



Intrinsic Focal Electromagnetic Induction, a Mechanism of Neurological Symptoms

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Abstract

The physics of Electromagnetic Induction (EMI) is reviewed and applied to the anatomy and neurophysiology of the human body. The neuron, the primary cell of the nervous system, coexists with a myriad of vascular structures and would be susceptible to EMI. When the neuron's electrical impulse, the action potential, traversing an axon with deficient myelin, intersects the electromotive fields of a blood vessel, a conductor, EMI could occur. By the laws of physics governing this phenomenon, a new current, inductance, would be produced and shared throughout the blood vessel and back into the axon source of the original current. Medical history and the study of physics support this phenomenon as the mechanism of the pain in trigeminal neuralgia, tic douloureux. Other neurological syndromes, such as seizures associated with arteriovenous malformations and causalgia seen after nerve injuries in the extremities may share this mechanism.

Keywords

Electromagnetic Induction; Neurological Syndromes; Trigeminal Neuralgia; Tic Douloureux; Seizures; Causalgia

This review applies the physics of electromagnetic induction to the anatomical and physiological features of clinical syndromes and demonstrates the feasibility of this phenomenon as a mechanism of neurological symptoms.

In a 1955 medical school physiology class, Dr. Merrill Spencer and Dr. Adam Dennison lectured and demonstrated their invention, a blood flow meter that combined their knowledge of the anatomy and physiology of the vascular system and the physics of electromagnetic induction (EMI). Previous research had established that blood was a good conductor of electricity and this property was exploited in the design of their instrument [1]. When the magnetic

field of the instrument was applied across an intact blood vessel, the movement of the blood inside the vessel created inductance which altered the magnetic field of the instrument. This change was calibrated to reflect the blood flow [2].

The lecture captured my attention and I pondered the billions of neurons with their innate ability to generate and propagate an electric current coexisting in the human body with a myriad of blood vessels. Could intrinsic focal electromagnetic induction occur in the human body?

Later as a neurosurgery resident I encountered patients with trigeminal neuralgia, tic douloureux.

They suffered cataclysmic paroxysms of distressful, horrible, stabbing, flashing, shocking, shuddering, lightning-like, electrical pains which were precipitated by the slightest stimulus of a specific spot, a trigger zone, commonly located in a division of the trigeminal innervation of the face. A refractory period, an electrical phenomenon, would follow a paroxysm of the symptoms. The mechanism of the pain was unknown, but the electrical character of the syndrome was inescapable. Could this be an example of intrinsic focal EMI?

A review of the physics of EMI as discovered by Faraday (1831) and refined by Maxwell (1854), revealed that four criteria are necessary for EMI: an electric current, a conductor, proximity, and motion [2-7]. These criteria are the design requirements of generators, motors, starters, transformers, and most electrical applications.

The anatomical and physiological features of the dorsal root entry zone of the trigeminal nerve satisfy these criteria. The axons carrying action potentials (current) are tethered as they enter the lateral pons and cannot escape the encroachment (proximity) of blood vessels (conductors). The constant motion of the blood flow inside the vessels completes the criteria for EMI. Additional factors which would abet this phenomenon are the naturally deficient myelination of the axons in this transition zone and the law of physics which states that inductance is inversely proportional to the square of the distance between the current and the conductor [2-7]. When the electromotive force fields of the current source interact with the fields of the conductor, EMI produces a new current, inductance. This new current is shared throughout the conductor and back into the original current source [3]. The inductance would flow from higher to lower voltage and follow the paths of least resistance, the vascular structures and the existing neural pathways. The inductance would convey into the brainstem nuclei and tracts of the trigeminal nerve and would pass to the thalamus and finally to specific cortical sensory neurons [8].

The cortical neuron learns by repetitive stimuli to recognize an individual axon's action potential signal

as a specific sensation from a particular location. This information is then communicated to thousands of cortical neurons for interpretation and response [8]. When, instead of the expected signal, the inductance, an aberration, an electrical storm, arrives, the cortical neuron passes this erroneous information on to a multitude of cortical neurons. The patient experiences this as the cataclysmic symptoms of tic douloureux.

Many centuries of medical and surgical history reflecting the efforts of physicians treating tic douloureux, have taught us that surgical interruption or damage of the afferent nerve is effective treatment, but sacrifices the function of the damaged nerve [6,9,10]. The medical treatment with a modicum of success is the use of anticonvulsants, such as carbamazepine, which alters the electrical properties of the neuron [11]. These therapies remove, or alter, the current criterion of the EMI phenomenon. A further review of history reveals that tic douloureux will not occur if any one of the required criteria is not present.

In 1925 Dr. Walter Dandy surgically explored the cerebellar-pons angle and noted the complex arrangement of the vascular and neural elements at the dorsal root entry zone of the pons. He considered this to be the site of origin for tic douloureux and performed a selective rhizotomy [5]. This removed the current criterion and arrested the tic but sacrificed the function of the nerves severed.

Forty years later Dr. Peter Jannetta using the advantages of magnification and illumination provided by the operating microscope developed his microvascular decompression (MVD) operation for tic douloureux. He moved the encroaching vascular structure away from the nerve without damaging the nerve. The procedure removed the proximity criterion for EMI and successfully stopped the tic douloureux without sacrificing the function of the nerve [12-14].

For over fifty years I shared a friendship with Dr. Jannetta, and we exchanged ideas and results as we struggled with tic douloureux. I employed most of the surgical modalities used for tic as they would come

into vogue, but never understood the actual mechanism of the pain. Peter thought it was the physical insult of the blood vessel on the nerve rootlets. As we ended our surgical careers, still challenged by this conundrum, we continued to study the mechanism of this painful syndrome.

I began by studying the difference in the pathology encountered in the trigeminal syndromes, neuralgia and neuropathy. Was it different for the two disorders? Was the site of the pathology different? Would some lesions cause neuropathy while others caused neuralgia? Why did the two different presentations rarely occur concurrently?

My surgical experience (1963-93) included 37 patients who had surgical intervention for trigeminal symptoms. These included 9 neuralgias (tic douloureux), 25 neuropathies, and 3 patients with symptoms of both disorders.

The pathology found in the 9 patients with tic douloureux was always in the dorsal root entry zone- 6 vascular encroachments, 2 meningiomas, and 1 metastatic melanoma.

In the 25 patients with neuropathy, the surgical pathologies were scattered from the dorsal root entry zone to Meckel's cave and included: 2 meningiomas, 2 acoustic neuromas, 2 neurofibromas, 1 arachnoid cyst, 8 epidermoid cysts (pearly tumors), 1 foreign body (bullet), and 1 boney mass. 15 of these surgical specimens were noted to be distinctly avascular lesions. The only vascular lesions were the two meningiomas which were located on the petrous ridge.

The 3 patients with both tic and neuropathy symptoms had vascular tumors (2 meningiomas, 1 metastatic melanoma) at the root entry site. These lesions would be both conductors and axon deforming masses.

In this small sample, tic douloureux patients had vascular encroachment, or a highly vascular lesion, at the root entry site. The neuropathy patients had

lesions which tended to be avascular scattered along the course of the nerve. This vascular distinction also characterizes the electrical difference between conductors and non-conductors [1].

These observations increased my suspicion that EMI could be the mechanism of the pain in tic douloureux.

An extensive survey of 5,056 patients seen at the Mayo Clinic (1976-90) with face pain revealed 2,972 diagnosed with tic douloureux. 296 had tumors as a cause of their symptoms. Meningioma, a very vascular tumor was the most common. 47% of the patients also had neurological deficits indicating trigeminal neuropathy. The conclusion was that a vascular tumor in the area of the dorsal root entry zone could cause tic douloureux [4].

Love and Coakham described demyelination in the axons in the dorsal root entry zone where vascular compression had occurred. They reported rapid recovery after decompression and attributed this to reduction of the spontaneous generation and ephaptic spread of impulses. In addition to the symptomatic relief, a recovery of nerve conduction across the area of decompression was noted [15]. Their findings are supportive that EMI may have been involved. The inductance could be the mechanism of the syndrome while the physical compression of the rootlets at the collision site may have contributed to previous existing myelin deficiency.

Other reports in the medical literature support this premise

A patient with an AVM in the cerebellar-pontine angle presented with tic douloureux. She was managed by an endovascular procedure which thrombosed the lesion. The malformation remains in place but the motion criterion for EMI has been arrested and symptoms of tic douloureux have stopped [16].

A patient presented with a bullet lodged in the dorsal roots of her trigeminal nerve as they entered the lateral pons. She had severe neuropathy but no tic douloureux. The bullet was a copper-clad, steel slug,

an excellent conductor, but was at rest and with no motion; the bullet did not cause tic douloureux [17].

Patients with seizures frequently have a vascular lesion in the cerebral area from which the seizure arises [18]. 23% of all cerebral tumors are accompanied by seizures and 26% of tumors with known high vascularity (meningiomas, metastatic melanomas, and cystic angiomas) present with seizures [19-21]. This proximity could initiate an EMI phenomenon.

In a large series of penetrating cerebral injuries, the incidence of post-traumatic seizures conforms more significantly to the cerebral vascular anatomy than to the topography of the injury. (Rish B, Caveness W, Poster Session, Seizures after penetrating cerebral injuries, CNS, San Francisco, 1972).

In my clinical practice I consulted on patients with post-thoracotomy intercostal neuralgia occurring when an injured intercostal nerve remained in the proximity of a vascular structure. The syndrome is resolved by surgical separation, or removal, of the components.

Symptoms accompanying multiple sclerosis (MS), other than the specific neuropathy resulting from the demyelination plague, may be due to focal EMI occurring at the site if vascular proximity exists. This explains the perplexing association of MS and tic douloureux presenting in younger patients or bilaterally [22].

The syndromes of reflex sympathetic dystrophy and complex regional pain present features that suggest an EMI mechanism [23]. This large group of disorders includes classical causalgia. This entity was originally described in detail by Dr. Weir Mitchell during the Civil War as a syndrome which followed a penetrating injury to an extremity which included damage to a major nerve anatomically associated with a vascular bundle [24]. We now appreciate the time lag of several weeks after the injury while the nerve undergoes demyelination before the symptoms

of causalgia appear, and the sympathetic display which results from the sharing of the inductance occurring at the injury site into the sympathetic nerves of the vessels involved. Surgical separation of the injured nerve from the vessel relieves the causalgia.

Amputees with stump pain symptoms present the same anatomical problem and are managed using the same surgical rationale. These problems became much less frequent as general and orthopedic surgeons learned to separate the neural and vascular elements carefully during the amputation procedure. (Unpublished, Rish B, Experience at USNH, Bethesda, MD, 1967-68, 1971-73).

For years Dr. Jannetta did not accept this ethereal, invisible mechanism for the pain of tic douloureux and considered the premise a critique of his MVD procedure. Later as he performed MVD on the VII nerve for hemifacial spasm, he alternately moved the vessel away from the nerve and then gently repositioned it against the nerve. His anaesthesiologist reported a stop and restart sequence of the facial spasms. Hearing this report Dr. Jannetta repeatedly moved the offending artery away from the nerve and returned it into proximity with the nerve without any deformity or agitation. As the facial spasm stopped and restarted in a time sequence matching his manipulations, he realized an ethereal and invisible phenomenon was transpiring. He became suspicious that a mechanism other than the physical insult of the artery against the nerve may be present [25]. Later I had the opportunity to repeat this demonstration in my own operating room.

Many vociferous discussions ensued between us before Peter came to realize that this phenomenon was not contradictory, but was supporting validation, of his MVD. He had successfully removed the proximity criterion for EMI. Then, embracing the premise, he began to prepare a Letter to the Editor expressing his support of the premise which had been reported in 2015 [17]. This letter was not completed before he died in 2016. (Personal communication, Diana Jannetta, March 2019). His work and friendship are acknowledged and appreciated.

Conclusion

This clinical review, a merger of anatomy, neurophysiology, pathology, and the physics of electricity, supports the premise that focal intrinsic electromagnetic induction is the mechanism of the symptoms of tic douloureux. There are also other neurological syndromes in which EMI may be the inciting mechanism.

Further data delineating the physics of intrinsic focal electromagnetic induction in the physiological scenario will promote better therapeutic intervention.

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