

<b>Editorial</b>
------------------

## Neurogliamodulation

---

Howard S. Smith, MD

---

**T**he field of neuromodulation continues to blossom especially with respect to its use in efforts to achieve analgesia. The journal *Neuromodulation* as well as the recently written text: *Neuromodulation* (Eds. Krames E, Hunter Peckham P, Rezai A from Elsevier) scheduled to come out in May 2009 are evidence that neuromodulation is an important part of clinical medicine and pain medicine that needs to be appreciated. Neuromodulation for analgesia may be utilized in both the peripheral and central nervous systems (Fig. 1). However, at least with respect to pain medicine, the term neuromodulation may be too restrictive and this author postulates that a potential mechanism which may partially contribute to the analgesia achieved from spinal or supraspinal electrical stimulation could, in fact, come from electrical stimulation of glial cells or gliamodulation.

Electrical stimulation of glial cells on the border between the alveus of the rat hippocampal CA1 region and stratum oriens evoked inward currents through several routes involving glutamate receptors and inward rectifier K<sup>+</sup> channels (1). Moreover, electrical stimulation resembling in vivo activity evoked long-lasting depolarization (1). Yamazaki et al (1) performed dual, whole-cell recording on CA1 pyramidal neurons and oligodendrocytes in efforts to examine the modulatory effects of oligodendrocytes on neuronal activities. Direct depolarization of oligodendrocytes shortened the latencies of action potentials evoked by antidromic stimulation (1). These results indicate that oligodendrocytes increase the conduction velocity of action potentials by a mechanism additional to saltatory conduction and that they have active roles in information processing in the brain (1).

A category of glial cells has been identified that can fire action potentials, and their excitation is driven by synapses from axons (2). This finding may have relevance to excitotoxicity in ischemia, but the normal function may be to regulate myelination according to functional activity in axons (2). Action potential propagation through CNS axons can be rapidly regulated by oligodendrocytes (2). Mature oligodendrocytes in the rat hippocampus are depolarized by theta burst stimulation of axons, and when the oligodendrocytes are depolarized by current injection in paired whole-cell recordings with CA1 pyramidal neurons, the latency of impulse conduction through the axons it ensheathes rapidly decreases (2). This suggests a dynamic role for myelin in regulating impulse transmission through axons, promoting neural synchrony among the multiple axons under the domain of an individual oligodendrocyte (2).

Thus, it is proposed that neurogliamodulation may be a broader and more accurate and precise term to use for spinal or supraspinal electrical stimulation when utilized in efforts to achieve analgesia.

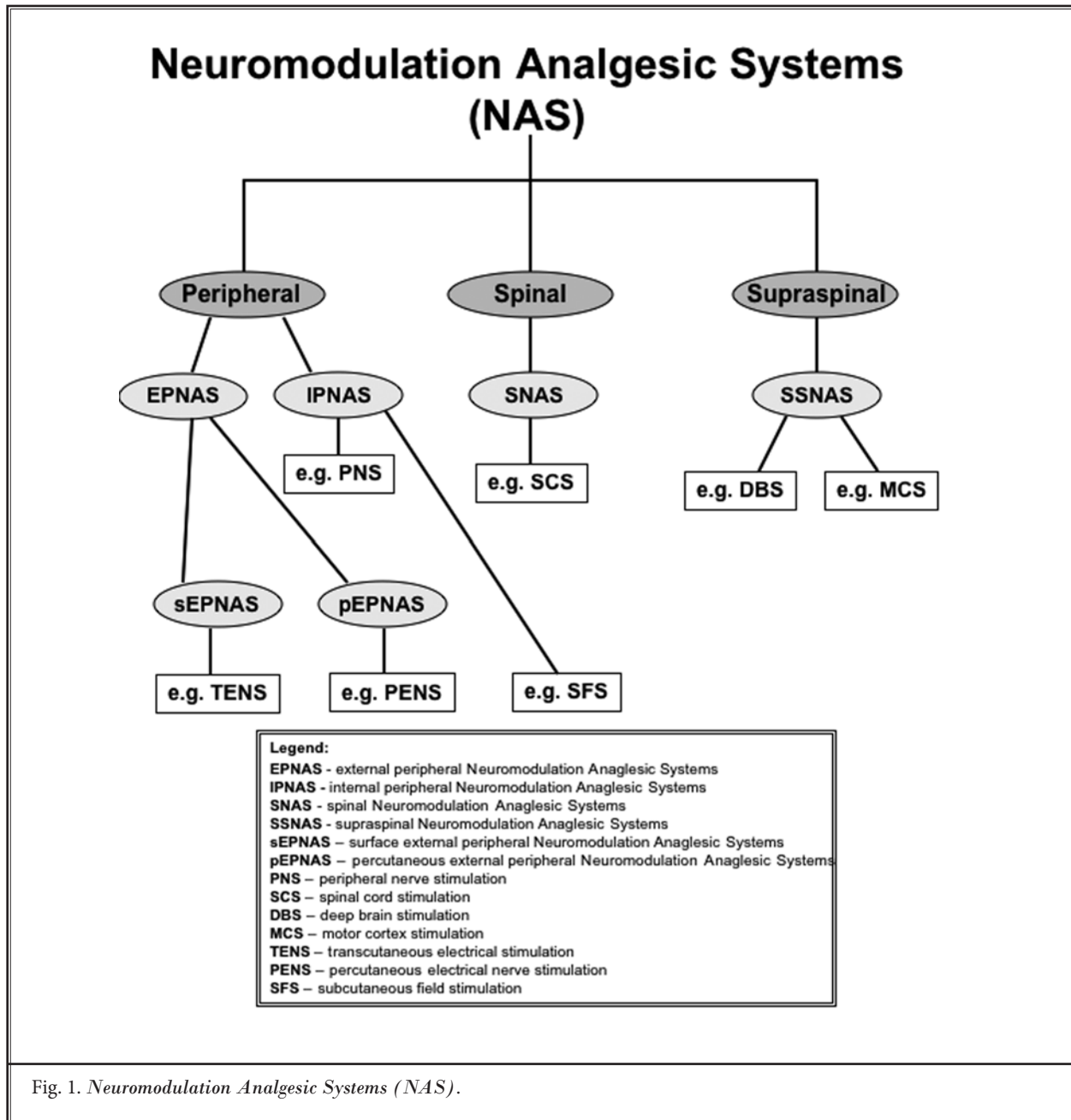
---

From: Albany Medical College Department of Anesthesiology,  
Albany, NY.

Dr. Smith is Editor-in-Chief of the *Pain Physician* journal and Associate Professor and Academic Director of Pain Management for Albany Medical College Department of Anesthesiology, Albany, NY.

Address correspondence:

Howard S. Smith, MD  
Associate Professor & Academic Director of Pain Management  
Albany Medical College  
Department of Anesthesiology  
47 New Scotland Avenue; MC-131  
Albany, New York 12208  
E-mail: smithh@mail.amc.edu



### REFERENCES

1. Yamazaki Y, Hozumi Y, Kaneko K, Sugihara T, Fujii S, Goto K, Kato H. Modulatory effects of oligodendrocytes on the conduction velocity of action potentials
2. Fields RD. Oligodendrocytes changing the rules: action potentials in glia and oligodendrocytes controlling Action potentials. *Neuroscientist* 2008; 14:540-543.