Prevention and Management of Nerve Injuries in Thoracic Surgery

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KEYWORDS

• Peripheral nerves • Anatomy • Physiology • Thoracic surgery • Complications
• Peripheral nerve injury

KEY POINTS

- Understanding of the basic anatomy and physiology of the peripheral nerves of the chest is important for thoracic surgeons.
- Peripheral nerves may be injured during an operation by partial or complete transection, thermal insult, crush, stretch, or exposure to a toxic environment.
- Progress has been made in the field of nerve repair and regeneration, but there is no treatment that is as reliable as avoiding these injuries.

INTRODUCTION

Nerve injuries have the potential to cause substantial morbidity after thoracic surgical procedures. For the most part, these injuries are preventable, provided that the surgeon has a thorough understanding of the relevant anatomy and follows important surgical principles. When nerve injuries do occur, it is important for the surgeon to recognize the options available in the immediate and postoperative settings, including expectant management, immediate nerve reconstruction, or auxiliary procedures to mitigate the consequences of loss of the damaged nerve's function. This article covers the basic anatomy and physiology of nerves and nerve injuries, an overview of techniques in nerve reconstruction, and a guide to the nerves most commonly involved in thoracic operative procedures.

ANATOMY AND PHYSIOLOGY OF NERVES AND NERVE INJURY

Extensive knowledge of the complex peripheral nervous system is unnecessary for the practice of safe thoracic surgery. However, surgeons should have an understanding of the basic anatomy and physiology of peripheral nerves. This understanding helps to eliminate surgical techniques that put nerves at risk for injury, and fosters an appreciation for the natural history of nerve injuries and nerve repair.

Anatomy and Physiology of Peripheral Nerves

The peripheral nervous system is composed of 2 cell types: neurons and neuroglia. An individual neuron consists of a cell body and an axon. Multiple dendrites are associated with the cell body and transmit synaptic information inward, whereas a single axon conveys information away from the cell body. Neuroglia exist to provide the scaffolding and nutrient milieu for neurons. In some cases neuroglia produce myelin, a substance that insulates the long axonal processes of most neurons and improves the speed of conduction. Myelinated and unmyelinated axons are invested in several layers of connective tissue called endoneurium, perineurium, and epineurium. The epineurium contains the vascular and lymphatic

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network that supplies individual neurons. The viability of the epineurial layer is a key determinant to how well a nerve may recover after injury, with or without repair.

Neurons can be classified in multiple ways. Neurons belonging to the somatic nervous system provide voluntary skeletal motor control and precise sensory information, whereas neurons belonging to the autonomic nervous system provide involuntary motor and sensory information to and from the viscera and smooth muscle. Within the autonomic nervous system, a further subdivision exists between sympathetic ("fight or flight") and parasympathetic ("rest and digest") neurons. These distinctions are of little importance to surgeons with respect to the handling of individual peripheral nerves, because most peripheral nerves contain neurons of all different types enclosed within a single epineurial sheath. Injury to a nerve is likely to affect all neurons equally.

Classification of Peripheral Nerve Injury

A peripheral nerve may be damaged in several ways during the course of an operation, including by partial or complete transection, thermal insult, crush, stretch, or exposure to a toxic environment. The pattern of injury that results from these injuries exists along a spectrum with important implications for immediate management and prognosis for recovery. The severity of peripheral nerve injury is determined first by whether or not there is axonal damage, and then by the degree to which there is connective tissue damage. A classification of peripheral nerve injuries was originally proposed by Seddon in 1943, expanded by Sunderland in the 1950s, and is still widely in use today.

- Neurapraxia (Sunderland class I injury) refers to temporary interruption of normal nerve conduction without axonal or connective tissue damage. Neuropraxial injuries are the mildest form of peripheral nerve trauma. Usually the cause is mild crush, stretch, or thermal injury to the nerve that in turn causes demyelination. The mechanism by which demyelination is triggered is not understood fully, but may be related to transient ischemia or local inflammatory response. However, because the axon itself remains intact, full recovery of nerve function is expected in days or weeks.
- Axonotmesis (Sunderland class II injury) is defined as axonal injury without damage to the connective tissue scaffolding of the nerve. Typically, this injury is caused by prolonged stretch, crush, or ischemia with resultant axonal damage. An axon that is damaged cannot repair itself and undergoes a process of Wallerian degeneration, whereby the distal part of the axon degenerates and the resultant products are scavenged by macrophages. Renervation of the target organ can be achieved by collateralization or axonal regeneration. Collaterization occurs if only some of the axons leading to a target organ are damaged, allowing the tissue innervated by the remaining axons to hypertrophy and preserve the function of the organ. If the majority of axons have been destroyed, then regeneration must take place for function to be maintained. This is possible because the connective tissue scaffolding of the nerve remains intact. After a period of Wallerian degeneration, axons begin to regrow at rate of 1 to 3 mm per day along their endoneurial tubes, nourished by the surrounding epineurium. If they reach the target organ within 12 to 18 months, renervation can occur, albeit with somewhat abnormal conduction. Beyond that window, a process of denervation-related fibrosis makes muscle recovery unlikely; however, some sympathetic and sensory nerve targets have been shown to maintain renervation potential as far out as 3 years.
- Neurotmesis (Sunderland class III/V injury) is axonal damage along with some degree of connective tissue damage. If the endoneurium alone is disrupted, then axonal regeneration may be possible. However, if damage extends to the epineurium (ie, complete transection of the nerve), surgical repair is required.

Special consideration exists for nerve injuries caused by thermal insult, local anesthetic toxicity, or exposure to damaging environmental factors. Thermal injuries are unique in that the degree of destruction to both the axons and the connective tissue scaffolding of the nerve may be more severe than is perceived by the surgeon. Total disruption of the epineurial layer with subsequent fibrosis may occur in the absence of obvious physical signs of damage. For this reason, thermal nerve injuries are often not identified at the time of surgery. If they are, however, the safest course is to resect the affected nerve along with proximal and distal margin and reconstruct the remaining tissue. The severity of local anesthetic toxicity may also be difficult to judge in the immediate period. Infiltration of local anesthetic agent directly into the epineurial layer typically results in neuropraxial injury. However, if high enough concentrations are achieved axonal death and even connective tissue damage are possible. Last, experimental