Neurolysis

A Brief Review for a Fading Art

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KEYWORDS

• Spasticity management • Neurolysis • Hypertonia

KEY POINTS

- For the clinician attempting to address the focal manifestations of hypertonia without surgical intervention, there have been 2 primary options: neurolysis or chemodenervation.
- Before the introduction of the botulinum toxins, neurolysis was the only focal spasticity treatment option available to the previous generation of physical medicine and rehabilitation practitioners.
- The goal of this chapter is to present the historical events and describe the treatment approaches that were common prior to the use of botulinum toxin. Case studies will be presented to provide support for the importance for the continued use and training in this technique.

Video content accompanies this article at http://www.pmr.theclinics.com/.

The key to the successful treatment of patients with spasticity is contingent upon choosing appropriate interventions tailored to meet the needs, goals, and specific presentation of each patient. The physician's toolbox to address spasticity has remained relatively unchanged over the past 30 years. Specifically, for the clinician attempting to address the focal manifestations of hypertonia without surgical intervention, there have been 2 primary options: neurolysis or chemodenervation. Chemodenervation using botulinum toxin has become the primary treatment choice to address focal hypertonia across all diagnostic classes. The past decade alone has seen nearly monthly journal articles exploring the different formulations, dosing, and refinements of technique and localization. Although the attention and accolades for chemodenervation using botulinum toxin are warranted, this article focuses on the procedure whose role over the past decade has diminished. Before the introduction of the botulinum toxins, neurolysis was the only focal spasticity treatment option available to the previous generation of physical medicine and rehabilitation (PMR) practitioners.

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The term "nerve block" refers to the process of applying chemical agents to various nerve structures to intentionally interfere with nerve conduction. Nerve blocks were initially performed to address painful conditions such as cancer-related pain. Nerve blocks to manage spasticity typically focus on the intramuscular nerve (motor point), motor branches of nerves, or peripheral nerves. The blocks can be further subdivided based on the intended duration and mechanism of action.

Diagnostic nerve blocks (anesthetic nerve block)

- Term used when a short-acting anesthetic is placed over the nerve
- Typical agents used include lidocaine (0.5%–2.0% concentration) and bupivacaine (0.25%–0.75% concentration)
- Mechanism of action: works by interfering with the ion channels on the axon by decreasing membrane permeability to sodium ions. This prevents depolarization of the membrane, which interrupts signal transmission. Block occurs without residual damage to the nerve.
- Onset of action: within 5 minutes (lidocaine) to 15 minutes (bupivacaine)
- Duration of effect: approximately 1 to 3 hours for lidocaine, 4 to 6 hours for bupivacaine
- Common uses:
  - Evaluation of the functional benefit from spasticity
  - Improve comfort and ease of serial casting
  - Investigate extent of soft tissue contracture
  - Assist in planning for more permanent procedures

Therapeutic nerve blocks (neurolysis):

- Term used when applying longer-acting agents.
- Agents and the most commonly used concentrations include phenol 3% to 6% and ethyl alcohol 10% to 50%.
- Mechanism of action: destruction of nerve tissue via protein necrosis, the higher the concentration the more tissue damage noted.
- Onset of action: anesthetic effect occurs immediately, maximal neurolytic effect 24 to 48 hours.
- Duration of action: Variable based on type of block and amount placed. Generally, 2 to 6 months expected.
- Common uses: In conjunction with botulinum toxin or alone to address significant postures, such as the equinovarus foot, adducted hips, internally rotated shoulder, flexed elbow and wrist.

HISTORIC MILESTONES IN MEDICINE RELATED TO SPASTICITY AND NEUROLYSIS

- 1860s: Sir Joseph Lister began using phenol as an antiseptic agent during surgery.
- 1903: Schlosser reports injection of alcohol into nerves for the treatment of neuralgia and other painful conditions.
- 1912: May publishes results of functional and histologic effects of intraneural and intraganglionic injections of alcohol of various concentrations in cats. Concludes that the alcohol causes local necrosis and fibrosis when placed over a peripheral nerve. Partial regeneration of the nerve was noted.[1]
- 1919: Liljestrand and Magnus report reduction of triceps rigidity in a decerebrate cat after an intramuscular injection of procaine.
- 1959: Nathan and Kelly separately described techniques for Intrathecal phenol injections to treat spasticity.[2,3]