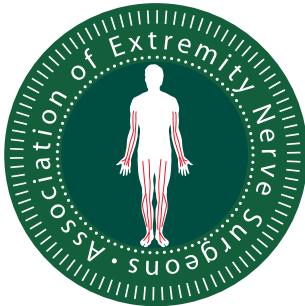


Clinical Practice Guidelines

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The Association of Extremity Nerve Surgeons



Clinical Practice Guidelines 2014

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Disclaimer

These recommendations are based on our professional experiences and current available research. They are not intended to replace current standards of care, but are given to provide insight and knowledge into the subject of nerve surgery.

The Protocols and Guidelines Committee of the Association of Extremity Nerve Surgeons (AENS) has formulated this document based on more than a decade of professional experience and current available research. AENS is a component society of the APMA and was founded to promote the collaborative study and development of medical research regarding the treatment of extremity nerve diseases. Membership is inclusive of MD, DO, DPM, PHD, and medical students interested in furthering the dissemination of current knowledge and development of basic medical research in peripheral nerve disease.

The Association of Extremity Nerve Surgeons adopts the position that outcomes of peripheral nerve surgery are highly dependent on scientific understanding of peripheral nerve physiology, diagnostic acumen, post-operative management, knowledge of nerve anatomy, and surgical technique and handling. Many of our members are involved with education and residency programs throughout the United States to help further this understanding. After careful review of overall knowledge on nerve diseases, the AENS suggests that formalized training during and beyond residency is imperative for successful patient outcomes.

- The Protocols and Guidelines Committee, AENS

Peripheral Nerve Surgery Tissue Handling and Post-Operative Management

Magnification

When performing peripheral nerve surgeries, adequate visualization is imperative. You must be able to visualize internal anatomical structures, to differentiate between nerve structures and other similar tissues, and to clearly see the peripheral nerves and their small branches so they may be preserved. To do this, we recommend using loupe magnification of 3.5x or greater. We recommend this, despite the dearth of published studies addressing the question of whether or not it provides better surgical outcomes, due to the fragility of the nerves and their structures. Proper visualization is essential in peripheral nerve surgery, as is proper tissue handling.

Dissection Technique

It is crucial that proper dissection technique be used. When handling tissue, the incision placement should be planned based on knowledge of the anatomy and pathology being treated. The initial skin incision should be down to and through dermis only – no deeper. Blunt dissection should then be carried to the level of the pathology to preserve the integrity of the nerve and its branches. Dissection with a scalpel should be avoided whenever possible.

When handling the tissue, avoid grasping or pulling the peripheral nerves with any instrumentation. Avoid traction, compression, grasping, or crushing of the nerves. Nerves are highly susceptible to traction and compression type injuries.^[1] Atraumatic technique should be used when performing nerve dissection.^[1-3]

Hemostasis

When performing peripheral nerve surgeries, it is imperative that there be adequate visualization. To ensure this, an extremity tourniquet should be used in most operations. We also recommend bi-polar cauterization be used. Mono-polar cauterization is

highly discouraged due to its uncontrolled extent and degree of tissue destruction. Meticulous hemostasis provides ideal operative recovery.^[4] Poor hemostasis is often associated with greater scarring.^[5]

Skin Closure

Unlike traditional layered closure, in peripheral nerve surgery we recommend minimal subcutaneous suturing as reapproximating deep facial layers can re-entrap released nerves.

Post-Operative Care

It is recommended that early mobilization take place following peripheral nerve surgery to encourage neural gliding, while also avoiding adhesions.^[6] Except in the case of primary nerve repair, post-operative splintage should be avoided when performing peripheral nerve surgery.

It has been shown that staging surgical procedures for cases with multiple pathologies can be advantageous. A nerve procedure can be performed after a pathologic fusion or primary repair. Due to the fact that the outcomes of peripheral nerve surgeries is highly dependent on post-operative management, we recommend staging procedures that involve multiple pathologies with a follow-up plan to include early mobilization.

It is important to note that repaired nerves have been found to heal at variable rates for sensory and motor function, ranging from 5mm/day for sensory nerve and 1.7mm/day for motor nerve. Generally this is in the order of 1mm/day and 1"/month, and may be accompanied by paresthesias.^[6, 7] An increase in pain following nerve decompression surgery can occur, which is generally temporary and due to neuronal regeneration. The post-operative pain management protocol should include patient education and appropriate analgesic medications.

Diagnosing Peripheral Nerve Disease

Favorable peripheral nerve intervention outcome is first dependent on an accurate diagnosis using proper techniques.

Clinical Evaluation

Clinical evaluation is essential in diagnosing peripheral nerve disease. A comprehensive history citing onset, timing, progression, presence or absence of burning or lancinating pain symptoms, etc. should be recorded. A thorough peripheral nerve evaluation should then be performed, which always should include localization of pain. Use of the Wartenberg pinwheel (Figure 1) exam, presence or absence of provocation signs, sensory testing, testing for hyperalgesia, pathologic reflexes, and motor strength evaluations are also highly valuable. Further evaluation can include urine toxicology and a psychosocial assessment.



Figure 1: The Wartenberg pinwheel is a valuable handheld exam tool for dermatomal and specific peripheral nerve trunk evaluation. It is very helpful in bilateral diagnostic testing and should be performed with the patient's eyes closed.

Biomechanical Exam

A biomechanical exam can be used to evaluate the forefoot nerve entrapments and pressures. Examples of biomechanical examination are: too many toes sign, Silfverskiold test, gait analysis, evaluation of range of motion, and so on.

Physical Exam

Silfverskiold Test:

Increased forefoot pressure has been documented to lead to nerve entrapment syndromes and nerve pain.^[8] However, equinus is often underappreciated and not recognized as contributing to increased forefoot pressures that can result in peripheral nerve pathology and symptoms. When attempting to diagnose an equinus deformity, the Silfverskiold Test is a valuable resource. Equinus should be evaluated and addressed to improve treatment outcomes.

Tinel's Sign:

The Tinel's Sign, or the Hoffmann-Tinel Sign^[9-11], is a widely accepted diagnostic exam and has been shown to be a valuable predictive indicator of nerve entrapment. It is thought to represent axonal sprouting in a nerve recovery process. It is easily performed in a clinical setting and should be included in any nerve evaluation. The test should be performed with gentle percussion over the entrapped or injured nerve with the examiner's finger. Use of a neurological hammer is not advised, as this may produce a false positive exam.

Provocation Sign:

A provocation sign can also be used to detect nerve entrapment. Moderate digital pressure is applied at the suspected site of pathology, which will often elicit withdrawal, discomfort, alarm, or verbal responses from the patient. Mulder's is an example of a provocation sign.

Semmes Weinstein Monofilament:

The Semmes Weinstein Monofilament examination can be used to measure cutaneous sensation. However, the classic 5.07g SWMF testing lacks the sensitivity and specificity to make accurate nerve diagnoses. When patients are unable to feel the 5.07g filament, severe nerve damage has already occurred.[12, 13]

Two-Point Discrimination and PSSD:

Two of the more accurate semi-quantitative exams currently available for research, as well as clinical evaluation, are Two-Point Discrimination and Pressure Specified Sensory Device (PSSD). These exams are a valuable resource that can be used to evaluate nerve density and axonal loss.[11, 13-20]

Electromyogram (EMG) and Nerve Conduction Studies (NCS):

Electrodiagnostic testing is a valuable resource to evaluate muscular response to nerve stimulus and has been found to be diagnostic for lumbar radiculopathy. EMG's are very specific but may lack sensitivity for focal nerve entrapments. And, although neurology specialists consider EMG to be the gold standard exam, the presence of a negative electrodiagnostic study does not necessarily rule out nerve pathology. Clinical evaluation is essential for an accurate and complete diagnosis and necessary to propose appropriate treatment.

Epidemal Nerve Fiber Density (ENFD) and Intraepidermal Nerve Fiber Density (IENF):

Although the reference standard for diagnosing painful small fiber neuropathies is ENFD by skin biopsy, the relationship between ENFD and neuropathic pain is still unclear.[21]

ENFD biopsies are of unknown value in the evaluation of degree of neuropathy and effectiveness of nerve therapies, but can be extremely helpful in delineation of small fiber neuropathy. The ultimate role of these tests in the determination of peripheral neuropathy is yet to be determined. Therefore, they should be interpreted only in conjunction with good clinical examination.

"The presence of diffuse swellings on IENFs has been shown to predict the progression to overt neuropathy in patients with HIV, diabetes, or other causes of small fiber neuropathy, and to correlate with paresthesia." [22] If a normal ENFD test is found in patients with peripheral neurologic symptoms, there should be high suspicion of nerve entrapment.

Imaging:

Radiography, ultrasound, MRI, and neurography can be useful in demonstrating such pathologies as nerve entrapment, nerve gliding, nerve enlargement, space occupying lesions, Morton's Entrapment, Tibial Nerve Entrapment, and muscle denervation. When considering any lower extremity nerve pathology, examination of the architecture of the extremity (both clinically and radiographically) should be done to determine its role in the pathology.

Diagnostic Nerve Blocks:

Diagnostic peripheral nerve blocks can be an extremely valuable tool if properly performed with a thorough understanding of peripheral neural anatomy. Specific nerve blocks in small volume should be performed proximal to the suspected site of nerve damage. Relief following a diagnostic nerve block is usually indicative of the site of neural pathology.[23]

Appendix 1 - 3.5 min Physical Exam (AR)

Denervation

Denervation is the interruption of nerve impulse to and from an organ or body part. This may be due to nerve disease, nerve damage, chemical toxicity, personal injury, or intentional disruption. The principles of tissue handling do not differ in denervation procedures from any other neurological procedures. Gentle nerve and tissue handling must be utilized.

Current Methods for the treatment of denervation:

Current methods of denervation treatment include cryoablation and radio frequency ablation, alcohol injections, and surgical resection. Aside from surgical resection, all other methods damage tissue in a relatively blind manner without absolute control and may not be a permanent resolution of symptoms. Neuro-destructive procedures may be useful on nerves that are already damaged; however, they should not be used as initial treatment for entrapment neuropathy. Ultrasound is beneficial for guiding non-surgical percutaneous denervation techniques and has demonstrated efficacy in musculoskeletal techniques similarly.[24-29]

Ablation:

Cryoablation (cryotherapy) should be used with extreme caution, as the amount of literature in the lower extremity is limited. If cryotherapy is used, it should ideally be performed with open technique rather than percutaneously for optimal results.[30]

Radiofrequency ablation has use in the lower extremity, but must be done with caution as this procedure has the potential for thermal necrosis of the adjacent tissues. Judicious use of fluoroscopy and other visualization techniques is advised while utilizing radiofrequency ablation. Our clinical experience over the last decade has shown efficacy – but further research in this technique is needed.

We do not recommend ablation in the primary treatment of Intermetatarsal Entrapment (Morton's Neuroma").

Alcohol injections:

The literature regarding alcohol injections is equivocal. There may be some short-term positive effect, but long-term effect is poor for this therapy.[31] Some of the literature recommends using 30% alcohol solution to get effective results.[32] However, there is not enough data to support the use of alcohol. As a general rule, we do not advocate the use of alcohol injections.

Surgical Resection:

Neurectomy can be effective in difficult cases but must be used with extreme caution. Surgical resection of the nerve ending without muscle implantation has a high propensity for painful neuroma formation.[33-36] When a painful nerve has failed other surgical interventions a neurectomy can be performed. Various methods of implantation have been reported in the literature. Proper identification and isolation of the offending nerve, followed by proximal transection and subsequent implantation into an available muscle belly, will yield the most successful results in minimizing painful stump neuroma formation.

Graft:

A graft may be used in cases where there has been a prior injury to a nerve in the lower extremity. Nerve grafting has proven useful in treating peripheral nerve defects,[37-39] but should only be performed by surgeons with appropriate training and/or experience.

Diabetic Polyneuropathy

In its 14-year history AENS has seen nerve decompression produce dramatic clinical benefit in diabetic peripheral neuropathy patients.[40-44] We believe the clinical and laboratory evidence strongly indicates the frequent nerve entrapments seen in diabetic sensorimotor polyneuropathy (DSPN) are a secondary pathology which frequently accompanies DSPN, is responsible for many serious complications which are frequently responsive to safe and effective surgical neurolysis. Nerve decompression surgery has been found to produce balance improvements, which may aid in fall prevention, decreased ulcer formation and recurrences, improvements in pain, and recovery of protective sensation.[45, 46].

Diabetic Sensorimotor Polyneuropathy (DSPN)

Diabetes is eventually complicated by neuropathy in 50-60% of cases, and 20% experience mild to severe pain. The dogma of hypothetical etiopathogenesis is that DSPN is a “length dependent axonopathy” first appearing in the legs due to their most extended distance from the spinal cord cell body. Later, arm symptoms can appear to generate the classical picture of “stocking glove anesthesia”. No mechanism has been proposed to explain how axonal length could be involved in producing this picture.[47] But this hypothesis fails to explain the common occurrence in diabetes of nerve entrapment syndromes, the asymmetry of sensory change and absence of global uniformity in limb sensibility loss often found by careful neurological exam.

The AENS finds evidence that nerve entrapments so frequently found in diabetes more often represent single or multiple metabolically induced nerve trunk entrapments in areas of fibro-osseous anatomic tunnels.[48] Such entrapments can easily produce a “stocking-glove” sensory loss. Many biochemical mechanisms are thought to contribute to the development of peripheral neuropathy in the diabetic patient, with one of the most prominent being the involvement of intraneuronal sorbital accumulation with attendant osmotic driven fluid accumulation and nerve enlargement.[49-52] Matched with

accumulation of advanced glycosylation end products, which shrink and stiffen fibrous tissue, the end result is fat nerves unable to glide and function in tighter anatomic “napkin ring” structures like the carpal, cubital, medial and lateral plantar, and tarsal tunnels. Entrapments are also common for the radial nerve at the distal forearm, common peroneal or fibular nerve at the fibular neck, the deep peroneal nerve under extensor hallucis brevis tendon on the dorsal foot, or superficial peroneal nerve as it exits the anterior or lateral muscle compartments into a subcutaneous position in the distal leg. Pain and loss of sensation are the common presenting symptoms in these superimposed entrapments of DSPN.

Neuroactive drugs have been found to be beneficial for many patients, but those with demonstrable nerve entrapments should be considered for decompression surgery.[53, 54] Masking neuropathic pain with long-term neuroactive (gabapentinoid) medications can delay definitive treatment such as decompression surgery. Chronic focal nerve compression leads to further axonal degeneration, which will threaten surgical outcome.[41] Early decompression is optimal.

There are copious laboratory and clinical findings, which provide evidence of connections between diabetic neuropathy, pain and nerve function loss, which can be relieved or prevented by nerve decompression at entrapment sites.[40, 42-46, 55-60] Baltodano, et al[61] have reviewed and done a meta-analysis of the subjective symptomatic pain and sensibility benefits to be found with nerve decompression of these superimposed entrapments. Many academics view this hypothesis and its reported results as likely presenting evidence only of placebo effects and observer bias.

Objective measures of outcome can rebut or negate the placebo/bias critiques. Level II EBM reports show objective benefits after nerve decompression in balance improvement, elimination of dangerously high perineural pressure, protection against initial diabetic foot ulcer development (DFU), DFU

recurrence risk, lower extremity amputations, recovery of nerve conduction velocity and evoked muscle EMG motor potential.

Evidence indicates that using nerve decompression will minimize neuropathic DFU recurrence by over 80%. [46, 61, 62] There is also evidence that nerve decompression is protective against initial primary DFU in advanced DSPN in Tinel-positive patients. [44] Therefore, consideration of using nerve decompression to protect against recurring DFU and progression to amputation is warranted. Evidence of improved transcutaneous oxygen levels post-nerve decompression may mean that less severe neuroischemic DFU cases can also be protected. [59]

Diagnosis

Diagnosis of superimposed nerve entrapment in diabetic patients with DSPN relies on elimination of other causes of the neuropathy you have diagnosed clinically with medical history, laboratory tests and careful neurological exam. If good control of hyperglycemia and other medical co-morbidities do not resolve symptoms adequately, if ankle edema is absent and a Hoffman-Tinel sign is found over any entrapment site, then nerve decompression can be considered for therapeutic resolution of symptoms or protection against the cascade of foot complications like DFU or Charcot neuroarthropathy which can complicate DSPN.

Operative Technique

Nerve decompression in the lower extremity usually includes external neurolysis of the four tibial nerve branches in the tarsal tunnel area, the common peroneal (fibular) nerve at fibular neck, and deep peroneal nerve under extensor hallucis brevis. [62] Many surgeons also decompress superficial peroneal nerve as it transits the leg fascia of the distal leg. Usual meticulous nerve and tissue handling under loupe magnification is employed. Tourniquet use is optional for the peroneal nerve and branches if adequate anesthesia, perfect visualization, and meticulous hemostasis can be achieved, but this is difficult for the tarsal tunnels. Post operatively, guarded weight bearing activity is mandatory to maintain nerve gliding and avoid adhesion formation. Suture removal is delayed to 3 weeks post-surgery to avoid the ankle wound dehiscence, which can occasionally occur. Wound infection is quite unusual following nerve decompression surgery.

After nerve decompression, the VAS pain scores are reduced from average levels > 8 to < 3. Two-point sensibility often returns to normal. Ulcer recurrence risk becomes <5% per year. Ninety percent have major pain reductions and 70-80% have durable sensory recovery. [63]

Morton's Entrapment

"Morton's neuroma", as it is often referred to, is not a true neuroma in the sense that no nerve damage has occurred to the nerve. A true neuroma can only occur after damage to a nerve has occurred. However, "Morton's neuroma" is an entrapment syndrome manifesting itself as a painful neuralgia and sometimes with a loss of sensation. Therefore, "Morton's Neuroma" should actually be referred to as an intermetatarsal nerve entrapment, or "Morton's Entrapment."

Histologic findings:

Histologic findings of Morton's Entrapment are variable, ranging from no measurable pathology to perineurial fibrosis, and are not consistent with a true neuroma where there is a proliferative process rather than a degenerative one which is caused by focal nerve entrapment. A true neuroma demonstrates tangled axonal regeneration and is a pain generator resulting from a damaged nerve. These changes are rarely seen in Morton's Entrapment.[64-69]

Diagnosis:

Diagnosis is primarily dependent on subjective symptoms and physical examination findings. Common physical findings may include splaying of digits, Mulder's sign, Gauthier's sign, Tinel's sign, and impairment of web space or toe tip sensation.[70, 71] While clinical testing may confirm the diagnosis, it is most useful in differentiating between a nerve entrapment and other possible pathologies such as plantar plate injury, capsulitis, tarsal tunnel entrapment, ankle equinus, etc. X-ray, MRI, US, NCS, and toe tip sensation are all viable forms of diagnostic testing that can be performed.[71-77] Additionally, diagnostic injections with a small amount of local anesthetic can help localize the pain generator and differentiate between single and adjacent interspace entraptments.

Treatment:

Nonsurgical:

Accommodative techniques are often used to provide comfort and relief to those with mild

symptoms of Morton's Entrapment. Initial pain management in these cases may include conservative treatments such as padding, shoe gear changes, and orthoses. Steroid injections may also provide temporary relief of symptoms but have no demonstrative long-term efficacy, and should be avoided due to collateral damage to the adipose tissue and adjacent structures such as the plantar plate.[78] Another treatment to avoid is that of alcohol injection; these are destructive of neural and surrounding tissue and have poor long-term results. In all cases, neural destructive procedures of a focal nerve entrapment should be avoided.

Surgical:

If a brief trial of accommodation is unsuccessful in mild cases, then a surgical decompression of the nerve is appropriate. In severe cases, early surgical intervention will optimize outcomes. There is no other human nerve compression that is primarily treated with nerve resection.[79] It is important to note that excision of an entrapped nerve can release a hurricane of central nervous system physiological ramifications. Therefore, initial management of Morton's Entrapment should be decompression, rather than excision of the nerve. Various surgical methods have been described as yielding favorable results.[79-88]

In the event that decompression surgery fails and symptoms return, secondary neurectomy is appropriate. Specific surgical techniques can be guided by the surgeon's training and experience.

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Appendix 1

3.5 min neuro exam

Recognition/function (take a pen and ask what it is and what do you do with it) – Cerebral function
- Normal is reported as, “Normal recognition and function exam.”

Dysdiadochokinesia – (have pt. do the finger touching with the dominant hand and look for difficulty initiating the movements and sustaining the movements) - Extra pyramidal (ie. Parkinsons)
- Normal is reported as, “No dysdiadochokinesia.”

CN II-XII

2- visual acuity
5 – facial sensation to light touch
7 – facial symmetry
8 – hearing to conversational speech and finger rubbing
9,10 – palate elevates symmetrically
11 – shoulder shrug strength is normal
12 – tongue protrudes in midline and is without atrophy or fasciculations

Dysmetria – Cerebellar/Cerebral Ataxia
CN 3,4,6 continued at the same time (extra ocular muscles) -
Also watching for intention tremor (Cerebellar) - Saccades
- Normal is reported as, “ Cranial Nerves 2-12 Intact. No dysmetria noted.”

Sensory/Cerebellar ataxia – finger→ nose with eyes closed
- Normal is reported as, “No Sensory ataxia.”

DTR – Lower motor neuron/ reflex arc for S1 – note presence of areflexia or hyperreflexia
- Normal is reported as, “Achilles reflex 2/4.”

Straight Leg Raise Test – nerve root impingement
- Normal is reported as, “Negative SLR test.”

Heel/knee shin – Cerebellar/distal sensory ataxia
- Normal is reported as, ”Coordination normal for heel/knee/shin testing.”

Babinski – Upper motor neuron in spinal cord or pyramidal system. [If this stroking of the skin is uncomfortable to the patient, it is known as allodynia. This is indicative of small fiber pathology (C-fiber) and is reported as, “Allodynia demonstrated.”]

- Normal is reported as, “Babinski downgoing.”

Clonus – Upper motor neuron (ALS, stroke, MS)
- Normal is reported as, “Clonus absent.”

Proprioception – posterior column/peripheral nerve – (be sure to only hold on to the sides of the toe.)
- Normal is reported as, “Proprioception intact at 1st MP joint.”

Sharp/dull – A-delta sensory fiber

- Normal is reported as, “Sharp dull intact at all dermatomes of feet.”

Monofilament – A-beta fiber – specific dermatome – (use 1 gm monofilament to screen for individual nerve pathology)
- Normal is reported as, “1 gm monofilament sensation intact at all dermatomes of feet.”

Tuning fork – A-beta fiber – global loss

- Normal is reported as, “Vibratory sensation intact at the 1st metatarsal head.”

Tinels – Entrapment neuropathy

- Normal is reported as, “Negative tinels at (site).”

Mulders – entrapment of the peripheral nerve in the intermetatarsal spaces.

- Normal is reported as, “Negative Mulder’s in the (site) intermetatarsal space.”

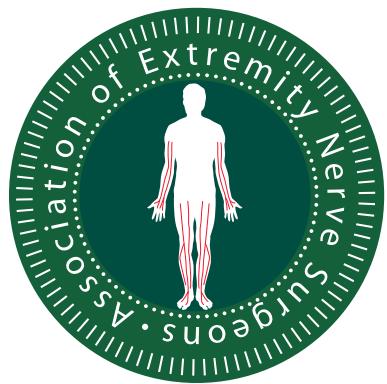
Hot/cold – C-fiber – with eyes closed the patient must determine if the handle of the tuning fork is cold or hot. Reapply the handle to the other foot and ask the same question. If any wrong answers, then there is C-fiber pathology.

- Normal is reported as, “Hot/cold sensation intact to feet.”

Notes

Notes

Notes



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