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In Philosophy of Nerves: Pain First!

In Peripheral Nerve Injuries (PNIs), pain is the initial symptom and the opening clinical manifestation. Pain is the nerve's cry against a hostile, injurious environment; it is the first reaction. It is also a detection and warning mechanism. On one hand, it is evidence of an assault. On the other hand, it is proof of the structural integrity of the pain-transmitting nerve itself, and likewise, proof that its defensive mechanisms are functioning properly. A nerve experiencing pain is an intact nerve—but only temporarily.

Why pain first?

Regardless of the cause of nerve injury—whether inflammatory in origin or a compressive force—it ultimately culminates in a strangulating compression that suffocates the nerve. The inflammatory edema accompanying both inflammatory and compressive events will exert its effect on the nerve. At this point, the least fortified structural components of the nerve become the easiest and first targets of the pathogenic agent. Here, pain-transmitting axons take precedence because they are the most fragile and the quickest to be affected by the pathogenic agent.

Pain sensation is transmitted by two types of axons: Unmyelinated axons (slow-conducting), known as C-type axons. And myelinated axons (slow-conducting), termed Delta (δ) axons. Both are thin-walled axons with low resistance to external pressures, though they differ in their degree of sensitivity to compressive forces.

As for C-type axons, they are enveloped by a single, uninsulated cellular membrane. This membrane is known for its thinness and limited resilience to both internal and external pressures. Conversely, Delta-type axons possess a myelin sheath of minimal thickness. Certainly, Delta axons exhibit greater tolerance than C-type axons to external and internal stresses—but only to a finite degree. Beyond this threshold, their resistance collapses, and their lumen is compromised. They then succumb like their predecessors, amplifying the manifestation of pain.

Critical Note:

On the significance of the myelin sheath in neural transmission, consult my article:

The Nerve Transmission through Neural Fiber: Personal View vs. International View

The Bitter and the More Bitter

Due to external compressive forces, pressure initially rises within pain-transmitting axons. Thus, pain becomes the opening act of the clinical presentation. When compression persists and/or intensifies, pain-transmitting axons sustain significant damage, halting neural conduction within them. Consequently, pain vanishes as nociceptive input from periphery to center ceases.

Hence, in peripheral nerve injuries, pain dominates the clinical picture as long as the nerve retains its capacity to cry out and protest. Conversely, pain may disappear when injury becomes chronic. Pain vanishes when compressive forces solidify and their destructive dominance prevails. Pain fades when its transmitting axons degenerate. Pain disappears when prognosis worsens. In this context, pain's presence is bitter, but its absence is more bitter.

The Black Sequels

When compressive forces persist and/or intensify, the defenses of the most resilient axons progressively crumble. These consist of axons transmitting touch and temperature sensation, or motor axons carrying voluntary movement commands and spinal reflex responses. Thus, after pain comes sensory chaos. Then follows motor weakness and disruption of spinal reflexes.

This is a profoundly logical sequence: Motor axons transmitting voluntary and reflex commands have the largest diameter, thickest walls, and greatest resistance to external compression. Hence, symptoms and signs of their injury appear last in the clinical findings. Their presence signals the depth and gravity of neural damage.

Perfect Your Timing

Pain is a glaring symptom we must recognize. Whenever pain appears alone—without sensory or motor accompaniments—the prognosis is at its best. If pain coincides with sensory changes, prognosis worsens. If it coincides with motor dysfunction, prognosis darkens further still.

Act swiftly for the nerve's sake. Sharpen your ears to its whispers. Attend to its

confession. Answer its plea; redress its grievance. Conservative treatment must be uncompromisingly strict. Avoid analgesics whenever possible—they may suppress the nerve's fury and silence its voice, but they do not heal it. If neurological distress persists beyond three months, intervene—explore the nerve surgically and liberate it from its constraints.

Do not hesitate to operate to rescue the nerve, even if electromyography (EMG) shows no pathological findings. EMG abnormalities lag behind the nerve's true suffering. How could it be otherwise? Electroneurodiagnostics do not align with the true mechanics of neural transmission.

Until you discover a more efficacious approach, adhere to the strategy of repeated clinical examination. And do not forget yourself. Listen to your inner voice—to your intuition—for they still hold the primary role here. Draw deeply from the wellspring of your experience. Long companionship with nerves imparts their fundamental alphabet and grants you keys to their rich universe. Thus, you enter their intimacies and perceive what remains veiled to many others—a privilege bestowed only upon you.

Critical Note:

My perspective on Electroneurography is profoundly divergent. Read my article:
Electroneurography vs. Neural Reality: Hidden Fallacies in Nerve Conduction Studies

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In other contexts, you can also read the following articles:

- *DOI The Spinal Reflex, New Hypothesis of Physiology*
- *The Hyperreflexia, Innovated Pathophysiology*
- *DOI The Spinal Shock*
- *The Spinal Injury, the Pathophysiology of the Spinal Shock, the Pathophysiology of the Hyperreflexia*
- *DOI Upper Motor Neuron Lesions, the Pathophysiology of the Symptomatology*
- *The Hyperreflexia (1), the Pathophysiology of Hyperactivity*
- *The Hyperreflexia (2), the Pathophysiology of Bilateral Responses*
- *The Hyperreflexia (3), the Pathophysiology of Extended Hyperreflex*

- *The Hyperreflexia (4), the Pathophysiology of Multi-Response Hyperreflex*
- *The pathophysiology of Triple flexion Reflex*
- *The Clonus, 1st Hypothesis of Pathophysiology*
- *The Clonus, 2nd Hypothesis of Pathophysiology*

DOI *The Clonus, Two Hypotheses of Pathophysiology*

DOI *The Nerve Transmission through Neural Fiber, Personal View vs. International View*

- *The Nerve Transmission through Neural Fiber (1), The Action Pressure Waves*
- *The Nerve Transmission through Neural Fiber (2), The Action Potentials*
- *The Nerve Transmission through Neural Fiber (3), The Action Electrical Currents*
- *The Function of Standard Action Potentials & Currents*
- *The Three Phases of Nerve transmission*

DOI *Neural Conduction in the Synapse (Innovated)*

DOI *Nodes of Ranvier, the Equalizers*

- *Nodes of Ranvier, the Functions*
- *Nodes of Ranvier, First Function*
- *Nodes of Ranvier, Second Function*
- *Nodes of Ranvier, Third Function*
- *Node of Ranvier, The Anatomy*

- *The Wallerian Degeneration*
- *The Neural Regeneration*
- *The Wallerian Degeneration Attacks Motor Axons, While Avoids Sensory Axons*

DOI *The Sensory Receptors*

DOI *Electroneurography vs. Neural Reality: Hidden Fallacies in Nerve Conduction Studies*

DOI *Piriformis Muscle Injection: Personal Approach*

- *The Philosophy of Pain, Pain Comes First! (Innovated)*
- *The Philosophy of the Form (Innovated)*
- *Pronator Teres Syndrome, Struthers-Like Ligament (Innovated)*
- *Ulnar Nerve, Congenital Bilateral Dislocation*
- *Posterior Interosseous Nerve Syndrome*
- *The Multiple Sclerosis: The Causative Relationship Between The Galvanic Current & Multiple Sclerosis?*
- *Cauda Equina Injury, New Surgical Approach*

DOI *Carpal Tunnel Syndrome Ends Its Adherence: Complete Median Nerve Transection*

DOI *Biceps Femoris' Long Head Syndrome (BFLHS)*

DOI *Barr Body, The Whole Story (Innovated)*

- *Adam's Rib and Adam's Apple, Two Faces of one Sin*
- *Adam's Rib, could be the Original Sin?*
- *Barr Body, the Second Look*

DOI *Who Decides the Sex of Coming Baby?*

- *Boy or Girl, Mother Decides!*
- *Oocytogenesis*
- *Spermatogenesis*
- *This Woman Can Only Give Birth to Female Children*
- *This Woman Can Only Give Birth to Male Children*
- *This Woman Can Give Birth to Female Children More Than to Male Children*
- *This Woman Can Give Birth to Male Children More Than to Female Children*
- *This Woman Can Equally Give Birth to Male Children & to Female Children*

- *Eve Saved Human Identity; Adam Ensured Human Adaptation*

DOI *COVID-19: Beyond the Crisis—Is It Targeting Our Genes?*

DOI *Fibromyalgia*

- *Mitosis in Animal Cell*
- *Meiosis*

- *Universe Creation, Hypothesis of Continuous Cosmic Nebula*
- *Circulating Sweepers*

- *The Black Hole is a (the) Falling Star?*

- *Pneumatic Petrous, Bilateral Temporal Hyperpneumatization*

DOI *Congenital Bilateral Thenar Hypoplasia*

DOI *Ulnar Dimelia, Mirror hand Deformity*

DOI *Thumb Reconstruction Using Microvascular Second Toe to Thumb Transfer*

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DOI *Liver Hemangioma: Urgent Surgery of Giant Liver Hemangioma Due to Intra-Tumor Bleeding*

DOI *Free Para Scapular Flap (FPSF) for Skin Reconstruction*

DOI *Claw Hand Deformity (Brand Operation)*

DOI *Algodystrophy Syndrome Complicated by Constricting Ring at the Proximal Border of the Edema*

DOI *Non- Traumatic Non- Embolic Acute Thrombosis of Radial Artery (Buerger's Disease)*

DOI *Isolated Axillary Tuberculosis Lymphadenitis*

DOI *The Iliopsoas Tendonitis... The Snapping Hip*

To read the article in Arabic, click on →

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DOI *The Delirium of the Economy*

DOI *Ovaries in a Secure Corner… Testicles in a Humble Sac: An Inquiry into the Function of Form*

DOI *Eve Preserves Humanity's Blueprint; Adam Drives Its Evolution*

DOI *The Manufacture of the Unconscious*

DOI *The Ballad of Eternity*

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