

The Association of Extremity Nerve Surgeons



Clinical Practice Guidelines v. 2.0 - 2020 -

Stephen L. Barrett, DPM¹, D. Scott Nickerson, MD², Andrew Rader, DPM³, Nathan L. Larson, DPM⁴, Peyman Elison, DPM⁵, Peter J. Bregman, DPM⁶, Robert Parker, DPM⁷, Jim Anderson, DPM⁸, Sequioa DuCasse, DPM⁹, George Zislis, DPM¹⁰, Porscha J. Bailey, DPM¹¹, Aaron K. Ruter, DPM¹²

¹ Chair, Protocols and Guidelines Committee (2014, 2019), Atlanta, GA

² Protocols and Guidelines Committee (2014, 2019), Big Horn, WY

³ Protocols and Guidelines Committee (2014, 2019), Jasper, IN

⁴ Protocols and Guidelines Committee (2014, 2019), Bellingham, WA

⁵ Protocols and Guidelines Committee (2014), Surprise, AZ

⁶ Protocols and Guidelines Committee (2014), Las Vegas, NV

⁷ Protocols and Guidelines Committee (2014), Houston, TX

⁸ Protocols and Guidelines Committee (2014), Fort Collins, CO

⁹ Protocols and Guidelines Committee (2019), Atlanta, GA

¹⁰ Protocols and Guidelines Committee (2019), Marietta, GA

¹¹ Protocols and Guidelines Committee (2019), Marietta, GA

¹² Protocols and Guidelines Committee (2019), Crown Point, IN

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] In nerve decompressive surgeries, post-operative splintage should be avoided.[7]

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 are 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, 8] 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 implementation of multimodal evidence based perioperative analgesia.[9]

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.

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.

Laboratory Testing

Initial screen testing may include the following: Complete Blood Cell count with differential, Complete Metabolic Profile, Serum Vitamin B12, Thyroxine and Thyroid Stimulating Hormone, Rheumatoid Factor, Erythrocyte Sedimentation Rate, Antinuclear Antibody, Serum Protein Electrophoresis[10-12], and Vitamin D levels.[13, 14]

Physical Exam

Silfverskiold Test:

Increased forefoot pressure has been documented to lead to nerve entrapment syndromes and nerve pain.[15] 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.



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.

Tinel's Sign:

The Tinel's Sign, or the Hoffmann-Tinel Sign[16-18], 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.[19, 20]

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.[18, 20-27]

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.

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

An ENFD biopsy is a cost-effective low risk procedure and a minimally invasive reliable diagnostic tool. This is especially beneficial in patients with complaints of symptoms consistent with small-fiber neuropathy.[28] A 3-mm punch biopsy can be taken from any location on the body and is typically performed on sites of interest.[29] Quantitative analysis of small nerve fibers using bright-field immunohistochemistry or indirect immunofluorescence is then performed. The number of fibers traversing the dermoepidermal junction is calculated through standardized means, and documented as the number of intraepidermal nerve fibers per millimeter.[29, 30]

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.[31] A positive biopsy (decrease or absence of small fibers) is consistent with small fiber neuropathy(rare)[32], chronic nerve compression, and mixed-fiber neuropathy (common).[33-36]

ENFD biopsies are of unknown value in the evaluation of degree of neuropathy and effectiveness of nerve therapies. Additionally, they are not helpful in assessment of the etiology of neuro-pathy, 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, as management of small fiber neuropathy will depend on the underlying etiology with concurrent treatment of accompanying neuropathic pain.[29]

"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 parasthesia." [37] 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.

Ultrasonography can be a valuable tool in diagnosing neural pathology and entrapments. Ultrasound allows assessment of both size and quality of the neural tissue. Most of the causes of neuropathy are from entrapment or external compression.[38] Ultrasound imaging should be combined with clinical examination for diagnosis of peripheral nerve pathology including entrapment and peripheral nerve injury syndromes.[38, 39]

Diagnostic Nerve Blocks:

Diagnostic peripheral nerve blocks can be an extremely valuable tool if properly performed with a thorough understanding of peripheral neuroanatomy. Nerve blocks performed prior to treatment can help in predicting efficacy in surgical intervention.[40, 41] 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.[42] Ultrasound guidance has been found to improve accuracy and specificity in diagnostic nerve blocks.[38, 39, 43-45]

Appendix 1 – Focused Neuro Physical Exam

Denervation

Denervation is a nerve destructive procedure and should not be implemented in cases where the pathology is identified as an entrapment.[46, 47]

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.

Cautions in denervation:

Denervation is a nerve destructive procedure and should not be implemented in cases where the pathology is identified as an entrapment.[46, 47] Neuro-destructive procedures may be useful on nerves that are already damaged; although the decision to perform a denervation should be made with expert care as these procedures can be associated with a higher level of chronic post-surgical pain when compared with nerve decompressions.[48] Neuro-destructive procedures should not be used as initial treatment for entrapment neuropathy.[46] There may be a higher association of chronic post-surgical pain and development of sympathetic maintained pain with destruction of nerves that have a larger cutaneous neural distribution.[46, 49]

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. Ultrasound is beneficial for guiding non-surgical percutaneous denervation techniques and has demonstrated efficacy in musculoskeletal techniques similarly.[50-55]

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.[56]

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 Nerve 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.[57] Some of the literature recommends using 30% alcohol solution to get effective results.[58] However, new research has shown the use of 30% EtOH does not create any measurable change in the histology of nerve tissue.[59] There is also moderate risk of necrosis of surrounding tissues.[60] 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.[61-64] When a painful nerve has failed other surgical interventions a neurectomy can be performed. Various methods of implantation have been reported in the literature.[41, 65] 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.[66]

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,[67-69] but should only be performed by surgeons with appropriate training and/or experience.

Diabetic Polyneuropathy

New anatomic clinical knowledge has led to a recognition that diabetic neuropathy is not only the metabolic disease classically described as stocking - glove anesthesia. A new understanding of the common existence of secondary physical nerve trunk entrapments creates the opportunity to attack the frequent pain, balance loss, and serious foot complication cascade of ulceration, recurrences, sepsis, amputations, and early mortality. We are finding that outpatient surgical nerve decompression is almost always an effective therapy to minimize or avoid these serious complications.

In its 14-year history AENS has seen surgery for nerve decompression produce dramatic clinical but scantily recognized benefit in diabetic peripheral neuropathy patients.[70, 71] We believe that clinical and laboratory evidence strongly indicates that the frequent nerve entrapments seen in diabetic sensorimotor polyneuropathy (DSPN) patients are a secondary, metabolically induced, physical compression pathology which frequently accompanies DSPN, are responsible for many serious complications, and are often responsive to safe and effective surgical neurolysis. Nerve decompression surgery (ND) has been found to produce improvements in pain, recovery of protective sensation, balance recovery which may aid in fall prevention, decreased foot ulcer formation and recurrences, avoidance of amputations and improved mortality risk.[72-74]

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 metabolically induced “length dependent axonopathy”, first appearing in the legs due to their most extended distance from the spinal cord cell body. Later, arm symptoms may appear, to generate the classical clinical picture of “stocking glove anesthesia”. No mechanism has been proposed to explain how axonal length could be involved in producing this picture. 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 in careful neurological exam.[75, 76] 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.[77] Such entrapments in combination can easily produce the classically described “stocking-glove” sensory loss. Several biochemical mechanisms are thought to contribute to the development of peripheral neuropathy in the diabetic

patient. One of the most prominent is the development of intraneural sorbital accumulation with attendant osmotic driven fluid accumulation and nerve enlargement.[78-81] 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, tarsal, and medial and lateral plantar tunnels. Entrapments are also common for the radial nerve at the distal forearm, common fibular (peroneal) nerve at the fibular neck, the deep fibular nerve under extensor hallucis brevis tendon on the dorsal foot, or superficial fibular nerve as it exits the anterior or lateral muscle fascial 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,[82, 83] but those with demonstrable nerve entrapments should be considered for decompression surgery. Masking neuropathic pain with long-term neuroactive (gabapentinoid) medications can delay definitive treatment such as decompression surgery. Chronic focal nerve compression can lead to further axonal degeneration, which will threaten surgical outcome. Early decompression is optimal.[84, 85] There are copious clinical reports which provide evidence of connections between diabetic neuropathy, pain relief, nerve function loss, and foot complications, all of which are reported to be relieved or prevented by nerve decompression at entrapment sites.[86] Baltodano et al 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.[70]

An elegant[87, 88] series of experiments in laboratory rats with induced diabetes and sciatic nerve compressions have shown histologic, pain behavior and electrophysiologic changes which closely mimic clinical human findings. Diabetes induction brings on

allodynia, Wallerian Schwann cell degeneration and demyelination, plus slowed nerve conduction velocity which worsen over time and with induced sciatic compression. These changes all recover over a 12 week period after sciatic decompression, though not fully to normal levels.

Many academics view this nerve compression hypothesis and its reported relief of subjective pain as likely presenting evidence only of placebo effects and observer bias. Cornblath's Level IV evidence expert opinion polemic[89] seems to dominate attitudes despite being non-dispositive and outdated by subsequent published evidence. Two thorough reviews[90, 91] find scientific evidence still inadequate to allow a recommendation to use nerve decompression for pain relief, although use for neuropathic diabetic foot ulcer(DFU) is declared to be better supported.[90] A subsequent Rozen et al prospective, randomized control trial[92] now indicates with Level I evidence that excellent and long lasting pain relief can be expected after nerve decompression in DSPN.

Objective measures of outcome can also rebut or negate the placebo/bias critiques and skepticism of ND surgery for relieving subjective DSPN pain. Level II EBM reports show objective benefits after nerve decompression in balance improvement, elimination of dangerously high perineural pressure, protection against initial DFU development and recurrence risk, reduction in lower extremity amputations, recovery of nerve conduction velocity and evoked motor potential muscle EMG.[86] Evidence indicates that using nerve decompression will minimize neuropathic DFU recurrence by over 80%.[73, 93-95] There is also evidence that nerve decompression is protective against initial primary DFU in advanced DSPN for Tinel-positive patients.[95, 96] Furthermore, a report of improved transcutaneous oxygen levels post-nerve decompression may mean that less severe neuroischemic DFU cases can also be protected.[94] A clever Markov analysis among patients with DSPN and superimposed nerve compression predicts surgery is more effective than current treatments at preventing several serious co-morbidities and is associated with lower mortality and greater long-term economic benefit.[74]

Therefore, consideration of using nerve decompression for DSPN pain relief and to protect against recurring DFU, progression to amputation, and early mortality seems well warranted. After nerve decompression, the VAS pain scores are reduced from average levels > 8 to

< 3. NCV improves. Two-point foot sensibility and protective sensation often return to normal.[95] Ulcer recurrence risk becomes <5% per year. Ninety percent have major pain reductions and 70-80% have durable sensory recovery.[70, 92]

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 percussion sign is found over any entrapment site, then nerve decompression can be considered. There are no known contraindication to this recommendation connected to clinical parameters of patient age, gender, diabetes type, disease duration, BMI or Hgb A1c.[97] Diabetes patients can expect therapeutic resolution of painful symptoms and protection against the cascade of foot complications like DFU, amputation, early mortality and possibly the Charcot neuroarthropathy which can accompany DSPN.[74, 98]

Operative Technique

Nerve decompression in the lower extremity must be tailored to the individual patient presentation. This may include external neurolysis of the tibial nerve and its three branches in the medial ankle's tarsal tunnel area, the common fibular nerve at fibular neck, and deep fibular nerve under extensor hallucis brevis.[99] Many surgeons, to avoid residual lower leg ache and cramping, also decompress the superficial fibular nerve as it exits the leg fascia of the distal leg into the subcutaneous tissue. Some cases may also require addressing symptoms arising from entrapment of the proximal tibial nerve at the soleal sling.[100] The usual meticulous nerve and tissue handling techniques under loupe magnification is employed. Tourniquet use is optional for external neurolysis of the common fibular 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 ambulation is mandatory to maintain nerve gliding and avoid adhesion formation while limiting wound healing risk. Suture or staple removal can be delayed up to 3-4 weeks post-surgery to avoid the ankle wound dehiscence, which can occasionally occur. Contrary to expectation in diabetes foot surgery, wound infection is quite unusual following the nerve decompression procedures.

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 a common plantar digital (intermetatarsal) nerve entrapment, or “Morton's Entrapment.”

Histologic findings:

Histologic findings of Morton's Entrapment are variable, ranging from no measurable pathology to perineural 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.[70, 72, 85, 99, 101-107]

Diagnosis:

Diagnosis is primarily dependent on subjective symptoms and physical examination findings. Common physical findings may include splaying of digits, Mulder's sign, Gauthier' sign, Tinel's sign, and impairment of web space or toe tip sensation.[108, 109] 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, elongated 2nd or 3rd metatarsal, tarsal tunnel entrapment, ankle equinus, etc.[15, 110, 111] X-ray, MRI, US, NCS, and toe tip sensation are all viable forms of diagnostic testing that can be performed.[109, 112-117] Additionally, diagnostic injections with a small amount of local anesthetic can help localize the pain generator and differentiate between single and adjacent interspace entrapments.

Treatment:

Nonsurgical:

Accommodative techniques are often used to provide comfort and relief to those with mild symptoms of Morton's Entrapment. Initial treatment in these cases may include shoe gear changes but other conservative treatments such as padding and orthoses have been shown to be minimally effective.[118-120] 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.[121] In all cases, neural destructive procedures of a focal nerve entrapment should be avoided.[122]

Surgical:

If a brief trial of accommodation is unsuccessful in mild cases, then a surgical decompression of the nerve is appropriate. Structural considerations should be addressed which may contribute to the influence of symptomatic intermetatarsal nerve entrapment. In severe cases, early surgical intervention will optimize outcomes. There is no other human nerve compression that is primarily treated with nerve resection.[123] 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.[123-132]

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.

Use of High-Resolution Ultrasonography

High resolution ultrasound can provide valuable diagnostic information and aid in effective treatment of peripheral neuropathology.

The value and necessity of ultrasound guidance for the administration of peripheral nerve blocks both from a therapeutic and diagnostic standpoint is now well established. Implementation of ultrasound guided infiltrations in a clinical setting has been shown to improve accuracy in diagnosis and outcomes while improving patient safety.

Advances in the quality and affordability of high-resolution ultrasound imaging have improved the accuracy of diagnosis in cases of peripheral nerve pathology. Because there is a significant amount of variability of lower extremity peripheral neuroanatomy, it becomes challenging to diagnose peripheral nerve pathology solely by clinical and electrophysiological examinations. Sonographic imaging allows for precision in determining anatomic peripheral nerve location and aids in evaluation of nerve morphology.[45]

The clinician should look for changes in nerve size, focal encroachment, and difference in echogenicity to appreciate the quality of the nerve. The adjacent abnormal structures, such as hypertrophic scars, can provide additional clues for nerve compression. This

is particularly useful in dynamic examination. Palpation of the target nerve by the transducer to reproduce the symptom is helpful for diagnosis. Comparison with the same nerve at the contralateral side is also useful.[38]

Ultrasound imaging during an injection can decrease the potential for damage by direct needle touch or through compression by an adjacent hematoma/thrombosis.[133] Real-time direct visualization allows for agents to be administered with less risk for inadvertent intravascular infiltration. This visualization also allows the clinician to administer the smallest effective amount of the local anesthetic and reduces the likelihood of a confounding finding (usually a false positive) where too much agent floods the anatomical area and involves more than the desired nerve to be addressed.[134, 135]

It is recommended that clinicians implement high resolution ultrasonography as a standard tool to diagnose peripheral nerve pathology as well as in the administration of diagnostic and therapeutic infiltrations.

Complex Regional Pain Syndrome: Peri-Operative Management

Surgery on the patient with complex regional pain syndrome may be necessary and requires careful perioperative management.

An overview of concepts regarding surgery on CRPS patients

Complex regional pain syndrome (CRPS) is a debilitating disorder characterized by widespread, chronic pain and is divided into two subsets: CRPS I and CRPS II. Surgery on an extremity affected with CRPS is generally avoided because of the risk that the symptoms will recur or worsen. Unfortunately, as many as 6% to 10% of patients with CRPS may require surgery on the affected extremity. In CRPS II cases where an isolated pain generator can be identified surgery need not be avoided with proper perioperative management.[136, 137] Elective procedures should be delayed until acute CRPS symptoms have subsided. However, certain scenarios require immediate surgical care.

EVIDENCE BASED POINTS

Pre-operative workup

- Surgical management of patients with CRPS requires a team approach.[138] It is imperative to coordinate with the physician who is actively managing the patient's CRPS. If a patient does not have an active pain management specialist, consultation with a pain management specialist should be sought prior to operating. The surgeon should coordinate with the pain management physician as well as anesthesiologist.[138]
- There is no standard perioperative approach for preventing the development of, or managing existing cases of, CRPS during surgery and the postoperative period. The timing of surgery, choice of the anesthetic technique, use of prophylactic medications and supplements, as well as postoperative pain management, are among the main factors that should be considered.[139]

Preoperative interventions

- Plan to decrease operative and tourniquet time. There is a positive correlation between tourniquet time and the development of CRPS.

- Tissue ischemia should be minimized.
- Choose a minimally invasive approach.
- Administer calcitonin 2-4 days preoperatively.[138]
- Administer gabapentin 300-1200mg 1-3 hours preoperatively.[140]
- Administer Vitamin C 2000mg on the day of surgery.[141]
- Preoperative Ketamine infusion should be discussed with pain management and anesthesia.[139]
- Low dose naltrexone is used in treating patients with CRPS. It should be held at least 24-36 hours before surgery to ensure that any opiate medication administered from anesthesia will produce desired effects.[138]
- A perioperative stellate ganglion block has been shown to reduce the recurrence of CRPS in patients undergoing hand surgery.[137]

Intraoperative interventions

- Consider spinal or epidural anesthesia. Evidence shows decreased recurrence of CRPS from 72% to 10%.[138]
- Consider IV regional anesthesia (IVRA) with lidocaine and clonidine. Evidence shows reduced CRPS recurrence from 74% to 10%.[137]
- Continuous infusion devices for delivery of local anesthetic should be placed proximal to the site of surgery and remain in place during the immediate postoperative period.[139]

Post-operative care/interventions

- Post-operative pain control is imperative.[138]
- Continue calcitonin up to 4 weeks postoperatively.[139]
- Continue Vitamin C 1000mg for 50 days.[141]
- Patients should be allowed to resume their preoperative CRPS medications as early as oral intake is tolerated.
- Early mobilization and rehabilitation should be done as soon as possible postoperatively.[138]

Tarsal Tunnel Syndrome: Peri-Operative Management

Tarsal tunnel syndrome is complex and variable from patient to patient. Multiple etiological factors must be considered in making an accurate diagnosis and planning appropriate treatment. It is a more common lower extremity nerve entrapment than has been appreciated in earlier literature.[142, 143] In order to obtain the best possible outcome of tarsal tunnel surgery, the clinician must consider multiple factors including metabolic status, biomechanical structural influences, extent of focal nerve entrapment, vocational demands, and patient expectations.

Diagnosis and Work-up

A complete history of present illness in combination with a thorough physical exam is essential in making an accurate diagnosis. There can be external compounding factors such as work environment, daily activity levels, and foot structures that will influence the patient's symptoms and potential outcomes.[144, 145]

Diagnosis of tarsal tunnel syndrome must also include assessment of both intrinsic and extrinsic factors. Metabolic syndromes such as diabetes, pre-diabetes, elevated BMI, inflammatory conditions, and toxins in the body have been shown to influence the entrapment potential within the tarsal tunnel.[146-149] Space occupying lesions such as cystic structures, enlarged venous plexus, tumors, hypertrophic muscle bellies, and osteophytic growths can decrease space within the tarsal tunnel and impinge the involved neural structures.[150] Other causes can include biomechanical deformities such as calcaneal valgus or varus, excessive pronation, equinus and tibial positioning.[144, 145] Injury, complications from external bracing and casting, and external compression also can play a role.[150] Evidence has shown earlier intervention is correlated with improved surgical outcomes.[151]

It is important to have a sound understanding of the neuroanatomy in the area. The symptoms of tarsal tunnel syndrome can be expressed through one or multiple nerve entrapments. The tibial nerve has multiple sites of entrapment: proximally at the level of the soleal sling, at the level where the medial gastroc-soleus muscular junction where the nerve exits the deep muscle layer, and at the flexor retinaculum.[152] The medial calcaneal nerves, medial plantar nerve, and lateral plantar nerve then branch from the posterior tibial nerve and can

become entrapped themselves within their own individual tunnels.[153] Each of these entrapment sites can present as true pain generators under the umbrella of "tarsal tunnel syndrome". Differentiation of proximal tibial nerve entrapments from distal entrapment at the medial ankle and porta pedis can improve surgical outcomes. Thorough clinical evaluation can be augmented via the use of diagnostic blocks, ultrasound evaluation, MRI, and neurosensory testing.[12] Due to the high level of false negatives, as high as 50%, seen with traditional electrophysiological testing, such as NCV and EMG, there is questionable value in utilizing these except in cases where comorbidities such as lumbar radiculopathy are present.[12, 24, 154-156]

Careful consideration should be taken when considering surgical intervention in patients with severe lymphedema, morbid obesity, vascular compromise, and an elevated international normalized ratio (INR) over 3.0.[12]

Surgical Treatment

Accurate incision placement is imperative when performing decompressive surgeries. Bi-polar cautery should be utilized instead of monopolar cautery due to increased risk of neural injury.[157]

Due to the variable neuroanatomical presentations, and the need to assess the nerve tissue fully, loupe magnification should be used. In some cases intraneural monitoring and/or nerve stimulation can be useful. Care should be taken to avoid perforation of venous structures within the tunnel. If this occurs, it must be addressed with adequate hemostasis prior to wound closure. If a tourniquet is being used, ideally it should be released prior to skin closure to ensure hemostasis and prevent

hematoma/thrombosis formation if there is any question about adequate hemostasis by the surgeon.

Revisional tarsal tunnel decompressions are demanding and are associated with lower success rates.[158] These procedures should only be performed by surgeons with appropriate peripheral nerve surgical training and experience.

Extensive clinical experience has shown that reapproximation of deep fascial layers must be avoided as this can compromise surgical outcome due to recreation of the original entrapment. Precise skin closure is imperative for prevention of post-operative wound dehiscence and to minimize scar

formation. This can be accomplished in multiple ways but ischemogenic suturing techniques should be avoided. Surgical staples may provide improved closure and because of high incidence of dehiscence in this area can be left in place for 3-4 weeks post-operatively.[12]

Post-operative Care

Early mobilization and nerve gliding in the immediate post-operative period is imperative to minimize scarring and adhesions which can lead to recurrence of the nerve entrapment.[158] Post-operative splintage, casting, or CAM walking boot should be avoided.[7]

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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.”