AMMAR YASEEN MANSOUR

The Wallerian Degeneration

Attacks the Motor Axons of Injured Nerve, while Conserves the Sensory Axons

N.B. the Arabic version of this article is the reference, read it on the following links:

التَّنكُّسُ الفالِيريُّ، يهاجمُ المحاورَ العصبيَّةَ الحركيَّةَ للعصب.. ويعفُّ عن المحاور الحسِّيَّة

It is my personal view of the Wallerian degeneration, and of the neural regeneration process in both the motor and the sensory axons, vis-à-vis the traditional view.

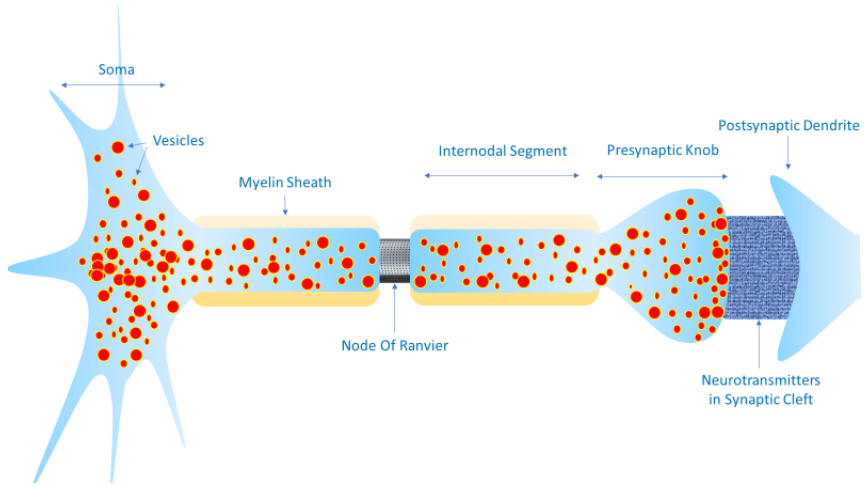
These readings are principally based on my post-surgical findings of many surgical repairs of peripheral nerves. In some of which, I noticed a rapid return of an appreciable protective sensation in the nerve-dependent areas regardless of the site and the date of nerve injury. In three subsequent figures, I will illustrate my point of view.

1. The Wallerian Degeneration

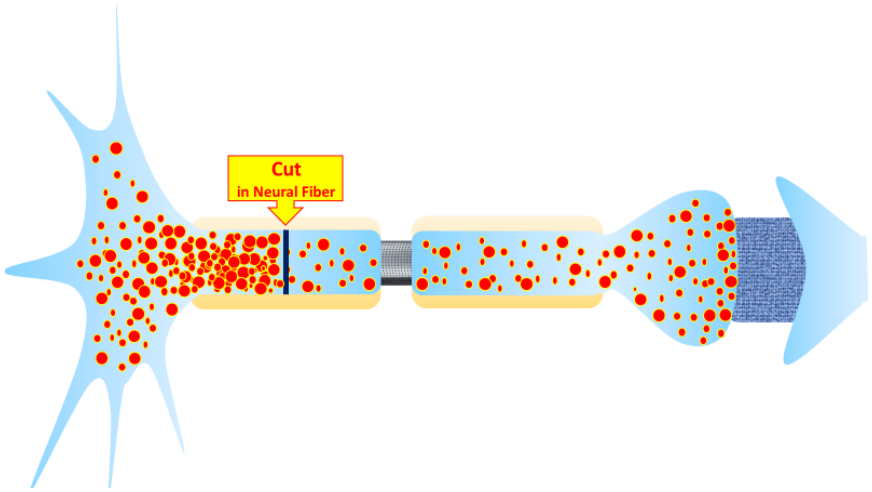
For mor details concerning this item, see the linked video:

Personally, I do believe the Wallerian degeneration to be an act that exclusively concerns the motor axons, while conserves the sensory axons. Moreover, I do refer the Wallerian degeneration to the vesicles' disappearance, and the neurotransmitter's disappearance as well, from the distal segment (distal to of the site of injury) of the injured motor axon and from the synaptic cleft respectively.

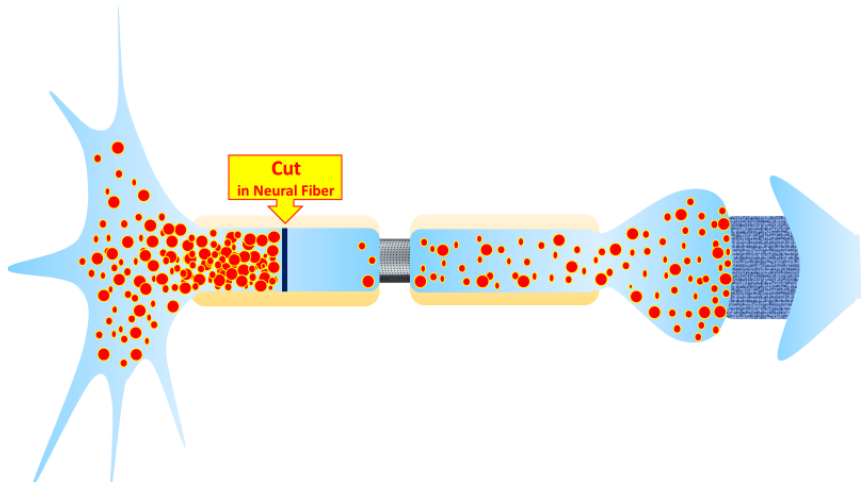
The Wallerian degeneration is a slow process, and is totally influenced by the velocity of vesicles' migration (1-3 mm/ day), as well as the distance from the site of injury to the terminus in the synapses and the synaptic clefts; ***figure (1).***


Soma
Vesicles
Myelin Sheath
Internodal Segment
Presynaptic Knob
Postsynaptic Dendrite
Node Of Ranvier
Neurotransmitters
in Synaptic Cleft

A


Cut
in Neural Fiber

B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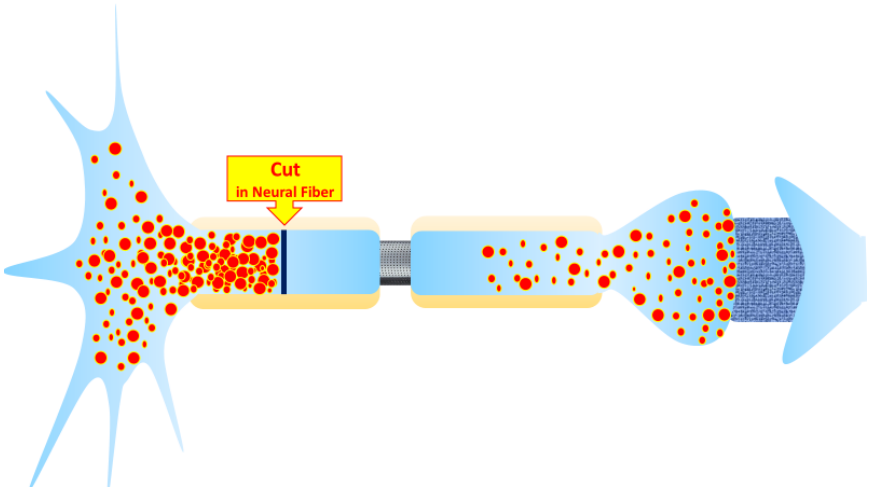

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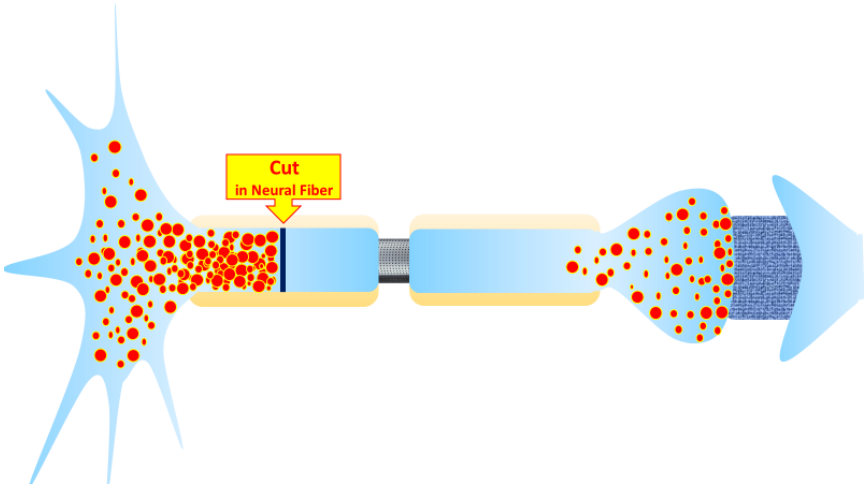

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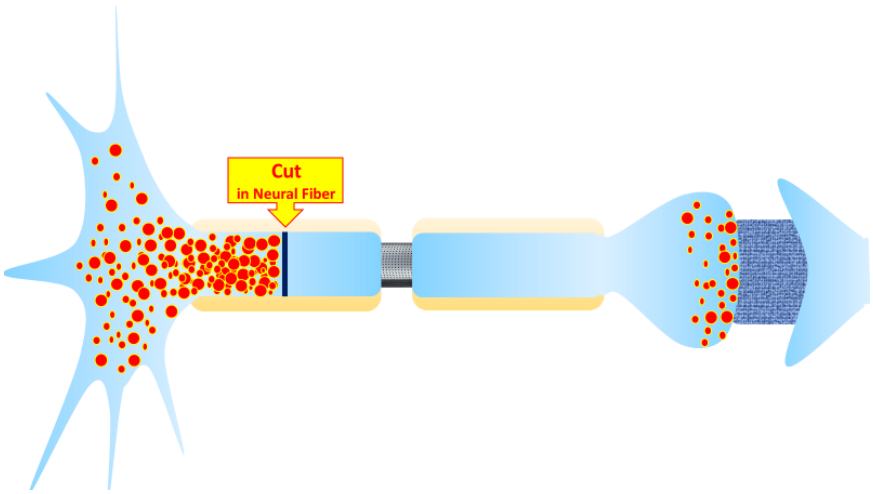

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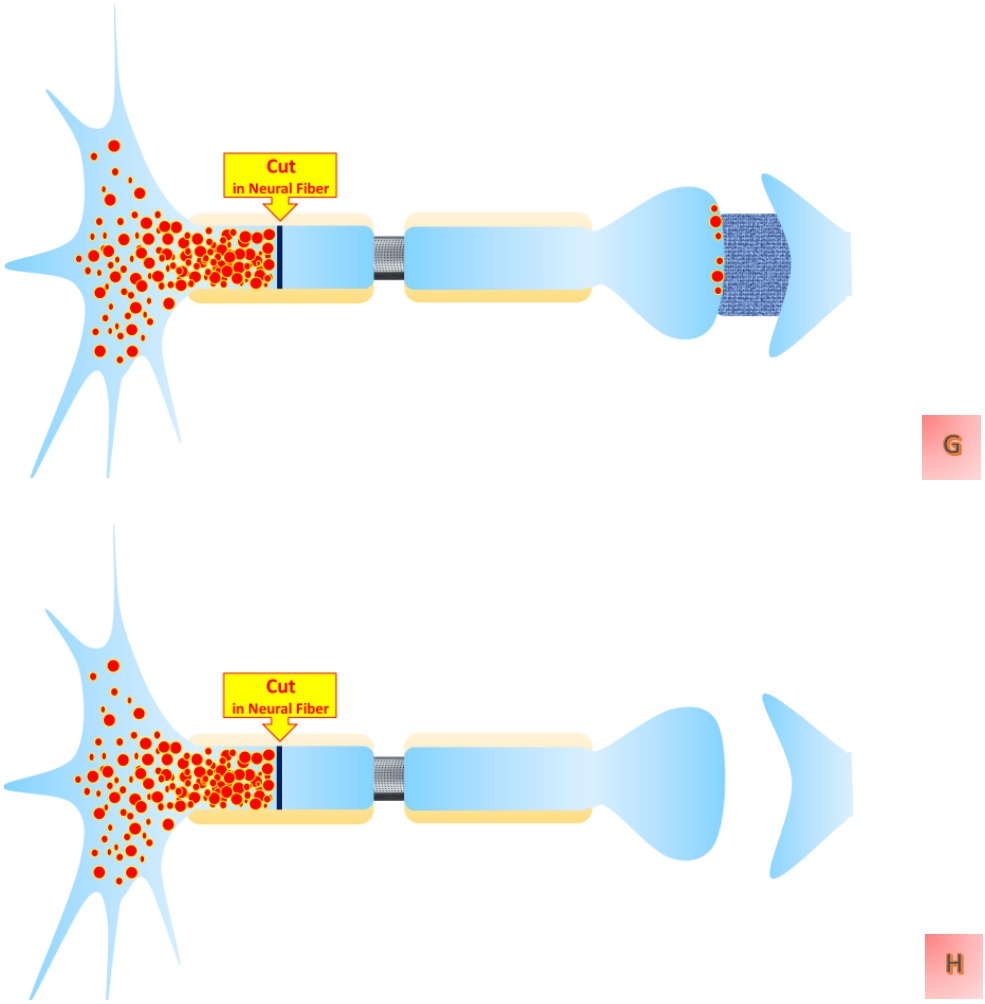


Figure (1)
The Wallerian Degeneration in the Motor Axon

For mor details concerning this item, see the linked video:

Figure (A): *The functional anatomy of a normal axon and a normal synapse. At rest as well as in action, the vesicles slowly migrate (1-3 mm/ day) from the site of fabrication in the cell body (the Soma) toward the theatre of their action in the synapses. In the synapses, the vesicles continuously inject their burdens of neurotransmitter into the synaptic cleft. The neurotransmitter fills the space of the synaptic cleft, rendering it good conductor to the electrical current. Such an act is repeated at rest and in action as well. So that, the synaptic cleft uninterruptedly is ready to pass the electrical currents to another side of the synapses.*

Figure (B) &(C) &(D) &(E) &(F): *After nerve injury, the vesicles' migration stops at the site of injury. Since, the vesicles' fabrication in the cell body continues, the vesicles gradually accumulate in the proximal portion of the motor axon (i.e., proximal to the site of injury). These restrained vesicles stay waiting the natural or surgical restoration of the axon continuity in order to pass into the distal portion of the axon (i.e., distal to the site of injury). However, in the distal segment of the injured axon, the vesicles continue their migration toward the synapses.*

From proximal to distal, the act of vesicles' disappearance announces the beginning of Wallerian degeneration process.

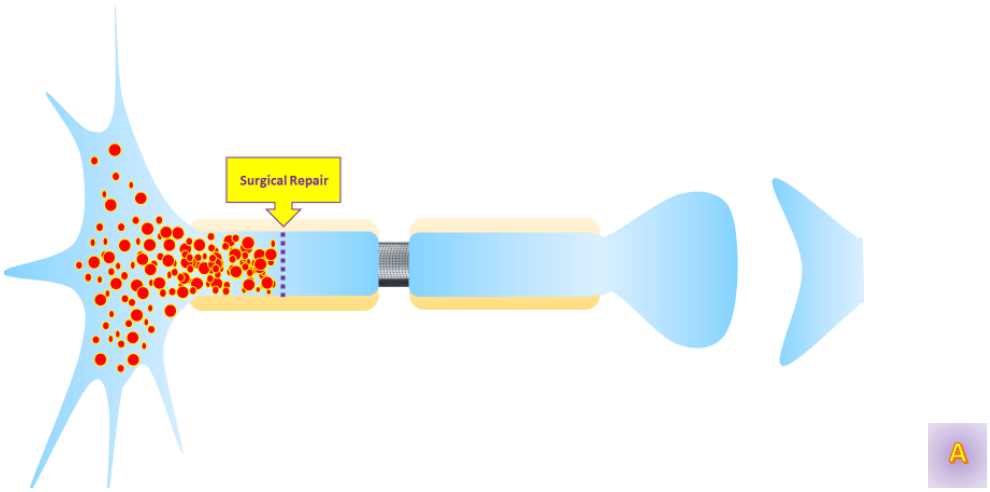
Figures (G): *In the synapse, the vesicles are gradually consumed, then disappear from the total distal portion of the axon. Since the neurotransmitters are still present, the synaptic cleft remains apt to pass the electrical impulse to another side of the synapses. In such a way, some responses in the electromyogram do exist indicating incomplete Wallerian degeneration.*

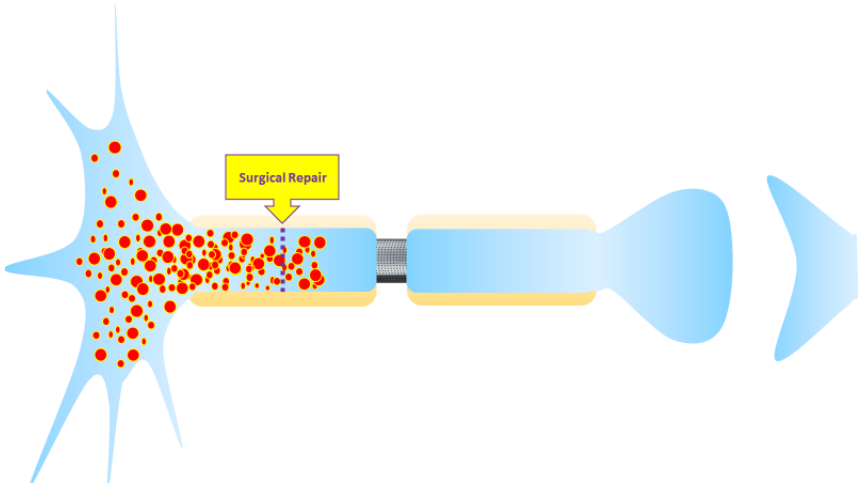
Figure (H): *Herein, one could officially announce the end of process of Wallerian degeneration. No more vesicles nor neurotransmitters exist in the lumen of the distal portion of the injured axon and in the synaptic cleft respectively. No more responses could be detected on the Electromyograph.*

2. The Neural Regeneration

For mor details concerning this item, see the linked video:

Personally, I do consider the neural regeneration to be strictly identified by the reappearance of vesicles in the distal portion of the injured axon. Precisely, I do consider the start point of neural regeneration to be when the restrained vesicles traverse the suture line for the first time. While, the end point of neural regeneration should be when the migrating vesicles refill the synapses, and afterword inject their burdens of neurotransmitter into the synaptic clefts; ***figure (2).***





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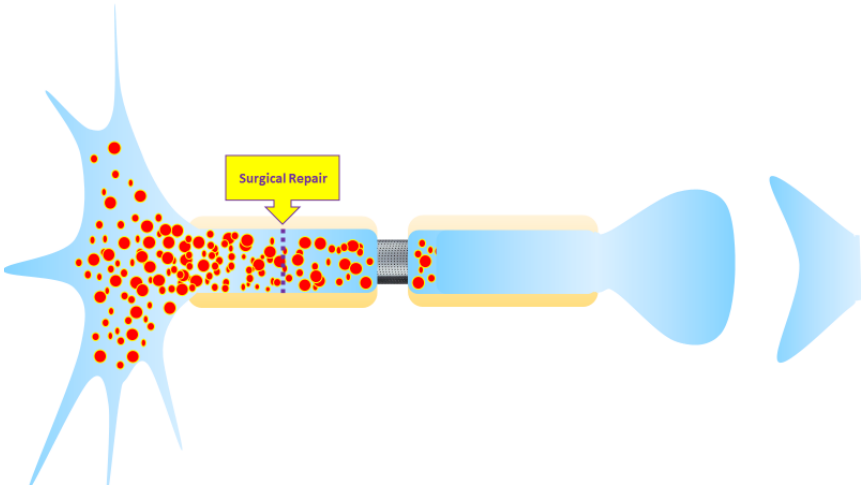

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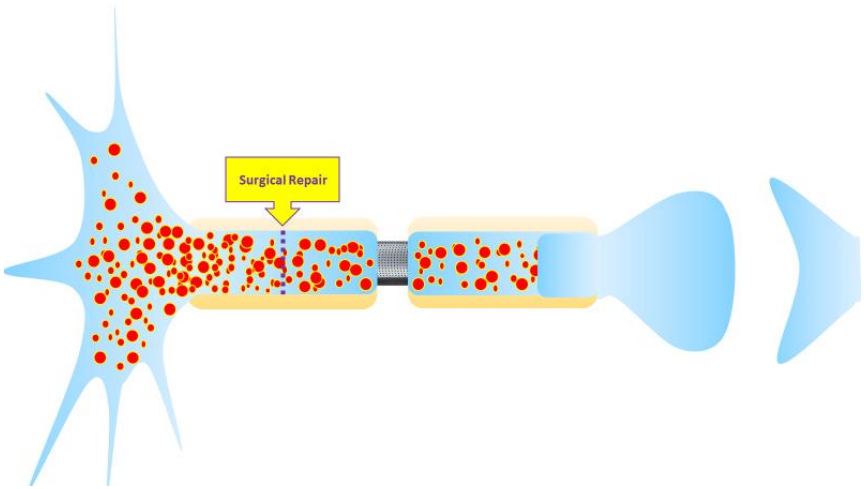

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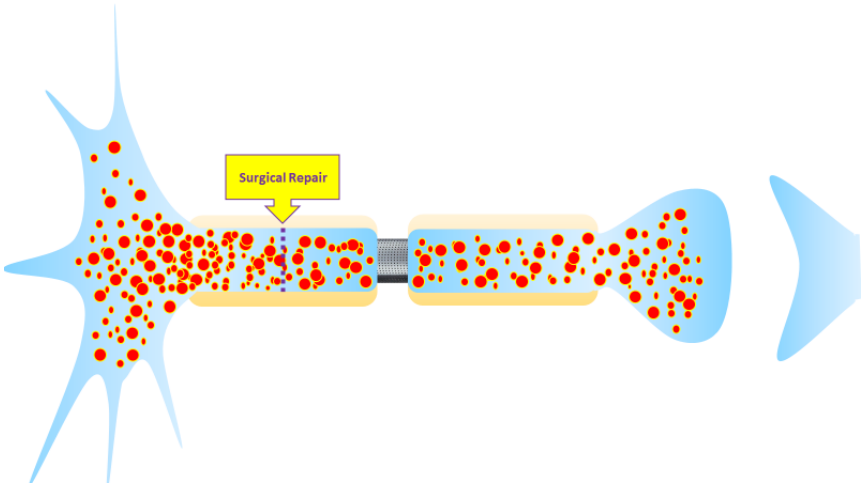

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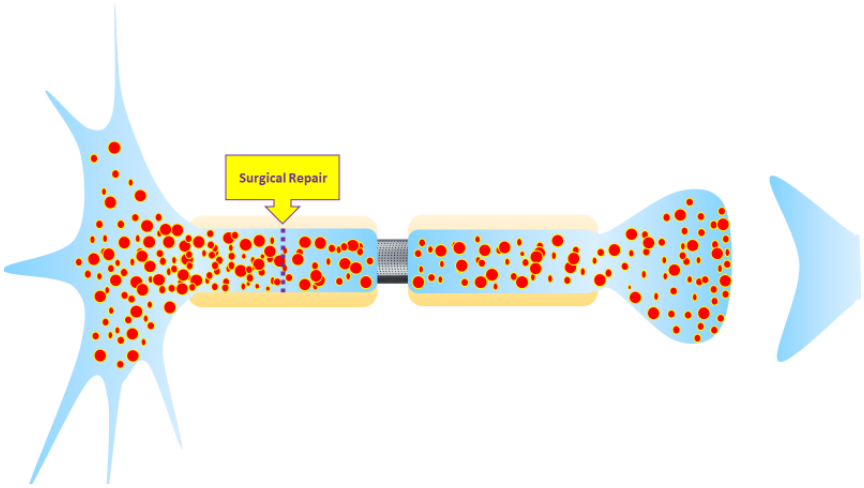

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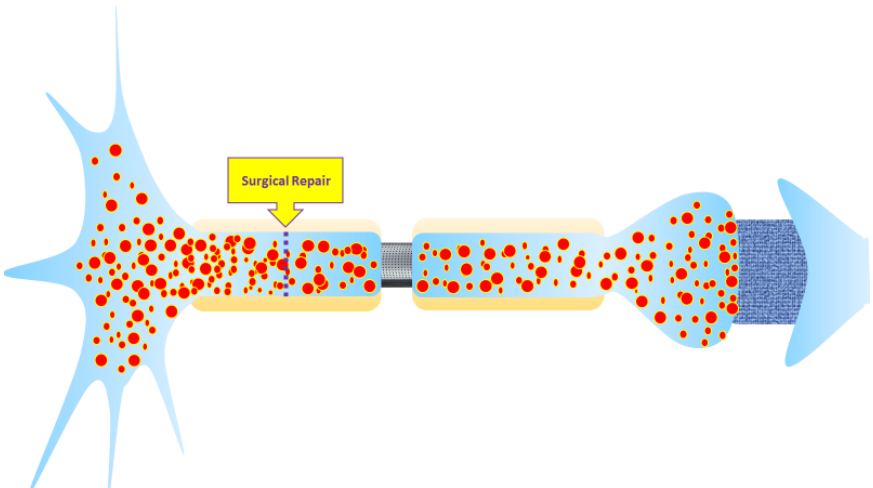

G

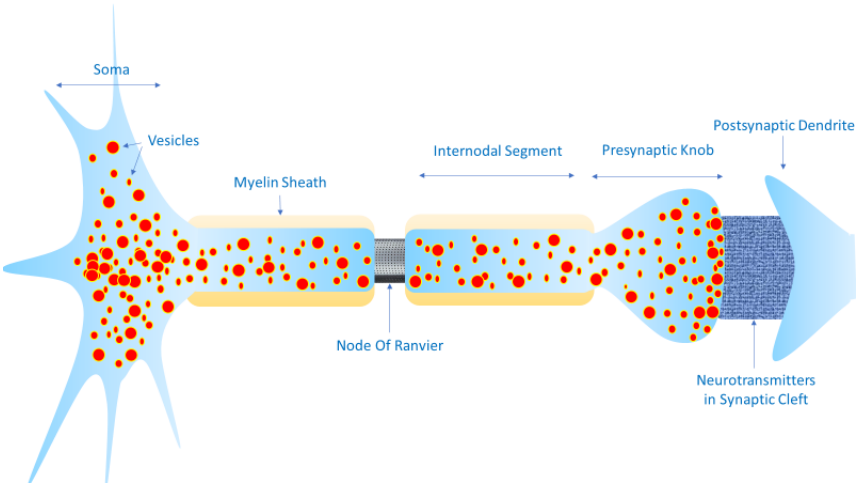

Figure (2)
The Neural Regeneration

For mor details concerning this item, see the linked video:

Figure (A): *After the restoration of the continuity of the axon, the restrained vesicles regain liberty and escape the suture line declaring the beginning of the neural regeneration. These recently liberated vesicles slowly invade the lumen of the distal portion of axon looking forward the synapses in the periphery. The neural regeneration progress in a slow manner due to the slow rhythm of vesicles' migration toward the synapses.*

Figures (B) &(C) &(D) &(E) & (F): *The recently liberated vesicles as well as the new born vesicles keep moving toward the synapses in a rhythm of 1 to 3 mm/day. It happens that the vesicles do occupy the lumen of the Knob of the synapses, however, they do not yet inject the neurotransmitter into the synaptic clefts. Hence, we do not expect obtaining any response on the Electromyograph. The process of neural regeneration is still missing its final step.*

Figure (G): *The final step of the neural regeneration comes when the vesicles sufficiently inject the neurotransmitter into the synaptic cleft. The neurotransmitter renders the synaptic cleft good conductor of electricity. Henceforth, we could obtain some responses on the Electromyograph.*

Figure (H): *The functional anatomy of a normal axon and a normal synapse.*

3. The Neural Regeneration in the Sensory Axon

Since the Wallerian degeneration, and the neural regeneration, are strictly linked to the Vesicles' disappearance and the vesicles' reappearance inside the distal portion of the injured axon, respectively, I do believe the Wallerian degeneration does not exist in the sensory axons; ***figure (3).***

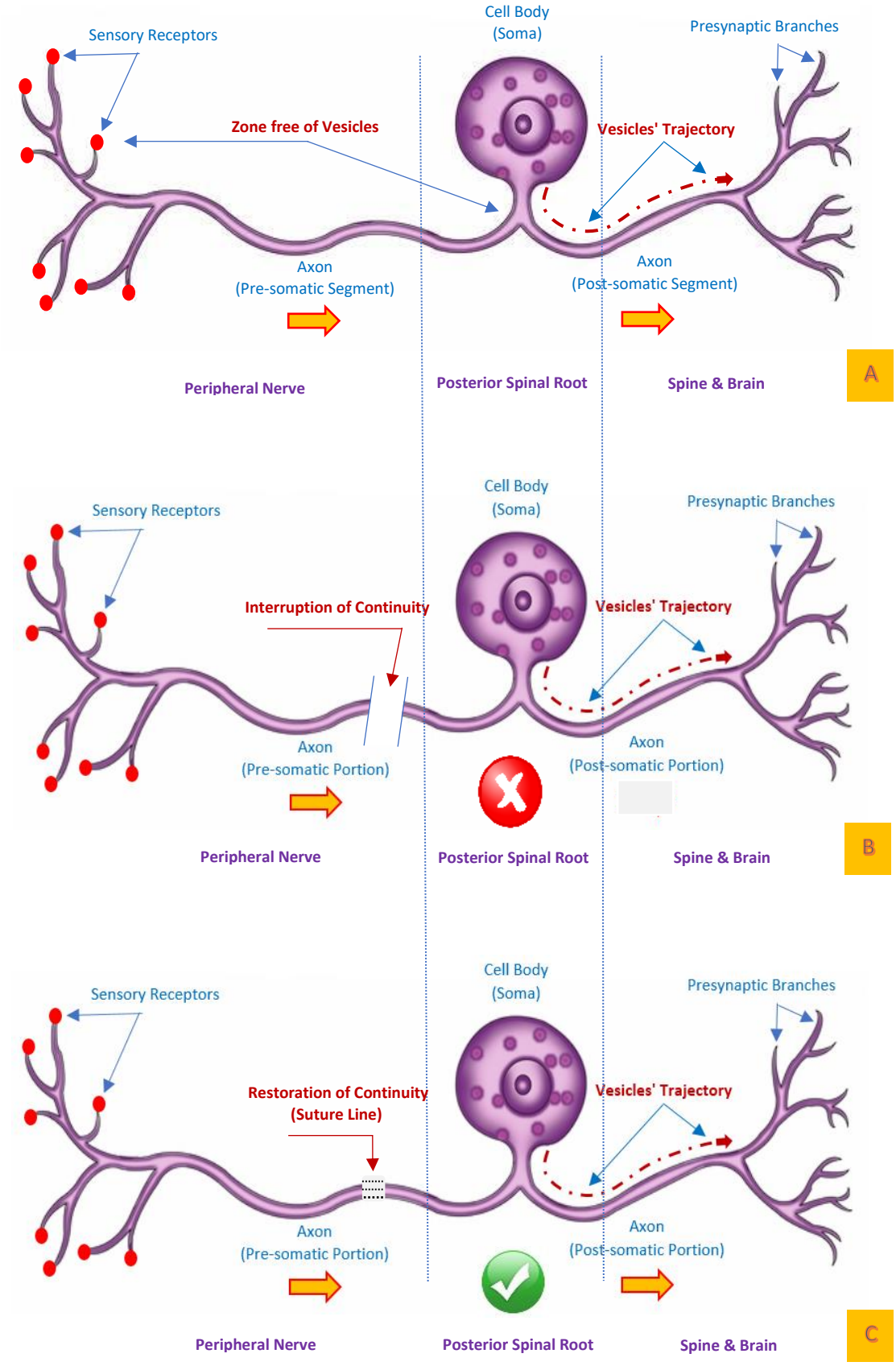
Sensory Receptors
Cell Body
(Soma)
Presynaptic Branches
Zone free of Vesicles
Vesicles' Trajectory
Axon
(Pre-somatic Segment)
Axon
(Post-somatic Segment)
Peripheral Nerve
Posterior Spinal Root
Spine & Brain
A
Sensory Receptors
Cell Body
(Soma)
Presynaptic Branches
Interruption of Continuity
Vesicles' Trajectory
Axon
(Pre-somatic Portion)
Axon
(Post-somatic Portion)
Peripheral Nerve
Posterior Spinal Root
Spine & Brain
B
Sensory Receptors
Cell Body
(Soma)
Presynaptic Branches
Restoration of Continuity
(Suture Line)
Vesicles' Trajectory
Axon
(Pre-somatic Portion)
Axon
(Post-somatic Portion)
Peripheral Nerve
Posterior Spinal Root
Spine & Brain
C

Figure (3)
The Neural Regeneration of the Sensory Axon

Figure (A): *The sensory neuron has some anatomical specificities. The sensory axon has two distinguishable portions; the pre-somatic portion and the post-somatic portion. The pre-somatic segment of the sensory axon connects the sensory receptors to the cell body (the Soma), while the post-somatic segment relates the soma to the dendrites. The pre-somatic portion is an essential element of the peripheral nerve, while the post-somatic portion resides within the posterior spinal root or merges within one of the ascending sensory tracts. The pre-somatic portion has no vesicles within its cytoplasm, while the post-somatic portion forms the unique pathway of these vesicles.*

Figure (B): *After peripheral nerve injury, only the pre-somatic segment of the sensory axon is damaged, while the post-somatic portion is quite conserved. Since, there is no vesicles within this portion of the sensory axon, the famous Wallerian degeneration does not occur. The injury simply induces an interruption of axon continuity, hence stops the neural conduction via the axon. No other structural changes could be detected.*

Figure (C): *Upon restoration of the axon continuity, the current of neural conduction rapidly regains its patent canal. Therefore, the sensory afferent impulses directly pass into the post-somatic portion. Since, the vesicles' life- cycle, and consequently the neurotransmitter's life-cycle, have not endamaged by the trauma, the neurotransmitter will be always present in the synaptic cleft. Thus, the sensory afferent impulses could fluently pass to another side of the synapses.*

This innovated conception is consistent with my personal post- surgical findings. In many cases, the patient regains his (her) good protective sensations shortly (in few days, even directly) after the surgical repair of the injured nerve (s). Some of these injuries were old and at high position (ex. at wrist or forearm).

4. The Conclusions

- *The Wallerian degeneration is strictly related to the vesicles' disappearance, and the consequent neurotransmitter's disappearance, from the distal portion of injured axon and from the synaptic cleft respectively.*
- *Since there are no vesicles in the peripheral sensory tracts and they are only present in the motor axons, the Wallerian degeneration is a pathological process exclusively concerns the motor neurons.*
- *In the sensory axon, the life cycle of both the vesicles and the neurotransmitter are out of danger in the peripheral nerve injuries. Therefore, the sensory axons rest free of a such pathological process called the Wallerian degeneration.*

- *The neural regeneration is strongly related to the return of both the vesicles and the neurotransmitter to the synapse and to the synaptic cleft respectively.*
- *The neural regeneration is a slow process due to the slow rhythm of vesicles' migration toward the synapse (1-3 mm/ day).*
- *Regarding the important role of the vesicles' reappearance in the healing process, the neural regeneration should be restricted to describe the return of the neural conduction in the motor axons only. Therefore, the neural regeneration of the motor axons is a slow process (1-3 mm/ day). Personally, I did not see any rapid return of movement in any of my surgical cases. However, we could expect a rapid return of function in the sensory axon, wherein the vesicles have no role in the healing process.*

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In another context, one can also read:

- *Neural Conduction, Personal View vs. International View (Innovated)*
- *Upper Motor Neuron Lesions, Pathophysiology of Symptomatology*
- *Neural Conduction, Action Pressure Waves (Innovated)*
- *Neural Conduction, Action Potentials (Innovated)*
- *Neural Conduction, Action Electrical Currents (Innovated)*
- *The Function of Action Potentials (Innovated)*
- *The Three Phases of Neural Conduction (Innovated)*
- *Neural Conduction in the Synapse (Innovated)*
- *Sensory Receptors*
- *Nodes of Ranvier, the Equalizers (Innovated)*
- *Nodes of Ranvier, the Functions (Innovated)*
- *Nodes of Ranvier, First Function (Innovated)*
- *Nodes of Ranvier, Second Function (Innovated)*
- *Nodes of Ranvier, Third Function (Innovated)*

- *Node of Ranvier.. The Anatomy*
- *The Philosophy of Pain, Pain Comes First! (Innovated)*
- *The Philosophy of the Form (Innovated)*
- *Spinal Injury, Pathophysiology of Spinal Shock, Pathophysiology of Hyperreflexia*
- *Who Decides the Sex of Coming Baby?*
- *Spinal Shock (Innovated)*
- *The Clonus (Innovated)*
- *Hyperactivity Hyperreflexia (Innovated)*
- *Hyperreflexia, Extended Sector of Reflex*
- *Hyperreflexia, Bilateral Responses*
- *Hyperreflexia, Multiple Responses*
- *Nerve Conduction Study, Wrong Hypothesis is the Origin of the Misinterpretation (Innovated)*
- *Wallerian Degeneration (Innovated)*
- *Neural Regeneration (Innovated)*
- *Wallerian Degeneration Attacks Motor Axons, While Avoids Sensory Axons*
- *Barr Body, the Whole Story (Innovated)*
- *Boy or Girl, Mother Decides!*
- *Adam's Rib and Adam's Apple, Two Faces of one Sin*
- *The Black Hole is a (the) Falling Star?*
- *Adam's Rib, could be the Original Sin?*
- *Pronator Teres Syndrome, Struthers Like Ligament (Innovated)*
- *Function of Standard Action Potentials & Currents*
- *Posterior Interosseous Nerve Syndrome*
- *Spinal Reflex, New Hypothesis of Physiology*

- *Hyperreflexia, Innovated Pathophysiology*
- *Clonus, 1st Hypothesis of Pathophysiology*
- *Clonus, 2nd Hypothesis of Pathophysiology*
- *Clonus, Two Hypotheses of Pathophysiology*
- *Hyperreflexia (1), Pathophysiology of Hyperactivity*
- *Hyperreflexia (2), Pathophysiology of bilateral Responses*
- *Hyperreflexia (3), Pathophysiology of Extended Hyperreflex*
- *Hyperreflexia (4), Pathophysiology of Multi-Response Hyperreflex*
- *Barr Body, the Second Look*
- *Mitosis in Animal Cell*
- *Meiosis*
- *Universe Creation, Hypothesis of Continuous Cosmic Nebula*
- *Circulating Sweepers*
- *Pneumatic Petrous, Bilateral Temporal Hyper pneumatization*
- *Ulnar Nerve, Congenital Bilateral Dislocation*
- *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*
- *Piriformis Muscle Injection_ Personal Approach*

- *Eve Saved Human's Identity, Adam Ensured Human's Adaptation*
- *Corona Virus (Covid-19): After Humiliation, Is Targeting Our Genes*
- *Claw Hand Deformity (Brand Operation)*
- *Corona Virus (Covid-19): After Humiliation, Is Targeting Our Genes*
- *Barr Body; Mystery of Origin & Ignorance of Function*
- *The Multiple Sclerosis: The Causative Relationship Between The Galvanic Current & Multiple Sclerosis?*
- *Liver Hemangioma: Urgent Surgery of Giant Liver Hemangioma Because of Intra-Tumor Bleeding*
- *Cauda Equina Injury, New Surgical Approach*
- *Ulnar Dimelia, Mirror hand Deformity*
- *Carpal Tunnel Syndrome Complicated by Complete Rupture of Median Nerve*
- *Presacral Schwannoma*
- *Congenital Bilateral Thenar Hypoplasia*
- *Biceps Femoris' Long Head Syndrome (BFLHS)*
- *Algodystrophy Syndrome Complicated by Constricting Ring at the Proximal Border of the Edema*
- *Mandible Reconstruction Using Free Fibula Flap*
- *Non- Traumatic Non- Embolic Acute Thrombosis of Radial Artery (Buerger's Disease)*
- *Isolated Axillary Tuberculosis Lymphadenitis*
- *Free Para Scapular Flap (FPSF) for Skin Reconstruction*
- *Three Steps of Neural Conduction*

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