Management of Neuromas of the Upper Extremity

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KEYWORDS

• Neuroma • Management • Surgery

KEY POINTS

- Neuromas of the upper extremity are common, and their treatment can prove challenging. A multitude of operative and nonoperative techniques have been described with varying degrees of efficacy.
- Diagnosis of neuromas is based on physical examination findings and can be aided with the use of selective anesthetic injections.
- Several oral medications have been used in treating neuropathic pain, with anticonvulsants appearing to be the most efficacious.
- The underlying principle of all operative treatment is to remove the nerve or neuroma from any persistent source of mechanical irritation.
- Operative techniques can be divided into 4 categories: resection alone, resection with subsequent nerve grafting or primary repair, containment of the neuroma, or translocation of the nerve.
- The chosen method of treatment depends on the location and type of nerve as well as the injury sustained.

INTRODUCTION

After a nerve sustains a partial or complete injury, it possesses an intrinsic reparative capacity to establish continuity with its distal end. The ensuing proliferation of disorganized axons, myofibroblasts, endothelial cells, and Schwann cells can result in the formation of a neuroma (**Fig. 1**).[1,2] Reports of incidence range from 4% to 25%.[3–5]

Neuromas were first described by Odier in 1811,[1] with patients presenting with disabling pain or loss of motor function. Numerous treatments have been described, with varied success. In this article, the pathophysiology of neuromas, their clinical manifestations, and the role of current nonoperative and operative treatments are reviewed.

CAUSE AND PATHOPHYSIOLOGY OF NEUROMAS

The underlying cause of all neuroma formation is a degree of nerve irritation or injury (**Fig. 2**). Acute injuries are typically iatrogenic or traumatic. Examples of common iatrogenic injuries include damage to the superficial sensory branch of the radial nerve during dorsal exposures of the wrist or distal radius[6,7] or to the palmar cutaneous branch of the median nerve during carpal tunnel surgery.[8] Traumatic injuries generally result from lacerations to digits or the hand, which can result in stump neuromas. Subsequent repair of lacerations or replantation of digits with nerve repair can also result in neuroma formation, particularly if the segmental defect is long, excessive scarring occurs, or a mismatch in size exists between proximal and distal segments.[9]

When transected nerves are not repaired, end neuromas may result from fascicular overgrowth. When a nerve is injured or cut, signals travel retrograde through the proximal axon to the cell body to stimulate a reparative response.[10] There is a milieu of host signaling factors at the site of injury to direct the response, including substance P, calcitonin gene–related peptide, and mast cells,

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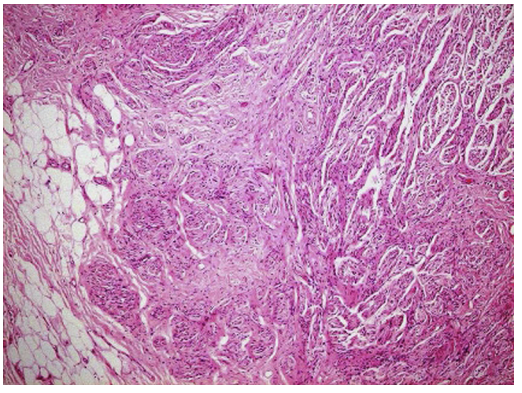

Fig. 1. Sciatic nerve neuroma resulting from above-knee amputation. (*From* Kitcat M, Hunter JE, Malata CM. Sciatic neuroma presenting forty years after above-knee amputation. Open Orthop J 2009;3:126.)

all of which may function to enhance the regenerative process.[11] Various neurotrophic factors, including neuropoietic cytokines, fibroblast growth factors, and neurotrophins, may also be involved. Within the distal segment, Schwann cells and macrophages begin to phagocytose myelin through the process of Wallerian degeneration.[12] The Schwann cells align themselves along the basal lamina of the distal segment to form bands of Bungner, which help to guide the regenerating proximal segment. This is accompanied by an up-regulation of corresponding neurotrophic factors and neurite growth-promoting factors,[12] including neuropoietic cytokines, fibroblast growth factors, and neurotrophins.[13] Within the proximal segment, several sprouts form from the regenerating axon, each with a growth cone on its end that attempts to identify the suitable distal neural tube to guide regeneration.[14] When the regenerating unit cannot identify its corresponding distal segment, elongation cannot occur and an end neuroma forms (**Fig. 3**).[15]

Neuromas may also arise from crush or stretch injuries of the nerve, which remains in continuity. Maintenance of the basal lamina allows for organized regeneration to distal targets,[12] but fascicular escape can still occur through disruption of the perineurium.[13] This subsequent spilling of the fascicles allows disorganized neuroma formation,[16] resulting in a neuroma in continuity.

Inflammation around a nerve can induce scar formation and accounts for a third mechanism of neuroma formation, even in the absence of direct neural injury. Scar tethering of nerves, or traction neuritis, results in activation of the nerve secondary to inflammation, irritation, or mechanical shearing.[17] Nerves of the upper extremity, specifically the digits, are at risk of mechanical irritation, given their proximity to the skin.

Despite their formation, not all neuromas develop painful symptoms.[2] Prediction of pain after partial or complete nerve transection is notoriously difficult, in part because of its multifactorial cause. Neuropathic pain is influenced by the presence of mechanical or chemical irritation, development of local scar tissue (ie, traction neuritis), and dysesthetic sensory symptoms.[1,13] In particular, proximal neuronal activity within the dorsal root ganglion, spawned by the injury, can contribute to the disabling dysesthetic pain. The type and size of nerve that is injured also influence the size and likelihood of neuroma formation. Injuries that occur more proximally lead to larger neuromas as a result of increased axoplasmic flow.[18] Nerves with a higher ratio of fascicles to epineurial tissue are more likely to form neuromas, because it is easier for the fasciculi to escape.[13]

DIAGNOSIS OF NEUROMAS

A history of sharp trauma, crush, or stretch injury along with a thorough physical examination can help diagnose neuromas. Pain related to a single peripheral nerve distribution, with or without accompanying numbness or diminished sensation, may also be useful in localizing the lesion. However, overlapping innervations of adjacent nerve

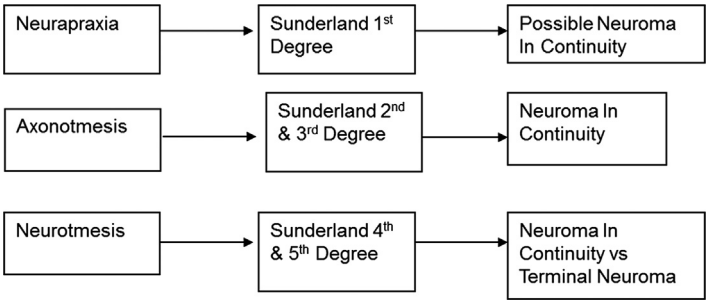

Fig. 2. Comparison of Seddon and Sunderland's classification systems and their relations to neuroma formation.