

The Definition & History of Myalgic Encephalomyelitis (M.E.)

*This monograph was prepared for the Amsterdam M.E. Conference September 2015. It has not been adequately spell and grammar checked by my staff. For this I apologize to the readers. There are undoubtedly errors also in composition but the story is essentially valid, as is the much overlooked history Of Myalgic Encephalomyelitis.
Byron Hyde MD, The Nightingale Research Foundation.*

The Nightingale Definition of M.E.

Briefly, Primary Myalgic Encephalomyelitis (M.E.): is like poliomyelitis. In its epidemic form it is a two stage chronic post-infectious and autoimmune disease. However, where polio primarily injures the pons and spinal cord, M.E. injures the cerebral cortex causing deregulation of the Central Nervous System. Like Poliomyelitis, most M.E. first and second stage patients recover. Those that do not, give rise to a deregulation of many CNS functions causing chronic and variable disabilities. These disabilities may involve any or all of recuperative, cognitive, intellectual, gastric, bladder, motor, sleep, temperature, pain and vasomotor regulation abilities.

The primary symptoms of M.E. are all related to the deregulation of the cortical hemispheres. M.E. is variable depending upon the degree of CNS injury and is diagnosed on the basis of two necessary measurable scientific findings.

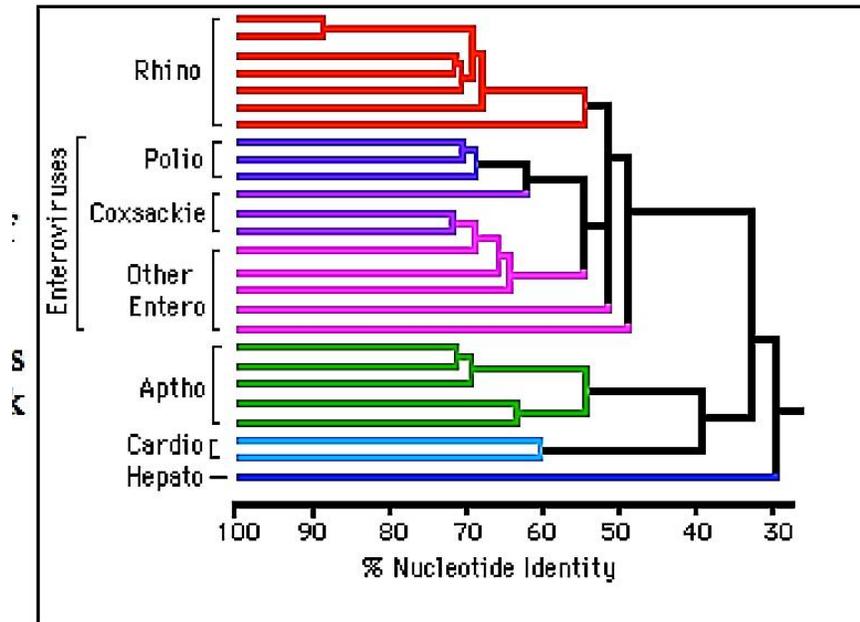
1. **Necessary Finding 1:** A chronic, measurable, encephalopathy always involving the Limbic System and other areas of the Cerebral Cortex, and in worse cases, basal ganglia injury. These injuries can be measured by Brain SPECT with adequate software.
2. **Necessary Finding 2:** Either proof and recovery of Enteroviral Infection at onset of illness or recovery of capsid protein from the gastric mucosa in chronic illness.

Secondary Myalgic Encephalomyelitis: is a similar CNS injury with chronic deregulation of the Cerebral Cortex without recovery of enterovirus. It is usually caused by various neuro-toxic chemical injuries commonly seen in agricultural or golfing workers exposed to pesticides and herbicides. Less frequently it is seen following CS gas exposure in the military and police, or from turbine lubricant nerve

toxin degradation products (TCP-TriCresylPhosphate) seen in airline pilots and flight attendants.

A Short History of Myalgic Encephalomyelitis Or The Fourth Polio

1. **The Two Stepbrothers, Polio and M.E.:** Until the introduction of effective Salk polio immunization, M.E. occurred simultaneously with Polio epidemics. Both polio and Myalgic Encephalomyelitis are caused by enteroviruses. This is possibly due to different individuals response to similar but different or the same enteroviruses. We know that all enteroviruses have a capsid end that continually mutates and even the three major polio-viruses, Lansing, Leon and Brunhilde, mutate. No polio virus at the beginning of an epidemic is the same as the “same” virus at the end of an epidemic. This is true of the probable several enteroviruses that cause Myalgic Encephalomyelitis.



Post-Polio Network, Western Australia

2. **Epidemic Myalgic Encephalomyelitis (M.E.):** Epidemic M.E., like epidemic poliomyelitis has a **two-stage incubation period**. The first stage is 3-5 days, then the initial respiratory track, gastric or pain symptoms commence. Depending upon the author, the second stage begins in 4-15 days, when the paralytic features of lower central nervous system symptoms begin (bulbar & spinal cord) in Polio or when the cerebral cortex features of Myalgic Encephalomyelitis occur. In a way, it is unfortunate that the term Myalgic was attached to the term Myalgic Encephalomyelitis as it was by both Ivar Wickman (polyneuritic) in 1895 and as it was by Melvin Ramsay in the 1950s. If that was unfortunate, Stephen Straus directing attention to “fatigue” in Chronic Fatigue Syndrome was a unmitigated disaster. It is true, than many patients with M.E. begin with severe malaise and an incredible variety of pain syndromes. It is true that Myalgic Encephalomyelitis patients have fatigue, but so do most patients with major chronic pathologies. The problem with fatigue is it is virtually indefinable and universal. With a single word, Wickman, Ramsay and certainly Strauss, sent the researchers down the wrong road. The main diagnostic entry word into Myalgic Encephalomyelitis is “Encephalopathy”. It always has been, that, and the effects of this encephalopathy.
3. **Second Stage of Polio:** In polio signs and symptoms of the second stage are heralded by flaccid paralysis with microvascular injury originating primarily from the bulbar and pontine brain through the spinal cord.
4. **Second Stage of Myalgic Encephalomyelitis:** In Myalgic Encephalomyelitis, the signs and symptoms of the second stage are heralded by initial injuries to the upper central nervous system, primarily injuries occurring above the bulbar brain and manifested by deregulation symptoms, including pain, profound malaise and a multitude of other CNS and vascular symptoms. Unfortunately, it became customary to call these relapses. They were never true relapses, the second stages never went away.
5. **Myalgic Encephalomyelitis has a characteristic brain map that varies with the degree and location of CNS injuries:** Myalgic Encephalomyelitis, as in many known diseases, can vary from mild to major to infrequent death. The degree of injury is easily observed on adequate brain mapping. The areas always injured include the temporal lobe and the limbic system with either spotty or severe injury to the rest of the cerebral cortex (brain). The patient’s symptoms can be easily declared simply by using the old but somewhat inadequate Brodman map applied to the brain SPECT using Dr. Ismael Mena’s brain SPECT software.
6. **The Chronic Viral Nest:** Just as varicella virus appears to find a chronic home in the posterior root ganglions of the spinal cord, M.E., an enterovirus, takes its chronic home in the gut. Myalgic Encephalomyelitis in part represents and can in part be confirmed by the chronic enteroviral gastric

infection, potentially causing continual seeding to the brain and spine and other organs. Dr. John Chia has superbly demonstrated this with his immunoperoxidase staining for enterovirus capsid protein and double stranded RNA.

7. **The Two Stage Vasculitis:** Myalgic Encephalomyelitis, as in Poliomyelitis, is a vasculitis affecting neurons and other organs. The nature of this vasculitis is probably both infectious and autoimmune. In M.E. a secondary vasculitis exists caused by brain deregulation, in some cases mildly and in some cases seriously injuring the function of the peripheral arterial system.
8. **Polio and M.E. Rarely Kills, But in Polio, the Second Stage Bulbar and Spinal Flaccid Paralysis is Dramatically Visible for Life to the World, whereas in the Second Stage of M.E. Only the Patient is Aware of the Disastrous Cortical Effects of Their Injury:** As in poliomyelitis, except for the bulbar area injuries, the patient rarely dies, (less than 0.5-1% of polio victims die). Like poliomyelitis only a few patients injured by the enteroviruses causing M.E., become symptomatic and the rest may become immune. In both polio and M.E. enteroviral infected patients, clinically, we only see the tip of the iceberg, those who have suffered major injury. In M.E. what the patient complains of and what the physician frequently does not see, is major deregulation of the upper brain's cortical functions, including (a) physical, (b) intellectual, (c) autonomic, (d) vascular, (e) emotional, and (e) pain regulation features.
9. **Repair and Recovery:** Some patients, depending upon the degree of injury, particularly surviving children and adolescents, have a more elastic central nervous system (CNS) and are more capable of rebuilding the control areas of the various injured CNS sites and systems into different brain areas. Since the negative effect on the Cortical Central Nervous System is through an injury of the micro-vascular system, ANY stress on that microvascular system can potentially further deregulate an already unstable system. One of the greatest stresses which are rarely if ever discussed in M.E. is the terrible effect of anxiety caused by sudden poverty, a sudden inability to work, an isolation from one's friends to be independent, to support or help support one self and one's family. The healthiest post M.E. patients I have met with are those some 40-50 medical doctors who have received immediately, a suitable and liveable pension.
10. **Like Dr. Ivar Wickman in 1907, I believe that Myalgic Encephalomyelitis, is the Fourth Polio.** It is a polio which would not occur today, if the capsid data of the other enteroviruses had been included in Jonah Salk and Albert Sabin's immunizations.

Chronic Fatigue Syndrome: This was a false definition, I believe, based upon Steven Straus's theory that the Lake Tahoe epidemic and other cases of Myalgic

Encephalomyelitis were caused by Epstein Bar injury. Epstein Barr has an incubation period of circa 40-days; it can never be an epidemic disease. You can observe this contention to be so in the references at the end of their definitional paper. Their references only includes papers on Epstein Bar. Few of the experts on this definition ever before or after ever published on M.E. or CFS. I have never decided if this fabricated definition was simply an example of gross stupidity on the part of the definitional committee or if it was a technique to purloin 30 million dollars from NIH research funds by the committee members. This definition should be forgotten and buried. It is not that chronic fatigue patients do not exist, they do, but they represent multiple misdiagnosed minor and major medical diseases. **No disease exists if the physician cannot illustrate this disase with patho-physiological injuries. Common symptoms such as fatigue do not demonstrate a diagnosable disease state.**

A Short History of Myalgic Encephalomyelitis

The following paper was prepared by Byron Hyde MD for the Dutch M.E. Association (M.E./CVS Vereniging) in September 2015.

“Historically, and until the introduction of Jonas Salk’s immunization in 1955, epidemic Myalgic Encephalomyelitis had been so closely allied to paralytic poliomyelitis epidemics that most learned and senior physicians on both sides of the investigations of these much disputed epidemics have not been able to distinguish, one from the other. Experts fought as to whether they were dealing with different aspects of the simultaneous occurring polio epidemic or cases of Myalgic Encephalomyelitis. It does not seem that they did not ask themselves, whether these were mixed epidemics of two or more different viruses and what part resulted from the infective nature of polio and other entero-viruses or the autoimmune injuries created by these viruses, or both.

*I can only assume that until 1955 and the introduction of effective polio-immunization; all polio epidemics were **mixed epidemics** of various polio and entero-viruses. It may have been the multiplicity of entero-viruses that caused various disease states including Paralytic Polios, Myalgic Encephalomyelitis, Guillain Barre Syndrome or different patient’s reactions to the same virus. The only other reasonable possibility is these historical epidemic patients were victims of both the deadly infectious aspects of entero-viruses and at times, a devastating autoimmune reaction to these same viruses or both possibilities. Whether it was infectious, autoimmune or both, these epidemic entero-viruses attacked both the individual’s nervous systems and their vascular systems. Ultimately, in the worse cases these infections and autoimmune reactions disorganized the entire human condition, at times, these viruses killed.*

Although other viruses and organisms can do the same thing, by far the multiplicity of injuries were probably due to enteroviruses. Curiously, we owe much of our lack of knowledge today, of both Myalgic Encephalomyelitis and Guillain Barre Syndrome, which occurred simultaneously to the incredible successes of Jonas Salk and Albert Sabin immunizations. After their incredible successes, thinking the problem was now solved, tens of thousands of enteroviral researchers were let go, fired or sent into other disciplines and our knowledge of enteroviral pathology quickly withered on the vine. Today, in 2015, with the resurgence of paralytic disease and deaths due to various enterovirus, the funders of medical science may once again look to a prevention and cure to Myalgic Encephalomyelitis.”

Amsterdam Conference: Byron Hyde MD, September 2015

In the 1895 and then the massive 1905 Stockholm polio epidemic involving 1031 injured or dying patients, Dr. Ivar Wickman noted several different injuries that included:

- 1. Lethal and paralytic spinal and*
- 2. Bulbar poliomyelitis,*
- 3. Guillain-Barre Syndrome and two another injuries he called,*
- 4. The encephalitic &*
- 5. The polyneuritic forms of polio.*

The latter, groups 4 & 5 were not followed up, possibly since no lethality is mentioned to have occurred. However Wickman does give a very convincing list of symptoms and findings that sounds identical to Myalgic Encephalomyelitis. His description became more consistent with Myalgic Encephalomyelitis following the 1905 epidemic.

Wickman also noted injuries in this epidemic included the vasculature: "the capillaries are often greatly distended and tortuous"; "the blood vessels are ... dilated and congested." This vascular aspect is still true of Myalgic Encephalomyelitis and was noted by Dr Eric Ryll in the 1975 Mercy San Juan Hospital epidemic in Sacramento. These vascular difficulties have been consistently documented in my M.E. patients.

Thermogram



Healthy Father

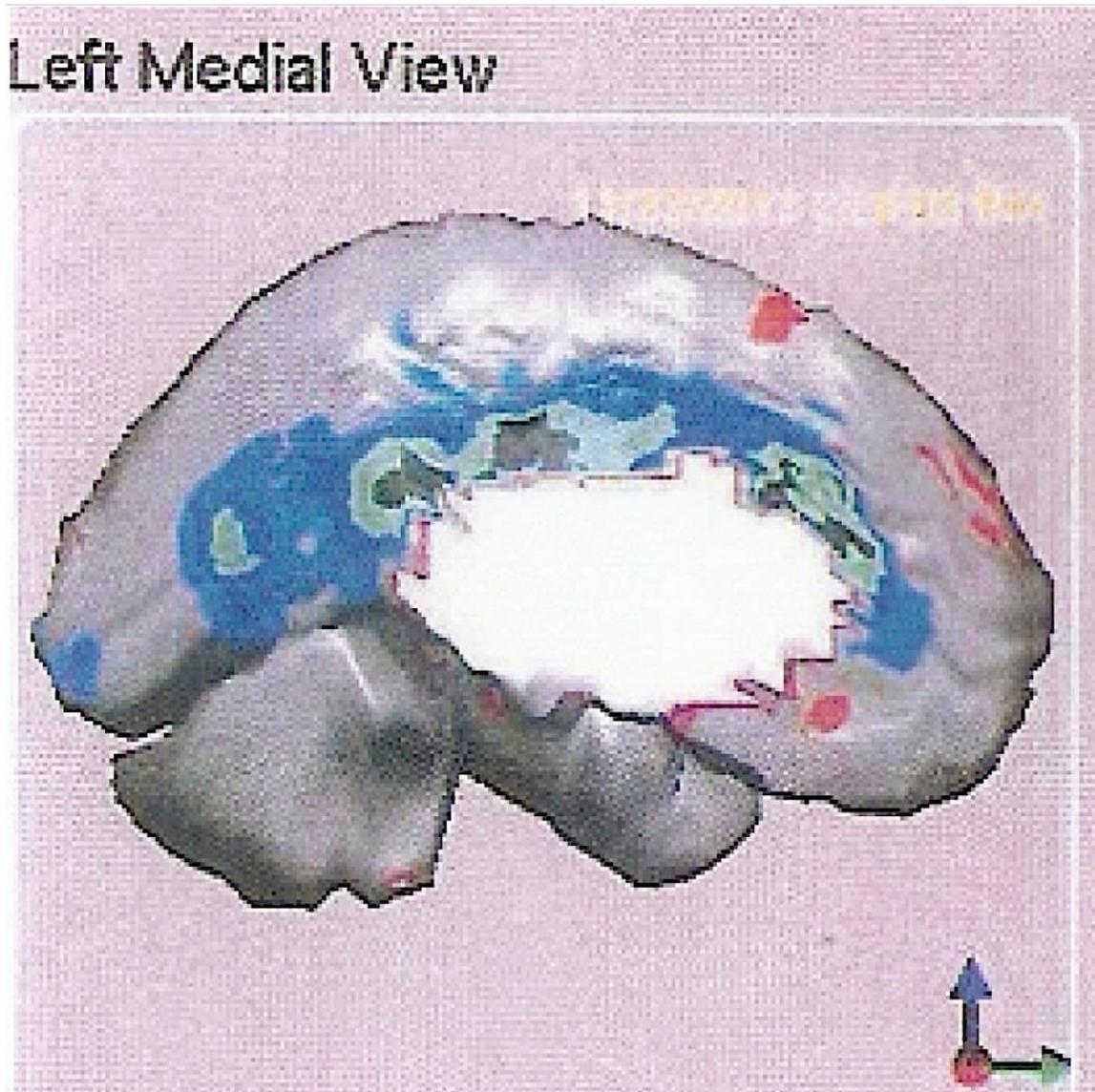
Daughter with severe

RAYNAUD'S PHENOMENA: is typical in many M.E. patients with vasoconstriction. This causes blanching, cold and pain of the extremities. Many M.E. patients so injured are unable to adjust for changes in ambient temperature. This girl has minimal circulation in her fingers, hands and arms. This is due to an injury of a particular area of the central nervous system. Temperature is controlled by the preoptic area of the anterior hypothalamus, which in turn is part of the limbic system and in particular the anterior cingulate. I have never seen a brain map of a Myalgic Encephalitic patient where the limbic system is not injured. The limbic system is always injured and gives rise to many of the difficulties of M.E. patients. The degree of limbic injury varies greatly between patients.

Any patient with poor temperature control has probably injuries either to this area of the brain or the thyroid functions or both. In thyroid function testing it is not sufficient

to only consider TSH hormone, thyroid-stimulating hormone. TSH is almost always normal in major changes of the thyroid and its function.

The following brain map of a young 39-year-old teacher who fell seriously and chronically ill with Myalgic Encephalomyelitis. This demonstrates both anterior and posterior cingulate (limbic system) hypo-vascularization. (Significant decreased circulation.) This is a medial brain view, as if the brain was sliced through, separating the brain into two pieces, left and right.



Dr Wickman also quotes Harbitz and Scheel who state in cerebral cases, "the Sylvian fossa and the central ganglia are more intensely inflamed." It is these same areas, seen on SPECT brain scans in M.E. patients, which tend to be most injured. These are the

brain areas in large or smaller part, responsible for memory, intellectual abilities, and general coordination of all body functions. Although injuries are variable, this area is always injured in brain maps of M.E. patients.

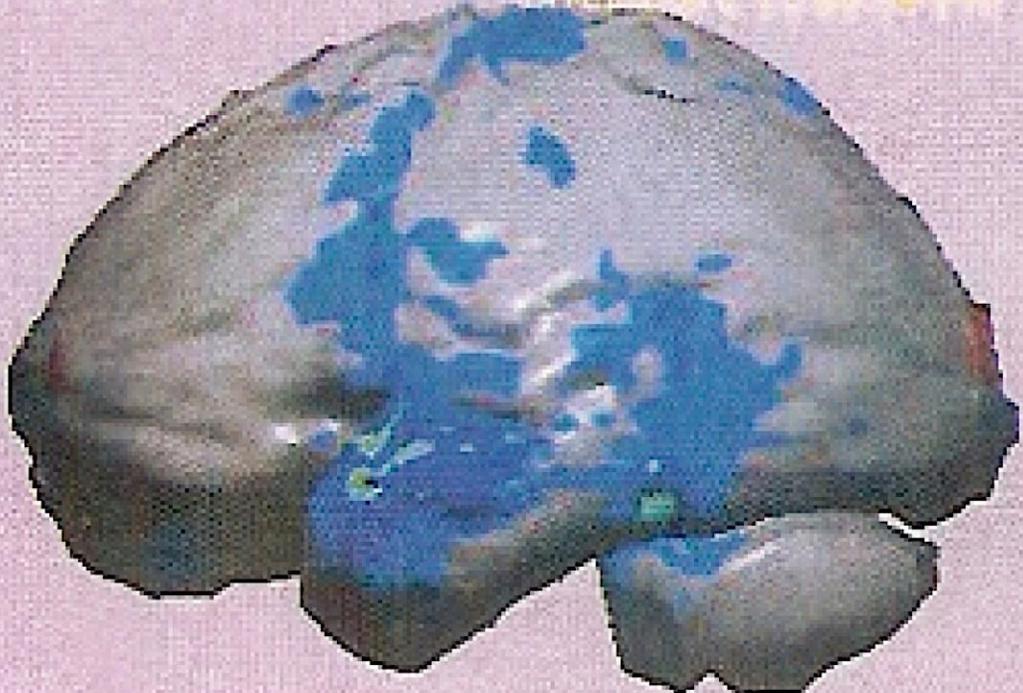
This is the external view of the same teacher's brain (left cerebral cortex) demonstrating major dysfunction of the entire anterior temporal lobe, the motor cortex and both the Brocha's area and Wernicke's area in the posterior temporal lobe. The anterior part of the temporal lobe does not contain memory but is essential for memory retrieval. Invariably when this area of the cortex is physiologically injured, so is the cingulate area of the limbic system. The posterior frontal lobe shown here is also injured and that is the area for the initiation of voluntary movement, often called the motor cortex Brocha's area controls speech production and when this is seriously injured they have difficulty forming words. In this patient, only Broca's area 44 is injured and Broca 45, immediately anterior has been spared. Wernicke's area (Brodmann 22) is the area traditionally thought of as controlling the understanding of speech. Many acutely ill patients cannot follow television or radio programs because of damage to this area. Such was the case in this teacher.

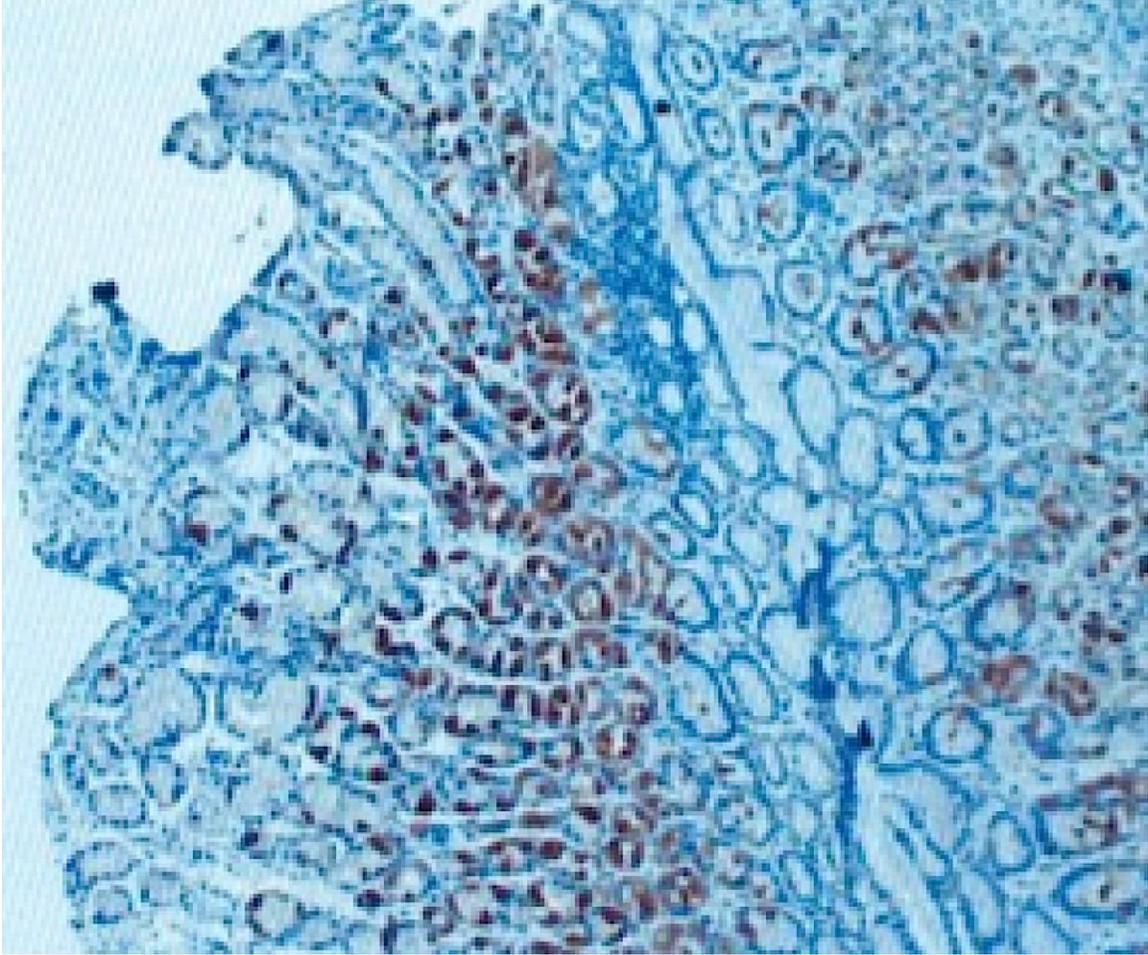
It is of interest that Dr. Ivar Wickman developed dysphonia; this may have represented a similar area injury.

Left View

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Executive Function: The ability to plan, organize, and execute tasks. It involves working memory, attention, and problem-solving skills.





Evidence of chronic enterovirus capsid protein in the gastric mucosa of a typical M.E. infected patient.

Amsterdam Conference: Byron Hyde MD, September 2015

Dr. Ivar Wickman's report on the 1905 Stockholm area epidemic, released in 1907 in Swedish and in English translation in 1913, noted one of the major differences between paralytic spinal and bulbar poliomyelitis, and what he called Superior or Medin's polio, (what we today call Myalgic Encephalomyelitis, M.E.) is that paralytic poliomyelitis attacks the brainstem and the spinal cord and anterior horn cells, whereas Superior or what we call M.E. attacks the microvascular of the cerebral cortex. The only point to

resolve is whether M.E. is due to the infectious nature of various enteroviruses, or the autoimmune injuries provoked by the same family of enteroviruses.

Amsterdam Conference: Byron Hyde MD, September 2015

"The history of poliomyelitis is a history of errors." Professor Emeritus, Dr. Hans Eggers of Institut für Virologie der Universität zu Köln: 1999

Chapter One

To Understand Myalgic Encephalomyelitis, it is essential to begin with the history of at least four principal epidemics. So I will start this paper with a brief history of each of four of the more than 60 such epidemics:

1934 California Epidemic,
1947-1949: Akureri, Iceland Epidemic,
1955-56: Royal Free Hospital and Cumberland Epidemics,
1984: North America Pandemic, Lake Tahoe, Carolinas, Canada.

The 1934 Los Angeles and California Epidemic

It is generally believed that the first scientifically documented incidence of **Epidemic Myalgic Encephalomyelitis** occurred in a mixed large epidemic of paralytic poliomyelitis in California in 1934 and Myalgic Encephalomyelitis. However, as a polio epidemic it was unusual in several ways but particularly in the low rate of paralysis and deaths. In the few autopsy cases, damage to the anterior horn cells and the vascular system were noted. Poliovirus was recovered in autopsy material and transferred to monkeys, which in turn were paralyzed. But many, possibly a majority of the cases, were more consistent with Myalgic Encephalomyelitis. It is estimated that approximately 100,000 patients fell ill from San Francisco to San Diego, a major city just above the Mexican border. What ever this epidemic was created both panic in the public and a controversy in the medical community that remains today.

Convalescent Serum: The first failed polio immunization: Medical publication interest however centred around the staff of the Los Angeles County General Hospital when 192 physicians and hospital health care workers fell ill following an immunization of what was thought to have been an injection of **sterile** immune **convalescent serum** taken from what was believed to have been recovered polio patients. It was never resolved whether their illness was caused by the

immunization or contact with the thousands of patients who appeared at the doors of the hospital in 1934. Battle lines drew up in the medical community with some physicians such as Gilliam, insisting it was a form of polio. The small group of physicians sent from Yale, thought otherwise, and although it is not said in words, if you read their correspondence, one obtains the opinion that the men from Yale felt this epidemic was a joke and was more likely hysteria. However, the patients did not get better. **What is the likely truth?** The California – Los Angeles epidemic was probably a typical mixed paralytic poliomyelitis / Myalgic Encephalomyelitis epidemic

What we do know is that James P. Leake, Medical Director of the United States Public Health Service tried to prevent publication of Alexander Gilliam's report on this epidemic. (*A. Gilliam related this to Dr. A. Shelokov Sr., then, Shelokov was the foremost M.E. authority in the United States.*) The County of Los Angeles Authorities and the insurance industry did their best to prevent any settlement of the claim for damages. However, by Gilliam removing certain unknown items from the report, and by threatening to go public if the report was not published, it was eventually published in 1938 in its revised form as Public Health Bulletin No. 240.

Nevertheless, the battle continued in the courts until in 1938, four years after the epidemic, the 192 injured hospital staff, received a financial settlement sufficient for each to have purchased two significant houses in Hollywood California. (*This is equivalent to several million dollars each.*) The 192 staff members were also obliged to not discuss this settlement with anyone. However, when neurologist, Dr. Alberto Marinacci took over the care of these L.A. Hospital patients, and some of the patients did discuss the settlement with him. Years after Dr. Marinacci retired, he invited me to come to see him at his home in Santa Monica, where I met his colleague who had continued to attend these patients and it was then I examined two of the remaining hospital patients. It was very obvious, 55 years later in 1989; these patients were still disabled with classical chronic Myalgic Encephalomyelitis.

In the Public Health Bulletin 240, Gilliam labelled the report as, *an Epidemic, diagnosed as Poliomyelitis*. Dr Leake however states, *“the absence of typical histories of poliomyelitis is striking”*. *So began the fight against Myalgic Encephalomyelitis*.

One logical conclusion is the 1934 epidemic was a mixed epidemic of Paralytic Poliomyelitis and Myalgic Encephalomyelitis. This was the later view of Dr. Gilliam when he discussed the 1934 epidemic with Dr Shelokov. They both presumed it was a mixed epidemic. Neither knew of a previous similar reported epidemic.

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1947-1949: Akureri, Iceland Epidemic

Even serious epidemics tend to be missed when they occur in the general public, with individual cases, sporadically seen by individual busy physicians who tend not to communicate with each other. Also, not just in physicians but in all persons, if you do not aware of something, often you do not see it. For instance, if you awake at 5 – 6 am you will see Nightingales in our garden. My wife had seen these birds but had not recognized them. She never knew we had nightingales. So it is with disease.

Serious epidemics tend to be observed when either the public are alarmed as in Myalgic Encephalomyelitis or when the epidemics occur in hospitals, school residences, in busses, aircraft and in military barracks. Historically, almost all Myalgic Encephalomyelitis Epidemics have been documented in institutions or enclosed spaces as noted.

Like the Cumberland epidemic to follow, this epidemic began in the boarding school in Akureri, Iceland, was initially diagnosed as poliomyelitis and rapidly spread to the adult public in the community. Various reports state 465-488 persons fell ill, but as in Los Angeles the rate of permanent paralysis was relatively low and the death rate was only 0.8%. Dr Björn Sigurðsson, MD, & PhD Princeton U., and director of the Institute of Experimental Pathology investigated this epidemic. Unfortunately, as in the Los Angeles, and the Royal Free Epidemics, this came to be considered by some to be mass hysteria. The patients became ashamed of being injured and tended to hide their illness.

Of significant interest, Sigurdsson, reports that a major epidemic of paralytic poliomyelitis struck all of Iceland a few years later except for Akureyri and the surrounding area. Serum tests on the Akureyri patients demonstrated they had been infected with this poliovirus but no one in the Akureyri area where the 1947-1949 epidemic struck, fell ill. What ever the virus was that caused the 1947-1949 Akureyri epidemic, it was close enough to the poliovirus that it gave immunity to the area.

Sigurðsson, with funds through Princeton, made several attempts to grow the virus and failed. However in his memoirs, a collection of his records published following his early death, he mentions he discover evidence of enteroviral infection as a cause of this epidemic.

The Akureyri epidemic was important for one other feature, although few young children fell ill during this epidemic, three children did fall ill and over the next few months developed Parkinson's Disease, an injury to the basal ganglia, and died.

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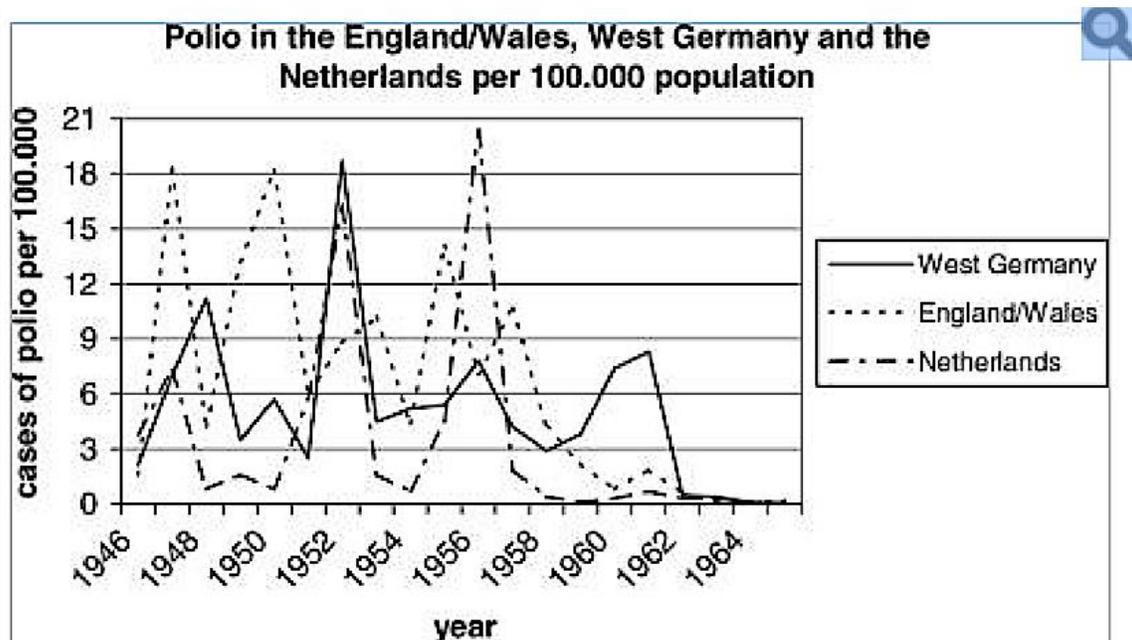
The Royal Free Hospitals Epidemic

& The 1955-1956 English Pan-Epidemic

It is generally believed that the name, **Myalgic Encephalomyelitis**, was first created following the 1955-1956 epidemic that in London, first appeared in Hampstead and then spread to the Royal Free Hospitals and nurses residences in central London. The initial name given was **Benign Myalgic Encephalomyelitis** since so few died or were paralyzed.

Unbeknownst to Dr. Ramsay, one of the senior physicians in Infectious Diseases at The Royal Free Hospital and his colleagues, this was a simultaneous pan-epidemic covering at least the north of England. It occurred simultaneously in Dalston, Cumberland in northwest England. There, as in Akureri, it began in a boy's boarding school and rapidly spread into the adult community. During the same time period, the disease also struck in Northumberland in the area around Newcastle-upon-Tyne, where according to Dr John Richardson, who knew nothing of the epidemics in Cumberland, 60 miles to the west of him or the epidemic in London, he reputedly also named the disease Myalgic Encephalomyelitis. In London this epidemic occurred at the same time as increased cases of paralytic poliomyelitis also became evident. From the following chart, it is obvious that there was a dramatic upsurge of polio in all of England in 1955-1956. Once again, it appears to have been a mixed epidemic of poliomyelitis and what today we call Myalgic Encephalomyelitis.

See graph below: Ulrike Lindner, (University of Koln) & Stuart Blume, Cambridge Med His. 2006, Oct



Back in the Royal Free Hospital, this contagious illness soon turned out to be very much less than benign, the epidemic disease was then renamed, possibly by Dr. A. Melvin Ramsay, Consultant Physician to the Infectious Diseases Department of the Royal Free Hospitals in London to **Myalgic Encephalomyelitis**. The public press however called it Royal Free Disease.

The Royal Free Epidemic was remarkably similar to the Los Angeles Epidemic, except at the Royal Free, they did not know the L.A epidemic had ever existed. Similarly to the Los Angeles epidemic, the Royal Free epidemic, began in the community and rapidly spread to the physicians, nurses and health care workers of the Royal Free Hospitals and Nurses residence in central London. Dr. Ramsay was not aware at the time of the simultaneous Cumberland and the Newcastle epidemics.

The similarities of the two epidemics are striking. Both of these epidemics occurred simultaneously during paralytic poliomyelitis epidemics where few patients were paralyzed and fewer patients than usual died. Also, there was another curiosity in the both epidemics, instead of the primary victims being children as was most common in paralytic polio epidemics, it was the adults who fell ill in large number.

The same thing happened in Cumberland, although the epidemic began among children in a residential school, when it broke out into the community, it mainly struck down adults. The only deaths recorded were also adults in Cumberland.

The Mixed Nature of the Epidemics: What should have attracted wider attention in both epidemics is the mixed nature of both epidemics, where both paralytic Poliomyelitis and Myalgic Encephalomyelitis occurred at the same time. Like the 1934 epidemic, this 1955 epidemic occurred at the same time the Salk polio immunization was released in the United States. An event that not only changed the world but generally also prevented the appearance of any new Mixed Epidemics of Paralytic Poliomyelitis and Myalgic Encephalomyelitis.

The investigation of the Royal Free epidemic was effectively crushed by an Oxford PhD student who visited the hospital for 3 hours one day, never saw or interviewed a single patient and then wrote a thesis calling it mass hysteria. His PhD proctor just happened to be a lawyer who I understand did insurance industry cases in court. Almost immediately upon publication of the thesis, this obscure Oxford thesis found its way to the front page of Time Magazine. It is enough to make one paranoid. When I interviewed him at his home years later, I asked to see his investigational reports. He said he had none. I asked him why he wrote this thesis. He said he had help and it was easy. Who helped you, I asked. He wouldn't say. There is no way I can tell why this epidemic and the others represent such an apparent threat. Or is it, the medical world needs a whipping boy?

1984-1992: North America Pandemic

In August 1984, a North American Epidemic occurred and was reported in Incline Village, by Daniel Peterson and Paul Cheney. The epidemic was rapidly hidden and immediately became a cause for major anxiety, probably as much among the insurance industry as it was to the local physicians and the public. It was also reported in the Carolinas by Dr Grufferman and by myself in both Montreal and in Ottawa in Canada. None of the three groups were aware of the other three epidemic pockets of the same disease.

However the fight concerning the 1984 epidemic in Incline Village, Lake Tahoe Nevada was most curious. I was told that the town councillors of Incline Village threatened to vote their congressman out of office if he did not cancel the Centres for Disease Control investigation. The epidemic was bad for business. Incline Village was a holiday resort town covered with golf courses and toys for the wealthy of California and Nevada. Reputedly the Federal Congressman, concerned for his job, put a stop to the investigation. I was also told, the researchers that came to investigate from The Federal Government, Centres for Disease Control, and then played golf. Their technicians took blood samples. They then retired back to Marietta Georgia to the CDC without any federal follow-up. The investigation, which would have provided us with so much useful information, was largely halted.

The two major on-site physicians attempting to treat the schoolgirls where the epidemic started in August 2014, Dr. Paul Cheney and Daniel Peterson were also sidelined. They attempted to publish learned papers on this epidemic with **The New England Journal of Medicine**, one of the world's best medical journals. Their paper went through a peer review process with a secret three-member committee. At least one of those secret members was Dr Steven Straus of NIH, who was largely responsible for funding the Lake Tahoe investigation that I was told, vetoed the paper. This information was given to me by a good friend, one of the other secret peer review committee members.

Straus was also the head of the research funding committee of the NIH. It is my understanding that no significant NIH funds, perhaps no NIH funds were ever given to those local physicians who were attempting to investigate this epidemic. This is a tragedy of democratic system.

One of the Lake Tahoe physicians examining the patients then developed a cardiac disease necessitating a heart transplant. Cardiomyopathy and early death is the result of a typical enteroviral infection. One thing is certain; the Lake Tahoe Epidemic occurred as a typical late summer enteroviral epidemic, causing a typical enteroviral illness, Myalgic Encephalomyelitis.

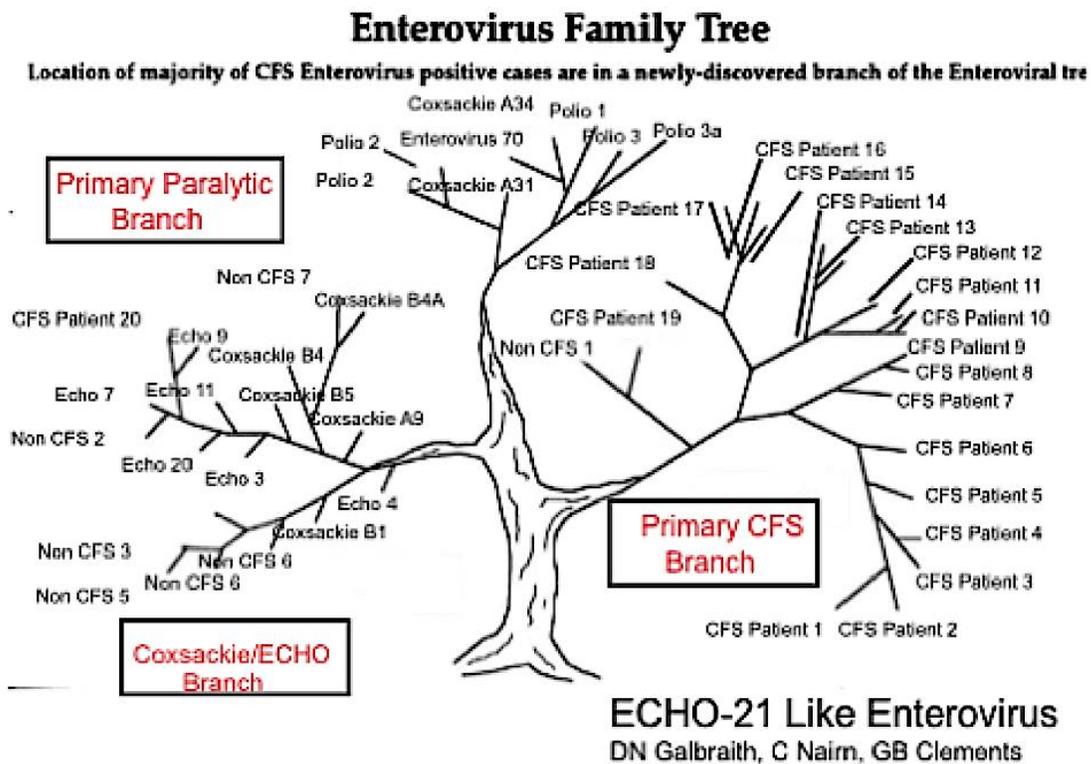
This epidemic covered all of North America and involved not only the Incline Village high school students in a girl's basketball tournament, travelling by bus in late August in the Lake Tahoe area. Like the Cumberland and Akureyri epidemics, it soon spread into the teachers and adults of the community.

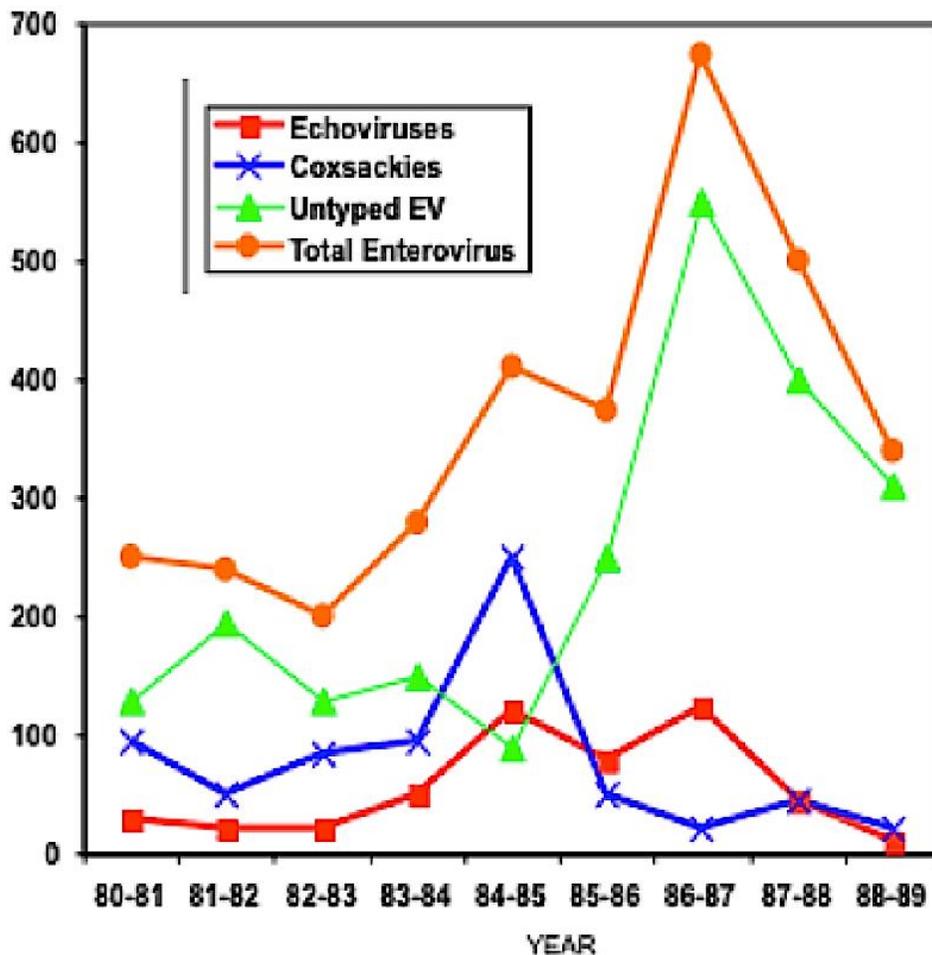
The same epidemic also ripped through a North Carolina symphony orchestra. They also were travelling closely packed and travelling by bus giving statewide concerts. It was reported by Dr. Seymour Grufferman at the University of Pittsburgh School of Medicine. I understand, no funds were given for follow-up research either by Strauss's NIH funding committee.

Also occurring in Montreal, where an epidemic among physicians and other health care workers occurred in late August. It also hit my patients in Ottawa in an epidemic that extended from August 1984 and continued through decreasingly until the early 1990s.

In Ottawa, we recovered entero-viruses as a cause of this epidemic, but not one enterovirus, but several. This work was done at Ruckhill Hospital in Glasgow. See their results below on a tree graph.

The Ontario Government also recovered enteroviruses during this period. See their charts below.





Work of senior virologist, Dr. B. McLaughlin of the Ontario Ministry of Health in Toronto, covering 700 serum samples from across Ontario.

The tree chart prepared by Rds. DN Galbraith, C Nairn and GB Clements from the blood samples of 100 of our Ottawa and Ontario based patients demonstrates this was a multi-enteroviral epidemic all occurring at roughly the same time. The important fact is that this epidemic occurred simultaneously in Lake Tahoe, in the Carolinas, in Montreal and in Ottawa all starting at the same time, August 2014.

What can we say about these four epidemics of Myalgic Encephalomyelitis?

1. Each epidemic began in late Summer or early Autumn,
2. Four of them began in institutions, schools or hospitals where they would be month visible, the Montreal and Ottawa groups were first observed in a hospital party and in a local community,

3. Government funding avoided those physicians who knew most about the first hand aspects of their epidemics,
4. No serious research was ever given to any of the individual physicians who were most knowledgeable of the patient base,
5. All of the four epidemics mentioned were consistent with an enteroviral infection,
6. No long-term follow-up publications of any merit were produced on any of these epidemics, some due to lack of government funds, some due to the shaming of the concerned physicians.
7. Each of the four, the LA, the Akureyri, the Royal Free, and the Incline Village epidemics and their local investigators were branded as hysteria and this was taken up by the press.

I must say, I have always found it curious that hysteria has an incubation period of 3-5 days. I have always found it curious that hysteria begins most often in the north temperate globe, in the late summer and early autumn. I have always found it most curious, that hysteria, certainly among adults, occurs most frequently among health care workers and those associated with the teaching professions.

A Complex Question: I have also, always wondered if it is strictly coincidental that those most frequently observed as falling ill with Myalgic Encephalomyelitis are also (a) those adults most frequently **immunized** or is it (b) these are also the individuals most frequently in contact with **infectious disease** or (c) those most frequently involved with long hours of **physically and intellectually exhausting professions** and activities.

Chapter Two

The First Major Epidemic: The first documented history of Myalgic Encephalomyelitis **may have** started with a small epidemic in Stockholm, Sweden in 1895 where only 21 persons fell ill with paralytic poliomyelitis and possibly something else.

However, the first precisely documented cases of Myalgic Encephalomyelitis, although by another name, first occurred in the aftermath of a much larger mixed epidemic that occurred in the late summer and early autumn of 1905, also in the Stockholm area. On this occasion, 1231 patients fell ill, many died, many had Landry's Polio (Lou Gheric disease), what today is called Guillain Barre syndrome, but there was also something else that appears to be Myalgic Encephalomyelitis.

I believe the reader cannot understand Myalgic Encephalomyelitis without first understanding the history of epidemic poliomyelitis, what was then in 1881, a totally new disease, Epidemic Paralytic Poliomyelitis. The important word here is *Epidemic*.

Prior to the introduction of the Salk polio immunization in 1955, polio epidemics appear to have been closely allied with the history of Myalgic Encephalomyelitis.

We are accustomed to believe paralytic polio was known since 1400 BC in Egyptian history. However, the wall painting often shown could easily have been caused by an arrow or a spinal injury. Up until the introductions of mumps and diphtheria immunization, paralytic injuries resembling paralytic poliomyelitis occurred routinely with mumps, diphtheria and syphilis as well as other infections. Facial paralysis was more common in mumps but both mumps and diphtheria were associated with diaphragmatic and peripheral paralysis and death. Paralysis of arms and legs was common in children for centuries but they were almost always isolated events, never seen in significant clusters or epidemics. Paralytic disease was relatively rare prior to 1881.



An Egyptian stele thought to represent a Polio victim. 18th Dynasty (1403 - 1365 BC).

The 1881 Epidemic: Although there may have been other incidences in the United States or elsewhere, the first well-documented Epidemic of Paralytic Poliomyelitis was recorded by a Swedish physician, Dr. Bergenholtz, in an epidemic in 1881. This novel epidemic occurred in a single room schoolhouse straddling the border of Sweden and Norway. It involved 23 students and teachers and was recorded in the Swedish medical archives. (18 in Sweden & 5 in Norway) Neither the Scandinavian nor the world's physicians appeared to notice.

This epidemic had no apparent association with Myalgic Encephalomyelitis.

The 1890 Epidemic: Then a second epidemic of paralytic poliomyelitis occurred in 1890 in Stockholm involving 44 patients, reported and well described by Dr. Oscar Karl Medin (1847-1928). He believed this epidemic was a non-contagious disease (*not spread from person-to-person*) and caused by *miasma*, or toxic night air. This was 1890 and although it was hard to believe, few *responsible* and *serious* physicians believed in the new fangled infectious disease theories of Koch and Pasteur.

From his descriptions, this epidemic investigated by Medin, also had no apparent association with Myalgic Encephalomyelitis.

First Mention? “A Painful Encephalomyelitis”

The 1895 Epidemic of Paralytic Poliomyelitis and Possibly Myalgic Encephalomyelitis: Then the world changed, another smaller epidemic reputedly of poliomyelitis involving only 21 patients occurred in Stockholm in 1895. This epidemic was investigated by Medin's 23-year old pupil, Otto Ivar Wickman. When this third small epidemic struck, epidemic paralytic poliomyelitis was still relatively unknown and except for these three epidemics I have mentioned, they were so rare that when similar epidemics started to bubble up in other countries, many world physicians and the press referred to what we call polio as *The Swedish Disease*. However, Dr Ivar Wickman believed in the infectious theories of Dr. Robert Koch in German and Dr. Pasteur in France. This unacceptable idea that the Swedish epidemics were of contagious origin did not appear to make him a friend of the head of the paediatric department of the university, Dr. Oscar Medin. It is at this point, in the descriptions of Dr. Wickman that we first hear of epidemic Myalgic Encephalomyelitis.

In describing the 1895 polio epidemic in Stockholm, Wickman had no preconceived notions of what he was seeing. Medin, the head of the department, insisted this was caused by *miasma*, toxic night air, a theory believed by most physicians at the time and presumably by Dr Medin's board. Dr. Wickman, Medin's student believed in the unacceptable infectious theories of disease of Koch and Pasteur. Wickman's radical views were not to help him with advancement in the University.

However, Wickman had several other advantages and it helped than no one previously had ever examined in detail an epidemic of paralytic poliomyelitis. These, never-before-seen paralytic polio epidemics were not only something startling new, there were also, no preconceived ideas of how to frame the investigation and what an investigator should see. This was a great advantage to Wickman.

1. Wickman was obviously brilliant, young, and,
2. He obviously didn't fill the role of sycophant, of Medin, his boss.
3. Unlike Medin he believed in the infectious theory of disease.
4. He took good histories and carefully examined each of the 21 patients,
5. Most important, he had no preconceived ideas of what he was to see.
6. He was examining something new with wide-open new eyes.

When we examine any situation, any subject or specimen, reconceived ideas alter what any of us tend to perceive as reality. All physicians are exhaustively trained to absorb medical textbooks, historically known "facts", (which very infrequently are in error).

In 1895, Ivar Wickman examined and reviewed the patients from the three Swedish epidemics and in particular he examined the 21 epidemic patients from 1895. His descriptions of the patients in this latter epidemic are still valid today. The injuries he found in this epidemic included:

1. Paralytic spinal polio-myelitis: these patients tended to be without pain,
2. Paralytic bulbar polio-myelitis, where death was a common outcome,
3. A painful (Poly-Neuritic) **Encephalomyelitis, (Wickman's description),**
4. **Wickman** was also the first to note the seasonal incidence of these epidemics, that they occurred in late summer and early autumn,
5. Wickman was also first to note a number of abortive and non-paralytic cases.

At this time Dr. Wickman did not give great details on what he meant by this painful, poly-neuritic form, but it could be translated into a painful encephalomyelitis. Looking at the term, it is about as close as one can get to the expression, Myalgic Encephalomyelitis. Of course it is not a certain diagnosis but it is beginning to hint at M.E. Wickman's view becomes much more interesting with his investigation of the next, the third Stockholm epidemic that struck both the Stockholm, Sweden area and Oslo, Norway area simultaneously in the late summer of 1905.

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Professor Emeritus, Dr. Hans Eggers of Institut für Virologie der Universität zu Köln correctly notes: "*the history of poliomyelitis is a history of errors.*" Up to 1881 paralytic polio had never been observed in epidemic in form; there were isolated

cases only. These isolated cases of paralytic disease were, as previously mentioned, and possibly correctly attributed to mumps, syphilis, diphtheria and bacterial meningitis. Also, as mentioned, their mode of transmission was generally believed by physicians and the public to be due to *miasma*, (toxic night air from decaying organic matter). There was another prevalent concept of disease.

(For interesting back ground on polio see Dr Eggers talk: [Milestones in Early Poliomyelitis Research \(1840 to 1949\)](https://www.jvi.asm.org/content/73/6/4533.full) [jvi.asm.org/content/73/6/4533.full](https://www.jvi.asm.org/content/73/6/4533.full))

The fault dear Brutus, is not in our stars: As mad as it may sound now, what today we know as infectious disease was believed to come from Miasma or the **influence** of the stars, an invisible liquid from the heavens. This was the origin of the term **influenza**. These two highly accepted theories of disease-spread, Miasma and the stars, caused people to shut up their windows at night to protect themselves from these toxic night air substances. Miasma caused Florence Nightingale, a strong believer in this theory, to develop and promote conditions of proper sanitation, to get rid of sewage that caused this decaying organic matter that in turn caused miasma. The decrease in death rate following Nightingale's practices supported her and the public's belief of miasma as the cause of disease. It is hard to imagine today that these two theories, believed by the public and physicians alike, continued well into the early 20th century. (*See also: Susan Sontag's book: Illness as Metaphor 1978.*)

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The 1905 Stockholm Epidemic: 1031 causalities

This was the first of the great polio epidemics that soon put the world into a state of understandable panic. It was also the beginning of the end for the **miasma theory of disease** so firmly held by Dr. Medin. In the 1905 epidemic a total of 1031 patients fell ill. Many, but not all were paralyzed, many died, but many had other associated illnesses, similar to what he saw in the 1895 epidemic. Some of the paralytic cases were different from what is called polio today, but several other occurrences were also noted and I will describe them as Wickman set it down. His findings reinforce the concept that polio epidemics were never just poliovirus epidemics alone. I believe these early epidemics were mixed epidemics of different enteroviruses or autoimmune reactions to one or more enteroviruses. No other concepts can explain Wickman's findings.

Paralytic Polio Epidemics Are Typical Examples of Mass Hysteria:

Paralytic Poliomyelitis is a typical case of mass hysteria: It should be of great interest to those readers chronically injured by Myalgic Encephalomyelitis, and those physicians who study this disease to remember Dr. Dejerine's words. In 1905, Dejerine was rightfully considered to be one of France's great neurologists; he was also a believer in psychotherapy. In 1905, on hearing of this unparalleled massive

Swedish epidemic, Joseph Jules Dejerine, (1849-1917) described those who manifested paralysis in the 1905 epidemic as a classical epidemic of mass hysteria.

Unfortunately Dejerine's reputed mass hysteria was now beginning to spread over all of Europe, North America and the world. The number paralyzed or dying continued to rise into the thousands and tens of thousands every year, until millions were stricken and died. This too began to change the perception of what a polio epidemic actually constituted.

Although the 1905 epidemic is referred to as the Stockholm Epidemic, it actually occurred in an area of more than 50 km surrounding Stockholm. Many of these injured patients were relatively inaccessible, tucked away in local hospitals or in country homes. Ivar Wickman, travelling in horse and buggy, set out to interview and examine every one of the 1031 patients and their homes despite the fact they were significantly separated. Dr Egger's notes: *"He tracked by meticulous investigations the spread of infections in small parishes, mostly living in isolated widely dispersed homes.* He noted the common source of the epidemic as Traestena's school." Professor Eggers notes: *In Traestena, 26 persons out of the town of 500 had significant paralysis but the large percentage of the townspeople had abortive and nonparalytic cases."* With this view, Wickman posed a startling new view of disease and its spread. Apparently healthy people can be the main conduit of disease.

As mentioned earlier, Wickman was doubly fortunate in that he had no preconceived view of what to look for, what to describe or what he would find. No one had previously performed such a detailed patient and patient environment examination and published it.

Again, in 1911, Ivar Wickman announced several aspects of this epidemic disease that was, in part to change the world and, most unfortunately for Myalgic Encephalomyelitis patients, was totally forgotten to medical science. Wickman noted that these diseases, polio was a biphasic disease. Some of his findings are as follows:

1. The infectious agent causing this epidemic had a definite incubation period: The time passed from first contact - to first symptoms of initial illness, was a short 3-4 days, (BH: This suggests one infectious organism or a family of very similar infectious organisms. This is consistent with enteroviral infections, a virus not yet known at that point.)
2. The time from first contact, to the first symptoms of major disease, was 6-8 days, then paralysis or other chronic signs and symptoms, if any, were to begin to appear. *(Today these onset figures for the second phase are most frequently estimated to vary from 7-21 days until paralytic features emerge. It is impossible to state whether the 21 days is due to well carriers infecting a new patient.)* The paralytic polio death rate is estimated to be 2-5% for children and

15-30% for adults. The death rate for adult bulbar poliomyelitis patients is estimated to be 25-75%).)

3. **Abortive Poliomyelitis:** In the majority of patients, major symptoms of illness did not appear, not then, not ever. This group, Wickman called abortive poliomyelitis. They and the well carriers made up the majority of the patients. *(No previous physician had ever made such a startling discovery of how infectious disease might be spread.)*
4. **Well-Carriers:** In a significant majority, no first symptoms or secondary symptoms appeared, yet from his interviews and examinations, it became obvious that these totally, apparently well persons, not only carried the virus but unknowingly transported the disease to many of the next victims, he called them well-carriers,
5. It was obvious that there was an organism, an infectious microbe that caused the disease and to which the majority of people appeared either immune, or had some natural resistance. These were both the abortive and the well carriers.
6. Wickman demonstrated with his maps that this was an infectious illness, and it was transported from the school to a person's house and from there to another house and the route of this disease could be shown by following the local road systems, from one victims house to the next,
7. Dr Ivar Wickman demonstrated by his maps that the various associated illnesses were caused by well carriers, more than obviously infected persons.
8. Wickman went to each house where a patient had resided and examined and interviewed all of the cohorts, even those who had no obvious signs of illness.

What Features Did Wickman Describe As Occurring in the 1905 Stockholm Epidemic?

Wickman described six different types of associated illnesses all of which occurred with this epidemic. He referred to the various illnesses associated with this epidemic as:

- A. Spinal Paralytic Poliomyelitis,
- B. Bulbar or Pontine Poliomyelitis,
- C. **Superior, or Medin Poliomyelitis,**
- D. Landry's Poliomyelitis, (which came to be called ascending or Guillain-Barre Syndrome)

Ivar Wickman, was an unsung medical hero, a genius who revolutionized western medicine by noting the majority of the individuals he investigated totally recovered after a minor illness, or were not ill at all, but could carry and transmit the infectious disease to others, causing paralytic poliomyelitis. Today, the next two items E & F might not be noticed by the reader, but they were revolutionary and did revolutionize the theory of infectious disease.

- E. Abortive Poliomyelitis,
- F. Well Carrier Poliomyelitis, (These were patients who always remained healthy but who unknowingly, carried the infection from one patient with any of the above symptomatic diseases, to the next.)

It is estimated today, only 0.5-2% of persons infected with poliovirus develop paralytic poliomyelitis or die once infected. The majority are as stated by Wickman, above, not seriously injured. We also know that other enteroviruses, other than polio 1, 2 and 3 cause paralytic diseases resembling paralytic poliomyelitis in form and pathology. Recent cases in Canada and the USA include Enterovirus 68 and 71. In 2014, M. Lang and A Mirand et all reported a similar case in central France.

From Dr. Ivar Wickman's publication on this epidemic, it is not possible to know whether he believed one microbe caused these different forms or illness or there were several different microbes associated with one epidemic. We assume that Dr. Wickman believed the epidemic was caused by one organism but if so, this organism had several different effects on the patient, possibly depending upon the resistance of the infected host. But is this true?

It is of interest that Wickman's Type C Polio (see above) was called both **Superior and Medin Polio**, (Superior meaning: affecting the central nervous system superior to the spine and brain stem (the cerebral cortex: where memory, sleep, decision making and intellectual facilities and even the immune system are controlled: as in Myalgic Encephalomyelitis), and did not significantly or irreversibly damage the Bulbar or Pontine regions of the brain stem or the spinal cord. In other words Superior Poliomyelitis primarily affected the thinking brain, the Cerebral Hemispheres rather than the primitive brain below it. Curiously he also named this form of polio, Medin, after his medical director Karl Medin.

Wickman's Signs and Symptoms of Superior Polio

*Let us now go to the signs and symptoms of what Ivar Wickman called Superior or Medin Poliomyelitis. Any reader with disabled with Myalgic Encephalomyelitis, and any physician studying this disease will note the following as a classical example of Myalgic Encephalomyelitis. It is essential to remember, this, **the first major description of a disease resembling Myalgic Encephalomyelitis**, is a symptomatic description of an illness, which occurred during the first recorded major paralytic poliomyelitis epidemic.*

Superior or Median Poliomyelitis

(Findings resembling Myalgic Encephalomyelitis, described by Ivar Wickman occurring during the 1905 Stockholm region paralytic poliomyelitis epidemic.)

(Please note, that in no place did Wickman state that each of these signs and symptoms, that he mentions, were present in a single patient. These findings may or may not have occurred in any single patient but many of his non-paralytic polio patients obviously had these findings. In his monograph, attention is given to cases resulting in death and permanent paralysis.)

1. An incubation period of approximately 3-5 days.
2. Fleeting palsies, (a type of partial paralysis or weakness often with shaking or tremors.),
3. Pain syndromes & malaise including onset headaches & head pain,
4. Nausea & gastric symptoms,
5. Tenderness of muscles resembling influenza,
6. Cognitive or psychic dysfunction,
7. Protracted prostration, (extreme exhaustion or lack of energy or power),
8. Subnormal temperatures, (& *difficulty with cold & heat perception*),
9. Transient aphasia, (*difficulty in saying words, dysphonia*),
10. Bladder symptoms, (*interstitial cystitis, painful sex*),
11. Hypotonus, (*loss of elasticity of muscles & arteries*),
12. Paresthesias, (*abnormal sensations, tingling, burning or pricking that can be transient or chronic*)(*pins & needles, formication, a limb falling asleep*,
13. Sweats,

Note: *I have inserted the explanatory details in italics and within parenthesis. These are my additions to the notes of Dr. Wickman.*

In truth Wickman's publication was a monumental document and proved Wickman's theory that paralytic poliomyelitis was caused by a non-bacterial, infectious microbe, often passed on by well carriers. It is hard to imagine today that anyone would have doubted Wickman's theory of infectious disease causing paralytic poliomyelitis, but most physicians and the public did doubt the infectious theory of disease up to this time. This was one of the great turning points of modern medicine and it occurred in 1907 with the publication of Wickman's monograph. It is interesting, that I can find no medical journal, which at that time was ready to publish his revolutionary infectious theory concepts. I assume they do exist in Swedish or German.

Almost immediately following his publication, Wickman's infectious disease theories were substantiated.

In December 1909, Karl Landsteiner, Erwin Popper and Constantin Levaditi, using the Chamberland porcelain filter, (invented in 1884), isolated an organism smaller than any known bacteria, (Bacteria could not pass through this filter.), and injected this bacterially sterile material from the brain and spine autopsy of a recently deceased paralytic poliomyelitis child into various animals including: rabbits, guinea pigs, mice and 2 monkeys. None of the laboratory animals fell ill except one monkey. However, one monkey on day six did fall ill and on day eight it died. Autopsy demonstrated it had the classic injury to anterior horn cells that has since defined paralytic poliomyelitis.

However it was not until 1949 that John Enders, Thomas Weller and Frederick Robbins grew poliovirus for the first time. It was only with this discovery, that an immunization could be manufactured successfully.

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Major Questions We Have to Consider

So let us stop for a few minutes to consider what we are actually reviewing.

First, there are several significant unanswered questions that arise from Wickman's and Landsteiner's discoveries of polio being an infectious disease and there was an associated "virus" that caused this major paralytic disease.

Wickman and Landsteiner's discoveries were not only revolutionary but were the explosive beginning of the history of viral illness. Immediately following their discoveries, a rapid worldwide escalation of paralytic poliomyelitis epidemics and deaths began in earnest.

This all started with the massive 1905 Stockholm polio epidemic, which was obviously from Wickman's description of Polio, Guillain Barre and Myalgic Encephalomyelitis like illness, a combined epidemic.

Not counting well carriers and abortive poliomyelitis cases, according to Wickman chronic illness in this epidemic came in at least four distinct forms. (Wickman actually subdivides the clinical expressions of disease even further.) But let us start with the 4 major types of chronic diseases noted by Wickman. During this massive polio epidemic, among the 1031 cases Wickman noted:

1. Spinal Paralytic Poliomyelitis,
2. Bulbar or Pontine Poliomyelitis,
3. Superior, or Medin Poliomyelitis,
4. Landry's Poliomyelitis, (which came to be called ascending or Guillain-Barre Syndrome) (Of the 159 patients who died during the first two weeks of the

1905 epidemic, 45 conformed with the picture of Landry's disease (32 ascending, 13 descending)

If the cause of this epidemic was a single virus subtype, how could one virus cause such different diseases? Wickman tells us that:

1. All 4 forms of this epidemic disease occurred in the same epidemic,
2. We can assume, as later authors did, (at least **until 1935**), that one sub-microscopic agent, one virus was responsible for this epidemic paralytic and lethal disease.
3. That this **single virus** caused at least four major disease variants listed as:

(1) Spinal Poliomyelitis &

(2) Bulbar Poliomyelitis:

(3) Landry's Poliomyelitis, now called, Guillain-Barré Syndrome:

(4) Superior, or Medin Poliomyelitis: (What we call, Myalgic Encephalomyelitis)

It is understandable that those physicians who believed in the infectious disease theory of illness and knowing that one bacteria caused one disease would assume one virus would also cause one disease. Until 1931, no one believed that there was more than one virus causing poliomyelitis. It took over 25 years to disprove this one virus theory. That was with tens of thousands of researchers in most countries of the world and millions of dollars/euros pouring into the research funds.

Then in 1931 two Australian physicians, Frank Burnet and Jean Macnamara discovered that there were two anti-genetically different polio-viruses causing paralytic disease. They were not believed.

In America, oblivious to the above discovery, in 1935, thirty years after the 1905 Stockholm epidemic, the researchers were still stuck on the one-virus-theory of poliomyelitis, and discounting Burnet and Macnamara they developed the second attempt at an anti-polio immunization. The first was **convalescent serum** prepared from the blood of recovered polio victims and used in the 1932 Los Angeles County Hospital epidemic with disastrous results.

In 1935 Maurice Brodie at New York University and John Kolmer at Temple University in Philadelphia both developed a one-virus immunization. It was a disaster and resulted in the death and paralysis of several individuals, followed by the tragic suicide of Dr Brodie himself at the age of 36.

It was not until 1951 that Jonas Salk and others proved that there were three poliovirus serotypes. They were named *Lansing* after material from a young man in Michigan, *Leon* after a boy who died in Los Angeles, and the third, *Brunhilde*, after a chimpanzee, and now generally referred to as poliovirus type 1, 2 and 3.

For those wondering why we don't know more about Myalgic Encephalomyelitis, remember, it took 1,400,000 dollars in 1951 to settle the question concerning the multiplicity of polioviruses. This had nothing to do with the cost of trying to find a cure. Today that would translate into more than \$13,000,000. Who is going to dedicate funds and expertise of that magnitude into a disease that most physicians do not believe exists, and if they do believe in it, kills so very few of its victims to cause governments to invest funds into Myalgic Encephalomyelitis? Then a new facet to this mysterious disease epidemic disease Wickman described arose.

Autoimmune Disease

Today, the problem is more complex since one of the significant 4 disease spectrums and one of the major causes of death and paralysis in the 1905 Stockholm epidemic was Guillain-Barré Syndrome (GBS).

After more than a century of research by thousands of brilliant minds world wide, and millions of dollars or euros spent, we still are not totally sure of what this disease entity represents. All we can say is that Guillain-Barré Syndrome (GBS) is not a single disease state, caused by a single infectious agent or a single immunization. It appears to be the body's primary **inappropriate immune response** to any of a multitude of infectious agents or immunizations that result in the devastating autoimmune injury of the peripheral nervous system and too often. Paralysis and death. It is also possible that the individual's nervous system has somehow been pre-sensitized, primed, by any number of infections such as Campylobacter jejunii or influenza virus or even certain immunizations. For a while, it was even questioned whether aspirin might be a trigger.

So the polio virus, if there was only one enterovirus that caused the 1905 Stockholm epidemic, the virus or viruses that gave rise to Spinal and Bulbar Poliomyelitis, as an infectious agent, may not have directly caused either the Superior Polio (Myalgic Encephalomyelitis) or the Landry's Polio (Guillain-Barré Syndrome: GBS).

In other words, the virus or viruses may not have caused an infectious disease in some patients. Instead, the virus or viruses may have triggered a deadly autoimmune disease causing the Guillain-Barré Syndrome, or a less deadly autoimmune reaction causing chronic Myalgic Encephalomyelitis-like disease. Medicine, even with the best of minds and the best of research funds is not so simple and definitely not as easy a profession as some patients believe.

It is also possible that two mechanisms may be in play, (a) entero-viruses in most cases of Myalgic Encephalomyelitis as well as (2) autoimmune disease. I am certain from examining scores of chronic Myalgic Encephalomyelitis patients, that some of the most injured patients also have a significant personal and family history of multiple autoimmune diseases. I know from examining hundreds of Myalgic

Encephalomyelitis patients who fell ill, immediately after Recombinant Hepatitis B immunization (usually within 1-5 days after the second or third immunization) and to a lesser extent, influenza immunization, that autoimmune reactions rather than the immunization itself or a virus can severely injure the patient causing major chronic illness and at times, death.

Unfortunately, the Federal authorities are even less compelled to investigate deaths after immunizations in case it causes panic among the public and when they do investigate, in my experience, they tend not to be serious investigations.

So I ask a slightly altered question. Was the cause of the 1905 paralytic polio epidemic in Stockholm in which the patients presented with multiple different severe but characteristic illnesses due to one or to several different several viruses? Or was the cause due to a combination of both infections and autoimmune reactions to one or more viruses the cause of some of the epidemic injuries that occurred? This question is still relevant today with Myalgic Encephalomyelitis patients. It remains unanswered.

The question becomes even more complicated. The three paralytic polioviruses identified by the Jonah Salk team in 1951 were members of the enterovirus family. No one knew this family of viruses existed prior to 1948-49 when Dr. Gilbert Daldorf, while investigating a polio outbreak in Coxsackie New York, found in the faeces of a polio injured child, a new enterovirus, he named Coxsackie virus after the town on the Hudson River where the virus was recovered. Today, including the three polioviruses, there are well over 100 other enteroviruses, each causing various kinds of destruction. To the best of our knowledge, these are not stable viruses but mutate constantly inside the body and probably outside of the body. They are also viruses that can be cultured in the cloaca of water birds, ducks, and geese

Then in 1951, researchers recovered a new virus, an ECHO enterovirus in the stools of a child just east of Coxsackie New York State, just across the river in Massachusetts.

Today we know that Coxsackie A2, A5, A9, B4, ECHO 5, 6 and 22 have each been recovered in Guillain Barré Syndrome deaths and paralyzed patients. (Let me remind you what I have already written: two of the major illnesses described by Wickman occurring in the 1905 Stockholm epidemic was Superior Poliomyelitis (Myalgic Encephalomyelitis) and Landry's Poliomyelitis (Guillain Barre Syndrome today). A total of 159 patients died during the first two weeks of the 1905 polio epidemic and 45 conformed to the picture of Landry's disease.

Today, because they die, physicians and researchers are still trying to figure out what causes Guillain Barré Syndrome when they already know it was caused by enteroviruses in 1905. Because Myalgic Encephalomyelitis patients die so

infrequently, although we have known for years that enteroviruses are the cause, no one is able to do autopsies on them to find the microscopic pathologies underlying this disease state.

Death and paralytic poliomyelitis is not restricted to Lansing, Leon and Brunhilde polio enterovirus strains. The last major poliomyelitis epidemic in the Soviet Union occurred in the 1950-1960s period and was caused by Coxsackie A 7 and then apparently disappeared. Enterovirus D68 2014-2015 is today causing paralytic disease in both children and adults in western United States and Canada. However, enteroviruses including types 70, 71, 89, 90, 91, 96, 99, 102, and 114 can all cause paralysis. Enterovirus 3-6 can cause hand foot and mouth disease and these viruses also can cause paralysis and possibly Myalgic Encephalomyelitis.

Approximately 70% of all meningitis cases are due to Coxsackie viruses, Meningitis can lead to death, to paralysis and permanent cognitive dysfunction.

These enteroviruses are all similar that they differ only by approximately 1% or less and this difference resides in the capsid end of the virus. Nor are these viruses stable. They continually mutate to become different strains. Even within the body, one infected, the virus continues to shift its genetic structure, making it impossible for the immune system to fix on it and destroy the disease-causing virus. (Leonard Archard of Charing Cross Hospital in London demonstrated this in in 1989.)

These 100 plus different enteroviruses, a little like influenza viruses, lack genetic stability. They are constantly mutating. So much so, it is a small miracle that Salk and Sabin ever produced a successful immunization. The less expensive Sabin oral polio immunization is a point. It is a live virus that has been passed through so many laboratory growth cycles that it is no longer paralytic. However when given to an individual, it confers immunity to the wild poliovirus, **BUT**. In the intestinal canal of the recipient, this tamed poliovirus mutates. At times this mutant virus becomes paralytic again. Given to a child, it passes through the child's gut, mutates and can then paralyze or kill the parent. For this reason the Sabin oral polio immunization is rarely if ever used in North America.

Coxsackie Viruses:

There remain many quandaries.

(1) The last major paralytic poliomyelitis epidemic in Russia was not caused by the Lansing, Leon or Brunhilde polioviruses but a Coxsackie virus,

(2) Guillain Barre Syndrome (GBS) is today considered to be not a single disease state, but represents a primary inappropriate immune response causing injuries to the peripheral nerves. It has been associated with Campylobacter jejunii and other microbes, to influenza and to immunizations but it is highly unlikely any one of these causes can define this often-devastating syndrome.

Was the cause of the 1905 Stockholm epidemic that brought the initial wave of epidemic death, epidemic paralytic polio, epidemic Guillain Barre Syndrome and epidemic Superior Polio (Myalgic Encephalomyelitis), one or several different enteroviruses or just different immune system responses to a single virus? Was this epidemic and every other epidemic of such enterovirus disease, at least up to the introduction of polio immunization in 1955, caused by one or more entero-virus. If was only one of the three accepted polio virus, how is it that Guillain Barre Syndrome and Myalgic Encephalomyelitis continues?

Today, in the post 1955 post Jonas Salk and Sabin world there has been little mention of epidemic Guillain-Barre Syndrome except in Northern China. With the exception of recent incursions of a very few cases of wild polio from Pakistan in southwest, China has been free of epidemic polio for many years.

Chapter Three

Myalgic Encephalomyelitis & Severe Adverse Effects of Immunization

I have found that some of the most severe cases of classical Myalgic Encephalitis appear immediately (1-4 days) after receiving Recombinant Hepatitis B immunization (90-95%) and to a much lesser extent to influenza immunization (5-10%). The problem is obviously not contained in the immunization so why should this occur. How can this be?

Please do not think immunizations are generally dangerous. They are not. Without immunizations, up to half your family or the people you know would have died or have been so severely injured you might never have met them. In fact, you might not be among us. However we should review immunizations in general and their relationship to Myalgic Encephalomyelitis type disease.

Precaution: There are at least two problems in discussing negative effects of immunizations.

1. The first is that many people will say they cause more problems than they solve. This is false and they then become involved in anti-immunization campaigns that many people believe them, they stop immunizing their children and consequently, end up killing and maiming innocent children.
2. The second is caused by the Government and the College of Physicians and Surgeons in each country. These bureaucracies are so pro-immunization that they will not discuss adverse effects. When a physician does discuss them,

both government and college immediately attack the physicians. Many physicians who criticize immunizations will lose their license or be involved in years of exhausting interminable attacks and investigations. Governments will organize false research demonstrating there is no problem with immunizations. Doctors will lose their license to practice.

The fact is, for every person injured or killed by immunization, 10,000 are saved. It is not those who are killed who are important to the governments, it is the survivors. Without immunization, hundreds of thousands of disabled people would have to be cared for. That will cost governments many billions of euros. So of course they are realistically pro-immunization. Do the math. Then you will understand the government's position.

However, lets discuss adverse effects to immunization and its association with Myalgic Encephalomyelitis because they appear to provoke disastrous effects in some patients.

There are at least two serious problems with immunization and they are:

1. **Temporary immobilization of the immune system:** this can allow benign viruses to become chronic and not be recognized by the immune system. This can be avoided.
2. **The Creation of Sensitivity:** The failure of immunization to immunize but to create sensitivity, which may result in severe autoimmune disease. This can be avoided in some instances and cannot be avoided in others.

1

The British Study on Immunization

Causing Paralytic Polio: England, Wales and Scotland: Prior to successful polio immunization in 1955, three separate studies were conducted by the British Government Health Services in 1947. These studies were carried out independently in England, in Wales and in Scotland. It was discovered that in the 30 days following **any immunization**, there was a marked increase in the incidence of paralytic poliomyelitis.

The government agencies also found that that the incidence of paralytic polio gradually increased during the first 14 days following any immunization and then began to taper off at day 16 and by day 28, the incidence of paralytic polio in the previously immunized individuals fell to pre-immunization days. Obviously, it was not the immunization that was causing the surge in paralytic polio cases. It was the

fact that the patient's immune system was busy with the effects of the recent immunizations, independent of whatever kind of immunization.

Remember what happens in polio: For approximately every 200 patients who are infected with poliovirus, only one will fall ill with symptoms. The rest will either have prior immunity or their immune system will safely defend itself against this polioviral infection. When the patient was immunized with anything, their immune system was too busy caring for the recent immunization. This is lesson that too many physicians ignore when giving immunizations.

2

Dr. Alberto Marinacci & How to Cause Myalgic Encephalomyelitis

Alberto Marinacci: When I went to see Dr Marinacci in California, (who had taken care of the Los Angeles Myalgic Encephalomyelitis patients until his retirement in the 1970s), he asked me a question. It is a routine question that I was taught to ask patients when I was studying at the University of Toronto Medical School. In interviewing a patient, one of the questions you must ask is: What makes your symptoms worse, what makes them better? Essentially, Dr. Marinacci question was a variant of that question.

How do you cause a patient to fall ill with Myalgic Encephalomyelitis?

Truthfully, at the time I didn't know what he was talking about. Easy he said. I will always remember his question and answer. It is something that all doctors should burn into their minds.

"It's easy to cause Myalgic Encephalomyelitis," he said. You take a patient who is exhausted from his or her work. They decide they have to get away, to have a holiday far from the strains of work and family obligations. They decide they are going on holidays, often to a third world country in Central or South America. They say:

"I cannot afford to be ill Doc. As soon as I come back I have a mountain of work, so shoot me up with every immunization you can think of."

"So the doctor does just that. Gives the patient a series of immunizations and a week later they get on the plane. They may be sitting beside someone with a common cold. They pick it up. They may arrive in the Dominican Republic and pick up some exotic virus their immune system has never previously encountered. However the result is the same. Their immune system is so involved with taking care of the recent immunizations that they have no defence system for the new virus. This virus becomes chronic, and if it happens to be a neurotropic virus, they develop Myalgic

Encephalomyelitis. If it happens to be a gastric bug, they develop gastritis. Its as easy as that."

"The trick is, never give any patient any immunization within the 30 days prior to them flying off. Do your immunization 30 days before or don't do it at all. Never give a patient an immunization if they are presently suffering from even a minor infection."

3

Dr. Aubrey Tingle, Chief of Viral Research, Vancouver Children's Hospital How to cause chronic rheumatoid-like arthritis

Sensitization Versus Immunization: In 1987 I took a flight from Toronto to Atlanta Georgia to attend a conference on chronic viral infections at the Centre For Disease Control in Marietta Georgia. It turned out to be a defining moment. At the meeting I was to encounter Dr. Alexis Shelokov, the most learned American concerning Myalgic Encephalomyelitis and his long time friend and associate, Dr. J. Gordon Parish the librarian of all Myalgic Epidemics. They were to change my life.

Serendipitously, on the same bus from the airport to the Centre for Disease Control was a man who I would have never known about, Dr Aubrey Tingle. He was the chief of Virology Research at the Vancouver Children's Hospital in Canada. He had written several papers on the adverse effects of immunization when improperly given. Here is a synopsis of what he had to tell.

Doctors are rightfully and significantly worried when they meet with a pregnant mother to be who is rubella negative. They know that the child can be severely handicapped or even killed if during the pregnancy the expectant mother contracts wild rubella (German measles). So the physician waits in fear until the delivery. Fortunately the mother did not contract rubella during the pregnancy. The child is perfect. It is a baby girl. So in the delivery room both the mother and the new baby is given an immunization against rubella. The mother goes home. The baby is breast-fed. All is fine. Both mother and child are perfect. But are they?

When the girl child is now 12-14, the girl is given a rubella booster and she then develops what appears to be severe rheumatoid arthritis. **What happened?** This is not an isolated case. Dr Tingle had possibly over 100 such cases.

What happened is this. The mother is given the rubella immunization. She breast-feeds the girl child. Her immune bodies in the breast milk protect the new baby against many or all of the infections that the mother may have had previously. However, her immune bodies prevent the newborn baby from seroconverting. The child becomes "sensitized" not immunized.

Skip forward until the girl has grown up to become a healthy 12-14 year old and she receives the booster of rubella immunization. But the girl is sensitized to rubella and she develops either rheumatoid arthritis or something that resembles it very closely. The girl then dies or is disabled for life with the equivalent of rheumatoid arthritis. This all occurred due to an immunization given to a breast feeding mother, 12-14 years earlier. It would not have happened if the rubella immunization were given once the mother had stopped breast-feeding.

The 1934 Los Angeles County General Hospital Epidemic: This has already been discussed, above, as following after immunization of the staff with convalescent serum from recovered patients. It was on this basis that the courts awarded the staff with a multi-million dollar settlement. It may also be the only clinical study where an actual virus was injected into approximately 200 hospital staff with an M.E.-causing-virus, which then resulted in classical Myalgic Encephalomyelitis.

Why am I telling you this?

Because that is exactly what happens when a patient is given a Recombinant Hepatitis B immunization (RHB) but she doesn't develop arthritis though some do. Possibly due to severe work or school fatigue at the time of the first RHB immunization, possibly due to a minor infection, possibly due to a quiescent enterovirus infection, the patient is **sensitized**, not immunized. A month later the patient receives the first booster immunization of RHB. Within 1-5 days she has not only Myalgic Encephalomyelitis, she has one of the most severe cases of Myalgic Encephalomyelitis you have ever seen. If she is lucky, she will die right away. I have had 3 such cases. If not, she will be most likely, permanently invalidated for the rest of her life. Who of the post RHB patients falls ill? Almost 80-90% are females. Almost 80-90% are health care workers or teachers, those with most contact with infectious diseases. Some have had prior RHB immunizations to which they didn't seroconvert but became sensitized.

These may or may not be enterovirus associated patients, but the effect is the same. A neurotropic antibody injures the patient's central nervous system. You can tell who these patients are but nobody does. If a patient has received one or more RHB immunizations and a viral study is done for surface antibodies to Hepatitis B and there are none. The patient is likely sensitized, not immunized. It is both amazing and tragic how many physicians and health centres continue to give RHB immunization when the patient has not seroconverted to RHB.

These post immunization RHB Myalgic Encephalomyelitis patients have at least three characteristics:

1. They tend to be women,
2. They tend to work in schools or health care,
3. They also tend to have a personal and family history of autoimmune disease.

4

The 1905 Paralytic Polio Epidemic in Stockholm and its Relationship to Myalgic Encephalomyelitis

You will remember that Wickman wrote, that during the first days of the 1905 Stockholm epidemic of paralytic poliomyelitis 1031 patients fell ill,

1. Many died,
2. Many were permanently paralyzed,
3. Many developed Landry's Poliomyelitis, now called Guillain-Barré Syndrome,
4. Many also developed a disease identical to Myalgic Encephalomyelitis,

They were all thought to be various forms or illnesses resulting from the same virus. However, subsequently:

Landry's Poliomyelitis, or Guillain-Barré Syndrome represented 45 of the 159 patients who died during the first two weeks of the epidemic. These 45 conformed with what was then called Landry's polio and what we call today, Guillain-Barré Syndrome.

Because Guillain-Barré Syndrome killed and paralyzed many of these epidemic patients, the world took note. In 1930, Hermon Cordinier in the Annals of Internal Medicine, stated:

"There is a remarkable difference of opinion with regard to the identity of Landry's paralysis: some contending that the type of paralysis described by Landry is a form of poliomyelitis, and others that Landry's paralysis is an independent symptom complex, which seems to have a special affinity for the peripheral motor neurons."

Reviewing the literature, Landry's or Guillain-Barré Syndrome is a rare and acute polyneuropathy that resembles a cross between Abortive poliomyelitis and severe Myalgic Encephalomyelitis. It is an autoimmune disease caused by the body's immune system attacking the peripheral nerves. Today, few die, but unlike Myalgic Encephalomyelitis up to 5% still die with the acute form of this syndrome. Some of the features aside from the rising paralysis are (a) dysautonomia, (b) disabling motor and sensory deficits, (c) progressive weakness, (d) numbness and parathesias very similar to Myalgic Encephalomyelitis.

Guillain-Barré Syndrome (GBS) is sometimes seen following immunization, but this is also true of Myalgic Encephalomyelitis. A large number of bacteria and viruses and medications have also been associated with GBS. None of these have demonstrated undeniable proof.

Today, over 110 years later, physicians and researchers are still unsure of what causes Guillain-Barré Syndrome and they have the advantage of doing post-mortem research on many of these unfortunate individuals. One thing they seem to agree upon is that Guillain-Barré Syndrome is most likely an autoimmune disease.

This leads us back to questions on Myalgic Encephalomyelitis and the original 1905 polio epidemic. I am certain that primary epidemic Myalgic Encephalomyelitis is caused by enteroviruses. I am certain that all polio epidemics were mixed epidemics of various polio and other entero-viruses. I am certain that the advent of Salk and Sabin immunization has just taken care of 3 of the hundreds of these constantly mutating enteroviruses. Lansing, Leon and Brunhilde. The question still remains: Is Myalgic Encephalomyelitis, an injury of the Central Nervous system, caused by:

1. The **infectious** effects of enteroviruses,
2. Or the **autoimmune** effects of enteroviruses.
3. And, if the chronic enterovirus infection Dr John Chia has demonstrated in the stomach mucosa of Myalgic Encephalomyelitis patients is treatable, can we cure these Myalgic Encephalomyelitis patients if an appropriate antiviral is produced?

Dr Charles Poser, Neurologist at Harvard, the professor who first involved me this discipline, was convinced that Myalgic Encephalomyelitis was an autoimmune disease and that if corticosteroids were given immediately upon the patient falling ill, they would not develop chronic Myalgic Encephalomyelitis. However it did no good attempting to treat these patients with corticosteroids when they were chronically ill.

Conclusions:

1. Like Dr. Ivar Wickman in 1905, I believe that Myalgic Encephalomyelitis is a polio- (enterovirus) family disease that attacks the cerebral cortex, the upper part of the central nervous system rather than the bulbar and spinal part of the central nervous system as in paralytic poliomyelitis.
2. Like the majority of British and Scottish physicians, who do not work for the insurance industry, and who have been involved in Myalgic Encephalomyelitis research and investigation, I believe that epidemic Myalgic Encephalomyelitis, like paralytic poliomyelitis, is a biphasic disease, with an incubation period of 3-5 days, and in epidemic cases is most prominent in late Summer and early Autumn.
3. Like Dr. Jay Goldstein and Dr. Ismael Mena, I believe that Myalgic Encephalomyelitis is a central nervous system injury, a limbic system and cortical encephalopathy as shown in the initial brain map.

4. Like Dr. Alberto Marinacci, I believe a **secondary form** of Myalgic encephalomyelitis can sometimes be provoked by injudicious immunization of any sort, given on the eve of travel,
5. Like the late Dr. Ismael Mena, I believe physicians can map, all Myalgic encephalomyelitis patients demonstrating chronic micro-vascular injury of the upper brain, the cerebral cortex, including the limbic system and basal ganglia. Like Dr. Mena I believe these injuries are vascular and can be shown to be made worse by injudicious or even what would be considered normal physical, intellectual and at times, sensory activity.
6. I believe, along with hundreds of US patients who have been awarded major financial compensation, due to chronic illness resulting from Recombinant Hepatitis B, that the injudicious use of Recombinant Hepatitis B can provoke a severe form of either Secondary Myalgic Encephalomyelitis or death.
7. I believe, *perhaps alone with the late Dr. Ivar Wickman*, that **Myalgic Encephalomyelitis is no more than a cortical form of polio-myelitis** and had we known more about enteroviral disease, Myalgic Encephalomyelitis, that virus or viruses would have been included in the Jonah Salk Polio immunization, and perhaps, just perhaps, there would have been no more Myalgic Encephalomyelitis.

Let me leave you with: Dr. Ivar Wickman's 1907 description of the fourth Polio, a major disease that occurred in the 1895 epidemic and the same 1905 deadly first major polio epidemic, along with (1) Spinal Poliomyelitis, (2) Bulbar Poliomyelitis, (3) Guillain-Barré Syndrome, and his description of the fourth polio which he called **(4) Superior or Medin Poliomyelitis but which he described as:**

1. An incubation period of approximately 3-5 days.
2. Fleeting palsies, (a type of partial paralysis or weakness often with shaking or tremors.),
3. Pain syndromes & malaise including onset headaches & head pain,
4. Nausea & gastric symptoms,
5. Tenderness of muscles resembling influenza,
6. Cognitive or psychic dysfunction,
7. Protracted prostration, (extreme exhaustion or lack of energy or power),
8. Subnormal temperatures, (& *difficulty with cold & heat perception*),
9. Transient aphasia, (*difficulty in saying words, dysphonia*),
10. Bladder symptoms, (*interstitial cystitis, painful sex*),
11. Hypotonus, (*loss of elasticity of muscles & arteries*),
12. Parathesias, (*abnormal sensations, tingling, burning or pricking that can be transient or chronic*)(*pins & needles, formication, a limb falling asleep*,
13. *Sweats*,

Dr. Ivar Medin, Stockholm, 1907