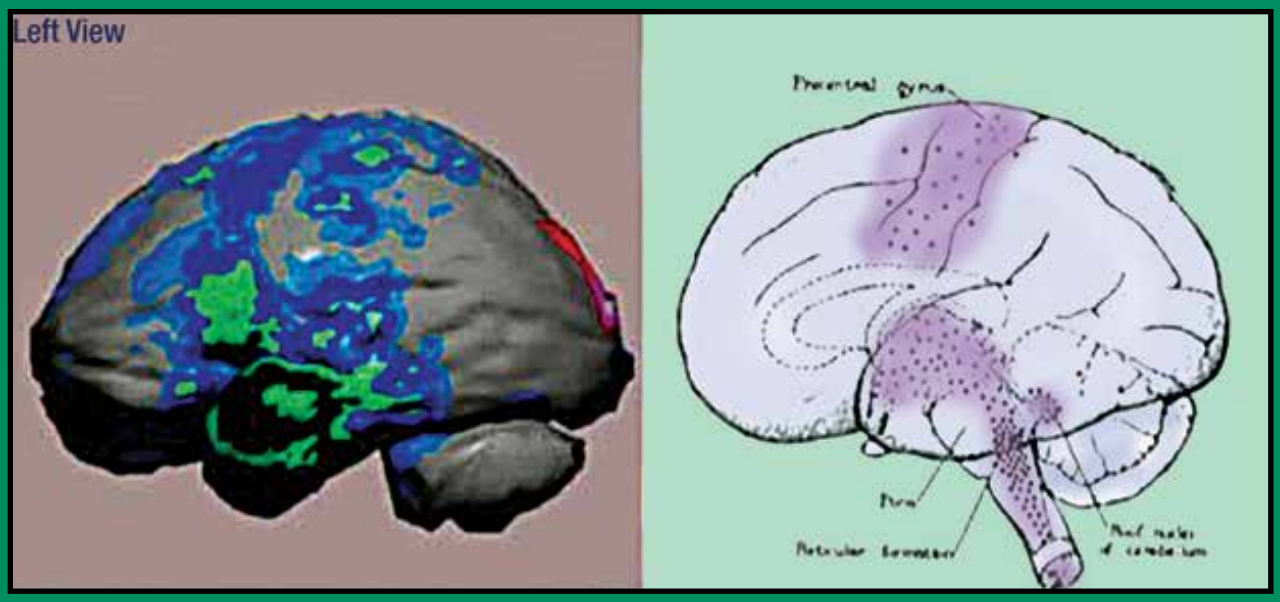
Two Chapters from the forthcoming book

Understanding Myalgic Encephalomyelitis

M.E. and Polio both injure the same Brain areas, except M.E. maims and Polio kills.



Severe M.E. Injured Brain Scan

Poliomyelitis Brain Autopsy

What you need to know about M.E. and CFS

and the New Paralytic Polios *(Acute Flacid Paralysis, or Acute Flacid Myelitis)*

threatening you and your children today

by Dr. Byron Hyde

The Nightingale Research Foundation

Chapter Eight

The Clinical Diagnosis of M.E.

Item 1:
Who Falls Ill with M.E.?

There has been considerable discussion concerning that approximately 80% of patients who fall ill with Myalgic Encephalomyelitis are females. There has been less talk about who falls ill. In my experience of investigating well over 1000 M.E. patients over the past thirty years, among adult patients are included:

a. a disproportionately large number of, nurses, physicians, psychologists, social workers and other health care workers, particularly women.

b. teachers, often those with students with hand, foot and mouth disease (an enteroviral disease) and other undiagnosed entero viral infections.

c. post-pubertal students who consistently burn the candle at both ends, living exhausting life styles and who tend to be in constant contact with infectious disease.

d. professional women who are often working well past exhaustion and are essentially doing two jobs – particularly if they have school children at home– and the children are in contact with other enterovirus-carrying school students.

The preponderance of teachers and health care workers are female, with added home duties often including children. If the teachers have a prior autoimmune intolerance, due either to history or non-stop exhausting work plus home duties that run down their immune systems they may be more illness intolerant. Thus it is understandable that up to 80% of M.E. patients are women.

Also, I have the opinion women may tend to be more bound by rules, one of which is to receive all appropriate immunizations. Immunization certainly protects patients from the targeted diseases, but not against other infections occurring at the same time as they receive immunizations.

In my experience prevalence of M.E. among professions also depends upon the catchment area. Government towns, for example, have a large number of bureaucratic workers.

Chronic Fatigue Syndrome patients do not necessarily demonstrate the same occupational characteristics. I believe their numbers generally lean to a more male than female distribution, but CFS also includes professions, with work and activities that are male-oriented. **Such as:**

1. military and war veterans with fatigue,
2. alcoholics with fatigue,
3. major depressive patients with fatigue,
4. post-traumatic patients with fatigue,
5. farmers, firemen and police officers with toxic chemical fatigue.

Major criteria or pre-conditions for M.E. are:

1. Acute onset,
2. A prior chronic immune exhausted state, plus,
3. Increased contact with infectious disease,
4. Onset during the late summer and autumn,
5. A history of autoimmune disease,
6. Recombinant Hepatitis B immunization in the ten days before they fell ill with M.E. (All immunizations cause the immune system work, and if the individual's immune system is occupied with the immunizing product, this tends to make the immune system less responsive to combat community infections during the first 30 days post immunization).
7. A history of a relative who had polio.

During the 1990s many patients still had a relative, parent or grandparent, uncle, or cousin who had experienced poliomyelitis. Despite the fear it generated, unlike tuberculosis at its apex, polio was not that common. It caused me to wonder whether patients with Myalgic Encephalomyelitis have a genetic sensitivity to enteroviruses.

Dr. Alberto Marinacci, who had taken charge of the 1934 Los Angeles County General Hospital staff and had himself fallen ill with M.E. during the epidemic, but who had recovered, told me the way to encourage the onset of M.E. was to give a series of immunizations to an exhausted health care worker or teacher, who was leaving the next day on a holiday, where they would be in contact with crowds in a bus or plane and, due to the immunization, had a short term compromised immune system. No one going on a holiday by plane or bus, particularly to a third world country, where they are likely to contact novel infectious elements, should receive any immunization, unless it is 30 days prior to departure. (See the British Government publication in The Lancet in 1956.[1]). For physicians who don't believe this association, the 1956 Lancet journal is essential reading.

1 Medical Research Council, UK. "Poliomyelitis & Prophylactic Inoculation". *The Lancet*, Dec. 15, 1956.

Item 2:
The Basic Characteristics of M.E.

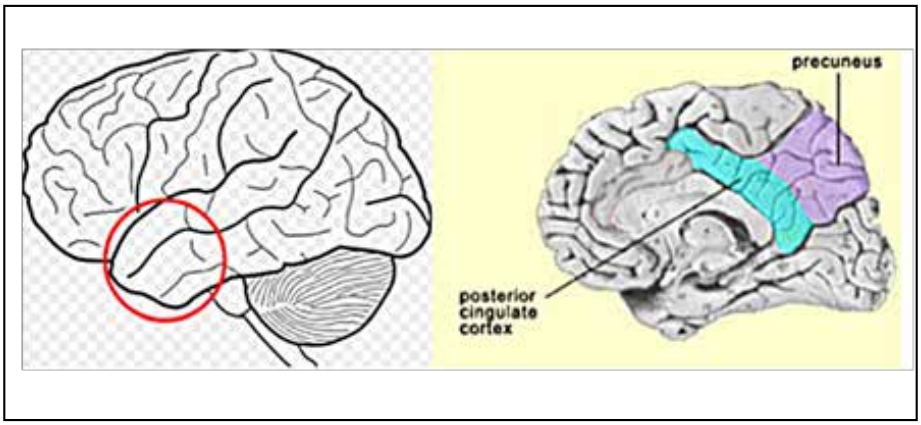
M.E. is the chronic stage of an encephalitic enteroviral infection of the central nervous system (CNS) similar, but less lethal than, that caused by polio enteroviruses 1, 2 and 3.

1. Myalgic Encephalomyelitis is a chronic, **and only**, enteroviral caused disease. The M.E. encephalitic features are less initially apparent, than in polio.
2. An incubation period of two to six days occurs before the first symptoms of illness appear.
3. In adults, as with polio, the illness may begin with the appearance of biphasic symptoms. The first phase may be missed in sporadic cases. In both illnesses the initial biphasic symptoms may be "cold like" or gastric but often these are so slight they may be missed.
4. The second phase of M.E. illness is the encephalitic, meningitic-like associated with muscular symptom phase followed weeks or months later by the cascading features (discussed later).
5. In northern latitudes M.E. occurs primarily from June to early November, peaking in late summer.
6. HMPAO brain SPECT scan demonstrates a permanent micro-vascular injury to the CNS from the first few days of acute illness.
7. Brain injury as seen on SPECT brain scans clearly with Segami Oasis software. **Injury in M.E. always involves the anterior left temporal lobe and left posterior cingulate gyrus** and frequently the left motor cortex. These hypo-perfusion CNS injuries are consistent with, and explains, the memory and muscle disfunction in M.E. patients.
8. Depending upon the severity of the illness, parts of the entire cerebrum may be involved. M.E., like Paralytic Poliomyelitis, is a neurological injury. In both M.E. and polio, the primary injury is a vasculitis. In polio, the main vascular system injury in the spinal column destroys motor neurons (anterior horn cells). In M.E. the primary injury is to the vascular system of the cognitive and administrative neurons in the brain. Segami brain SPECT scan examples in this book clearly illustrate these findings.

The painful vasculitis in M.E. also explains fibromyalgia. Fibromyalgia is the result of a vasculitis, NOT a rheumatological disease. This is why rheumatologists have never found any basis in their specialty to treat fibromyalgia patients and often refuse to see these patients.

As shown in this book, M.E. and polio are both the result of micro-vascular injuries, *(probably autoimmune and inflammatory)*, which damages essential neurological systems. Both are enteroviral injuries but the essential polio injury is to the vascular system of the brain stem and spinal cord, with a secondary injury to the brain as shown on the cover of this book.

M.E. and paralytic polio are in some ways mirror images. M.E. is the result of an injury to specific areas of the vascular system of the brain and cerebellum, and probably also a secondary vascular injury to the spinal cord. Except for location, this is not unlike paralytic polio where there is a vascular injury, primarily to the spinal cord and secondary injury to the brain. In other words, M.E. could be considered a form of non-paralytic poliomyelitis, as illustrated on the cover. M.E. appears to be the same as what was once referred to as missed poliomyelitis. M.E. occurred together with the early paralytic polio epidemics.



The red circle indicates the anterior left temporal lobe, the primary encephalitic injury site in all M.E. patients. This is the area of the brain essential to both retrieving stored brain memories and sending them on to the posterior cingulate lobe, to be then sent to appropriate administrative brain areas for action.

Item 3:
The Onset Phase of M.E.

Early Confirmation of M.E. :

1. Only during the first phase, as in polio or acute flaccid paralysis, is proof of enteroviral infection easily documented by raising titres or recovery of the actual virus.
2. Positive oligoclonal banding may be documented if the lumbar puncture is done in the first week of illness. Oligoclonal banding demonstrates and is consistent with neuronal injury in the anterior temporal lobe.

3. Abnormal brain SPECT scan can be observed in the first day of the clinical disease and these SPECT changes remain chronically present as seen on Segami oasis SPECT software.

Note: Only in this early phase is there a reasonable chance of potential remedial action. It is very important that physicians recognize M.E. during the first phase, because it may be the only time treatment may abort the second and chronic phase. Unfortunately, enteroviral titres are no longer available or covered by the health plans in many provinces and various American states. This is a major bureaucratic stupidity, leading to a failure to categorize both M.E. and the New Polio (Acute Flaccid Paralysis) patients in North America and Europe. Since no effective treatment has ever been developed to stop or alter the vasculitis in either poliomyelitis or M.E. the only partially effective treatment is ***total bed rest****, as suggested by Silas Weir Mitchell in the 1870s. Governments should support significant research in initial vascular treatment for polio and M.E, and research in general enteroviral immunization.*

The onset phase can last up to a week.

1. It is usually characterized by sudden onset of severe prostration, often with severe headaches and eye symptoms as described in *The Clinical and Scientific Basis of M.E. and CFS*[2]. There is often minimal physical signs of disease. Some cases exhibit marked temperature or cutaneous changes, or characteristics of other enteroviral diseases illnesses such as Enterovirus 71 (hand, foot and mouth disease), Coxsackie A (herpangina, which can be diagnosed visually), ECHO virus, Enterovirus 68 (causing the New Polio), or Acute Flaccid Paralysis or Myelitis. Recovering patients in polio epidemics, often known as missed polio patients, should be followed for evidence of M.E.-like disease. Unfortunately, although continually mentioned in the medical literature, these missed polio patients were never seriously researched.

2. The symptoms are often so severe, patients often develop a sense of fear and terrifying impending doom. They have multiple, seemingly inexplicable neurological and muscular symptoms and generally, few significant visual corroborative physical signs to support a diagnosis. The problem is compounded when the physician, failing to find obvious correlative physical or laboratory signs of disease, dismisses the illness as a hysterical reaction. Such physician "too rapid negation" should be considered a primary diagnostic defining symptom of M.E.

3. Often, severe head, limb, chest or gastric symptoms emerge with fleeting or migrating pain syndromes. These pains tend to resolve or decrease over the first few weeks or months, but infrequently can last for one or more years. In my experience, if the patient is treated with ongoing narcotics or corticosteroids at onset, the pain syndromes can become permanent. These M.E. patients tend to be highly sensitive, negatively so, to many medications.

2 "The Clinical and Scientific Basis of M.E. and CFS," (Hyde, Goldstein, & Levine, 1992)

4. This entroviral infectious onset phase can be followed by **(a)** a full or partial recovery, **(b)** a brief recovery, rarely of more than a few days before appearance of the chronic illness, **(c)** immediate onset of chronic disability, or **(d)** acute illness leading to death in the first weeks, although this is very rare except in poliomyelitis. Death has been reported in the literature in the Cumbria epidemic of 1955[3] and also by Dr. John Richardson in Northumbria and in several other epidemics. I am personally aware of only four deaths of M.E. patients in the past 30 years: a professional athlete with no sign of coronary artery disease who suffered a sudden cardiac death, and three young women who died in a severe, chronic and long-lasting bed-ridden state. It is not known if these two youths died of secondary infections from chronic inactivity.

Item 4:
M.E.'s Second or Chronic Phase

Note: In the chronic forms of M.E. described below, one of the physician's major roles, if they cannot significantly assist the patient, is above all to protect the patient from the avarice of the insurance industry by assisting the patient in obtaining their dissability pension.

Patients or physicians may become aware of the following symptoms:

1. Persisting physical prostration after negligible activity.
2. **Persisting pain, which significantly limits activity.** In many cases this pain decreases over time if the patient is not treated with chronic analgesics and narcotics, but it is difficult for both physician and patient not to be trapped into the prescribing of associated narcotic, sedative and analgesic medications causing dependence.
3. **Persisting cognitive difficulties** that may include sudden onset of (a) problems of verbal comprehension, recall and retention, (b) reading comprehension and retention difficulties, (c) auditory changes, loss of tonal appreciation, or (d) olfactory or auditory hallucinations.[4]
4. **Multiple other sensory abnormalities that should signify an upper Central Nervous System injury,** including visual phenomena: (a) distance appreciation difficulty, (b) tunnel vision, (c) facial agnosia (difficulty in recognizing faces), or (d) loss of night vision. Many of these findings can resolve over three to twelve months.
5. **A significant decrease in circulating blood volume** and signs of peripheral vascular disease. (This can be easily verified by SPECT blood flow testing.)

3 Wallis, A.L. "An Unusual Epidemic". The Lancet, 1955: vol. 2, no. 90. See also Wallis's thesis, University of Edinburgh, 1957
4 "The Clinical and Scientific Basis of M.E. and CFS," (Hyde, Goldstein, & Levine, 1992)

6. **Hyper-somnambulism,** which tends to be replaced by sleep reversals, various sleep dysfunctions and chronic unrewarding sleep. Hypnagogic and hypnapagogic changes occur infrequently. Almost all M.E. patients develop various sleep dysfunction states. Although many patients eventually recover and return to a normal sleep pattern, a few do not. (The habit-forming use of narcotics or analgesics and electronic media appears to perpetuate both chronic sleep and pain dysfunction.)
7. **Frequent physician-induced (iatrogenic) injuries**. Almost no primary care physician, general practitioner, neurologist or internist is equipped to appreciate or diagnose the second phase of this chronic CNS injury. After a few cursory tests and referrals resulting in negative brain scans – (CT, MRI or MS work-up)– the physicians tends to diagnose a psychiatric illness. Then they frequently prescribe multiple antipsychotic medications, sometimes corticosteroids and/or narcotics and physically injurious exercises, as promoted by the insurance industry. **This medical, diagnostic and treatment failure, for which the patient is frequently accused, is also diagnostic of M.E.** One of the worse iatrogenic injuries is caused by physicians frequently misdiagnosing M.E. as Lyme or psychiatric disease and instituting dangerous treatments, which have infrequently resulted in deaths.

The only accurate diagnostic brain imaging instrument that clearly reveals encephalopathy in Myalgic Encephalomyelitis, at all stages, are brain SPECT and brain PET with appropriate software.[5]

Item 5:
Fourteen Cascading Manifestations of M.E.

In addition to the persistence of chronic findings – particularly (a) the persisting rapid decay of intellectual and cognitive stamina, (b) the loss of physical stamina (endurance) following modest physical, intellectual or emotional activity, and (c) the unusually slow recovery following physical, intellectual or emotional exertion – any of the following abnormalities, (which may indicate a CNS deregulatory injury), can be anticipated to develop in M.E. patients. I include only the most frequently observed relatively common findings.

Note: (1) Pain syndromes may persist, but in general they tend to decrease and even disappear after six months to a year if the patient is not addicted to analgesics.

Note: (2) Sleep reversal and un-restorative sleep is the rule in most M.E. patients, particularly in the early months of the disease. In general it will persist if hypnotics, sedatives are used. If electronic media are used after normal evening sleep hours this may cause permanent sleep dysfunction which will never be overcome by

5 We use Segami Oasis software developed by Drs. Ismael Mena & Philippe Briandet.

narcotics or sedatives. The only treatment that works for M.E. sleep dissfunction is ***(a)*** *mobilising the patient during the day after the first six months, or earlier if able* ***(b)*** *avoiding all sedatives and narcotics* ***(c)*** *total restriction of nightime electronic media and* ***(d)*** *a supportive course of self-hypnotism or a good raja-yoga teacher.*

In both early and long-standing M.E., multiple autoimmune changes may develop. In chronic M.E. both patients and physicians should be aware of frequent findings that include an increased incidence of:

1. **Thyroid abnormalities: (a)** Ultrasound volume loss in patients who are not on thyroid medication can occur. *(The Mayo Clinic says normal thyroid volume is 6.5–10.5cc in women, 1cc larger in men)*, **(b)** multiple nodules as in Hashimoto's Disease can occur or **(c)** There is a marked increased incidence of thyroid malignancy found by ultrasound and needle biopsy in our patient samples. In our experience, thyroid blood tests are always normal in early thyroid cancer.[6] *All late stage M.E. patients require a thyroid ultrasound to find solitary malignant nodules with increased vascularization.*

2. **POTS.** Orthostatic dysfunction with **P**ostural **O**rthostatic **T**achycardia **S**yndrome **(POTS)** is routinely missed in these patients. Associated cardiac irregularities may require a pacemaker. A tilt table is not required to make this diagnosis. With the patient standing still, at attention, two inches in front of a couch or bed, with neither patient nor physician speaking, blood pressure and heart rate can be taken with a battery driven sphygmomanometer. From a few minutes, but normally by ten minutes, the immobile and standing POTS patient's heart rate will tend to rise to 100-150 beats or more per minute. Blood pressure may then collapse precipitously and the patient may suffer syncope. To avoid injury by falling, the physician can push the patient onto the bed or couch. *(This is a potentially dangerous test and should be conducted only by a physician).* **While testing a patient at John Hopkins Hospital, I witnessed a patient go into cardiac arrest during a similar test. Fortunately there was a cardiac team in attendance to restart her heart**. Severe dysautanomia or POTS is what probably leads to syncope in these patients, particularly while taking a hot shower where the peripheral vascular system expands, particularly in the legs, leading to decreased cardiac blood supply. Patient with POTS should take showers while seated on a stool.

3. **Palindromic arthritis:** The appearance of vague and highly variable on-off arthritic markers *(elevated anti-nuclear antibodies such as ANA, ENA, rheumatoid factor or an early associated elevated Erythrocyte Sedimentation Rate (ESR blood test.)* and associated non-specific rheumatoid-like symptoms. In my experience elevated pain is observed more frequently in patients who are HLA-B27 positive with or without ankylosing spondylitis, or particularly in Ehlers Danlos Syndrome. Both are complex, genetically derived, and may be associated with immune changes. This may also be a form of Still's disease also known as reactive arthritis.

6 In our experience all thyroid vascular tests are normal in early thyroid cancer. Only the presence of single hypervascular nodule may be indicative prior to needle biopsy.

4. **Quiescent hyper-flexible Ehlers Danlos Syndrome becoming severely symptomatic.** These patients will have had relatively symptomless hyper-extension phenomena (extreme flexibility of joints) prior to falling ill with M.E., but now become significantly disabled with M.E. symptoms. (This may be an important clue pointing to the mechanism of peripheral M.E. dysfunction.)
5. **Causalgia**, also termed **Complex Regional Pain Syndrome (CRPS)**. First to notice this in M.E. patients was a U.S. Civil War physician, Dr. Jacob Da Costa. Also Dr. Silas Weir Mitchell described both Causalgia and Neurasthenia, an early name for Myalgic Encephalomyelitis, in his 1877 book *Neurasthenia and Hysteria* describing this condition. He also wrote a bizarre treatment book, *Fat and Blood*, where he gorged these patients with excessive food and kept them almost as prisoners confined to their bed. The treatment was employed on Virginia Woolf, who was treated with Mitchel's treatment, and who may well have had M.E. She had very negative statements to make against Dr. Mitchell[7].
6. **Light Sensitivity.** M.E. **always** represents a chronic post-infectious injury to the left temporal lobe and the posterior cingulate gyrus of the limbic system. By 1987, the relationship was so consistent that Dr. Jay Cohen correctly referred to M.E. as a limbic system injury. Clinically, M.E. patients often suffer from extreme light sensitivity, possibly due to irritation of the visual pathway when images pass from the eye through the deep white matter of the inferior surface of the inflamed temporal lobe on its way to the visual cortex in the occipital lobe. Although this is more frequently found at the onset of illness, in some patients this light sensitivity can become a permanent disability.
7. **Adverse medication, food sensitivity and alcohol intolerances.** Prior to illness onset these either were not found or were minimal, but in chronic M.E. illness, they often become symptomatically increasingly difficult, sometimes seriously so. This may also be associated with taking long-term NSAIDS which permanently damage the arteries supplying the small and large colon *(as well as other arterial systems)*.
8. **Chronic gastric conditions associated with M.E.** As noted in '7' above, this is one of the most challenging of the cascading difficulties I have seen. Tests tend to be largely normal and if treatments work, they tend to do so only in the short term.
9. **Interstitial cystitis, interstitial vaginitis:** Often misdiagnosed as urinary tract infections, and often seen in polio patients. This can be chronic and unrelenting, and can be associated with dyspareunia.
10. **Severe iatrogenic stress and physical injurie**s resulting from **(a)** failure of insurance companies to compensate patients with disability insurance, resulting in poverty, **(b)** failure of most physicians to understand that M.E. is a serious and measurable injury, with major reactive anxiety occurring due to lack of physician

7 See Mrs Dalloway by Virginia Woolf

assistance **(c)** physician-induced narcotic dependency and injury as well as sedative and NSAID medication injury. **(d)** family break-up, in part because physicians often incorrectly inform other family members that M.E. is a hysterical disease state, **(e)** major weight gain from antipsychotic medications, or **(f)** further injury due to PACE treatment *(Pacing, graded activity, and cognitive behaviour therapy; a randomised evaluation),* which usually includes both cognitive behavioral therapy (CBT) and graded exercise therapy (GET). Insurance companies often recommended PACE treatment since true M.E. patients and many CFS patients become worse. When they with draw from the program, the insurance company may use this pretext to stop paying disability insurance **due to non-compliance**.

11. **Mitochondrial function injury.** *Note: At present I do not recommend testing for Mitochondrial function in North America, where this procedure may cost many thousands of dollars. It should not be done at the expense of the patient as there is still no adequate treatment. Dr. Sarah Myhill in Wales has written an excellent book on this subject and discusses how to best investigate this associated pathology and I believe, dissagrees with my impression. She is able to do this test in the U.K. much more reasonably.*

12. **Multiple social injuries, marital discord and separation, suicide and poverty from patients exhaustingly searching for a cure or a knowledgeable physician.** The causes are **(a)** incessant hope that an alternative medicine will help, unfortunately a few are dangerous and few are more than Band-Aid helpful, **(b)** incessant and expensive search for a physician who will promise recovery, **(c)** social disruption due to insurance companies that refuse or resist paying patients' disability insurance pension, and **(d)** insurance companies that do not allow partially recovered patients to return to work part-time and receive partial compensation. They then, often want the patient to return to full time work or resign. If at all possible such work accommodation should be made to these patients. Most M.E. patients don't wish to be excluded from a normal work life.

13. **Sectioning.** This is a cruel technique employed by the authorities and some physicians in the UK. Sectioning refers to the separation of M.E. children from their parents and their incarceration in psychiatric institutes or hospitals, where they were often placed in a PACE trial on psychiatric medications since it is falsely assumed these patients are psychiatric patients. Deaths have been associated. Years ago, Dr. Michael Cohen, paediatrician of Tarzana, near Los Angeles, believed some of the street children he had seen were simply abandoned M.E. children.

14. **Post-M.E. Syndrome:** Twenty-five to thirty-five years after falling ill, as if chronic M.E. illness was not bad enough, approximately a fifth of all M.E. patients have a worsening of their clinical disease. This condition is best described by Gareth Williams, Emeritus Professor of Medicine at Bristol University, in his extremely

well researched book on polio, *Paralyzed With Fear*. Williams is actually describing post-polio-syndrome (PPS); however, this is exactly what is happening to one in five of my older M.E. patients.

I could not better describe the condition occurring in some older M.E. patients than in the following description, extracted from Dr. Gareth Williams' book, Paralysed With Fear (page 35). His description of post polio syndrome is identical to that of post-M.E. Syndrome. Williams describes it as follows:

> *The cardinal features of PPS are the new onset of muscular weakness, pain and fatigue, together with physical and mental tiredness affecting the whole body. Sometimes there is deep burning pain and tenderness over tendons and other "trigger points". A flu-like exhaustion can be permanent and debilitating. Symptoms usually develop insidiously and are often made worse by exertion.*
>
> *The diagnosis rests solely on the patient's account of his or her symptoms and can be contentious. There are no distinguishing physical signs, nor any laboratory tests, which can distinguish these changes. Since two of the symptoms, anxiety and disturbed sleep, are common in older people they are often dismissed.*
>
> *The impact of PPS is highly variable, as is the way it is perceived by the medical profession. Some authorities dismiss PPS as an indolent condition. This view might not be shared by the many who coped well for 20-30 years after falling ill, but who now can no longer climb the stairs, or run their homes.*

Item 6:
The Reputed CFS PACE Treatment

Treatment: There can be no treatment for Chronic Fatigue Syndrome in any patient without first discovering the cause of the disability obscured by the term CFS. As repeatedly mentioned, there are multiple totally different pathological causes of CFS, therefore potentially multiple totally different treatments depending on the cause of the patient symptons, a few of which are malignancies.

If Michael Sharpe, Kim Goldsmith and Trudie Chalder's publication: The PACE trial of Treatments For Chronic Fatigue Syndrome is accepted, one would believe PACE is an accepted treatment of CFS[8]. Their publication begins with the following statement, and I quote:

"Chronic Fatigue Syndrome (CFS) is a chronic disabling illness…"

In this statement, the authors shy away from calling CFS either a disease or a syndrome. Yet CFS is not a disease. Is not really a syndrome either because CFS clearly represents what is probably well over 100 pathologies or diseases. You cannot have a single treatment for 100 plus pathologies. That is like prescribing the single product "x", shall we call it "aspirin" to treat every known malignancy in the book, which can cause fatigue, HIV, major depression etc, etc. and the list of possibilities is indeterminate. This is the basic idiocy and damage of PACE treatment.

If the treatment were as banal as a sugar tablet, it wouldn't be so bad. However, at the patient's insistence, I placed one of the senior Nursing Directors in Ottawa, whose physicians had her diagnosed CFS, into the equivalent of a PACE trial for GET, or graded exercise therapy. I didn't believe in this so I hospitalized her in the excellent Ottawa Rehabilitation Hospital for a month and let the specialists perform this "patient insisted" service. This graded exercise not only resulted in a disaster, but she had walked into the hospital and came home in an ambulance and ended up for subsequent years in total bed and house confinement. I and her physicians exacerbated her already severe injury by agreeing to her request. My experience with CBT, Cognitive Behaviour Therapy has been equally disastrous. Insurance companies regularly send patients with "CFS" for such combined treatment. The result in every one of my patients subjected to this treatment is they are made, not only worse, but when they are forced to leave the program, the insurance companies then stop paying their client their disability pension. The insurance company rational: Non-compliance with the treatment programme.

I wonder if insurance companies either intentionally or unintentionally "salt the mine," by placing a minor or non-illness patient in a PACE program who then miraculously gets better.

8 The PACE trial of treatments for chronic fatigue Syndrome : BMC Psychology 2019 7 :15 : https://doi.org/10.1186/s40359-019-0288-x (Published 12 March 2019

I have written to Dr. Sharpe in order to go to his place of work in England and question him. He does not reply, or should I say: He doesn't comply?

I am informed that Dr. Sharpe works for the insurance industry. I cannot confirm or deny this statement because he won't answer my communication.

The PACE trial including Graduated Exercise Therapy (GET) programs will not restore a real M.E. patient to health. Cognitive Behavioral Therapy (CBT), Antidepressant and other psychoactive medications, prescribed by hundreds of their physicians have not rendered any significant assistance to M.E. patients I have seen and if anything have represented a danger. Psychoactive medications are well know to work very well for many major psychiatric diseases; the very fact they don't work for so called CFS patients should be telling physicians something.

Item 7:
Potentiators

Potentiators are genetic and external factors that may promote the onset of M.E. or make the condition worse

Since 1984 I have examined countless patients with Myalgic Encephalomyelitis (M.E.) or individuals who believed they were disabled by this acute-onset illness. It became apparent in the first few years certain variables appeared to potentiate (increase) the frequency and/or severity of M.E.:

Genetic Potentiators Include:

1. Male-female divide.
2. Major prior allergic precondition, particularly childhood asthma.
3. Ehlers Danlos Hypermobility Syndrome and other collagen diseases.
4. HLA-B27 genetic anomaly.
5. Close relative with a history of paralytic poliomyelitis.
6. Multiple prior, and different enteroviral illnesses (raising the possibility of a specific gene weakness).
7. Multiple closely spaced repeat infectious diseases in the year prior to permanent illness onset suggesting the possibility of an immune system break down.

External Potentiators:

1. Returning to work or physical activity too soon after the initial viral injury.
2. Occupations with increased exposure to infectious diseases.
3. Illness onset within two weeks of Recombinant Hepatitis B immunization (RHB), which for a short period appears to lower the resistance to external infections. This is seen more often when the patient has had multiple RHB immunizations. If there is an untoward reaction to the first immunization the second will be worse and may trigger the chronic injury. If an immunization injury occurs it is usually within one week or less of the injection.
4. The organized attack by the insurance industry, above all upon women with M.E. and the CFS spectrum of diseases, which causes unusual and persistent financial stress, family and home disruption injury. I have seen infrequently, when an insurance physician tells the partner of the injured patient, "She is just being hysterical! She really is not ill!" This becomes an excuse for marital breakdown with the husband leaving.
5. The refusal of insurance companies to honour disability pensions paid for by women. Whereas, in my experience, insurers tend to immediately award physicians, members of parliament, and men with their disability pensions.

In the late 1980s, Dr Ismael Mena at the University of California, demonstrated that, in M.E. patients, both intellectual and exercise stress causes increased brain SPECT negative circulatory changes.

These potentiators, all observed from patient histories, will not necessarily be noticed if the physician or health care provider does not take a complete personal and family history. I totally missed the effect of Ehlers Danlos until I did a house call in Sydney, Australia, in 1989, and a young woman patient began my education in this significant potentiator of chronic post-infectious illness.

Discussion of Potentiators

1. Male-female divide

- It is a common stereotype that when a male falls ill with an infection, whether a minor cold or influenza or other significant viral or microbial illness, he tends to act or pretends to react much more severely than girls or adult females might with a similar infection. Could this be because men may have a more reactive immune system, which provokes a more explosive reaction? One possible explanation is that women are genetically engineered to have a lesser immune system reaction so they do not reject and abort a normal pregnancy.

- In my M.E. patient investigations, this difference between the sexes appears in several manners:
- Significantly fewer men develop M.E. than women. This may be due to the increased number of women, versus men, who work as health care workers and teachers. So it is possible men come in less contact with enterovirus infections in general.
- When my male patients fall ill with M.E., they appear to have a better ability to recover, partially or fully, than the majority of women.
- Female patients appear to develop a greater number of autoimmune side effects to M.E. illness than my male patients.
- Female patients also appear to develop a greater number of medication reactions than men with M.E.

2. Occupation of M.E. patients seen in Ottawa area

I live in a capital city where federal government employees number approximately 150,000 while primary, secondary and university teachers number approximately 10,000. So there are roughly 15 government workers for every teacher in the Ottawa region. Even if post pubertal students are included, the total number of teachers and post-pubertal students do not exceed the number of government workers.

Yet in my practice the cumulative number of health care workers, teachers and students with chronic M.E.-like illness significantly exceeds that of government workers. Although I do not propose this as a clear epidemiological study (judging by numbers), patients with M.E. or M.E.-like illness among teachers, students and health care workers greatly exceed those in government work. These more vulnerable professional groups may be associated with the following factors:

- Higher contact with infectious disease.
- Increased occupational fatigue due to longer hours of work or study.
- Increased physical and intellectual fatigue.
- Women in the work-force, work all day and go home to continue working, thereby possibly developing increased immune exhaustion.

It is easy to conclude those occupations most chronically over-fatigued and most in contact with infectious disease present a significantly larger percentage of patients with M.E.-like disease.

3. Major prior allergic precondition, particularly childhood asthma

Young or adult patients with a childhood history of major allergy conditions, particularly bronchial asthma, even if they have not had asthmatic problems for many years, appear to be more numerous than one would anticipate.

4. Ehlers Danlos hypermobility syndrome and other collagen diseases

The number of patients I have seen with a history of Ehlers Danlos syndrome or other collagen diseases significantly exceed what one would anticipate. I can only assume a genetic link with collagen diseases and immune response. Hypermobile Ehlers Danlos is not a common condition. It is reportedly seen in approximately 1/5,000 to 1/20,000 individuals in the general population, but occurs in up to 1/20 in my classic M.E. population[9]. This can only suggest that Ehlers Danlos hypermobile Syndrome sufferers share not just a genetic weakness for enteroviral infections but the gene source of EDS is possibly located in a place that normally controls or shares major immune control.

Many people with Ehlers Danlos have totally normal lives and activities. But following an acute infectious disease in late teenage years or early adulthood, they appear to develop an M.E. disability in far greater numbers and often with far more severity than one would anticipate. The increase is seen in both the number of patients and the degree of chronicity and disability, although there is wide variability in all acquired disease manifestations.

Unfortunately, unless a more precise definition of M.E. can be internationally accepted that is based upon enteroviral cause and verifiable scientific measurement outside of patient symptoms, and an acceptable genetic test for Ehlers Danlos hypermobility syndrome can be agreed on, I do not believe that an epidemiological study can be conducted with any precision.

5. HLA-B27 genetic anomaly

This is another common genetic anomaly which appears to be a potentiator in M.E. It is related to M.E., not necessarily in the numbers who fall ill, but to the degree of both pain and gastro-intestinal dysfunction. Its presence may also be a potentiator of muscle, spinal, joint and varied body and urethral pain. The presence of HLA-B27 may add to the degree of dysfunctional symptoms that increase an M.E. patient's difficulties.

This human genetic anomaly (HLAB27) is quite common in certain Scandinavian populations, although to my knowledge, these populations have not been shown to have an increased number of M.E. or M.E.-like patients. Once again it is not the numbers that appear important but the degree of disability. The epidemiological features once again cannot satisfactorily be clarified unless a more accurate M.E. diagnosis is accepted. The association of HLA-B27 with Reiter's Syndrome, in which several common infections trigger reactive arthritic pain and dysfunction in the presence of HLA-B27 and its variables, is well known.

9 https://ghr.nlm.nih.gov/condition/ehlers-danlos-syndrome

6. Immediate post-Recombinant Hepatitis B immunization (RHB)

The number of patients who die or fall seriously ill immediately after or within hours or a week of receiving Recombinant Hepatitis B immunization has already been mentioned. RHB immunization is a totally synthetic product resembling the capsule of the hepatitis B virus. This synthetic protein is a powerful immune stimulant against the presence of the viral envelope of Hepatitis B, thus preventing infectious Hepatitis B from maintaining its presence in the human body. Although certain countries such as the USA recognized the increased side effect to this immunization, and to my knowledge have paid millions of dollars in compensation to the injured patients or their families, they have done nothing to investigate, prevent or treat this apparent danger. **Note**: This is an essential immunization for any health care workers worldwide or any person coming into contact with human fluids. RHb immunization should come with an essential death or disability pension wherever it is given, as in the USA when the association can be proven.

7. Relative with a history of poliomyelitis or enteroviral illness

I first became aware of this relationship in 1984, just three decades after the last deadly North American wave of Paralytic Poliomyelitis in 1955. Many patients who had fallen ill with M.E. knew of a close relative who suffered paralytic polio, a closely related enteroviral disease.

I have had a very small group of patients who had repetitive enteroviral illnesses. One patient, whose great-grandparents died within a day of each other circa 1890 in what may have been an early Paralytic Polio epidemic, had **(a)** poliomyelitis as a child and **(b)** severe Bornholm Disease that required hospitalization for ten days, associated with a high fever and repetitive angina-like episodes that lasted a year. *(This is unusual, since most attacks are over in one to three days)*. Then **(c)** classic severe Myalgic Encephalomyelitis with a significant encephalopathy that required hospitalization; the patient was confined to bed for five months and years later continues to be partially disabled.

In this small but important group, one has to question whether some individuals had a genetic weakness for the entire family of enteroviruses.

8. I cannot underestimate the severity of what appears to be the organized attack by the insurance industry, above all upon women with M.E. and the CFS spectrum of diseases, often with the well-paid assistance of callous university-based physicians or researchers.

I have mentioned this previously but it is worth reviewing . Between 1983 and 1992 a major surge of enterovirus-based M.E. patients appeared at my office, including approximately 50 physicians. These physicians were an unusual though unintended study group, in that they were either well insured by their medical association insurance policy or not insured at all.

That a significant number of younger physicians were not personally insured is understandable. Coming out of medical school, these physicians wanted to pay off their accumulated enormous debt. Being young, they believed their chance of falling chronically ill was almost non-existent or they just never thought of chronic illness. This M.E. group tried to work as soon as they could stand, but soon collapsed. To the best of my knowledge, some declared bankruptcy and the majority never returned to work as physicians. I am not aware of what eventually happened with all of this group. However, few of those uninsured physicians who I followed, who tried to work, continued in practice.

The second group, those physicians insured with their medical association, who thus had water-tight policies or collective clout, immediately received insurance benefits without a legal fight or obvious insurance company reticence. M.E. during this period was particularly severe and many of these physicians were bedridden or housebound, in some cases for well over a year. They were then partially disabled for several years before slowly returning to work. Most of these physicians did eventually return to work, but often remained partially disabled and unable to undertake strenuous medical work such as orthopaedics or surgery that requires hours of standing and physical effort. At least one of these physicians changed his speciality to psychiatry to be able to sit and to see significantly fewer patients.

If this dialectic is transferred to the majority of M.E. patients who are female, the consequences are immediately understood. Disabled professional or semi-professional women, particularly in teaching and medical support services or in middle or upper-level bureaucracies – often with children, mortgages and life responsibilities – often showed a pattern similar to that of the uninsured physicians I once followed.

In some American states such as California an ombudsman can assist these disabled patients with the insurance companies' intransigence – but in the U.K., most U.S. States and in Canada, I am aware of no such protection. A Canadian disability benefit is available in all provinces except Quebec, where the insurance is monitored by the province and where it may be more difficult to obtain a pension due to presumed government financial difficulties. In any case, this is a subsistence pension and does not compare to what normal work insurance would cover.

Chapter Nine

How to Help Distinguish and Understand the Differences Between M.E. and CFS

Also

The Difference Between CFS and Chronic Fatigue

	Myalgic Encephalomyelitis (M.E.)	Chronic Fatigue Syndrome (CFS) and Chronic Fatigue (CF)
1	**Onset:** M.E begins as an acute onset post infectious disease. This results in a variable acute encephalitis. As in polio, also caused by an almost identical enterovirus, most patients recover. Also as in paralytic poliomyelitis, those who develop a persisting neuronal injury become chronically disabled, in polio with paralysis or death. In M.E. with both cognitive, motor and executive dysfunction.	**Onset:** Since CFS represents hundreds of possible different pathologies this may be either an acute or gradual onset group of diseases. Gradual onset may be observed more frequently.
2	**Nature of Onset:** This is often severe and very dramatic. There is often acute major head and body pain and disability. The severity and suddenness of the symptom onset, often causes an over-riding symptom driven fear. The motor and intellectual dysfunction collapse is dramatic, and, understandably, causes this fear. However a wide range of onset symptoms are possible in M.E.[1] Often patients can tell you the day and hour they fell ill.	**Nature of Onset:** Onset is more often gradual, with growing disability over weeks or months except in acute toxic injury or with other non-enteroviral infections, such as measles, varicella or EBV. Patients tend to be vague about the day, the month and at times even the year of illness onset. They will often mention two or more conflicting dates of onset or partial onset. Many patients who believe they have CFS actually have multiple different pathologies that together accumulate to form their now chronic illness. They should also be taken seriously. Sometimes these can be treated effectively **if the pathologies are searched for and discovered**.

1 See "The Clinical and Scientific Basis of M.E. and CFS," (Hyde, Goldstein, & Levine, 1992)

	Myalgic Encephalomyelitis (M.E.)	Chronic Fatigue Syndrome (CFS) and Chronic Fatigue (CF)
3	**Seasonality:** Onset is seasonally associated, usually in June to November and a small blip at Christmas in the north temperate climatic zones.	**Seasonality:** There is no seasonal association with CFS. Onset can be any month of the year.
4	**Cause:** M.E. is caused by any of a large number of clinically invisible and a few visible, enteroviral infections. These infections can appear either in the patient or in their close contacts. Although most M.E. causing enterovirus infections are relatively invisible, a few, such as hand-foot and mouth disease, herpangina, Apollo disease (conjunctivitis) are visible commencing with a typical associated stigmata. **Visible enteroviral diseases:** these include and are not limited to clinical disease or contact with **(a)** hand-foot and mouth disease (HFM), (Includes: Coxsackie A 16, EV 71), **(b)** Herpangina (Includes Coxsackie B & ECHO viruses), **(c)** poliomyelitis (missed polio), **(d)** Acute Flaccid Paralysis. This can be caused both by direct infection with the classical features of the disease or they can be secondary by association with a child who has one of these enteroviral diseases. Often, an enterovirus disease causing a classical illness as noted in a child, when transferred to an adult, may appear in a totally different form: eg HFM in a child and M.E. in the teacher or parent. In other words, a visible enteroviral disease in a child may transfer to an adult as a largely clinically invisible enteroviral disease such as M.E..	**Cause:** CFS is so diverse it is better described as a mythical invention since it is neither a disease, nor a pathology, nor a syndrome. CFS can be due to: **(a)** Various post infectious diseases. Those associated with severely elevated body temperature at onset causing brain injury are obviously more easy to recognize early on. EBV, Mumps, Varicella, measles and any historically normal childhood infection can all cause the typical published CFS features in adults. Childhood infections in adults over 25 often cause both lethal and sub-lethal injuries. Many diseases such as **syphilis, gonorrhoea, tuberculosis** are often quite invisible and can cause the typical CFS manifestations. Physicians have become blasé about testing for these once common serious infections. **(b)** Single or repeat physical trauma (e.g. concussion) as in sports and MV injuries, **(c)** A wide variety of toxic chemical exposures. **(d)** Within 1-7 days of Recombinant Hepatitis B immunization particularly, if this is followed by air or bus travel within the first few days of travel where minor infections in other passengers can cause a chronic infection in the recently immunized. **(e)** Some medications eg: Cipro family can cause major CNS injury, in addition to tendon rupture months after taking the medication. Some NSAIDS can cause severe vascular and cardiac disease. **(f)** Various genetic diseases. **(g)** Malignancies. **(h)** An accumulation of multiple pathologies in a single patient. The sheer extent of infections, traumas, genetic and other factors which can exactly replicate the 1988 & 1994 published symptoms of CFS is what makes any definition of CFS not only ridiculous and a waste of time but dangerous since most physicians tend to treat anyone with a diagnosis of CFS as an anxiety neurosis or a major psychiatric disease and then, not properly investigate what may be a treatable disease. NIH/CDC in the USA has wasted millions on this fictitious chimera called CFS.

	Myalgic Encephalomyelitis (M.E.)	Chronic Fatigue Syndrome (CFS) and Chronic Fatigue (CF)
5	**Epidemic & Sporadic:** M.E. exists as an epidemic or sporadic disease. Epidemics are usually observed and sporadic disease, although equally serious, often dismissed as anxiety.	**Sporadic:** CFS is primarily a sporadic finding. It is not known as an epidemic disease except during mass toxic chemical exposure: eg: the Bhopal disaster in India from Isocyanate pesticide.
6	**Location of Epidemic Onset:** Except in epidemics, M.E. is rarely recognized for what it is except with its chronic symptoms. Most epidemics occur in hospitals, military camps, residences, bus and travelling groups, when the large numbers make diagnosis easier.	**Location of Onset:** : CFS also is recognized by its chronic symptoms and is almost never related to any specific work or residential location unless it is due to toxic chemical injury.
7	**Biphasic Nature:** M.E. is a biphasic disease, particularly noticed during epidemics. After the initial infection, the patient may appear to have a partial recovery for one or more days prior to lapsing into the chronic phase.	CFS is not known as a biphasic injury:
8	**Research Basis:** For both identification and research, either recovery or evidence of associated enteroviral infection is important. In the chronic phase, months or years after onset, viral identification is not practically possible with present diagnostic capabilities. A consensus of factors must be taken as a guide. These factors are discussed in the previous chapter.	**Research Basis:** One cannot validly research CFS. Since CFS is neither a recognizable disease, pathology, or verifiable syndrome. **In all cases, CFS represents a missed diagnosis**. There can be no valid CFS publication or CFS research paper, other than to state CFS does not exist, either as a physical or psychological or psychiatric phenomena. **All CFS publications are without merit**. **All CFS treatments are without merit**.
9	**Sexual Bias:** Between 70 to 80% of all patients are female.	**Sexual Bias:** There is NO female sexual preference. Friedberg and Jason, In Understanding CFS ISBN 1-55798-511-1 suggest males may predominate.

	Myalgic Encephalomyelitis (M.E.)	Chronic Fatigue Syndrome (CFS) and Chronic Fatigue (CF)
10	**Who Falls Ill with M.E.:** There is a professional bias to M.E. **(a)** health care workers, physicians, nurses, support staff, **(b)** teachers, **(c)** post pubertal students. **(d)** working mothers with school age children demonstrate an undue preponderance, possibly due to increased infectious contact in these individuals. *(When younger children are so infected, and fall ill with M.E., their illness is often missed since they cannot explain what has happened to them).*	**Who Falls Ill with CFS:** No professional bias to CFS exists other than in those working in or exposed to a toxic chemical environment. (eg: firemen, military, police, commercial pilots, farmers, golf course workers, industrial workers.). Toxic chemical exposure is often missed, particularly in single cases or when the toxic chemical exposure is cumulative over a long period.
11	**Source of Most Symptoms:** 50% or more of the symptomology in M.E. is associated with abnormal CNS (brain and spinal cord) dysfunction including **(a)** memory, **(b)** motor & **(c)** administrative dysfunctions. The classical M.E. brain injury is associated with anterior temporal, limbic system (cingulate), motor cortex and vermix (cerebellum injury).	**Source of Most Symptoms:** Primary complaint is fatigue and often fibromyalgia, both of which can be caused by up to 100 or more different causes including medications. Part of the problem with diagnosing fibromyalgia is due to the fact, as in CFS, it can be due to hundreds of different causes. Fibromyalgia, like CFS, is not a disease but as in CFS appears in multiple conditions and after multiple medications.
12	**Coarse:** ttends to begin acutely and dramatically, associated with intense fear due to the severity of symptoms. In many cases the symptoms improve over the first months or year, almost always leaving residual permanent CNS driven disabilities. In severe cases, improvement is limited. In many patients a post-polio-like recurrence occurs in older age (60-70s).	**Coarse:** Except in cases of toxic chemical injury, and non-enteroviral childhood infections occurring in adults, onset tends to be benign or gradual. Depending upon the varius and multiple causes, those with a non-specific chronic fatigue can lead to (a) total recovery, (b) chronicity or (c) to death. Too often, CFS can be due to a progressive or lethal (i) missed malignancy, (ii) missed cardiac, (iii) missed vascular injury (in brain or torso) or (iv) missed medication injury. Note: 20% of our CFS patients have a missed, physically measurable, cardiovascular pathology.

	Myalgic Encephalomyelitis (M.E.)	Chronic Fatigue Syndrome (CFS) and Chronic Fatigue (CF)
13	**Cascading Features:** I believe these are due to CNS injuries, which trigger multiple cascading features as described in chapter 8. Some cascading features are due to sociological conditions as mentioned.	**There Are No Specific Cascading Features.**
14	**Recovery in Youths:** Many post pubertal youths recover totally if further injury is not caused by (a) narcotics, (b) anti-psychiatric medications, sedative and NSAID medications, (c) PACE program or other diseases and genetic anomalies .**Recovery in Adults:** Many adults will partially recover given sufficient physical and emotional rest and support early in the disease, but will rarely completely return to their pre-infection state. Physical, intellectual, chemical or psychological stress early in the disease can render M.E. worse	**Recovery:** Age is not a factor. Recovery is totally variable depending upon the cause of the symptoms. Some improve and some worsen depending upon the cause.
15	**Prevention:** In theory if a total enteroviral immunization were developed, this could prevent M.E. as it has prevented paralytic polio, but only from the 3 polio viruses included in the common polio immunizations. The problem with the polio immunization is that when invented in 1955, the total number of polio viruses were thought only to be three in number whereas there are are several with others evolving.	**Prevention:** CFS is not a single disease, or single pathology, so there can be no prevention or treatment unless the cause of the missed diagnoses is first discovered.

	Myalgic Encephalomyelitis (M.E.)	Chronic Fatigue Syndrome (CFS) and Chronic Fatigue (CF)
16	**Treatment:** (Also See Section on Treatment): There is no working medication treatment I am aware of for any enteroviral infection including poliomyelitis. To improve the acute CNS injury one would require: **(a)** a medication to offset the initial micro-vasculitis associated with the acute encephalopathy that causes the *(i)* variable memory, *(ii)* administrative and *(iii)* motor nerve dysfunction. **(b)** The same is true of paralytic poliomyelitis. No medication has ever been invented to counteract the initial vasculitis in the spinal column obstructing blood flow to the anterior horn cells, which in-turn innervates the peripheral nerves and causes paralysis or death. See Chapter on Treatment at end of this book for useful advice. There is still much to be done to improve patients dysfunction. Over 100 years ago, William Osler in his 1914 textbook of medicine advised: There is no effective medication for this disease. All one can do is to trust in god. This has not changed today	**Treatment:** No treatment, whether medication or PACE program by any person or company for CFS is valid. Anything you read regarding treatment is false. Patients who state they have got better from CFS with any given medication or treatment would undoubtedly have gotten better without this placebo. For those patients who miraculously improved on PACE, it would have been far better to prescribe a nice chocolate each day, for equal but a more enjoyable effect. Many treatments for CFS patients are inventions of charlatans. CFS represents hundreds of different undiagnosed illnesses, injuries or pathologies. No single cure has ever been devised or can be devised for hundreds of totally different pathologies. The same is true for the non-disease fibromyalgia.
17	**Chronic Disease Advise:** After 6 months to a year of illness and once the most severe symptoms begin to decrease, patients should begin a regime of gentle mobilization. Lying permanently in bed can be very dangerous. Kierkegaard, the Danish philosopher stated: you can walk away from any disease. Although he was a bit too optimistic his direction was good. However PACE treatment can be seriously dangerous. Disability doesn't mean death: some of the best books and discoveries ever made have been made by disabled individuals. Assistance by the patient's physician to obtain disability pension for M.E. disabled patients is one of the best helps a patient can receive.	**Chronic Disease Advice:** If you do not have M.E. your physician has not demonstrated the cause of your CFS illness. If you have still are diagnosed with CFS, your physician has missed your diagnosis. There is no such pathology as CFS and no effective treatment. The best advice is to find another physician to properly investigate you.

Dr. Byron Hyde

Byron Hyde is a leading worldwide authority on M.E. and CFS. For 35 years he has explored M.E. patients, and uncovered multiple causes of CFS. He has known and worked with the majority of the great M.E. researchers who have lived since 1934.

Byron Hyde is one of only two physicians invited to address the House of Parliament and the House of Lords' Committee on M.E. in London.

This book demonstrates fascinating evidence that M.E. is closely related to poliomyelitis, both in its genomic structure and its association with earlier polio epidemics. Byron Hyde has worked in an orphanage of abandoned polio children in Asia and knows the extreme end of the M.E. polio spectrum. Byron Hyde knew Jonah Salk and visited Albert Sabin in Washington DC, the inventors of two of the polio immunizations.

Byron Hyde has also interviewed and met several of the less loved denizens of the under-world of M.E. and CFS, including Stephen Straus, Colin McEvedy who called the Royal Free M.E. epidemic a case of mass hysteria. He knows Michael Sharpe and carried on a friendly communication with the equally charming and unfortunately, very dangerous Sir Simon Wessely.

Byron Hyde is the only living physician researcher who has visited and examined M.E. patients and spoke with physicians from M.E. Epidemics including: **(a)** the LA County Hospital epidemic, **(b)** the Akureyri epidemic **(c)** the Royal Free Epidemic, **(d)** the New Zealand and Australian epidemics and **(e)** examined the patients from the 1984 - 1995 epidemic in North America, the effects of which, have persisted until today.

Byron Hyde published the world's best-selling book on M.E., ***the Clinical and Scientific Basis of Myalgic Encephalomyelitis /Chronic Fatigue Syndrome*** which has sold 10,000 copies. He also published ***Missed Diagnoses***, discussing the multiple causes of CFS and Fibromyalgia. ***Understanding Myalgic Encephalomyelitis*** is an essential read for anyone interested in M.E. or the terrible diseases often hidden by the term CFS.

Dr Hyde and the Nightingale Research Foundation organized the first world symposium on M.E. in Cambridge University, England. ***Understanding M.E.*** is his third book on M.E., which is soon to be published.

Advanced orders are now being accepted throught the Nightingale Research Foundation web-site, **www.nightingale.ca**

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