Nightingale Research Foundation

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The Academy of Nutritional Medicine (AONM)

The Forgotten Relationship Between

A. Poliomyelitis, (polio),

B. Myalgic Encephalomyelitis (M.E.) and

C. the New Polios
(Acute Flaccid Paralysis – AFP)

My appreciation again to:

The Academy of Nutritional Medicine (AONM)

&

Clare Palmer and Gilian Crowther

1942 our house was bombed
I was alone with my sister in
a steel Anson shelter on
the ground floor in Hove.

Myself in 1942, in Sussex after the bombing.
I looked like trouble and undoubtedly, still am.

In 1944, I, and my little sister, sailed on the Cunard ship, RMS Aquitania from Scotland to Canada. By ten I was going to school in the morning and working 8 hours a day from 1 to 9 pm.

Polio: In 1948 I fell ill with paralytic polio. I spent the entire year in the hospital and home in bed followed by a year recuperating. It was great as I didn't have to go to school, but it was the beginning of my education in chronic disease.

Among my British family, only my mother and one cousin survived into the early 1940s.

Peter Nunn became Professor of Toxicology at London University on the Strand and
I became a physician in Canada.

My House and Office Entrance

The Unknown Diseases: Polio & M.E.

14th century BC, Amenhotep III of a reputed polio victim
This could have equally occurred due to birth trauma or many infectious diseases

Medin's 5 types of polio in 1887

1. Spinal Polio
2. Bulbar or Pontine Polio
3. Polyneuritic Polio
4. Ataxic Polio
5. Encephalitic Polio

1904-1905: World's First Mega-Polio Epidemic

Over 2000 children and adults injured.

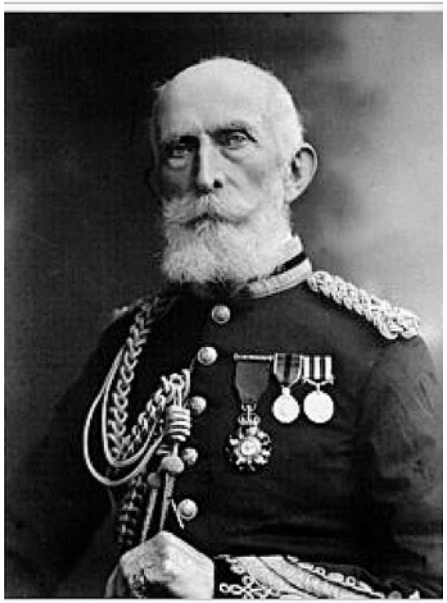
Ivar Wickman assigned to investigate

This was the first epidemiological study of a Polio epidemic.

Question?

- How you think some of the chief Medical experts diagnosed this epidemic of over 2000 dead or paralyzed or injured patients?

Dr. Jules Dejerine 1849-1917



Joseph Jules Dejerine

Dr. Dejerine, neurologist, psychoanalyst

- No such epidemic of Polio had ever occurred previously.
- Dejerine states, this was a classical case of mass hysteria.

Question?

How many M.E. patients

are there in the U.K.?

Dr. Otto Ivar Wickman (1972-1914)

Wickman described not 5 but 10 types of polio

1. Spinal Poliomyelitis
2. Landry's Ascending Paralysis (Guillain-Barre Syndrome)
3. Landry's Descending Paralysis
4. Bulbar or Pontine Polio
5. **Superior Polio (M.E.)**
6. Meningitic Polio
7. Ataxic Polio
8. Polyneuritic Polio (fibromyalgia)
9. Abortive Polio (M.E.0
10. Well Carrier Polio

Wickman's description of **Superior** polio in 1905

1: Incubation period of 3-5 days

2: Fleeting Palsies (muscle weakness, shaking, tremors)

3: Pains and malaise, headaches, nausea, gastric symptoms

4 : Tenderness of muscles resembling influenza

5: Cognitive or psychic dysfunction

6: Protracted prostration & exhaustion, lack of energy or power

7: Subnormal temperatures, cold and heat perception difficulty.

8: Transient aphasias

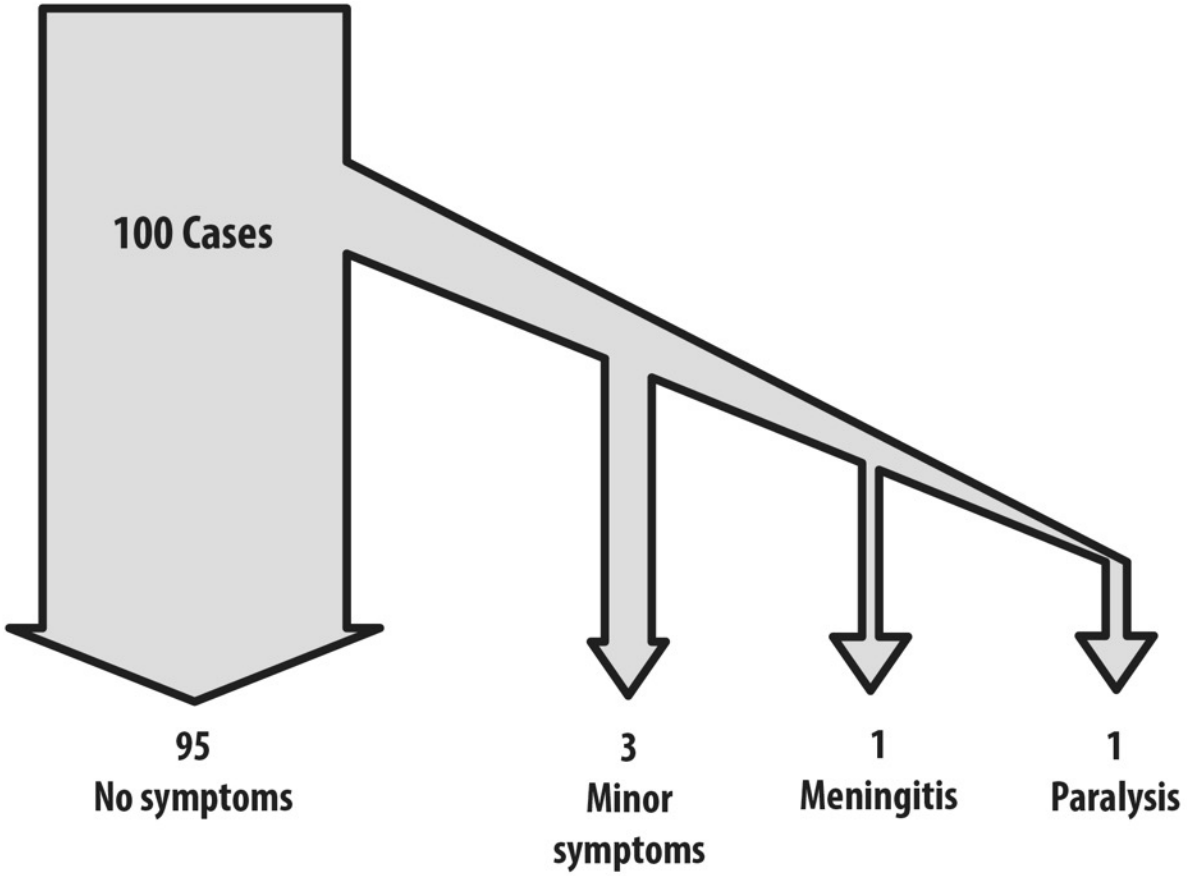
9: Bladder symptoms

10: Hypotonus (loss of elasticity of muscles and arteries (POTS)

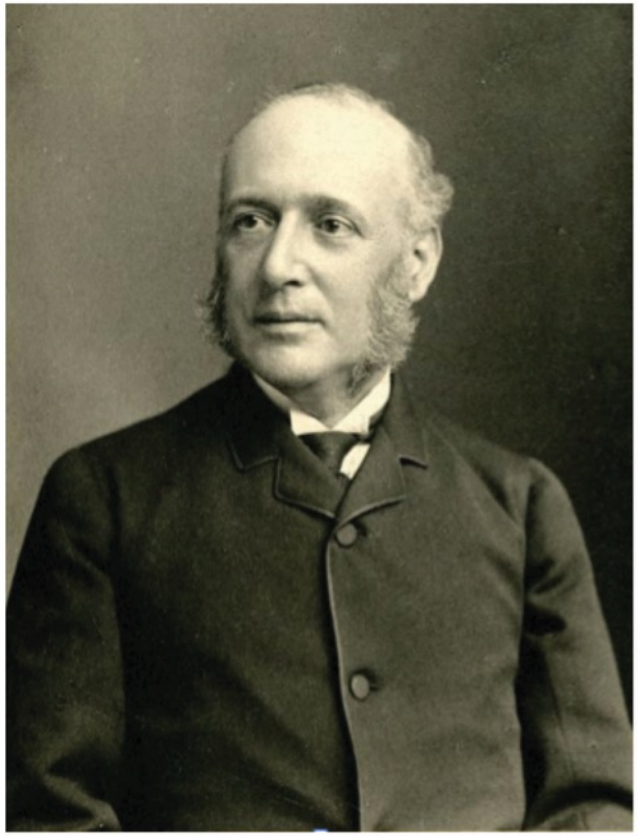
11: Sweats

12: Parathesias (tingling, burning, limbs falling asleep).

Polio & M.E. Infection
Both Result In The Following



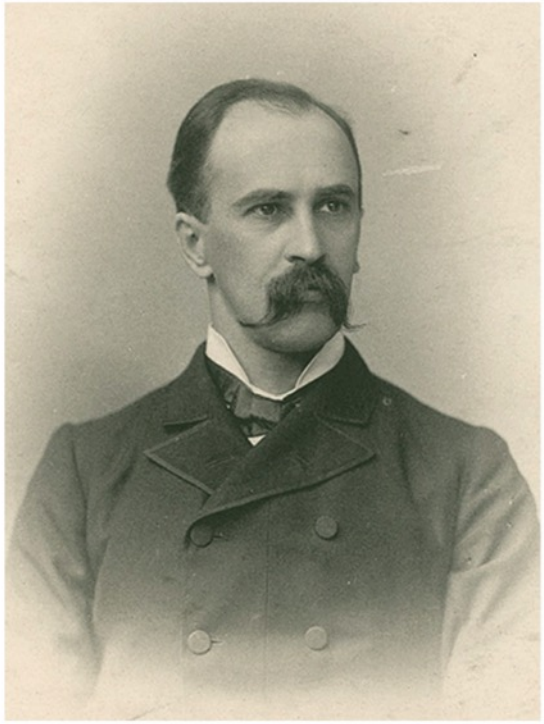
Dr. Jacob Da Costa, 1833-1900



Described orthostatic intolerance in Neurasthenia circa 1870

William Osler's 1849-1919

Founder of Johns Hopkins Hospital
Died as professor at Oxford University



1914: Osler stated Neurasthenia should not be confused hysteria!

Osler's 1914 Description of Neurasthenia

Neurasthenia follows an infection & may include:

- cerebral, cardiovascular and gastric symptoms
- A striking lack of accordance with complaints & physical findings
- The patient falls easy pray to charlatans and quacks

Osler's 1914 Description of Neurasthenia

- Imperfect sleep
- Inability to perform the normal mental work
- Difficulty doing addition
- Difficulty taking dictation or writing letters
- Disturbance of articulation or writing abilities
- Loss of fixed attention
- Hyperaesthesia related to pain
- Pressure points are painful
- Pains in the skin, eyes, joints, blood vessels

Osler's 1914 Description of Neurasthenia

- Head pains
- Anxiety in almost all patients
- Fear of approaching death
- Increased risk of suicide due to despair
- Hearing disturbances (hyperalgesia)
- Weariness on the least exertion
- Numbness and tingling
- Incoordination
- Muscular weakness can be extreme
- Gastralgia & gastric hyperaesthesia

Osler's 1914 Cardiovascular symptoms

- Tachycardia
- Pain in the heart area
- Palpitations
- Sweats
- Cold extremities
- Transient hyperaemia of the skin
- Increased risk of suicide due to despair

Osler's 1914 Treatment Suggestions

- Treatment with drugs should be avoided as much as possible
- The patient requires an understanding physician
- Have faith in religion and maintain spiritual guidance

Osler separates neurasthenia from psychiatric disease mentioning:

- the anxiety
- fear of death
- risk of suicide
- depression that comes with this illness.

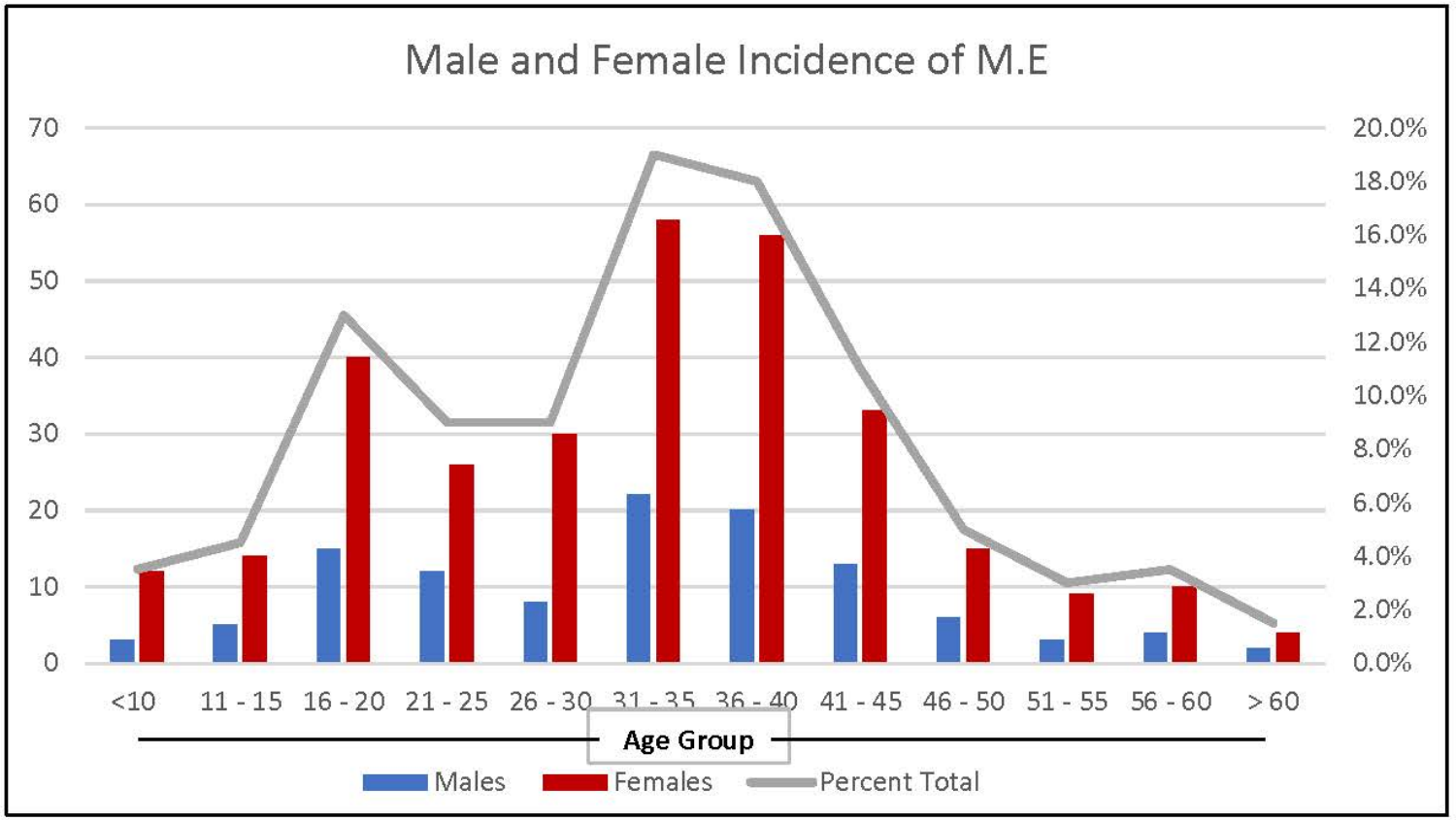
Who Falls ill With M.E.

1. Nurses, physicians & other health care workers, particularly women.
2. Teachers
3. Very active students
4. Professional women working well past exhaustion, doing two jobs –with children bringing home infections from school.

Explanation:

The EV that causes HFM disease, Gastritis or Pneumonia in a child, will not cause the same disease in an adult who may develop Polio or M.E.

Dowsett, Ramsey & Bell Incidence of 420 Post Enterovirus M.E. Patients - 1970



Dowsett, Ramsay & Bell – 1970 Signs & Symptoms In 420 M.E. Patients		
CNS or PNS Neurological Findings		
1	Muscle dysfunction	100%
2	Cognitive disturbance (memory, concentration, anomia, dyslexia)	77%
3	Headache	74%
4	Balance difficulties	72%
5	Autonomic dysfunction (circulation and thermoregulation)	69%
6	Auditory disturbances (hyperacusis, deafness, tinnitus)	69%
7	Sleep reversal: 64%	64%

Dowsett, Ramsay & Bell – 1970 Signs & Symptoms In 420 M.E. Patients		
CNS or PNS Neurological Findings		
8	Visual disturbance (loss of accommodation, photophobia, nystagmus)	62%
9	Paraesthesia	61%
10	Fasciculation, spasm, myoclonus	57%
11	Clumsiness	56%
12	Orthostatic tachycardia	21%
13	Non-bacterial conjunctivitis	16%
14	Positive Romberg sign	6%
15	**As in Polio, Paresis and muscle wasting =**	1%

Who Develops Chronic Fatigue Syndrome

1. PTSD military and war veterans with fatigue,
2. alcoholics with fatigue,
3. major depressive patients with fatigue,
4. post-traumatic patients with fatigue,
5. farmers, firemen and police officers with toxic chemical fatigue
6. Missed cardiac pathologies
7. Missed malignancies
8. Missed genetic illnesses, particularly with Ehlers-Danlos-Syndrome
9. Missed prescription drugs injuries: eg., Cipro anti-biotic family
10. Adult Encephalopathies following child-hood illnesses, EBV, measles, varicella.

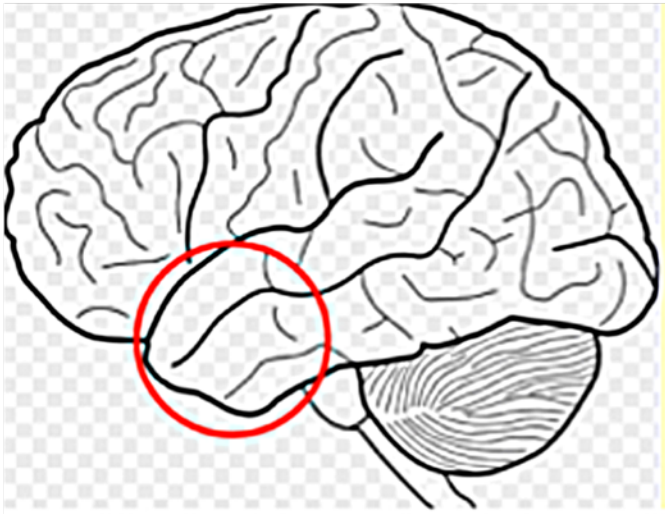
Some M.E. Pre-Conditions

1. **Acute onset** *(no CFS definitions mentions acute onset)*
2. A prior chronic immune exhausted state
3. Increased contact with infectious disease
4. Onset during the late summer and autumn
5. A prior history of auto-immune disease
6. Recombinant Hepatitis B immunization in the ten days before they fell ill with M.E.
7. A history of a relative who had polio.

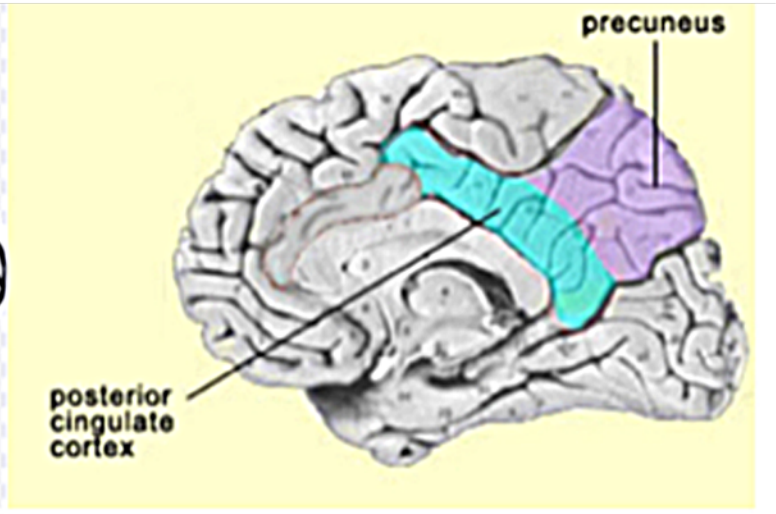
Basic Characteristics of M.E.

1. Only **EV** caused **acute** illness including Polio can result in M.E.
2. An often missed biphasic disease with an Incubation period of 2 - 6 days
3. The 2nd phase of M.E. is the encephalitic, meningitic muscular illness
4. Followed by cascading features in following months
5. Occurs **primarily** from June to November, peaking in late summer.
6. Brain SPECT demonstrates **permanent** CNS injury from first day of illness.
7. On Segami Oasis software. **Injury demonstrates anterior left temporal lobe & cingulate gyrus** consistent with memory and muscle disfunction.
8. Parts of the entire cerebrum may be involved.
9. Polio is a vasculitic injury primarily of the spinal column motor neurons.
10. M.E. is a vasculitic injury of the cognitive and administrative neurons.

Primary M.E. Brain Injury Sites

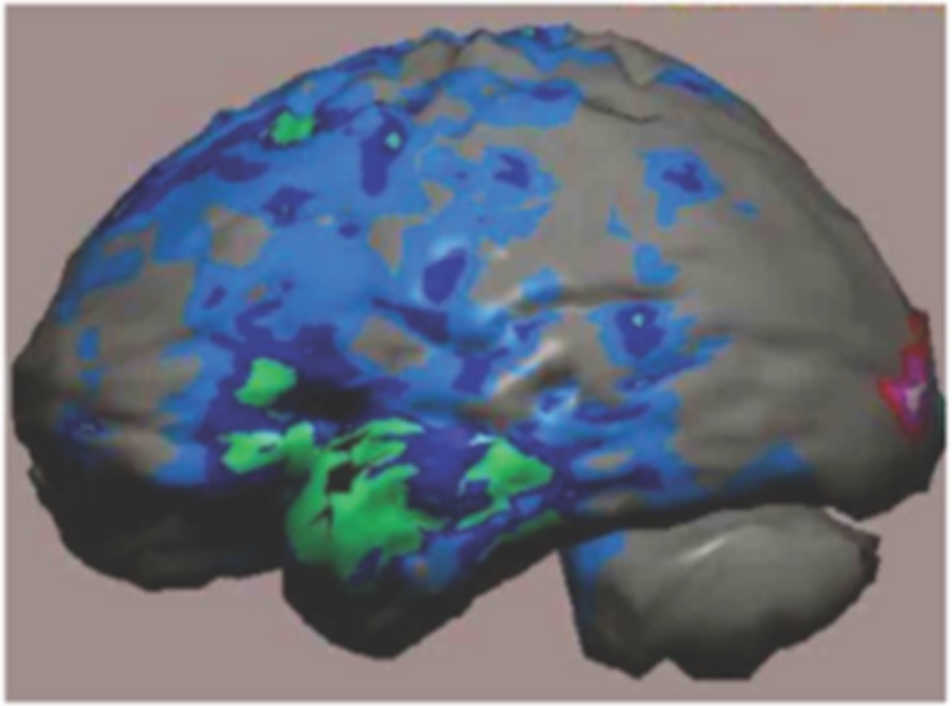


Anterior Temporal Lobe



Posterior Cingulate Gyrus

Segami Oasis M.E. Brain Map



M.E. patient brain, with anterior and Middle Temporal Lobe injury, plus Brocha and Motor Cortex injury

Question For Physicians and Health Care Workers only?

What is the primary cellular Pathology which causes Paralytic Poliomyelitis?

1871 Micro-Graph of Polio Spinal Injury

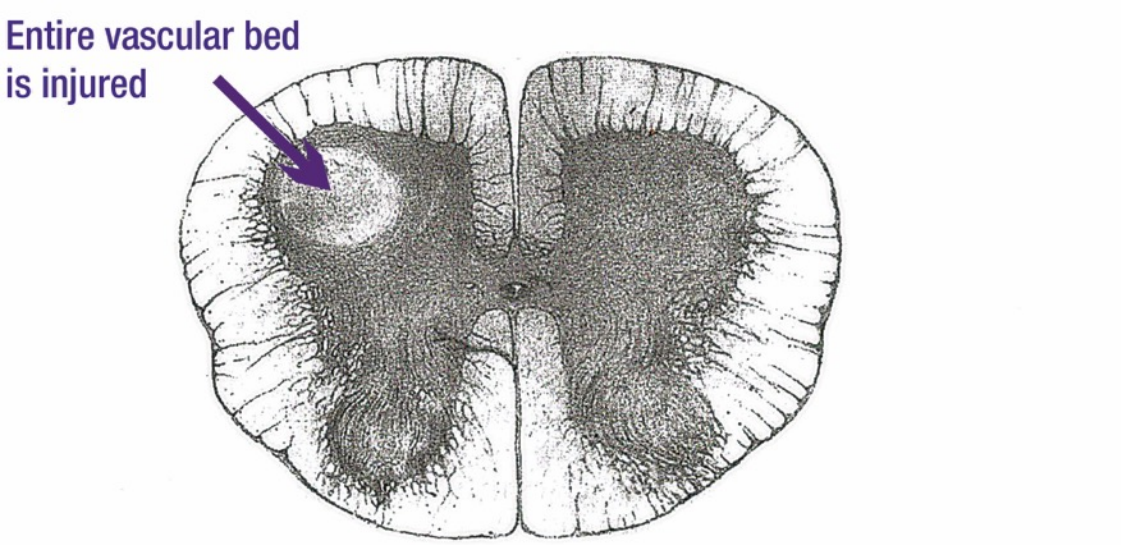
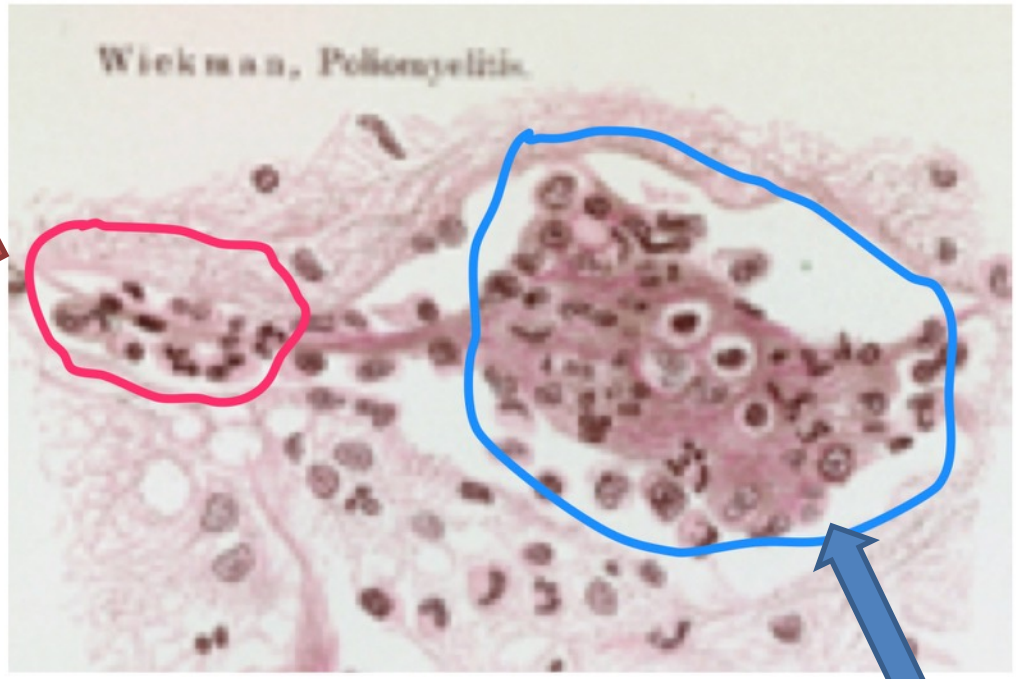


Figure 1.8 Cross-section of spinal cord, showing the anterior and posterior horns of the grey matter. The left anterior horn (top left in the image) contains a lesion of poliomyelitis. Reproduced from Roger H, Recherches anatomo-pathologiques sur la paralysie de l'enfance, Paris: Adrien Delahaye (1871), © Bibliothèque Interuniversitaire Santé, Paris

1911 Wickman's Micro-Graph of Polio Injury

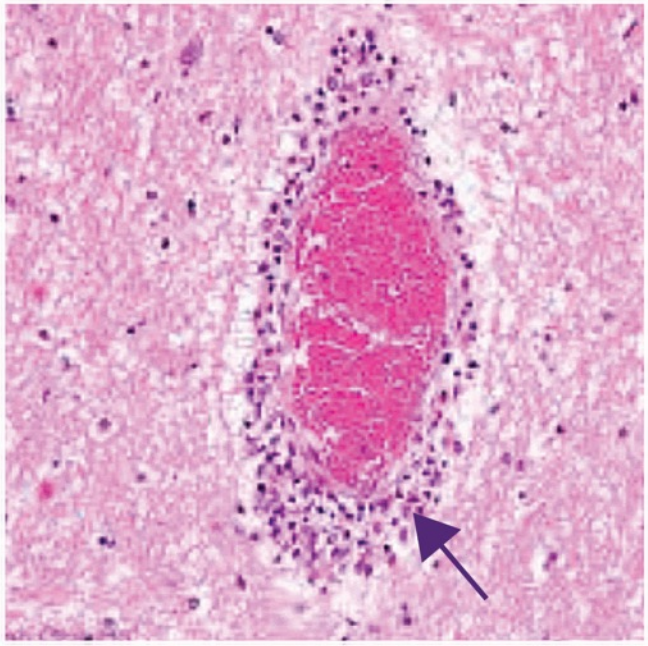
Vasculitis cutting off blood to Anterior Horn Cell



Notice: injury is cuffing, choking small capilliary which kills the Anterior Horn Cell

Anterior Horn Cell death cuts off peripheral nerve, causing paralysis

Blood Cell Cuffing, Strangling Blood Vessel



This is the primary pathology in both Polio and probably in M.E. with auto-immune capillary cuffing to motor neuron in Polio and cognitive and administrative neurons in M.E.

Question:
Who are our principal M.E. Patients?

1. 70-80% women, often with 2 jobs many with children bringing infections from school,
2. Nurses, physicians & health care workers,
3. Teachers,
4. Very active, over tired teen-age students,

Testing for M.E. at Onset Phase

1. Only at Onset Phase can EV infection be easily documented
2. Positive Oligoclonal Banding is seen on Lumbar puncture in 1st week
3. Abnormal SPECT Brain Scan is positive from day one

Classical onset symptoms

1. Characterized by sudden, severe prostration in June to November
2. Often severe, pharyngitis,headaches and eye symptoms
3. Patients often develop a sense of fear and terrifying impending doom
4. Often migrating severe head, limb, body and gastric pain develops

Onset Phase Can Evolve Into

1. Full or partial recovery
2. A brief recovery of a few days followed by chronic illness
3. Immediate onset of chronic disability
4. Rarely acute illness leading to death in the first few weeks
5. Rarely, immediate Parkinson's disease, even in children (Akuyeri Epidemic)

Second or Chronic Phase

1. Persistent physical prostration
2. Persisting pain
3. Persisting cognitive difficulties
4. Sensory abnormalities (visual, distance perception, tunnel vision)
5. Decreased circulating blood volume
6. Hypersombulism, followed by sleep reversal
7. Frequent physician induced iatrogenic injuries

Cascading Features of M.E.

1. Thyroid abnormalities
2. POTS
3. Palindromic Arthritis
4. Hyper-Flexible Ehlers-Danlos Syndrome becomes severely symptomatic
5. Causalgia (complex regional pain syndrome)
6. Light sensitivity
7. Adverse medication, food and alcohol intolerance

Continued next slide. . .

Cascading Features of M.E. Continued

8. Chronic gastric conditions
9. Interstitial cystitis, dyspareunia
10. Severe iatrogenic stress
11. Mitochondrial Injury
12. Multiple Social injuries, marital discord, suicide, poverty
13. Sectioning
14. Post M.E. Syndrome after age 60, as in post-polio syndrome

Internal Disease Potentiators

1. Male/Female divide: 80% are female, males have better recovery
2. Prior allergic pre-condition
3. Ehlers Danlos Hyper-Mobility syndrome and other collagen diseases
4. HLA-B27 genetic anomaly
5. Close relative with a history of Polio Myelitis
6. Multiple prior and different enteroviral illnesses
7. Multiple prior, closely spaced, repeat minor infectious diseases

External Disease Potentiators

1. Returning to work or physical activity too soon after onset
2. Occupations with increased exposure to infectious disease
3. Illness onset within 2 weeks of RHB immunization
4. Early sexual abuse
5. Alcohol
6. Organized attack by Insurance Industry
7. Insurance company refusal to honour women's disability pensions

The End

Louis Kurz (1835–1921)

The Battle of Missionary Ridge

Doulton Ceramics, Lambeth, London

Owned by English Great, Great Grand-Parents, the Nunns

By 1900 the company name was changed to Royal Doulton
And almost the entire family died from tuberculosis

By 1920 only two of the original family were still alive.
They all died of tuberculosis.
My grand-mother died in London in a psychiatric hospital
with rampant tuberculosis of the brain
And my mother was given away to a Scottish family.

My mother married Cyril Wrenn (RN), killed by the Germans in 1941 when his warship was torpedoed. His father, also Royal Navy, died in WWI at Scapa Flow.