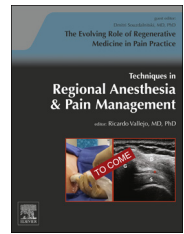


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# Peripheral nerve entrapment, hydrodissection, and neural regenerative strategies

Andrea Trescot, MD, DABIPP, FIPP<sup>a,\*</sup>, Michael Brown, DC, MD<sup>b</sup>

<sup>a</sup>Pain and Headache Center, Alaska

<sup>b</sup>Interventional Regenerative Orthopedic Medicine, Bellevue, Washington

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## ABSTRACT

Peripheral nerve entrapments are an underrecognized cause of pain and disability. Hydrodissection (perineural deep injections) is one of the techniques that can release the entrapped nerve. Furthermore, discussed are the techniques of neural therapy, neural prolotherapy (perineural injection therapy), and autologous platelet lysate, as well as the use of adipose-derived stem cells.

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## Introduction

Peripheral nerve entrapments are common but are often underrecognized sources of pain. An *entrapment neuropathy* is defined as a pressure-induced segmental injury to a peripheral nerve due to an anatomical structure or pathologic process.<sup>1-3</sup> The defining criteria of an entrapment, according to Kashuk,<sup>4</sup> include altered transmission because of mechanical irritation from impingement of an anatomical neighbor. Many nerve entrapments occur at areas where the nerve travels through a canal, channel, or tunnel, but they also occur because of trauma and potential scar “strangulation” of the nerve. The nerve has its own blood flow (*vasa nervorum*) as well as accompanying vascular structures. Compression of the nerve, whether intrinsic or extrinsic, can cause damage to the neurovascular structures, leading to ischemia of the nerve. Nerve entrapments can result in clinical symptoms that range from mild discomfort to numbness, paralysis, or incapacitating pain.

Injections have a unique role in the management of peripheral nerve entrapments. Injections can aid in diagnoses, but they can also treat the underlying nerve entrapment, presumably by the anti-inflammatory effect of injected

corticosteroids, hydrodissection of the constricting tissues, and the dilution and flushing out of inflammatory mediators. Precise and atraumatic injection techniques are essential to maximize the informational and treatment value of any nerve injection for peripheral nerve entrapment.

## Nerve entrapment

Nerve entrapments may be present in varying degrees, leading to a variety of clinical presentations. Nerves can be entrapped by several mechanisms, including mechanical, which may involve compression, constriction, overstretching, or edema. Entrapment may occur in tunnels (such as carpal tunnel syndrome) (Figure 1), between muscles (such as the axillary nerve) (Figure 2), around blood vessels (such as the occipital nerve) (Figure 3), between bones (such as Morton’s neuroma) (Figure 4), across joints (such as the superficial peroneal nerve) (Figure 5), through fascial penetration sites (such as the anterior cutaneous nerve entrapment syndrome) (Figure 6), or from external compression (such as common peroneal nerve entrapment by a cast) (Figure 7).

\*Corresponding author.

E-mail address: [DrTrescot@gmail.com](mailto:DrTrescot@gmail.com) (A. Trescot)

E-mail address: [DrTrescot@gmail.com](mailto:DrTrescot@gmail.com) (A. Trescot).

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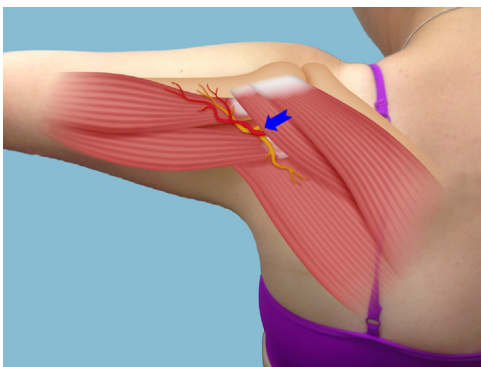


**Fig. 1 – Carpal tunnel injection under fluoroscopy, showing contrast in the carpal tunnel. (Image courtesy: Andrea Trescot, MD.)**

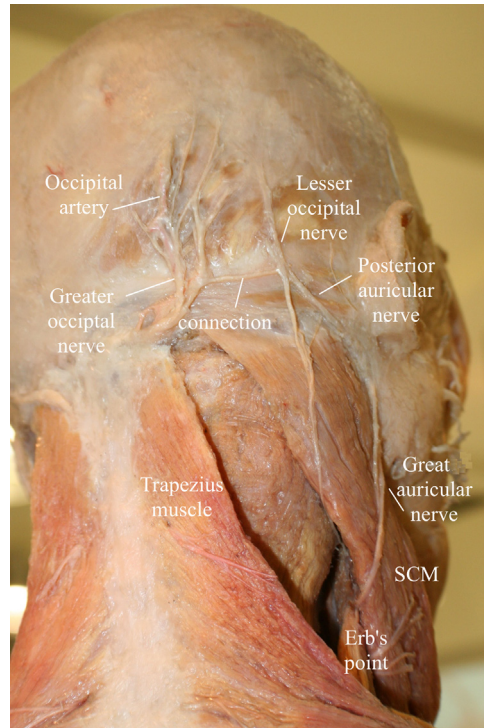
There are 2 major ways that the fascial penetration point can affect a nerve. Trauma to a nerve would cause edema, which can travel proximal and distal to the injury. When this swelling reaches the fascial penetration points, this can cause a self-strangulation of the nerve and decreased nerve growth factor (NGF) flow. Shearing at the penetration point can also cause entrapment, such as those seen during the distention of the abdomen during pregnancy, causing anterior cutaneous nerve entrapment syndrome.

Peripheral nerve entrapment can lead to or contribute to a wide variety of disorders (Table). In addition, painful conditions with well-described pathology such as complex regional pain syndrome (CRPS) or postherpetic neuralgia likely have a component of nerve entrapment, either as the initiating event (CRPS) or as a consequence of the pathology (postherpetic neuralgia).

The characteristic nerve pain (“neuropathic pain”) is described as burning, shooting, lancinating, or “electric shocks.” There may be allodynia (pain from typically non-painful stimulation) or hyperpathia (longer than expected



**Fig. 2 – Axillary nerve entrapment at the quadrilateral space with the arm in abduction, showing entrapment of the axillary nerve and artery (arrow) by the teres major and teres minor muscles. (Image courtesy: Andrea Trescot, MD.) (Color version of figure is available online.)**



**Fig. 3 – Anatomy of the occipital region. (Adapted and modified with permission from Bodies, The Exhibition.) Note the connection of the greater and lesser occipital nerves. (Image courtesy: Andrea Trescot, MD.) (Color version of figure is available online.)**

pain from a painful stimulation). This pain often increases over time because of “central sensitization.”

### Postoperative nerve entrapments

There are multiple mechanisms by which surgery can cause postoperative nerve entrapments. The pain can occur immediately after surgery because of nerve damage from retractors, scapel, or edema, or it may start weeks or even years after the surgery because of the scar cicatrix that gradually tightens around the nerve.

As an example, pain after total knee replacements can be caused by entrapment of the saphenous (SN) and infrapatellar saphenous (IPS) nerves. The SN, which is composed of sensory fibers from the L3 and L4 nerve roots, branches off the femoral nerve not far below the inguinal ligament (Figure 8), then descends through the anteromedial thigh with the femoral artery and vein to the adductor (Hunter’s) canal. The anatomical variability of the SN, and particularly the IPS,<sup>5,6</sup> increases distal to the vastoadductor membrane. In most people, 2 or more branches of the SN leave the adductor canal proximal to the joint line, the most anterior of which crosses the knee as the IPS (Figure 9) to innervate the skin below the patella and the anterior-inferior knee capsule. The most posterior branch continues as the distal saphenous (sartorial) nerve, traveling down to the ankle.

The IPS can be traumatized by the medial surgical retractors, and, with the knee in flexion during the procedure, the IPS can be under tension, causing a stretch neurapraxia.<sup>6</sup>

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