What's New in the Diagnosis and Treatment of Peripheral Nerve Entrapment Neuropathies

Charles P. Toussaint, MD, Edward C. Perry III, MD,
Marc T. Pisansky, BA, Douglas E. Anderson, MD*

KEYWORDS

• Peripheral nerve • Entrapment neuropathy
• Occipital neuralgia • Neurostimulation

Entrapment neuropathies are widespread and commonly debilitating clinical conditions that have a profound physical, psychological, and economic impact on the afflicted. In general, surgical interventions for these entrapment neuropathies carry a very favorable outlook, with relatively low risk of serious morbidity and high success rates for relief of symptoms and recovery of useful neurologic function. Following the lead of other surgical specialties, peripheral nerve surgeons in recent years have applied minimally invasive techniques to nerve entrapment surgery in hopes of reducing surgical morbidity and postoperative recovery times.

An entrapment neuropathy is defined as a pressure-induced injury to a peripheral nerve in a segment of its course due to anatomic structures or pathologic processes. Such pressure-induced injuries include a range of etiologies. Some patients have a predilection to entrapment neuropathies, related to congenital narrowing of the nerve's osseus tunnel or thickening of an overlying retinaculum. Inflammation or edema of adjacent structures, such as tendons, may reduce the size of the passageway for the nerve, and mechanical forces on the nerve can result in nerve compression. The effect of nerve compression is mediated by ischemia and edema; compression of the nerve results in disruption of the blood-nerve barrier and dysfunction of the intraneural circulation. In early stages of compression, morphometric

Department of Neurological Surgery, Loyola University Medical Center, 2160 South 1st Avenue, Maywood, IL 60153, USA
* Corresponding author.
E-mail address: dander1@lumc.edu

Neurol Clin 28 (2010) 979–1004
doi:10.1016/j.ncl.2010.03.017 **neurologic.theclinics.com**
0733-8619/10/$ – see front matter

changes may not be seen, but as ischemia persists, segmental demyelination occurs. At this point, injury is often reversible with treatment. Prolonged compression results in edema, which may result in epineurial fibrosis, further thickening the nerve. Damage to the myelin sheath and axonal disruption are end stages of chronic compression, resulting in irreversible nerve damage.[1] Not all nerve fibers are equally susceptible to pressure; larger fibers are more susceptible than small fibers and fascicular location within the nerve may also affect vulnerability, depending on the force vectors applied.[2,3]

THORACIC OUTLET SYNDROME

The term thoracic outlet syndrome (TOS) can describe multiple separate entities that result from compression of elements of the brachial plexus or subclavian vessels in their passage from the cervical and upper thoracic area toward the axilla and the proximal arm (**Fig. 1**). TOS is divided into 2 categories: the neurogenic type and the vascular type, each of which has 2 subtypes. Neurogenic TOS, one of the most controversial clinical entities in medicine, refers to a constellation of neurologic signs and symptoms related to compression or irritation of proximal elements of the brachial plexus. In general, neurogenic TOS is subdivided into 2 categories: true (classic) neurogenic TOS and disputed (common) neurogenic TOS. True neurogenic TOS arises from structural anomalies at the base of the neck; a prominent C7 transverse process or rudimentary cervical rib and associated fibrous bands extending to the first

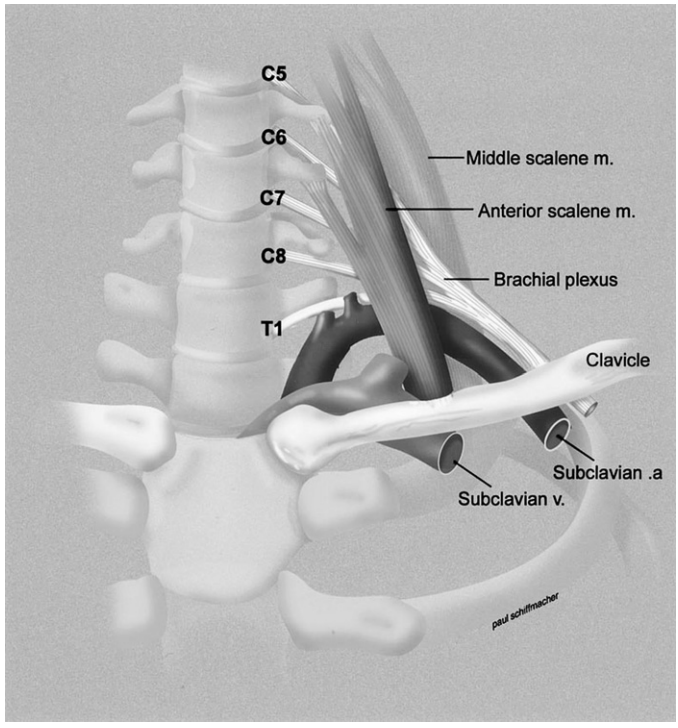

Fig. 1. An anatomic representation of the thoracic outlet illustrating the brachial plexus, subclavian artery and vein, musculature, and clavicle. (*From* Huang JH, Zager EL. Thoracic outlet syndrome. Neurosurgery 2004;55(4):898; with permission.)