

Tutorial 8: Saltatory Conduction

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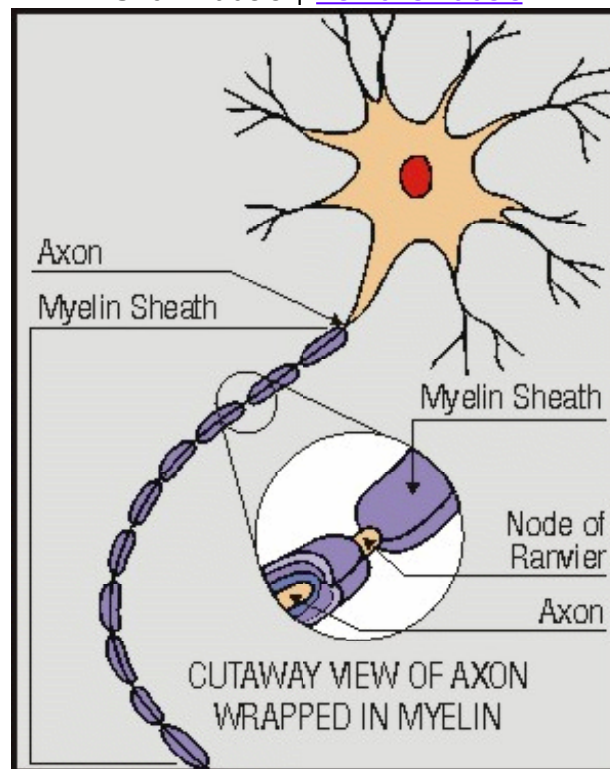


Figure 8a: Saltatory Conduction

[Intro](#) | [Figure 8a](#) | [Figure 8b](#)

[Part 1: Image-Mapped Tutorial](#)

[Part 2: Matching Self-Test](#)

[Part 3: Multiple-Choice Self-Test](#)

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Tutorial 8 discusses the anatomy and physiology that increase the rate at which an action potential is conducted down the axon. [Figure 8a](#) focuses on the relevant anatomy, while [Figure 8b](#) describes the physiological mechanism. *Saltatory conduction* (from the Latin word "to dance"), as this enhanced conduction is called, depends on the myelin sheaths introduced in [Tutorial 2](#). Saltatory conduction provides two advantages over conduction that occurs along an axon without myelin sheaths. First, it saves energy by decreasing the use of sodium-potassium pumps in the axonal membrane. Secondly, the increased speed afforded by this mode of conduction allows the organism to react and think faster.

Advanced

Multiple sclerosis is a common neurological disorder characterized by patchy demyelination of axons in the central nervous system (Giovannoni & Miller, 1999). The symptoms of this disorder reflect the disruption of normal function of the neurons affected by demyelination. These symptoms commonly remit for a period of time after their onset. The exacerbation and reduction of symptoms follow a cycle of demyelination and remyelination. It was not known until very recently, what mechanism was responsible for the remyelination process.

Until recently a precursor cell for the oligodendrocyte and Schwann cell had been identified in rodents,

but not humans (Engel & Wolswijk G, 1996). The identification of this so-called O-2A, proliferative progenitor cell both in human culture and in human brain white matter, indicates that this cell type may indeed underlie the fortuitous remyelination that may occur in multiple sclerosis (Scolding, Rayner & Compston, 1999). In yet another recent study this progenitor cell was transplanted into the spinal cords of rats (Brustle, et al., 1999; Ijichi, Noel, Sakuma, Weil & Tofilon, 1996). The transplanted cells promoted the growth of myelin in the rat nervous system. Science is moving closer to a possible remedy for multiple sclerosis and other demyelinating diseases.

Other neurobiological laboratories have focused on gaining a better understanding of the composition and function of oligodendrocytes and Schwann cells (Coetzee, Suzuki, Nave & Popko, 1999; Dupree & Popko, 1999; Dupree, Suzuki & Popko, 1998). These myelinating cells are composed of a number of different fatty, lipid molecules. Recent research indicates that the glycolipids, including galactocerebroside and galactosulfatide, are essential for saltatory conduction to occur. The deficiency of these particular glycolipids causes increased fluidity, permeability, and poor packing of the myelin layer of the axonal membrane underlying the myelin sheath. Identification and manipulation of the genes that produce these lipids may provide yet another avenue of treatment for demyelinating disorders.

We have known for some time that sodium channels are found in clusters at the Nodes of Ranvier, where they serve their purpose in saltatory conduction. Other recent research has found that sodium channels redistribute themselves along the length of the demyelinating axon (England, Levinson & Shrager, 1996). Since this redistribution underlies recovery of conduction along the demyelinating axon (albeit, inefficient conduction), scientists have studied the relevant mechanisms. This research has found that Schwann cells of the peripheral nervous system mediate a proliferation of sodium channels at the edge of the nodes acutely following the onset of demyelination (Novakovic, Deerinck, Levinson, Shrager & Ellisman, 1996). The redistribution of these sodium channels along the exposed axon over time is mediated by the neuron itself. Short of stopping the demyelination process or triggering the remyelination process, perhaps building on this knowledge science will be able to speed the recovery of conduction following demyelination.

References

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Suggestions for further study

SUGGESTED READINGS:

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