted.*

- Byron Marshall Hyde, MD, Ottawa - November 18, 2006

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*like the original definition of CFS produced in 1988 however, none of these definitions defines any distinct illness, including Myalgic Encephalomyelitis. (Hyde 2006, [Online]) All each of these flawed definitions 'define' is a heterogeneous **(mixed)** population of people with various misdiagnosed psychiatric and miscellaneous non-psychiatric states which have little in common but the symptom of fatigue. (Hooper et al 2001, [Online]) (Dowsett 2001b, [Online])*

This is why being diagnosed with any of the definitions of CFS is not a useful or meaningful diagnosis and why a diagnosis of CFS should never be accepted – by doctor or by patient – as an end point of the process of diagnosis."

The creation of the flawed disease category of 'CFS' (and the equally flawed government policies that have gone along with it) have had a devastating effect on hundreds of thousands of M.E. sufferers around the world, including young children. These very ill patients are often denied appropriate medical treatment and care, denied appropriate insurance entitlements and other medical benefits and are often accused of malingering by doctors, welfare agencies and the media (and in turn even their own friends and family). M.E. patients are also routinely recommended or forced to participate in inappropriate or harmful psychologically based interventions while basic appropriate medical care is withheld. These harmful interventions (and the lack of basic medical care) have had disastrous and long-term physical effects on many sufferers. In some cases this has resulted in ***death****. (Hooper et al. 2001, [Online]) (Hyde 2003, [Online])*

Patients with M.E. are not the only patient group to be negatively affected however. Other patient groups misdiagnosed as CFS are also denied appropriate diagnosis and treatment. They may also be subjected to inappropriate psychological interventions. Doctors, researchers and the general public are also negatively affected in various ways by this subterfuge (As explained previously in Smoke and Mirrors). The only groups which gain from the 'CFS' confusion are insurance companies and various other organisations and corporations which have a vested financial interest in how these patients are treated, including the government.

The only way forward for every group involved is that the disease category of 'CFS' must be abandoned*. (Hooper 2006, [Online]) Each of the patient groups involved must be correctly diagnosed and then treated as appropriate based on legitimate and unbiased science involving the SAME patient group. People with M.E. must be diagnosed and treated for M.E. Patients with depression should be diagnosed and treated for depression. Patients with cancer should be treated for cancer, and so on. Lumping these disparate patient groups together under a vague and meaningless category of 'fatiguing illnesses' (or CFS) only hinders each of the patient groups involved in their battle to regain their health. (Dowsett 2001b, [Online]) (Hooper 2006, [Online]) (Hyde 2003, [Online])*

What a diagnosis of 'CFS' actually means is that the patient has a gradual onset fatigue syndrome which is usually due to a missed major disease. 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. (Hyde 2006, [Online])*

Some of the illnesses commonly misdiagnosed as 'CFS' include:

- *Various post-viral fatigue states/post-viral fatigue syndromes (eg. following glandular fever/mononucleosis, hepatitis, Ross river virus, Q fever, flu, measles, chickenpox, herpes and many other infections)*

- *Fibromyalgia*
- *Candida*
- *Athlete over-training syndrome*
- *'Burnout'*
- *Multiple chemical sensitivity syndrome (MCSS)*
- *Multiple sclerosis*
- *Thyroid illness*
- *Adrenal insufficiency*
- *Localised and Metastatic malignancies*
- *Brain tumours, including astrocytomas, gliomas*
- *Transverse Myelitis*
- *Myopathic illnesses including:*
    - *Myasthenia gravis*
    - *Mitochondrial myopathies*
    - *Post-infectious polymyositis*
- *Vitamin B12 deficiency disorders:*
    - *Pernicious anaemia*
    - *Intentional dietary deprivation*
    - *Intestinal disease associated with or independent of M.E.*
- *Rheumatoid illness or lupus (SLE)*
- *Sarcoma*
- *Renal or liver disease*
- *Infectious illnesses including:*
    - *Toxoplasmosis*
    - *AIDS*
    - *Lyme disease (Borrelia burgdorferi)*
    - *Tuberculosis*
    - *Brucellosis*
- *Various psychiatric and social psychiatric states including:*
    - *Anxiety neurosis*

- *Uncomplicated endogenous or reactive depression*
- *Clinical depression*
- *Psychopathic personality disorder*
- *Post-traumatic stress disorder (PTSD)*
- *Schizophrenia and other psychiatric disease (Ramsay 1986, [Online]) (Ramsay 1988) (Hyde 1992, p 22) (Dowsett n.d.a. [Online]) (Hooper et al. 2001, [Online]) (Hyde 2003, [Online]) (Hyde 2006, [Online])*

This is of course not a comprehensive list. M.E. expert Dr Elizabeth Dowsett explains that, 'There are actually 30 well documented causes of 'chronic fatigue." (n.d.a. [Online]) It should also be remembered that although none of the CFS definitions define M.E., the majority of those with M.E. will be given a CFS diagnosis by default (due to the ignorance surrounding M.E., and the confusion with 'CFS'). Therefore the possibility that a patient misdiagnosed with CFS has authentic Myalgic Encephalomyelitis should also be investigated, along with these myriad other possibilities.

- *For details on the symptoms listed in the most commonly used CFS definitions, and the symptoms of many of the illnesses commonly misdiagnosed as CFS (as listed above), see part 2 of this text.*

Today patients with all sorts of different illnesses are commonly misdiagnosed as having 'CFS.' *Under cover of the bogus disease category of CFS, this diverse mix of patients are treated as if they each suffered the exact same specific illness. This is clearly unscientific, and unethical. These patients must be given the opportunity to be diagnosed correctly if they are to have any chance of appropriate treatment or recovery, not given a meaningless 'CFS' misdiagnosis. Patients with M.E. need this same opportunity. Treating this diverse and heterogenous patient group as if their illnesses each shared the same symptoms, aetiology, pathology and response to treatment is inappropriate and highly unlikely to benefit the health and wellbeing of any of the patient groups involved. Treating this 'CFS' group as if they each shared a specific psychological or behavioural illness is also clearly inappropriate. Aside from representing a heterogenous patient group, many (likely the vast majority) of those with the diagnosis are not mentally ill, and do not suffer from behavioural problems. (This includes of course, those patients with authentic M.E.) (Hooper 2006, [Online]) (Hyde 2006, [Online]) (Hooper et al. 2001, [Online])*

For the benefit of all of the patient groups involved, doctors must return to the age-old medical principals of correct diagnosis (a) careful history, (b) detailed physical examination and (c) appropriate investigation. *(Hyde 2006, [Online]) As Dr Byron Hyde MD explains:*

> *Although the authors of these definitions have repeatedly stated that they are defining a syndrome and not a specific disease, patient, physician, and insurer alike have tended to treat this syndrome as a specific disease or illness, with at times a potentially specific treatment and a specific outcome. This has resulted in much confusion. (2006, [Online]) Thirty years ago when a patient presented to a hospital clinic with unexplained fatigue, any medical school physician would have told the students to search for an occult malignancy, cardiac or other organ disease, or chronic infection. The concept that there is an entity called chronic fatigue syndrome has totally altered that essential medical guideline. Patients are now being diagnosed with CFS as though it were a disease. It is not. It is a patchwork of symptoms that could mean anything. (2003, [Online])*

Physicians who diagnose 'CFS' in any patient experiencing new onset fatigue without looking and testing for the true cause of the symptoms do their patients–and themselves–a great disservice. As Dr Elizabeth Dowsett explains,

"There is no such disease(s) as CFS' (n.d.a. [Online]) Some of the conditions commonly misdiagnosed as CFS are very well defined and well-known illnesses and very treatable – but only once they have been correctly diagnosed.

Some conditions are also very serious or can even be fatal if not correctly diagnosed and managed, including Myalgic Encephalomyelitis."

Every patient deserves the best possible opportunity for appropriate treatment for their illness, and for recovery. This process must begin with a correct diagnosis if at all possible. ***A correct diagnosis is half the battle won.***